



Asbestos exposure in New Zealand

1992 to 2005



Published by the Department of Labour
Wellington, New Zealand
October 2006

ISBN 0-478-28076-9

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→ INTRODUCTION

The two asbestos registers, the *Disease Register* and the *Exposure Register* have been in existence for 15 years. When established, certain aims were envisaged.

They were:

1. To raise the 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.

To a large extent these aims have been achieved. The recent publications by *Kjellstrom* and by *Smartt* on asbestos-related diseases, and the report in the Australian publication CCH OHS magazine June/ July 2004 *Asbestos: A Ticking Time Bomb* support the correctness of the New Zealand Government's initiative in 1990 to establish the Asbestos Advisory Committee. The committee in turn went on to publish the *Report of the Asbestos Advisory Committee to the Minister of Labour* in April 1991 and established the National Asbestos Registers in March 1992.

While it is clear that the legacy of disease from working with and exposure to asbestos in New Zealand is continuing and will do so for several decades to come, it is predominantly a legacy from the past. Exposure levels in New Zealand are decreasing and are largely confined to brake repairers, limo removers, demolition workers, carpenters, electrical, plumbing and building maintenance workers and asbestos removal workers.

However, it is important to remember that asbestos exposure still occurs at a high level in some parts of the world and that there are lessons to be learned from what is a world-wide asbestos epidemic.

The first is that there was a general lack of awareness of the effects of inhaling dust at work – regardless of the type of dust – and in spite of the fact that historical evidence went back over 2000 years.

The second was that too great an emphasis was placed on arguing whether the dust was a danger to health or not. Concepts that cement dust was safe and silica dust unsafe or that blue asbestos was worse than white asbestos and so on, distracted governments from the real issue, namely dirty workplace air.

Today we recognise that dirty workplace air is a major problem, both in terms of respiratory and general health, and that solutions are not complex. They involve the putting in place of good general and local exhaust ventilation and providing comfortable and effective respiratory protection. While relatively simple technically, the motive force must be 'the will to do it'.

Finally, the question must be asked of the registers 'where to from here?' It is clear that with the gradual reduction in cigarette smoking among the working population, the impact of dirty workplace air on respiratory health will become clearer with diseases such as welder's lung, asthma, chronic bronchitis and emphysema being recognised more clearly for their relationship to work practices and processes.

It may now be time to expand the asbestos registers to reflect the current situation in the workplace and to include these other occupational respiratory diseases.

Such a move would be in line with the growing recognition by government of the importance and seriousness of occupational disease, a recognition reflected in the 2004 report to the Associate Minister of Labour: *The Burden of Occupational Disease and Injury in New Zealand* published by NOHSAC (the National Occupational Health and Safety Advisory Committee).

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→ PART 1: REVIEW OF ASBESTOS-RELATED DISEASE NOTIFICATIONS

Summary

This report reviews a total of 926 cases notified to the National Asbestos Medical Panel for the period March 1992 to December 2005.

- 164 cases of mesothelioma
- 90 cases of lung cancer
- 205 cases of asbestosis
- 467 cases of pleural abnormalities.

Once again it is noted that the number of lung cancer cases is relatively small compared with mesothelioma cases. This suggests that the taking of a lung cancer history is still dominated by the smoking factor and occupation is ignored.

The transfer of asbestos from the workplace 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 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 JC and AD McDonald, 1998)

Results

The following figures are based on the 926 cases recorded over the period March 1992 to December 2005:

- 164 cases of mesothelioma
- 90 cases of lung cancer
- 205 cases of asbestosis
- 467 cases of pleural abnormalities.

Categories of disease

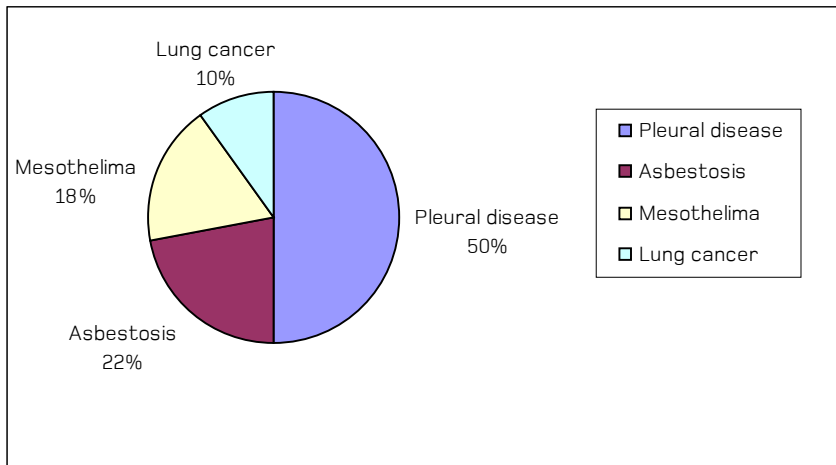


Figure 1: Categories of disease 1992-2005

Figure 1 shows the distribution of the four main diagnostic categories: mesothelioma, lung cancer, asbestosis and pleural disease. What is noticeable is that pleural disease is the main category and lung cancer is clearly under represented when compared with mesothelioma.

Notified asbestos disease by occupation

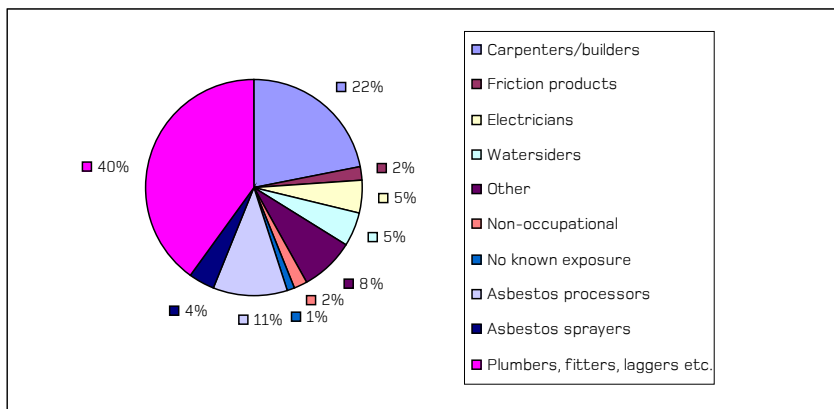


Figure 2: Notified asbestos disease by occupation 1992-2005

Figure 2 looks at occupation for the total number of notified asbestos disease cases. It is clear that carpenters, plumbers and electricians, etc. are together responsible for almost 66 per cent of all cases. These 'all-purpose' construction workers are an occupational category at risk, and particularly so because, unlike asbestos-cement workers, they are not always seen as an 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 women who wash their husband's asbestos-contaminated clothes. The 'no known exposure' category refers predominantly to mesothelioma cases where conclusive exposure histories were not available.

Pleural abnormalities

This category includes 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.

There were 467 cases reviewed.

- 456 were Caucasian
- 7 were Maori
- 4 were from Pacific Islands
- All but 5 were males
- The mean exposure index was 171, with a range of 6 to 708
- There were 48 smokers, 270 ex-smokers and 115 never smoked. (Accurate smoking histories were not available in 34 cases.)

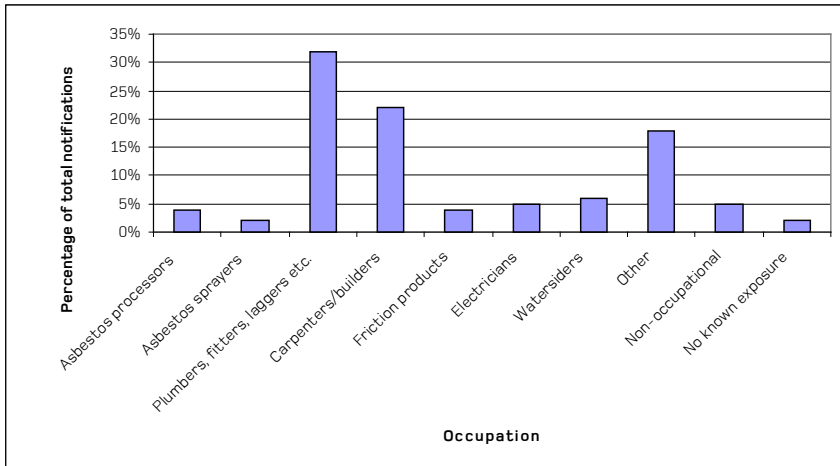


Figure 3: Distribution of pleural abnormality notifications by occupation

Asbestosis

The panel reviewed a total of 205 cases of asbestosis.

- 203 were Caucasian
- 1 was Maori
- 1 was from a Pacific Island.
- 190 were male; 15 were female
- The mean age at diagnosis was 66 (range 37-86)
- The mean number of years since first exposure was 39 (range 15-71).
- The mean exposure index was 194 (range 10-720).
- There were 19 current smokers, 147 ex-smokers and 33 never-smokers. (Accurate smoking histories were not available in 6 cases.)
- Radiological changes showed 101 cases with pleural plaques and/or pleural thickening.

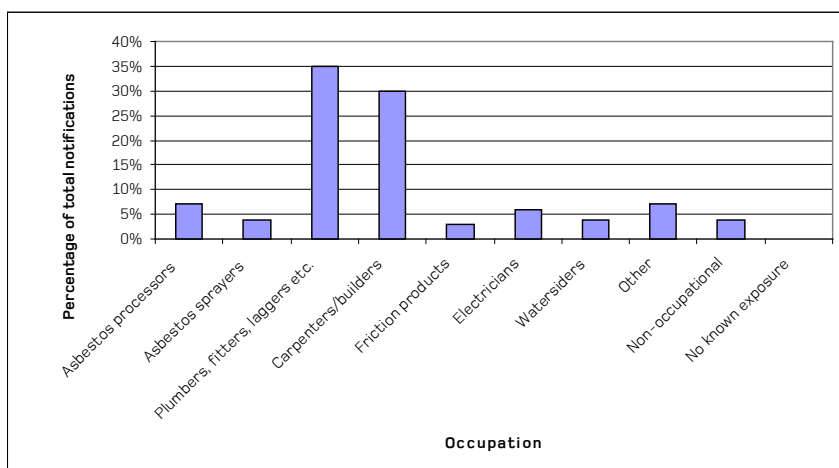


Figure 4: Distribution of asbestosis notifications by occupation

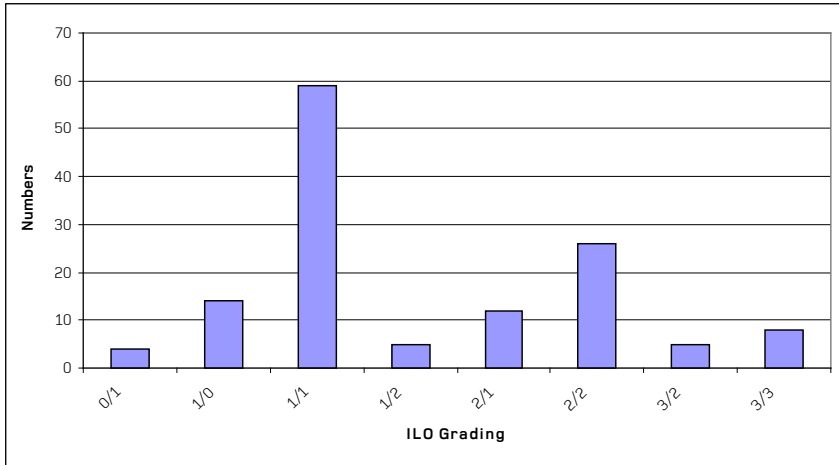


Figure 5: ILO grading of asbestosis cases (n=148)

Of the 205 asbestosis cases, 148 were categorised by ILO classification, others were categorised on the basis of CT, HRCT or pathology where available.

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.

Gilson defined asbestosis along the following lines:

- significant exposure to asbestos dust
- ILO Grade 1/0 or above
- bilateral crepitations
- restrictive lung function pattern.

This definition of *JC Gilson* in his review of asbestos-related lung conditions in the ILO encyclopaedia has been chosen by the panel for the reasons stated above 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 that not all criteria need to be met in all cases but that (a) is essential, (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 commonest pattern (about 40%), in about 10% of cases airway obstruction is the main feature and in the remainder a mixed pattern is seen.

In the 205 asbestosis cases:

- All had a significant exposure history with a mean exposure index of 194 (range 10-720).
- Mean latency was 39 years, with a range of 15-71 years.
- Most cases had an ILO rating of 1 /1 or greater.
- 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.

The numbers in the report are small but confirm that the classical restrictive picture does not dominate, with obstructive, mixed, and normal patterns all occurring.

Lung cancer

The panel reviewed a total of 90 cases of lung cancer.

- 85 were Caucasian
- 3 were Maori
- 1 was from Pacific Islands
- 1 was identified as 'other'
- 88 were males; 2 were females
- The mean age at diagnosis was 64 (range 42-80)
- The mean number of years since first exposure was 40 (range 17-62)
- The mean exposure index was 159 (range 12-565).
- There were 19 current smokers, 61 ex-smokers, 7 never-smokers and 3 unknown.

Histological classification revealed 42 cases of squamous cell carcinoma, 25 adeno, 12 oat cell, 4 undifferentiated, 1 bronchiolar-alveolar, 2 large cell and 4 cases where classification was not stated.

Tumour site was as follows: 43 upper lobe, 31 lower lobe, 8 middle lobe, and 8 were not stated.

Occupational distribution of lung cancer follows a similar pattern to both pleural plaques and asbestosis.

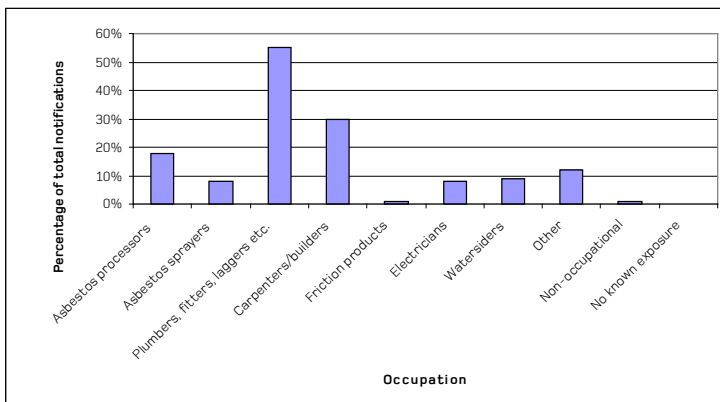


Figure 6: Distribution of lung cancer by occupation

Mesothelioma

The panel reviewed a total of 164 cases of mesothelioma.

- 156 were Caucasian
- 5 were Maori
- 3 were identified as 'other'
- 153 were males; 11 were females.
- The mean age at diagnosis was 63 years (range 35-89).
- The mean number of years since first exposure was 42 (range 12-74).
- The mean exposure index was 198 (range 1-780).
- There were 16 current smokers, 94 ex-smokers and 40 never-smokers (information for 14 cases was unavailable).

The three categories asbestos processors, plumbers /fitters /ladders 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 a 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 teenage girl she had washed the clothes of her older brother who was an apprentice in a railway workshop. Asbestos lagging was used in the repair and maintenance of the boilers, and apprentices frequently used the asbestos in "snowball fights".

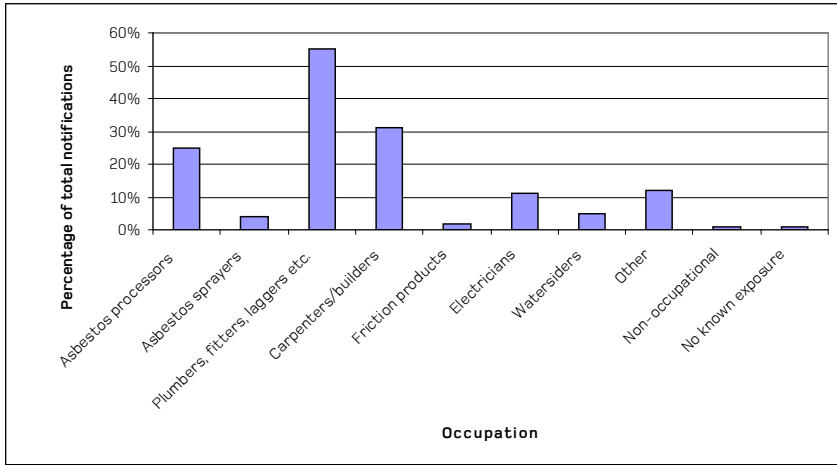


Figure 7: Distribution of mesothelioma by occupation

Discussion

It is recognised that the information recorded in the Disease Register underestimates the total burden of asbestos-related disease in New Zealand. This is a consequence of the voluntary nature of the register; the lack of understanding of work as a factor in disease causation by the medical profession and the failure of the Cancer Registry to code occupation in their data base. Nevertheless the Disease Register continues to serve a useful purpose. There is now a greater awareness of work as a factor in disease than in 1992 when the registers began. The Department of Labour has a greater commitment to the importance of occupational health, ACC employs a greater number of occupational doctors, and there is an increasing number of occupational nurses and safety officers working in the private sector.

While it would be unreal to see these developments as directly arising from the activities of the Asbestos Registers, the registers as part of the wider Notifiable Occupational Disease System operated by the Department of Labour 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 publication, *Lung Function Changes in Asbestos Exposed Workers with Pleural Abnormalities*, in 2000, indicated a clear dose response pattern with a reduction of FVC and FEV1 with increasing asbestos exposure and independent of smoking habit.

Asbestosis

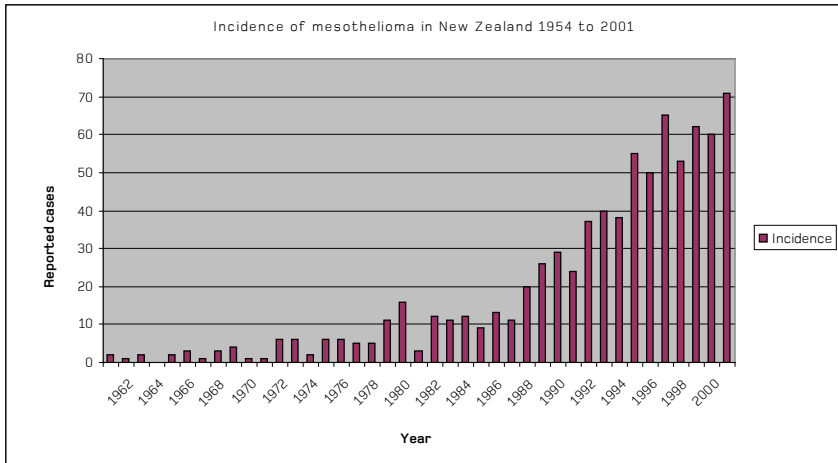
The increasing use of HRCT has resulted in the identification of minor degrees of asbestosis often with few if any symptoms and no disability. It is possible that these individuals will have a better long-term outlook although this is not yet established.

Lung cancer

The contribution of occupational asbestos exposure to the causation of lung cancer is well recognised as being underestimated and incorrectly attributed to smoking. One approach to this issue is to determine a ratio between mesothelioma and lung cancer on the grounds that most mesotheliomas are diagnosed and the majority are seen as caused by asbestos exposure at work. *Kjellstrom* in a recent paper on this issue noted that estimates can range from between 1 and 10 in other countries with the most likely ratio being 2. Thus one can postulate that a much higher number of asbestos-caused lung cancers are occurring than are currently being recorded in the Register or as pointed out by *Kjellstrom*, being accepted by the ACC.

Mesothelioma

Reported cases of mesothelioma have continued to rise in New Zealand over the past decade as illustrated in the graph below. It is important to note that the mesothelioma figures are based on the New Zealand Cancer Registry not on notifications to the Department of Labour. They are thus more complete but not up to date.



Conclusion

Asbestos-related occupational disease continues to disable and kill workers in New Zealand. The extent to which this is occurring can only be estimated but it could be in the vicinity of 200 or more each year. When this disease is put into perspective as part only of the total burden of occupational respiratory disease due to work it is clear that a major task lies ahead to clean up dirty workplace air. With the ban of smoking in the workplace the true extent of non-smoking related occupational respiratory disease will become increasingly apparent.

→ PART 2: NON-ASBESTOS OCCUPATIONAL RESPIRATORY DISEASE

Unlike asbestos-related respiratory conditions there has been less emphasis on the importance of occupational asthma, silicosis and work-related chronic obstructive respiratory disease in New Zealand until recent years. Nevertheless, there is a historical base to non-asbestos occupational respiratory disease as reference to the following brief summary indicates.

1938: *Silicosis report of interdepartmental committee* bulletin No 57, Department of Scientific and Industrial Research, Wellington.¹

This report noted that since the Miners' Phthisis Act of 1915, 1,576 miners had been granted pensions as a result of silicosis, 1,508 were described as gold miners and 68 as coal miners.

The 1940s: A record of tuberculosis morbidity in hospital nurses is recorded in the book *Challenge for Health*, by Dr F S McLean², and 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 1524 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 has seen a significant output of publications on non-asbestos occupational respiratory disease, to a large extent due to the energy and drive of Professor Neil Pearce of the Massey University Centre, together with that of Dr David Fishwick and Lisa Bradshaw. Over this period occupational asthma has been investigated nationally⁴ and among farmers⁵, sawmill workers⁶ and aluminium potroom workers⁷, while respiratory symptoms, lung function and dust levels have been

measured in the wood industry^{8/9}, plywood mill workers¹⁰, hairdressers¹¹, mussel openers¹², welders¹³⁻¹⁸, and quarry workers¹⁹. Of this group, welders in particular have been intensely investigated.

At a 2005 scientific meeting in Christchurch, *Rumball* outlined concern at exposure to beryllium among aircraft engineering and maintenance workers, as well as cleaners and even the families of the workers. His presentation reinforced, if reinforcement was necessary, that dirty workplace air remains a major work environment issue and its consequences occupational lung disease is a continuing health hazard. At the same meeting, *Short*²⁰ presented a paper indicating that woollen mill workers showed significant across-shift changes in FEV₁, and other lung function indices.

Occupational asthma

In the *Colt Lecture* given by Anthony Newman Taylor at the Ninth International Symposium on Inhaled Particles at Cambridge University in 2001²¹, Professor Taylor noted some pertinent points about this disease as follows:

- 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 higher 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 clean up dirty workplaces can thus be seen as a practical response to the accumulating evidence relating to the causes of work-related asthma. In New Zealand a debate frequently ensues between 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, as both work-aggravated asthma 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 and the possibility 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.

Chronic obstructive pulmonary disease

Chronic obstructive respiratory disease (CORD), or as it is now more commonly referred to as Chronic Obstructive Pulmonary Disease (COPD), “is the fourth leading cause of death worldwide”²². It is defined as a condition with airflow limitation which is not fully reversible and which is progressive and 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 is linked to the development of this disease. It is, thus, now recognised as an occupational disease in certain situations with likely additive effects occurring between smoking and some workplace exposures.

Contaminants of air, which have been associated in studies with work-related COPD, include welding fumes, silica, coal, oil mist, Portland cement, cotton, grain and wood dusts.²³

Diagnosis of COPD requires both a clinical and occupational approach, and as far as the latter is concerned, a careful occupational history is required. That is, a chronological list of jobs, what the job entailed, what respiratory exposures occurred, to what extent and for how long. The exposure index methodology used for asbestos and, as illustrated, is an example.

Silica and silicosis – one agent, many outcomes

In a review of the outcome of silica exposure Steenland²⁴ 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 currently 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 material²⁵. 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 0.2 mg/m³ (the current New Zealand workplace exposure standard).

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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 of asbestos.

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

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*, 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 yet they could well be the group at most risk and be the source of asbestos disease in the decades ahead.

Brake lining repair workers

Brake linings still contain asbestos. Such work is often carried out in designated 'small workplaces' and as such 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 where it can be high-level and short-term. Nevertheless these workers comprise an ongoing 'at-risk group'.

Building maintenance workers

It is of significance that the Health and Safety Executive (HSE) of Great Britain has recently published two related guidance booklets. One, *Introduction to Asbestos Essentials*, is specifically aimed at building maintenance workers.

The other, *Asbestos Essentials Task Manual*, 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 the like.

Demolition workers

The process of demolition is often carried out over a weekend and utilising casual labour. The presence of asbestos is not necessarily determined prior to the 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 the 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 as an ad hoc body 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 the 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.

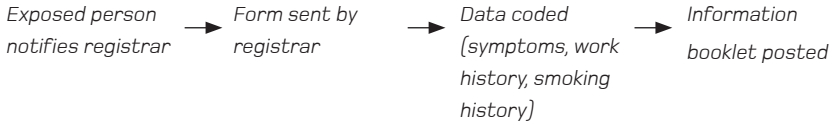
The system should allow movement of the name of a registered person from part 1 to part 2 of the register when indicated.

Notifications to part 1 of the medical register were to be made by those who felt that 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.

Asbestos Exposure Register



The Occupational Safety and Health Service of the Department of Labour (OSH - no longer exists as a separate brand entity), in association with the 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.

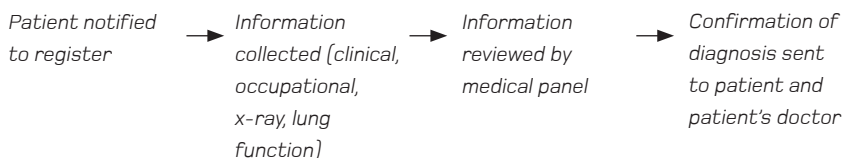
Notifications are directed to branch offices of the Department or directly to the registrar.

In recommendation 4, the committee had 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 this register. However, the huge response from those individuals who had been exposed made it impractical to screen registrants in this fashion.

Once a person has notified the Department of Labour that they have been exposed to asbestos, an asbestos exposure registration form is sent. The registration 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 that 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*.

Asbestos 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

Dr 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.

The first meeting of the panel was held in February 1992.

Associate 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, Dr Graeme Anderson.

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

In 2002 Ms Jenny West was the Registrar with Mr Dougal McNeill Registrar in 2004 and Ms Gail Abel in 2006.

Processes for registering people

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

Data collection

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

- a) An occupational health nurse visits the patient and conducts a health interview, a detailed occupational and social (including smoking) history.
- b) Relevant medical reports are obtained from general practitioners and physicians.
- c) 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.
- d) 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.
- e) Pathology and postmortem reports are reviewed where available.

Exposure Index

$$\boxed{\begin{array}{c} \mathbf{D} \\ \text{Exposure Index} \end{array}} = \boxed{\begin{array}{c} \mathbf{A} \\ \text{Years} \end{array}} \times \boxed{\begin{array}{c} \mathbf{B} \\ \text{Intensity} \end{array}} \times \boxed{\begin{array}{c} \mathbf{C} \\ \text{Degree} \end{array}}$$

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

Data assessment

The National Asbestos Medical Panel reviewed the information obtained, calculated an exposure index (see below) 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) = 5

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

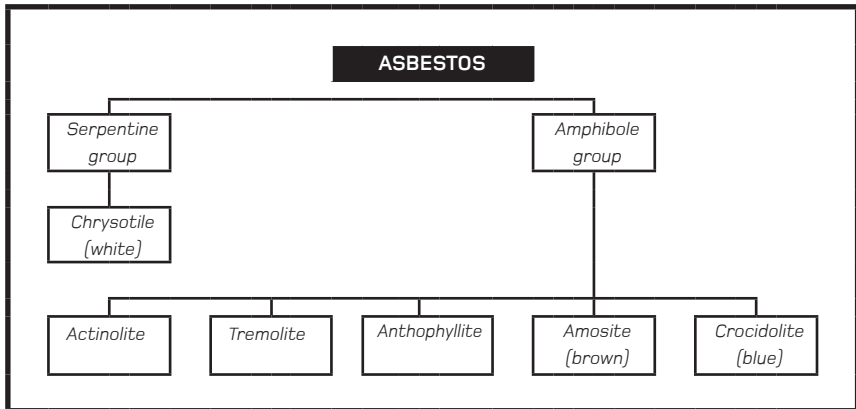
On the basis of the foregoing, the cases were placed into a primary diagnostic category of:

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

What is asbestos?

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

The figure below shows the different 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 make 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 out in the 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, can split longitudinally, are more resistant to body fluids so that they survive unchanged in the body for up to 40 years or more, and 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 and again the fibres remain unchanged in the body for years.

The uses of asbestos

The word asbestos was first coined by the Romans in the first century AD, but by that stage the material had already been in use for at least 2000 years. Archaeological evidence from Finland has shown that a type of asbestos was used to strengthen clay pots over 4000 years ago. The Roman writer Plutarch described in the first century AD a cloth woven from asbestos fibres that could be cleaned by immersion in fire, and when the Venetian explorer Marco Polo returned from the East 1200 years later he too described a fire suit he had seen there.

However, it was not until the late nineteenth century that asbestos began to be used on a large scale in the manufacture of many different items. From the 1880s it was used increasingly in the textile industry to produce incombustible products, also gland packings and other linings for machinery. The different types of asbestos had some remarkable properties - with their resistance to heat, friction and chemical decomposition combined with their fibrous nature; and as the industrial age - particularly the development of the automobile - gathered momentum early this century, asbestos began to be used in an increasing range of applications. World War I saw asbestos being used as thermal insulation in naval vessels and by the 1920s the substance was firmly established as a vital ingredient in the manufacture of friction clutches and brake linings.

Coinciding with this increased use was the discovery of large reserves of white and blue asbestos in Russia, Canada and South Africa. Reserves of both were also found in Australia. Asbestos became popular as the reinforcing material in asbestos cement products, including wall claddings and roofing materials, pipes and other building materials. At the same time it was used more and more in its raw state as an insulation material in buildings, around boilers, and as a fire retardant around structural steelwork.

The post-war years were the heyday of asbestos use in New Zealand and around the world. Each of the three main types of asbestos – white, blue and brown – were sprayed, and spread in what seemed to be an ever-increasing range of applications throughout industry, as part of machinery components, and, to a lesser extent, in homes. A United Kingdom report in the late 1970s estimated that about 3,000 manufactured products contained asbestos in one form or another. The same report said there were over 21,000 people in the United Kingdom alone employed in the manufacturing of products which contained some degree of asbestos, and about the same number employed in processes which were subject to the asbestos regulations of the time.

Asbestos use in New Zealand

Until just before the Second World War asbestos really only found its way into New Zealand in the form of manufactured items. Since that time, the only asbestos containing products that have been manufactured in any quantity in this country were asbestos cement building materials, such as roofing and wall claddings, pipes and other moulded products.

There have been two plants producing asbestos cement products. The first was established in 1938 at Penrose in Auckland, by the Australian company James Hardie Ltd. A second factory, operated by the local company Fletcher's, was established in the Christchurch suburb of Riccarton in 1943. Depending on the item being manufactured, they were made of a mixture of Portland cement, sand, and usually between 5 and 15 percent of either chrysotile, amosite or crocidolite – the asbestos acting as reinforcing because of its fibrous nature and its high tensile strength. The types of asbestos used varied. The bulk was the white variety, chrysotile, which was cheaper and more easily worked. Because the “best” blue crocidolite from South Africa was more expensive, it tended to be used only in products requiring greater heat tolerance or strength (such as in pipes expected to contain higher pressures or temperatures). A lesser quality crocidolite from the Wittenoom mine in Western Australia was also used to some

extent. Amosite, or brown asbestos was imported from Rhodesia (now Zimbabwe) and was only ever used in small quantities. The Auckland plant produced asbestos cement products until 1987 although from 1983 asbestos had been phased out of sheet products and included only in pipes. At peak production in the mid 1970s the Penrose plant employed up to 600 employees at any one time.

The Christchurch plant, called Durock Industries, operated until 1974. Estimates of the numbers employed over the life of the factory vary between 900 and 2000 and are confused by the fact that large numbers of casual workers were employed.

Another major use of asbestos was as the raw material for insulation products. This saw the various types of asbestos mixed with a binder and sprayed around boilers, pipes, ducts and other places where insulation against heat, or sometimes noise was needed. From the 1950s until the 1970s thousands of tonnes of asbestos were applied in this way, most notably in the power stations built in the period, but also in railway workshops, shipbuilding and maintenance and other large-scale industrial applications. Sprayed asbestos was also used extensively as a fire retardant for protecting structural steelwork. Usually the insulation material was applied by contractors who mixed asbestos from the bags or sacks it had been imported in, before spraying the mixture on to chicken wire reinforcing. been banned.

Other workplaces where asbestos was used included railway workshops boiler rooms, and in fact most of the country's major industrial complexes where insulation against heat was required. Some of the industrial applications were less obvious. For example, asbestos was commonly used in the brewing industry to filter beer from the 1920s until the early 1970s, and it was dropped into wine to act as finings and clarify the finished product. Another unusual use for blue asbestos was as a filtering component in gas masks of British manufacture that were standard issue for troops and others from the first World War until after the Second World War. An inner core of asbestos was surrounded by woollen wadding, and the item was standard issue to all New Zealand troops in danger of gas attack.

Before the Second World War, asbestos was not imported in its raw state in sufficient quantities to appear in the import statistics. With the beginnings of local manufacturing and the increase in post-war construction, more than 2,000 tonnes were being imported annually by the late 1940s. This continued throughout the 1950s with peaks of up to 5,000 tonnes in some years. Usage increased dramatically during the 1960s and until well into the 1970s with 5,000 tonnes being a minimum amount that was imported through those years, and the average being closer to 8,000 tonnes. Imported asbestos peaked in 1975 at 12,500 tonnes, although as recently as 1983, 3,000 tonnes were imported.

Throughout the 40 years asbestos was imported in large quantities, about two-thirds of the amount imported was chrysotile from Canada, with the balance being made up of different types from Australia, South Africa or, to a lesser extent, the United States.

Asbestos was only ever mined in small quantities locally, as chrysotile from a single mine near Takaka from the early 1950s until the early 1960s. It was of a low quality and had to be mixed with the imported material. In the late 1960s sizable deposits were surveyed near Dusky Sound, but for various reasons these were never exploited. Since 1984 the importing of blue and brown asbestos has been banned.

Key dates in the health history of asbestos

- 1898 Adelaide Anderson, DBE, MA, Principal Lady Inspector of Factories in the Home Office, noted that asbestos fibre inhalation in the workplace was injurious to the bronchial tubes and lungs. Referring to complaints from girls employed from 1898 onwards, she wrote: "The sharp jagged edge of the insoluble mineral dust has undoubtedly occasioned much illness and death from respiratory disease."
- 1906 Dr Montague Murray recorded fibrosis of the lungs in a 33-year-old asbestos cloth worker. He had been employed 10 years and was the sole survivor of 10 men who were at work in the carding room when he began his employment.
- 1924 Cooke coined the term asbestosis.
- 1928 Merewether and Price launched an extensive investigation into the disease and in 1930 published their results.
- 1931 Great Britain introduced Asbestos Industry Regulations.
- 1955 Doll confirmed the relationship between lung cancer and asbestos exposure.
- 1960 Wagner reported pleural mesothelioma among asbestos miners.

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

1971 Dr Allingham, who succeeded Dr Coplestone, reports on 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 crocidilite fibres) was reduced from 2 fibres/ml to 1 fibre/ml. Crocidilite 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.

1983 Asbestos Regulations replace 1978 regulations.

1983 *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.”

1984 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 public meeting in Christchurch.

1988 Barry Brown Secretary of the South Island Labourers Union arranges for Mavis Watson, occupational health nurse and Associate Professor Bill Glass from the Otago Medical School to interview ex-Fletcher workers from the Mandeville Street plant.

1988 Cate Brett – *Christchurch Star* – highlights the tragedy of asbestos use.

1989 Robin McKenzie, Engineer with NZED 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 WI. 103 workers contacted. 87 investigated.

47 cases of asbestos-related conditions (plus 7 probable cases). 12 deaths to date.

1990 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 Wl., 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.”

1991 *Report of the Asbestos Advisory Committee* presented to the Minister in April and Minister decides to implement the recommendations following Cabinet approval in August.

1991 Tim Frederiksen establishes 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 to administer the registers.

1992 ECNZ combines with the Department of Labour to publicise asbestos hazards.

1992 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.

1992 ACC lump sum entitlements ceased.(cons

1993 The first *Annual Report of the National Asbestos Registers* confirming 199 cases of asbestos-related disease.

1993 (March) Rights to initiate common law claims in regard to asbestos-related conditions ceased.

1993 ECNZ establishes model asbestos surveillance programme of current and past employees.

1993 Secondary cases of asbestos disease 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.

1994 Visit by Professor Margaret Becklake, International Asbestos Authority to advise on registers.

1995 *Asbestos Exposure and Disease: Notes for Medical Practitioners*, Department of Labour.

1995/96 The third annual report confirms 535 cases.

1996 *The Management of Asbestos in the Non-Occupational Environment*, 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 publish 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-significant 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.

1999 *Asbestos and the New Millennium*. A publication 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.

2000 *Increased Mesothelioma Incidence in New Zealand*. T Kjellstrom, P. Smartt. NZMJ, November.

2000 *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.

2001 Occupational Safety and Health Review of *Guidelines for the Management and Removal of Asbestos*.

2001 *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.

2004 *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: 1 P Smartt.

APPENDIX 3: MISCELLANY

Department of Labour publications on asbestos

1. *Safe Work on Asbestos-Based Floor Coverings*, 1985
2. *Talking Asbestos*, 1987
3. *What Every Manager Should Know About Asbestos*, 1991
4. *What Every Worker Should Know About Asbestos*, 1991
5. *What Every Home Owner Should Know About Asbestos*, 1991
6. *Safe Work on Asbestos-Based Floor Coverings*. Leaflet, 1991
7. *Audit of Floor Sanders and Work Practices Involving Asbestos-Backed Vinyl Sheetting in the Christchurch Area*. Occasional Paper Series No. 4, 1992.
8. *Recent Advances in Asbestos-Related Disease*. Dr Margaret Becklake, 1994. Special Report.
9. *Asbestos Exposure and Disease: Notes for Medical Practitioners*. 1995.
10. *Guidelines for the Management and Removal of Asbestos*. 1995.
11. *The Epidemiology of Mesothelioma in Historical Context*. J.C. McDonald and A.D. McDonald, 1998. Special Report.
12. *Respiratory Symptoms and Asbestos Dust Exposure*. Occupational Health Report Series: No. 2, 1997.
13. *Asbestos: A Deadly Dust. 50 Years of Asbestos Use in New Zealand*. Reprint from *Safeguard* magazine, December 1991, updated and reprinted 1999/2001.
14. *Mesothelioma in New Zealand*. Occupational Health Report Series No. 4, 2000.
15. *Lung Function Changes in Asbestos Exposed Workers With Pleural Abnormalities*. Occupational Health Report Series No. 6, 2000.
16. National Asbestos Registers, Annual Reports 1992, 1993, 1995, 1997, 1998, 1999, 2002, 2004 and 2005.

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