Office of the Prime Minister's
Chief Science Advisor

the ROYAL SOCIETY of NEW ZEALAND
TE APĀRANGI

Asbestos exposure in New Zealand:
Review of the scientific evidence of non-occupational risks

A report on behalf of the Royal Society of New Zealand
and the Office of the Prime Minister's Chief Science Advisor

April 2015

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8 April 2015

Hon Dr Jonathan Coleman
Minister of Health

Dear Dr Coleman,

The following report is provided in response to a request by the Ministry of Health in late 2014 to the Prime Minister's Chief Science Advisor (PMCSA) and the Royal Society of New Zealand (RSNZ) to review the available scientific evidence about health risks of casual exposure to asbestos in the non-occupational environment. The Prime Minister approved the engagement of the PMCSA. We were asked specifically to analyse data pertaining to risks from asbestos exposure to residents of older houses undergoing renovation and repair work, such as that which has been carried out and is ongoing in the aftermath of the Canterbury earthquakes. The complexity, urgency and scale of the rebuild in Canterbury resulted in some remediation activities involving asbestos being undertaken without full compliance with recommended safety procedures, and this has caused considerable concern among the public. The aim was to provide government decision makers with a comprehensive and up-to-date understanding of the possible levels of exposure encountered during these activities and their potential risks to health, so that reliable risk communication messages could be conveyed to the general public, and to assist further consideration of how to reduce future risks where they might be encountered.

Process

This scientific review was conducted in accord with a general process agreed between the Office of the PMCSA and the President of the RSNZ for such reports. The PMSCA appointed an experienced research analyst to undertake the primary research and literature reviews. Following an initial scoping that included an extensive reading of the literature (informal, grey and peer reviewed) on the subject, a draft table of contents was agreed between the PMCSA and the President of the RSNZ.

The RSNZ then appointed a panel of appropriate experts across the relevant disciplines that was approved by the PMCSA. A member of civil society with long experience in Canterbury issues, Hon Margaret Austin, CNZM, was invited to be an observer to the panel and to be included in the discussions and drafting to be sure that it met local community concerns and needs.

The research analyst in the Office of the PMCSA produced an early partial draft of the report that was presented to a meeting of the expert panel, and the input of panel members was sought both as to framing of the report and interpretation of the literature. Over the following weeks, the panel members joined in an iterative process with the research analyst to develop the report. In its advanced form all the members of the panel, together with the PMCSA and the President of the RSNZ, agreed via email exchange on the wording of the report and its executive summary. In this form it was sent out for international peer review by scientific experts in Australia and the UK. Representatives from the Ministry of Health were also provided with an opportunity to comment on the draft. Following receipt and consideration of all comments, the report and executive summary were returned to the panel for final review and approval.

Findings and recommendations

Like most developed countries, New Zealand has a legacy of asbestos use primarily in the construction industry that spans many decades. Despite cessation of the production and most uses of asbestos-containing materials (ACMs) in this country in the 1980s, the hazard remains in many buildings and homes that were constructed during the periods of heavy asbestos use. While no ACMs are manufactured in New Zealand, there may still be some importation, as this is not rigorously controlled. There are regulations covering exposure of workers to asbestos.

The evidence suggests that if bonded (non-friable) ACMs are maintained in good condition, they do not pose a health risk to building occupants. However, uncontrolled removal or repair of such materials, or their extensive deterioration may cause release of asbestos fibres, which are known to be hazardous if inhaled. The amount of asbestos released during work such as removal of sprayed-on asbestos coatings or during sanding of asbestos backing after lifting tile or vinyl flooring can be significant if proper procedures are not followed, but does not typically exceed workplace regulatory levels. Exposure levels associated with most home renovation activities are generally orders of magnitude lower than historical occupational exposures that are known to increase the risk of asbestos-related diseases.

The main potential outcome of concern related to such low exposures is mesothelioma, which is associated with much lower cumulative exposures to asbestos fibres than lung cancer or other asbestos-related lung diseases and cancers. Most asbestos-containing materials used in New Zealand houses contain mainly chrysotile asbestos, which confers a lower risk of mesothelioma than other asbestos types.

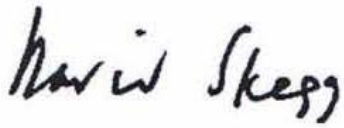
While there is no absolutely safe level of asbestos exposure, asbestos fibres in very low concentrations also exist in the natural environment, and therefore some exposure is unavoidable. The risk at very low exposure levels needs to be in put in the context of other inevitable risks, such as low-level radiation exposure during an aeroplane flight, for which no minimal safe dose is known.

The report concludes that remediation activities such as those that have taken place in Canterbury are unlikely to result in any significant increase in risk to homeowners and occupants of damaged houses, unless they were performing the work themselves, without taking proper precautions such as wetting the surfaces and using a respirator.

Although these conclusions should be reassuring for many home-owners, they do not provide grounds for complacency about the risks for people working with asbestos - including residents doing their own renovations. Messages about the importance of consistently taking adequate precautions when working with ACMs should be reinforced.

The report also notes that many countries have now banned the importation and continued use of ACMs and recommends that New Zealand should similarly consider introducing such a ban.

Yours sincerely



Sir Peter Gluckman
Prime Minister's Chief Science Advisor

Sir David Skegg
President, Royal Society of New Zealand

Acknowledgements

This report was commissioned by Sir Peter Gluckman, the New Zealand Prime Minister's Chief Science Advisor (PMCSA), and Sir David Skegg, the President of the Royal Society of New Zealand (RSNZ), at the request of the New Zealand Ministry of Health.

The report was prepared by Dr. Anne Bardsley, PhD, Research Analyst in the PMCSA office, working in collaboration with an Expert Panel appointed by the RSNZ. The report was peer reviewed by three international experts before its release. Advisors from the New Zealand Ministry of Health provided comments on an interim draft.

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Asbestos exposure in New Zealand:
Review of the scientific evidence of non-occupational risks

The purpose of this report is to provide a comprehensive and up-to-date understanding of the scientific evidence on the risks from casual asbestos exposure in the non-occupational environment in New Zealand, specifically addressing the level of risk to occupants of houses containing asbestos, and of exposure during renovations and repairs. The potential effects of events such as the Canterbury earthquakes and consequent rebuild on exposures and risk are considered. The intent of this report is to inform decision-making on asbestos management and consequent public health measures including risk communication to the public.

In order to assess asbestos risks in the residential environment, it was necessary to use the evidence base established by investigations in historical occupational settings, where asbestos exposure was very much higher and the association of such exposure with adverse outcomes was clear. Although the report discusses exposures that may be encountered by workers today who are involved in building construction, renovation, remediation and demolition, we caution readers not to treat the analysis of occupational risks as definitive; the information is provided to assist with understanding the non-occupational risks.

Executive Summary

Asbestos is a term referring to a group of related, naturally-occurring fibrous silicate minerals that have been mined extensively around the world and were once widely used industrially and in building construction because of their characteristic strength, pliability, insulating properties, and resistance to fire and chemical breakdown. Over time, asbestos was linked to a number of serious lung diseases and cancers in workers who were heavily exposed to its raw fibres in mines, mills, and factories producing asbestos products. Asbestos-related diseases were later observed in workers who regularly handled these products, and in people environmentally exposed to airborne fibre contamination near asbestos mines and factories.

Inhalation exposure to asbestos is now known to be a serious public health risk, with consequential disease liable to develop after a long latency period – the risk of which is influenced by the intensity (dose), the frequency, and the duration of the exposure (i.e. the cumulative amount breathed in). Although other routes of exposure are possible (e.g. dermal contact, ingestion), inhalation is the only route that has been established as causing harm. Fibrotic lung diseases (pleural changes and asbestosis), lung cancer, malignant mesothelioma, laryngeal cancer, ovarian cancer and possibly other cancers can occur 20 to 50 years after heavy exposure to asbestos fibres. The risk of developing disease from asbestos inhalation increases with increasing cumulative exposure. Efforts to reduce and ultimately to eliminate this risk have led to total prohibition of the production, importation and use of asbestos in many countries, and strict regulation of exposure of workers involved in repairing or removing asbestos-containing materials (ACMs). The presence of ACMs throughout many older homes and buildings means that the asbestos hazard still lingers, and non-

occupational exposure of the public is an ongoing risk, although the magnitude of this risk is not well characterized. This report aims to summarise the available evidence in order to inform policymakers and the public about the extent of risk from non-occupational exposure to ACMs in residential houses in New Zealand, and potential actions to be taken.

Asbestos exposure in New Zealand

Unprocessed asbestos was imported into New Zealand beginning in the late 1930s and building products composed of asbestos mixed with cement were produced over a 50-year period up until the mid-1980s. ACMs used in building construction were also imported from other countries. Many of these products were used in the construction of New Zealand houses between 1940 and 1990.

The incidence of asbestos-related diseases has been rising in New Zealand in accord with the expected latency from past heavy exposure of workers in the asbestos industry, and those working regularly with ACMs (e.g. construction workers). Although New Zealand lagged behind many other countries in dealing with the asbestos hazard, regulations on its use and on acceptable workplace exposure levels have ended the era of very high occupational exposure risk, and a decline in asbestos-related disease incidence is to be expected in the future. However the legacy of past asbestos use in New Zealand persists in the numerous ACMs that remain in place in older buildings and houses, including asbestos cement roofing, external cladding, internal wall linings, textured ceilings, vinyl flooring, and insulation around pipes and hot water heaters.

The necessity of large numbers of building and infrastructure demolitions as a result of the Canterbury earthquakes of 2010 and 2011 has increased awareness of asbestos, and the possibility of exposure to asbestos from ACMs in damaged older homes. There has been public concern that improper handling of asbestos in homes undergoing renovation and repair during the Canterbury rebuild may have exposed people to dangerous levels of airborne asbestos fibres. The main concern is exposure of the public to friable asbestos – that which is loosely bonded and can be crumbled or reduced to powder by hand pressure. Asbestos is considered non-friable if it is bonded within building materials and is therefore more resistant to mild abrasion or damage. Non-friable ACMs that are in good condition do not release fibres and do not pose a health risk, but they can become friable when damaged or weathered, or during remediation, repair or removal.

Risk characterization and assessment

Asbestos has been clearly shown to be a hazardous material with the propensity to cause cancer and other diseases in exposed individuals. The risks associated with asbestos depend on the extent and intensity of the exposure to the hazard and the possible underlying risk factors or susceptibilities of the individual. Risks also differ depending on the type of asbestos to which an individual is exposed. Asbestos fibres are naturally ubiquitous at very low levels in air and water, and therefore there are no completely unexposed populations. Nonetheless, there is no level of exposure that is known to carry no risk of asbestos-related disease.

Asbestos types and potency

All asbestos types can cause asbestos-related cancers. However, the different chemical composition and structures of the asbestos types affect their toxicity and persistence in lung and pleural tissues resulting in differences in carcinogenic potential. There are three common asbestos types that have been used industrially. Amosite and crocidolite are of the amphibole variety - they have straight fibre structures and are highly insoluble in lung fluid, and thus can persist in lung tissues for decades after inhalation. The third, and by far the most commonly used type in New Zealand, is chrysotile, which has a curly fibre structure and is relatively more soluble and more readily cleared from the lungs than the amphiboles. Estimates from different studies vary, but it is generally acknowledged that the cancer risk is higher from amphibole exposure than from chrysotile exposure. One estimate of the

ratio of the potency for inducing mesothelioma suggested that chrysotile is up to 500x less potent than crocidolite, and 100x less potent than amosite. Nonetheless, all forms of asbestos are considered to be carcinogenic, and therefore hazardous.

Dose, duration, and cumulative exposure

Epidemiological studies suggest that the level of risk of asbestos-induced cancer is directly related to the cumulative asbestos exposure received (the amount breathed in) over a period of time. This means that a small number of high-exposure incidents may confer roughly the same risk as a larger number of lower-exposure incidents. However, because of the long latency between accumulated exposure and cancer development, a given cumulative exposure accrued over a short period is expected to result in a higher risk of actually developing a cancer than the same exposure accrued over a longer period, if both exposures were to begin at the same time. This is because a substantial portion of the longer exposure will occur at older ages, when the potential to experience the full latency period is less likely.

Exposure level estimation

Asbestos is found in certain types of rock formations, and is present at very low levels in air and water as a result of natural erosion processes. However, industrial activities have greatly increased the levels of airborne asbestos fibres in some locations and situations. Environmental exposure has been high in the vicinity of working asbestos mines and factories. Levels are elevated around motorways and in cities, because of release of asbestos fibres from many automotive brake linings. The large amount of existing asbestos cement products making up the exterior cladding and roofs of many buildings and homes also contributes to a significant release of asbestos fibres into the total environment each year.

This report is primarily concerned with the airborne asbestos levels that may be found within homes where friable ACMs are present, and human exposures during repair or removal of such materials when the work has been carried out by others. The potential risk to building occupants posed by the presence of old ACMs has been the subject of intense debate, but studies suggest that undisturbed ACMs do not cause elevated airborne asbestos concentrations inside buildings. Fibre release episodes from small repair or maintenance activities or from random dislodging of ACMs also do not substantially increase average concentrations inside buildings, although they might result in exposure to an individual undertaking such work or present nearby.

Risks of low-level exposure

While the risk associated with working with raw asbestos or regularly handling ACMs as part of an occupation is relatively well understood, the level of risk arising from occasional, low exposures is more difficult to assess. The vast majority of data relating asbestos exposure to disease risk have come from studies of heavily-exposed groups in asbestos mining, milling, transport and manufacturing industries, or other occupational groups working with asbestos products (e.g. construction trades, ship builders, mechanics, etc.). Assessment of risks of low-level asbestos exposure has had to rely on extrapolation from studies of such highly-exposed workers in order to estimate risk for disease development in minimally-exposed non-occupational groups. A degree of uncertainty in assessing these risk levels is unavoidable, as knowledge of dose-response relationships at low exposure is limited by methodological and technical considerations.

In particular, the incidence of lung cancer attributable to asbestos exposure is difficult to quantify, because there is a substantial background incidence due to factors other than exposure to asbestos (mainly tobacco smoking). Whereas a substantially elevated incidence of lung cancer can be quantified in highly-exposed worker populations, any increase above background rates resulting from low-level, non-occupational asbestos exposure would be difficult to detect, and has not been

reported (though the risk should not be considered as nil). Current non-occupational exposure levels are also considered to be too low to cause asbestosis. Mesothelioma, which is a highly specific outcome of asbestos exposure, occurs at lower exposure levels than asbestosis or lung cancer and is the disease most likely to occur in relation to non-occupational exposures. This report thus focuses mainly on the risk of mesothelioma, as the low exposures to the general public of New Zealand today are not likely to increase the risk of any other asbestos-related diseases.

Reports of mesothelioma resulting from exposure to asbestos in the non-occupational setting have been increasing in many countries, although most involve environmental exposures related to residence near asbestos mines or factories. Exposure estimates have not been reported in such populations, so it is difficult to relate these risks to other non-occupational exposures, such as those encountered by occupants of houses with damaged or deteriorating ACMs or who have undertaken or been present during ACM repair or removal. The health risk to most building occupants appears to be very low. There is no evidence that a single peak in exposure of the kind encountered during maintenance or repair of ACMs significantly affects disease risk, although each incident of such exposure would add to an individual's cumulative exposure.

Risk assessment in the Canterbury Home Repair Programme

Earthquake damage to ACMs, as well as the removal and repair processes could cause release of asbestos fibres from previously non-friable materials, potentially resulting in elevated exposure and health risks. The use of proper abatement and cleanup procedures can effectively reduce these increased risks. For example, most asbestos removal procedures involve wetting the surface to reduce the release of dust. Dry scraping or sanding of friable ACMs should be avoided.

In the immediate aftermath of the Canterbury earthquakes, cleanup procedures and home remediation did not always follow appropriate guidelines for avoiding asbestos exposure. The level of exposure to workers and the public during this time is not known with certainty. A simulation study involving a small number of Christchurch houses was conducted to replicate typical exposures during removal work (in terms of duration and dustiness) that was carried out in the first year after the earthquakes, before stricter procedures for asbestos monitoring and abatement were fully operational. The resulting exposures were found to be well below the permissible workplace exposure standard even for full-time abatement work over a 3-year period, and it was therefore concluded the risk to occupants (who would have experienced only short duration exposures during this time) would have been extremely low.

Is the public at risk?

Assessment of the current scientific knowledge on exposure levels and risks associated with home remediation activities such as those that have taken place (and are still in progress) in Canterbury indicates that they are unlikely to result in a significant increase in risk to homeowners and occupants of damaged houses, unless they were performing the work themselves, without taking proper precautions such as wetting the surfaces and using a respirator. A simulation study showed that even in a scenario of uncontrolled removal of potentially friable ACMs by dry scraping methods, asbestos concentrations in air in the vicinity of workers' respirators did not reach regulatory levels. It is nonetheless very important that correct procedures for dealing with asbestos during remediation work are followed, and homeowners undertaking repair and renovation work themselves should be made aware of the potential hazard if asbestos is disturbed. Overall, the risk is considered to be low if proper precautions are taken, but it is recommended that repair or removal of friable ACMs are handled by professionals who are trained in the correct procedures. Neither alarm nor complacency about the level of risk to bystanders is warranted. While there has also been concern expressed about the dust present in the air in the immediate aftermath of the earthquake, data from major earthquakes elsewhere are reassuring.

Review methodology

This report set out to evaluate the peer-reviewed scientific literature on the health risks associated with asbestos exposure at the levels that may be encountered in the home environment in New Zealand, with specific reference to exposure type and duration in situations such as home renovation and/or repair, or during earthquake recovery.

Literature searches were undertaken (with no date limit) in Medline, EMBASE, the Cochrane library database, Scopus, and Web of Science in order to identify relevant studies relating to low-level, non-occupational asbestos exposure in the peer-reviewed scientific literature. The particular focus was on asbestos exposures to occupants of homes containing ACMs, and effects of renovation, repair, or removal of ACMs on airborne asbestos fibre levels. Very few studies were identified; therefore studies detailing occupational exposure levels and associated risks of asbestos-related diseases were used as a base for comparison and extrapolation to low-level exposure.

The review did not include studies relating to asbestos exposures (either occupational or non-occupational) from machinery insulation or friction products such as motor vehicle brake linings, although such products still exist in New Zealand and may contribute to occupational exposures in the mechanical trades, and environmental exposures to the public.

Reports and commissioned studies from recognized national and international bodies (NZ Ministry of Health, WorkSafe NZ, World Health Organization, International Agency for Research on Cancer, US Environmental Protection Agency, US Public Health Service, UK Health and Safety Authority, Safe Work Australia) were considered where relevant.

Asbestos exposure in New Zealand:
Review of the scientific evidence of non-occupational risks

1. Asbestos background

1.1 Types and characteristics

Asbestos is a general term encompassing a number of naturally occurring fibrous silicate minerals found in certain types of rock formations that are abundant around the globe. [1] The discovery of the many useful properties of asbestos, including high tensile strength, resistance to fire, very low thermal conductivity, and resistance to acid corrosion, led to its use as an insulating, fireproofing, and strengthening material in a vast number of industrial applications. [2]

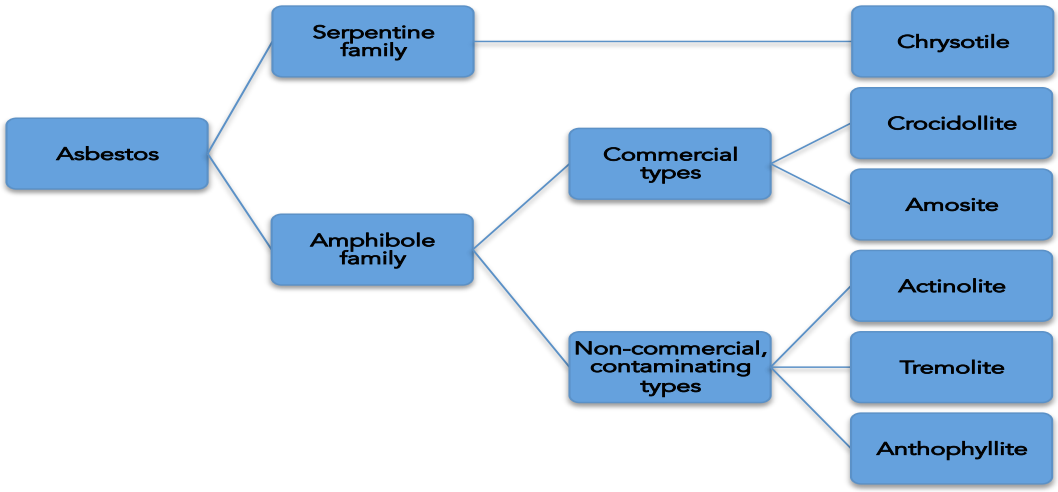
The 'asbestiform habit' refers to mineral crystals that grow in a single dimension, as opposed to random, multidimentional prismatic patterns. Asbestiform minerals form long, threadlike fibres that bend like wire rather than shattering under pressure. There are two 'families' of asbestos types; the serpentine family is characterized by curly fibres, and comprises a single member known as chrysotile asbestos. The amphibole group, characterized by long, straight, and thin fibres, consists of amosite, crocidolite, tremolite, anthophyllite and actinolite fibre types. The types of asbestos that were most commonly used in building products are chrysotile, amosite, and crocidolite, whereas tremolite, anthophyllite and actinolite are noncommercial contaminants. While the amphiboles share certain crystal features, all asbestos types differ in their chemical composition (see Table 1 and Figure 1). [3] The varying characteristics of the different asbestos types influence their effects on the human body (see section 3.1).

Table 1. Asbestos types and characteristics

Fibre type	Typical formula*	Description
Chrysotile	$Mg_3Si_2O_5(OH)_4$	Serpentine. White colour. Curly fibres, faster lung clearance. Fibres undergo longitudinal splitting
Amosite	$(Fe^{2+}Mg)_7Si_8O_{22}(OH)_2$	Amphibole. Brown colour.
Crocidolite	$(Na_2Fe_3^{2+}Fe_2^{3+})Si_8O_{22}(OH)_2$	Amphibole. Blue colour.
Tremolite	$Ca_2Mg_5\ Si_8O_{22}(OH)_2$	Amphibole.
Anthophyllite	$(Mg, Fe^{2+})_7\ Si_8O_{22}(OH)_2$	Amphibole. Brown colour.
Actinolite	$Ca_2(Mg, Fe^{2+})_5Si_8O_{22}(OH)_2$	Amphibole.

* there is variability in composition because the silicate framework can accommodate a mixture of many different ions

Figure 1. Asbestos types/families



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1.2 Historical use and hazard recognition

Inherent to virtually all innovations throughout history is the fact that while they are developed for a human benefit, they also carry potential risks of harm. [4] The industrial utilization of asbestos as a fireproofing material is a prime example of a technological advance that was developed to reduce a known risk – catastrophic fire - but was later found to carry considerable risks of its own. [5] Once referred to as the 'miracle mineral', asbestos is now known to be a human carcinogen, and therefore a public health hazard. Inhalation of its airborne fibres can cause pleural changes, asbestosis, lung cancer, and mesothelioma, depending on the intensity and duration of exposure. Asbestos exposure has also been associated with increased risk of laryngeal and ovarian cancers following heavy exposure.

Asbestos came into widespread use in the early 1900s, when fire risk featured prominently in the public consciousness. With the advent of new technologies using steam, kerosene and electricity, new fire hazards were emerging and fire was a constant threat. Experiences with catastrophic fires, involving hundreds of casualties in public buildings (theatres, schools, office buildings) and on ships, motivated the search for a building and insulating material that was non-combustible and had low thermal conductivity. Asbestos, long known for its strength and resistance to fire and chemical breakdown, seemed ideal. [6] It was mined extensively in several countries (Canada, South Africa, Australia, Russia, China, Brazil, Zimbabwe, Kazakhstan, and India) and came to have significant industrial and economic importance throughout the world. Russia is currently the largest producer of asbestos, followed by China, Brazil, and Kazakhstan. Canada, formerly one of the world's top asbestos producers and exporters, halted mining operations in 2011.

Reports of serious respiratory problems began to emerge in the early 20th century in asbestos miners and workers handling raw asbestos in the manufacture of asbestos products (textiles, insulation, building materials etc.). The first disease to be associated with asbestos exposure in the workplace

was termed asbestosis, a progressive scarring disorder (fibrosis) of the lungs. By the 1960s, a significant excess of asbestosis, as well as lung cancer and malignant pleural mesothelioma, had emerged in workers involved in installing and maintaining asbestos products, including plumbers, electricians, mechanics, ship builders and construction workers. [7] More recently, the consequences of asbestos exposure have been noted in people engaged in repair, renovation, and removal of ACMs. [8, 9]

The use of crocidolite asbestos, and the spraying of any type of asbestos, has been prohibited since 1986 under the International Labour Organization Convention No. 162, [10] but chrysotile asbestos continues to be used in asbestos cement products in a number of low- and middle-income countries. People all over the world are still being exposed to asbestos, not only in those countries where its use is still common, but also in those that have banned its use but still have vast quantities of ACMs present in public buildings and homes.

1.3 Hazard, exposure, vulnerability and risk

It is important to distinguish between hazards and risks and to understand the impact of exposure and vulnerability, because these concepts are critical for informed decision-making and risk communication. [4] A hazard is something with an intrinsic propensity to cause harm, whereas a risk is the likelihood that exposure to a hazard will result in harm. This likelihood is dependent on the vulnerability of the population, and their extent of exposure to the hazard. We can avoid the risks of hazards by reducing our exposure to them. A hazard with no exposure poses no risk.

The very high levels of exposure to asbestos that occurred in occupational settings before its hazardous properties were well known have cost many workers their lives, and others are still at risk of developing disease due to past heavy exposures. There is evidence that lower exposures, such as those that occur from encountering airborne asbestos fibres while living in the vicinity of asbestos mines and factories, and even brief but intense or intermittent non-occupational exposure can also increase the risk of asbestos diseases, in particular mesothelioma. No 'safe' lower limit of exposure has been identified with certainty – all exposures are thought to add to the overall risk of disease development – but the risk from a single, low-level exposure is considered to be extremely low. Awareness of the potential for exposure is nonetheless very important if risks are to be minimized.

Although work-related exposures have decreased, diseases resulting from exposure to deteriorating ACMs in older houses represent a potential public health issue for the future. There are reports of schoolteachers who have contracted mesothelioma for whom the likely contact was from friable in-place ACMs in schools [11, 12] Custodians and maintenance workers in public buildings have also developed asbestos-related diseases. [11] The problem of unrecognized asbestos exposure is an important health issue in settings where it is not controlled or not appreciated.

The risk to the general public depends not only on the effect of cumulative low-dose exposures, but also the relative vulnerability (susceptibility) of individuals to disease development. One factor influencing disease susceptibility is cigarette smoking, which greatly amplifies the risk of lung cancer associated with asbestos exposure beyond the combined effects of the individual risk factors. This means that smokers are much more susceptible to asbestos-induced lung cancer than are non-smokers. Smoking does not have an impact on the risk of mesothelioma or other asbestos-related cancers. There is some evidence of genetic susceptibility to mesothelioma; for example, *BAP1* gene mutations greatly increase mesothelioma risk in asbestos-exposed individuals. [13] This may partially explain why some individuals develop mesothelioma following low-level asbestos exposure, while

others with high-level exposure do not. [14] Very little is known about what other factors may influence susceptibility to these diseases, but it is clear that individuals exposed to the same asbestos hazard do not all respond in the same manner in terms of disease development.

The generally low exposures experienced today do not pose an increased risk for fibrotic lung disease (asbestosis), which requires very high-dose fibre inhalation to trigger its development. [15, 16] Levels of asbestos exposure in most contemporary environments are also not expected to result in a quantifiable increase in risk of lung cancer above the background incidence, though the risk should not be considered zero, particularly among smokers. The potential risk of developing mesothelioma, which is very strongly associated with asbestos exposure and has an otherwise low background incidence, remains an issue. Therefore this report will focus on the risks to the public of developing mesothelioma from exposure to asbestos in the non-occupational environment in New Zealand.

2. Asbestos-related diseases

All types of asbestos are known to cause fibrotic lung disease (asbestosis), pleural plaques, diffuse pleural thickening and pleural effusions, lung cancer, malignant pleural mesothelioma, laryngeal cancer and possibly other cancers with varying latency periods. The International Agency for Research on Cancer (IARC) has also accepted that there is sufficient evidence to indicate that women with a history of heavy occupational or environmental exposure to asbestos are at an increased risk of developing ovarian cancer. [17] The consequences of exposure are generally seen only many years after the exposure began, and often long after it has ended.

The earliest IARC report on asbestos in 1973 stated that all major commercial forms of asbestos can produce malignant mesotheliomas in animals, and that heavily exposed workers were at significantly increased risk of lung cancer and mesothelioma. [17] Asbestos has been listed in the US as a known human carcinogen since the first National Toxicology Program (NTP) report on carcinogens in 1980, [18] and is recognized by the WHO as one of the most important carcinogens worldwide, with a burden of disease that continues to rise despite declining industrial asbestos use. [8, 19, 20] The epidemiological evidence has only strengthened over time and there is currently overwhelming evidence that all commercial forms of asbestos fibres are causally associated with an increased risk of mesothelioma and lung cancer, despite ongoing uncertainty over the extent to which the various forms differ in potency. [21]

Most asbestos-related diseases are clearly dose related – their development depends on the intensity and duration of exposure. There remains some scientific uncertainty regarding the varying toxicities of chrysotile versus amphibole asbestos, as well as the risk of minimal exposure. To date no safe level has been convincingly demonstrated, but such a demonstration would be very difficult given that some very low level of exposure to asbestos is experienced by everyone. The major health concerns arising from asbestos exposure are detailed below.

2.1 Benign pleural disease

Benign pleural changes including diffuse pleural thickening, pleural effusion (fluid around the lungs), and pleural plaques are commonly observed in asbestos-exposed workers. Such changes are often asymptomatic, but can sometimes result in abnormal lung function or pain. Pleural plaques, which appear as discrete areas of thickening on the parietal pleura, are the most common manifestation of asbestos exposure. The incidence increases with increasing exposure duration, but may also occur after relatively low-dose exposures. Benign asbestos effusions are an early manifestation of asbestos disease, sometimes occurring within 10 years of exposure, but usually resolve within a few months. [22] These types of changes do not have any implications for the likelihood of developing an asbestos-related cancer, except by indicating that there has been exposure to asbestos.

2.2 Asbestosis

The most serious non-malignant asbestos-related disease is asbestosis. Asbestosis was first reported in the early 20th century as diffuse fibrosis leading to scarring of the lungs, resulting from inhalation of very high doses of asbestos fibres. Fibrosis progresses after cessation of asbestos exposure. As the disease progresses, the lungs contract progressively until they may no longer be able to expand with each breath sufficiently to support respiration. A high fibre concentration in the lungs is required for development of asbestosis, which was once frequent among heavily exposed worker populations. In fact, patients with asbestosis always have a history of high occupational asbestos exposure. [23] As a result of more stringent control of such exposures in the workplace, as well as the decreasing industrial use of asbestos, the incidence of this disease is now declining. It has never been reported as a consequence of casual or environmental exposure, and is not known to be an issue with current exposure levels either occupationally or involving the general public. [16]

2.3 Lung cancer

An increased incidence of lung cancer in asbestos workers was first suspected in the 1930s, but the linking of asbestos with excess occurrence of lung cancer was not fully appreciated until the 1950s, following publications by Doll [24] and Breslow [25] among others. Asbestos-related lung cancers are clinically indistinguishable from those due to other causes such as cigarette smoking. In the mid-1960s, Selikoff and colleagues reported an added effect of tobacco smoking on the risk of lung cancer in asbestos insulation workers. [26] The effects of smoking and asbestos exposure on lung cancer risk are synergistic, meaning that the combined risk for the development of lung cancer is significantly higher than the sum of the individual risks. Like asbestosis, lung cancer has mainly been observed in people with high occupational exposure to asbestos, rather than as a result of low-level environmental exposure. [21] Nonetheless, the risk should not be considered to be completely absent in the non-occupational environment, particularly among tobacco smokers, in whom the lung cancer risk is markedly amplified above that of non-smokers for the same level of asbestos exposure.

2.4 Mesothelioma

Mesothelioma is an uncommon, aggressive cancer of the mesothelium, which lines the pleural, pericardial, and abdominal cavities and the outer surface of the lungs, heart, and abdominal organs. The strong link between asbestos exposure and development of malignant pleural mesothelioma

was first made by Wagner in 1960 [27] and supported by the work of Selikoff. [28] In 1986 the US Environmental Protection Agency (US EPA) concluded that the risk of death from mesothelioma was directly related to the length of time since the start of a person's occupational exposure to asbestos. [29] The increasing incidence of mesothelioma since the mid-1970s follows the earlier trend of increasing widespread use of asbestos. The etiological link between asbestos and mesothelioma is now well documented, such that mesothelioma is considered a clinical sign indicating asbestos exposure, although there is a very low background rate independent of known asbestos exposure.

The crude background incidence rate for mesothelioma is estimated at ≤1-2 per million people per year. [30] Over the period 1994-2008, a total of 95,253 mesothelioma deaths were reported to WHO from 83 countries, equating to an age-adjusted death rate of 4.9 per million per year. The mortality rate more than doubled during the 15-year study period, probably reflecting both better disease detection and a real increase in incidence. The mean age at death was 70 years. [30]

A high incidence of mesothelioma was observed in men born around 1945-1950 throughout Western Europe, reflecting the extent of asbestos use in the 1960s and 1970s when this cohort was entering the workforce. [31] Mesothelioma does not just affect workers in the asbestos industry; it has affected brake mechanics (chrysotile was commonly used in brakes until mid-1980s in US), [32] railway workers, and construction trades. [33, 34] Many high-risk occupational exposures and activities have now ceased. A large proportion of people currently dying of mesothelioma have previously worked in building construction and maintenance, and this sector now constitutes the largest occupational risk group (see section 5 on exposures/risk assessment).

Most cases of mesothelioma are associated with asbestos exposure, but some are not. [35] The only other recognised risk factor for pleural mesothelioma is exposure to erionite, a naturally-occurring fibrous silicate mineral with similar structure to amphibole asbestos but different chemical and physical properties [36] Erionite is present in some volcanic ash deposits in New Zealand, Germany, Russia, Japan, Kenya, Turkey, Italy, and in the western United States. A very high incidence of mesothelioma was observed in the 1970s in several villages in Turkey, where erionite was present in zeolite stones used to build houses. The annual incidence was 800 cases/100,000 population, which is 1000 times the rate observed in the general population of industrialised countries. [37] The potency of erionite as a human carcinogen appears to be higher than that of asbestos, particularly for the development of mesothelioma.

While there is evidence that a true 'background' incidence of mesothelioma exists, [33] under-reporting of asbestos exposure and/or possible misdiagnosis of malignant mesothelioma (because the diagnosis can be difficult to establish) may account for some presumed non-asbestos related disease. [38] Because mesothelioma has been noted in individuals with relatively low exposure to asbestos, the incidence of this disease is considered the most sensitive indicator of asbestos exposure in a population.

2.5 Other cancers

Epidemiological studies have shown associations between asbestos exposure and cancers of the oropharynx, larynx, oesophagus, stomach, colon, rectum and ovary [39] In each case the evidence is less substantial than for asbestosis, lung cancer, and malignant mesothelioma. An IARC Working Group in 2012 concluded that a causal association is clearly established for cancers of the larynx and ovary [21]. Since inhaled asbestos fibres pass through the larynx, they may become deposited there. Asbestos fibres have been found in the ovaries of women who were exposed to asbestos either in an

occupational setting, or from residing in a contaminated asbestos mining area or living with an asbestos worker. However, the route by which asbestos fibres reach ovarian tissue has not been clearly established. [40] Causal associations between asbestos exposure and risks of other cancers have not been confirmed.

3. Mechanisms of asbestos toxicity

Asbestos fibres cause damage when inhaled into the lungs, where they can penetrate deep lung tissue and remain deposited for many years, exerting fibrotic, inflammatory and mutagenic/carcinogenic effects. These effects are modified by factors that determine the respirability (potential for inhalation into the small distal airways), bioactivity, and clearance of fibres from the lungs.

3.1 Determinants of toxicity

While all types of asbestos share the same hazards, i.e. the potential for lung cancer, asbestosis and mesothelioma, they have varying degrees of risk - the likelihood that disease or death from the hazard will occur. The physical and chemical makeup of fibres, including crystallinity, surface reactivity, and the presence of transition metals, determines fibre stability in the body and the biological response to the contaminant, and therefore influences the carcinogenic potential of a particular fibre type. [19] Crocidolite is an iron-rich asbestos fibre that is considered the most pathogenic for causing mesothelioma. [41] Critical determinants of asbestos toxicity are fibre dimensions, dose and durability.

Dimensions

For measurement purposes, asbestos fibres are defined as having a minimum length of 5μm and an aspect ratio (length to diameter) of ≥3:1. The most important property of asbestos for respirability is fibre diameter. Smaller diameter fibres (<0.5 μm) exhibit greater penetration to distal portions of the lung, because they can align longitudinally in small airway passages and reach the alveoli. Respirability and deposition are also determined by fibre length - although shorter fibres are respirable, they can be engulfed by macrophages and removed, whereas longer fibres cannot. [19] Animal studies demonstrate that long, thin fibres are more pathogenic than short, coarse/thick ones, [42] though fibres of all lengths have the potential for toxicity. [43]

Chrysotile fibres have physical characteristics that are unique among the asbestos types, and that greatly influence its aerodynamic properties and respirability. Whereas amphiboles exist as single fibres in air, chrysotile fibres tend to clump together, meaning they are less readily transportable to the deep lung airways compared with amphibole fibres.

Dose

The intensity and/or duration of exposure influences the capacity of macrophages in the lungs to engulf and remove fibres. Short but intense exposures can overwhelm the lungs' capacity for clearance, allowing more fibres to become deposited. However, even with low dose exposure, asbestos fibres can accumulate in the lungs over time, so the duration of exposure is an important factor in assessing the asbestos fibre lung burden.

Both cohort and case-control studies have demonstrated a dose-response relationship between asbestos exposure and risk of mesothelioma. There is no evidence of a threshold for the carcinogenic effect of either amphibole or chrysotile types of asbestos; in theory even very low doses could trigger pathogenic reactions in the lungs, eventually leading to cancer, but the risks increase substantially with increasing dose intensity and duration of exposure. It appears that mesothelioma can be triggered by lower exposures than those that lead to lung cancer or other cancers. In contrast, very high intensity exposures are required to trigger asbestosis. [16]

Durability/biopersistence

Fibre durability relates to how fast a fibre will dissolve in body fluids, and other factors that affect its persistence in body tissues. Most asbestos fibres do not dissolve readily in lung fluid. Chrysotile is the most soluble of the asbestos types because of its chemical composition: the magnesium hydroxide content of chrysotile is removed in solution in a time-, temperature- and pH-dependent manner, leaving an insoluble silica skeleton. The amphibole contaminant tremolite is the least soluble of asbestos types, and has been considered one of the most hazardous. The solubility of asbestos types decreases from chrysotile (most soluble) to tremolite (least soluble) as follows: chrysotile > amosite > actinolite > crocidolite > anthophyllite > tremolite [19, 44]

Once inhaled, all varieties of asbestos fibres become deposited throughout the respiratory tract, but often accumulate at bifurcations of larger airways, where lung cancers tend to initiate. Over time after exposure, the average length of retained fibres increases, and diameter decreases, meaning that longer, thinner fibres are cleared more slowly than shorter, thicker ones. [8] The straight, needle-like fibres of amosite and crocidolite asbestos can split longitudinally, becoming thinner, but otherwise are resistant to degradation and can remain in the body for 40 or more years. The very fine fibres can migrate through lung tissue into the pleura. In contrast, curly chryostile fibres tend to degrade chemically, therefore showing shorter residence time in the lung. These factors affect the biopersistence of fibre types, and have implications for their toxicity.

3.2 Biological mechanisms

While asbestos has long been classified as carcinogenic, [45] the exact mechanisms through which asbestos fibres exert their carcinogenic and other effects have not been fully elucidated. Some identified mechanisms include macrophage activation, inflammation, generation of reactive oxygen and nitrogen species (ROS and RNS), tissue injury, genotoxicity, changes in chromosome number, and altered gene expression affecting cell survival and proliferation. [21]

Carcinogenesis is a multistage process. Both direct and indirect fibre genotoxicity can cause mutations that allow the initial escape of cells from normal growth control and promotion and progression of tumour growth. Over time, a series of oncogenic events occurs that leads progressively towards more invasive cancer. The known synergism between asbestos and tobacco smoke for the development of lung cancer but not for mesothelioma suggests that the mechanism for carcinogenicity of asbestos fibres may differ in different target cells. [46]

4. Asbestos use in New Zealand

Asbestos importation to New Zealand began in the late 1930s and peaked in 1974, when the annual amount imported totaled more than 12,000 tons. Imports declined rapidly after this time. There was some limited mining of raw chrysotile asbestos near Takaka in the 1950s, but it was of poor quality and had to be mixed with imported asbestos. ACMs came into New Zealand before World War II as wall claddings, pipes, and cements. In 1938 and 1943 two ACM manufacturing plants were established in New Zealand (in Auckland and Christchurch). These industries mainly manufactured asbestos-cement building products containing around 5 to 15% asbestos. [47] From around 1960, the predominant asbestos type used in buildings in New Zealand and most other industrialized countries was chrysotile. Smaller amounts of crocidolite and amosite were used in building products prior to 1960. [48]

In addition to its construction uses, asbestos was used in New Zealand for machinery insulation, insulating tapes and cloths, gaskets and seals (particularly in the aviation and marine industries), and friction materials (e.g. brake linings) for motor vehicles. [49] This report will focus on exposures from products that were used in the construction of residential houses in New Zealand.

In terms of kilograms of asbestos used per capita per year, asbestos use in New Zealand was lower than in many industrialized countries until the 1970s-1980s, when per capita use exceeded that of the USA and the UK, though it remained substantially lower than in Australia, Canada, Germany, and Denmark. [21] The cumulative amount of asbestos imported into New Zealand over time totals more than 200,000 tons, much of which is still in place in buildings, homes, and machinery insulation. [47]

Despite the known health risks, and in contrast to many Western industrialized countries, the use of materials containing chrysotile asbestos is not yet banned in New Zealand, and import of such material is not strictly regulated. The importation of raw crocidolite and amosite asbestos was prohibited by a succession of temporary Customs Import Prohibition Orders (CIPO) beginning in 1984 for amosite and crocidolite and in 1999 for chrysotile. [49] The most recent CIPO expired in 2008, when it was effectively replaced by the Hazardous Substances and New Organisms (HSNO) Act 1996 approval process. All forms of asbestos are regarded as unapproved hazardous substances under HSNO, but are not strictly banned. Theoretically, approval could be sought from the New Zealand Environmental Protection Authority (NZ EPA) to import asbestos into New Zealand, if it could be shown that the benefits outweigh the risks and costs to the environment and public health, but such approval would be very unlikely. Nonetheless, it is possible that some ACMs containing chrysotile asbestos are still entering the country. [50] A recent inventory of product imports noted significant uncertainties and discrepancies in the data and suggested that there may be cases of imported products being incorrectly labeled as containing asbestos, and also of asbestos-containing products that have been declared as asbestos-free. [49] However, a survey that included building industry groups (NZ Building Industry Federation [BIFNZ], Claddings Institute of NZ, NZ Fibrous Plaster Association, Building Research Association of New Zealand [BRANZ]), found that there are very few current uses of ACMs, and in almost all cases (aside from replacement parts for some aircraft), substitutes for asbestos have been in use for a long time. The survey found no evidence or knowledge of imported products containing asbestos, or of any companies supplying ACMs. [49] This is, however, no guarantee that products imported from countries still manufacturing ACMs are asbestos free, whether or not they are labeled as such. Even where bans are in place, imports can slip through. For example, wall tiles imported into Australia from China in 2010 were found to contain tremolite asbestos despite this being a banned substance. [51]

4.1 Asbestos in New Zealand homes

Most New Zealand houses built in the 1940s-60s used tile or asbestos-cement sheet roofing. Asbestos cement was easily moulded, so was ideal for corrugated roofing (e.g. Super-six roofing). As well as being fire resistant, it was also inexpensive, durable, and easy to install. Asbestos-cement cladding in the form of sheets (e.g. Fibrolite) or planks (e.g. Hardiplank) was popular for the same reasons. Cement-based claddings that were installed before 1988 and have a corrugated profile or a dimpled back surface are likely to contain asbestos. Some claddings will last around 50-60 years and may still be sound if they are regularly painted. Uncoated claddings that have weathered or cracked may need to be encapsulated or replaced. [52]

From the 1950s through the 1970s, many asbestos materials were spray-applied, including textured decorative coatings on ceilings and walls that contained chrysotile asbestos. Although phased out from the late 1980s, such coatings are also still in place in many older homes and buildings. Other asbestos building products included vinyl sheet floor coverings ("lino") with a chrysotile paper backing, vinyl-asbestos floor tiles, sprayed fire protection, and roofing membranes.

Specific data on the asbestos content of ACMs imported and used in New Zealand houses is lacking, though it is clear that chrysotile was by far the most extensively used asbestos type. Some asbestos cement or tile products imported from other countries contained amosite and crocidolite in addition to chrysotile. After about 1960, crocidolite was unlikely to be present, but some amosite fibres could be found in ACMs used in the 1960s and 1970s. [53] The lack of certainty on importation and usage of ACMs suggests that a conservative approach to dealing with all ACMs is warranted.

Although asbestos insulation was used extensively in some parts of Australia and elsewhere, home insulation in New Zealand was relatively rare until the late 1970s. The first bylaw requiring insulation in new homes went into effect in Christchurch in 1971-1972 but it wasn't until 1978 that thermal insulation was required for new houses in the rest of the country. [54] Asbestos insulation was only used in commercial buildings in New Zealand, and is unlikely to be found in residential dwellings. [53] Most insulation in New Zealand homes is made of fibreglass or wool-based material rather than asbestos.

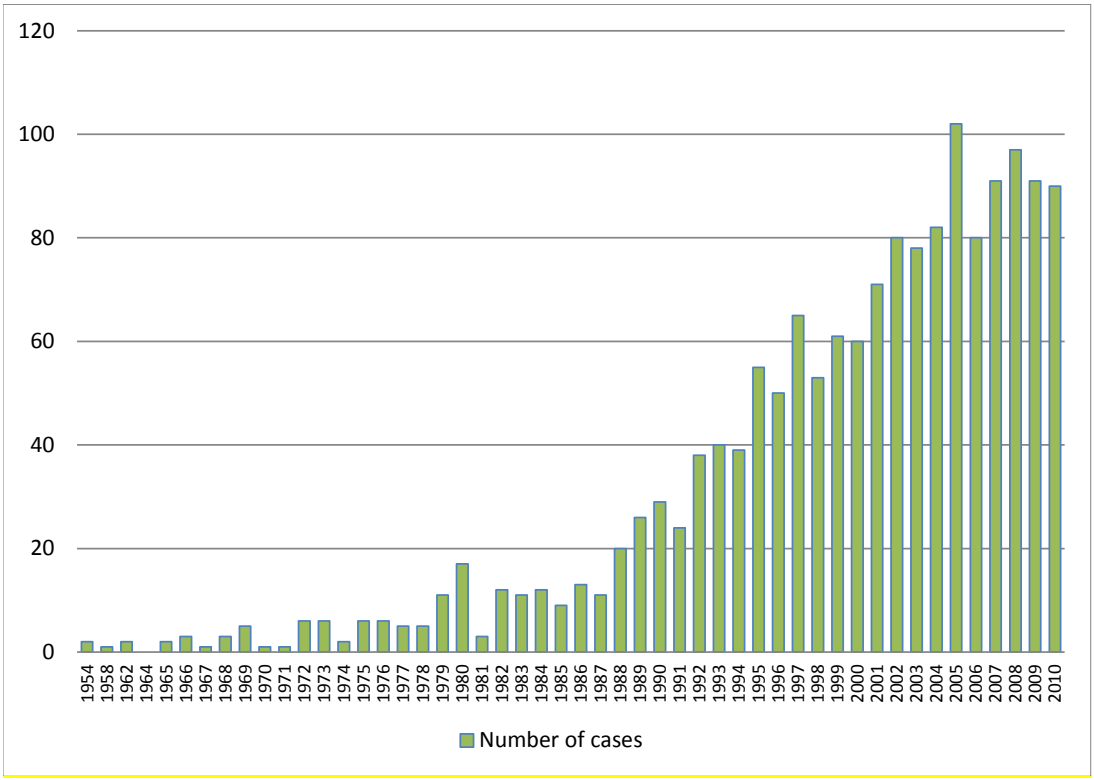
Asbestos products likely to be found in New Zealand in houses built between ~1940 and 1990:

- Profiled or corrugated cement sheets – roofing, wall cladding, weather-boarding, fencing
- Compressed and semi-compressed flat sheet board – as partitioning board, decorative panels, bath panels, soffits, linings to walls and ceilings
- Decorative textured ceilings and walls
- Bitumen-based waterproofing membranes – on flat or parapet roofs
- Asbestos-containing floor coverings –
    - Vinyl-asbestos tiles - chrysotile. Mostly laid on bitumen adhesives that also contain asbestos.
    - Asbestos-paper backed vinyl flooring (lino)

4.2 Asbestos-related diseases in New Zealand

A 1991 report to the Minister of Labour by an Asbestos Advisory Committee led by Professor Bill Glass [55] resulted in the establishment of two asbestos registers in New Zealand: the *Disease Register* and the *Exposure Register*, data from which are used to produce annual reports on asbestos and other occupational lung diseases. [56] The registers were established to raise national awareness of asbestos-related disease. Data from the registers show that mesothelioma incidence has been increasing in New Zealand since the 1970s, in parallel with past asbestos use (see Figure 2). [56, 57] Although the incidence of diagnosis of asbestos disease is continuing to rise, this mainly reflects the legacy of past occupational exposures at levels that are no longer experienced. The registers are based on voluntary notifications, and not all cases of mesothelioma are included, though the recent register data do not differ significantly from the New Zealand Cancer Registry (NZCR), for which notification is mandatory. [58]

Figure 2. New Zealand cases of mesothelioma 1954-2010 notified to the NZ National Cancer Registry (reproduced from the NZ Asbestos Disease Register Annual report 2012 [56] under Creative Commons License: Attribution-NonCommercial 3.0 New Zealand [http://creativecommons.org/licenses/by-nc/3.0/nz/legalcode])

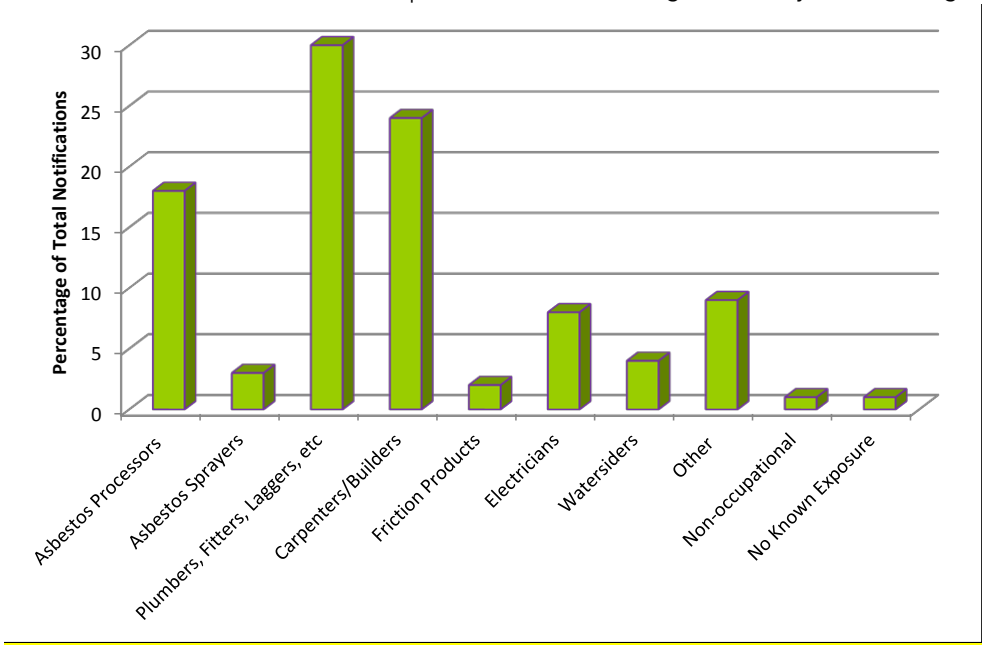


Mesothelioma is primarily a disease of older age; 49% of reported cases in New Zealand since 1994 were in people aged 70 or over. [56] NZCR data indicate that over 68% of individuals registered with mesothelioma in 2011 were in this age bracket. [58] Over 86% of cases were in men, as would be expected from the male-dominated asbestos worker population. Smartt [59] suggested that 20-40% of all adult men are likely to have had some past occupational exposure to asbestos, with over 8,000 having been directly employed in the asbestos industry, and 1,500 exposed in secondary industries utilizing asbestos products. Exposures to women have been mainly non-occupational.

In the 2012 and 2013 reports from the New Zealand registers, [56, 60] mesothelioma is reported twice as frequently as asbestos-associated lung cancer. A recent estimate of the ratio of asbestos-related lung cancers to mesothelioma deaths indicated that twice as many people die from asbestos-related lung cancer as from mesothelioma, [61] suggesting that attribution of lung cancer to asbestos exposure is under-reported in the register. The latest report provides data on notified cases of asbestos-related disease through 2011. The mesothelioma diagnosed currently will mainly reflect exposures in the 1960s and 1970s. The number of cases of mesothelioma reported to the register in 2011 was 78, down from 90 in 2010. [56] The same data are found in the NZCR, which tracks all cancer registrations and deaths in the country. [58] This translates to an annual incidence of mesothelioma in New Zealand of approximately 1.9 cases per 100,000 population (19 per million). Mortality data from the New Zealand Ministry of Health (NZ MoH) indicate that the crude death rate from mesothelioma in New Zealand in 2010 was 22 per million. [62]

Future asbestos-related cancers in New Zealand are projected to involve mainly people employed in building trades who had exposure to ACMs during construction, renovation and remediation projects. Approximately 25% of all deaths of males in New Zealand from 1991-1997 where asbestos was listed as a contributing cause were of workers in building trades. [47] Construction workers including carpenters, plumbers and electricians together represent 67% of all cases of mesothelioma notified in New Zealand (see Figure 3). Unlike asbestos workers of the past, these trades are not always seen as being at high risk, and precautionary practices to minimise potentially harmful asbestos exposures have not always been followed.

Figure 3. Distribution of mesothelioma cases by occupation in New Zealand, as reported in the NZ Asbestos Disease Register [56] (reproduced under Creative Commons License: Attribution-NonCommercial 3.0 New Zealand [http://creativecommons.org/licenses/by-nc/3.0/nz/legalcode])



As workplace exposures decrease (both from decreasing use of asbestos and increasing controls), asbestos-related disease resulting from non-occupational exposure is expected to make up a greater proportion of reported disease, but the absolute numbers will be much lower than they are currently. Only 1% of the reported asbestos-related disease in New Zealand in the 20-year period 1992 to 2012 was attributed to non-occupational exposure. This estimate was based on all categories of disease, including the more common non-malignant conditions (such as pleural disease and

asbestosis), and should be interpreted with caution. [56] Non-occupational exposure would include exposures from childhood where children were brought up in the home of an asbestos worker, and similar exposures to other family members. Such individuals are likely to have had frequent exposure to asbestos dust brought into the home on work clothing. There are as yet no data on exposure from home renovation associated with asbestos-induced disease in New Zealand.

4.3 Comparison with Australia

The environmental exposure situation in Australia is different from that in New Zealand. From the 1950s to the 1970s, Australia had the highest per capita rate of asbestos use in the world, which is now reflected in the country's high incidence of mesothelioma. Both amphibole (crocidolite and amosite) and chryostile asbestos were mined extensively in New South Wales (NSW), South Australia (SA), and Western Australia (WA). Crocidolite mining in Wittenoom, WA, dominated production until 1966. A ban was imposed on crocidolite use in 1967, but chrysotile continued to be mined in SA and NSW until 1983. Raw asbestos was also imported from Canada (chrysotile) and South Africa (crocidolite and amosite), and ACMs were imported from the UK, USA, Germany and Japan. Amosite asbestos was used in construction well into the 1980s in products such as cement board, and was used in friction materials and gasket products until late 2003. [63] Loose-fill crocidolite insulation was used in some houses, and wastes from asbestos plants were used in playgrounds, driveways, and park paths in some mining towns, most notably Wittenoom (crocidolite) and Baryulgil (chrysotile), [64, 65] exposing the general public to potentially dangerous airborne fibre concentrations.

The Australian Mesothelioma Surveillance Program began in 1980 seeking formal voluntary notification of mesothelioma cases and information on occupational and environmental exposure history. [66] In 2012 the Australian Mesothelioma Registry reported data on all people diagnosed with mesothelioma in Australia from 1 July 2010. [67] In the time period between 1 July 2010 and 31 December 2011, there were 942 diagnoses of mesothelioma (612 for the year 2011). The corresponding incidence rate of 2.7 per 100,000 (27 per million) is considered an underestimate.

The use of asbestos and exposure to the general public in Australia would appear to be higher than in New Zealand, thus it is surprising that the difference in reported incidence of mesothelioma in the two countries is not greater. In fact mortality data show a similar pattern. In 2010 there were 642 deaths from mesothelioma in Australia, giving a crude death rate of 29 per million. [68] In the same year there were 94 mesothelioma deaths in New Zealand, with a death rate of 22 per million. [62] The age-adjusted rates (WHO world standard population) were 17 per million and 14 per million, respectively.

As in New Zealand, the job types with the highest asbestos exposure likelihood, and the highest mesothelioma incidence, were in the construction and building trades, followed by electrical and related trades. [67] Where the Australian data differ markedly from that from New Zealand is in the proportion of mesothelioma patients whose exposure to asbestos was considered to be non-occupational (37%, compared with <5% in NZ). The New Zealand data cover a period of 20 years, whereas the Australian data refer only to recent mesothelioma diagnoses, though both would reflect exposures at least 20 years in the past. The differences may partly reflect environmental exposures in mining areas in Australia, which contributed to non-occupational asbestos-related diseases, particularly among women. [69] Self-reported exposure of 'do-it-yourself' (DIY) home renovators to asbestos has been documented in Australia [69] and may be associated with some of the increased risk of mesothelioma observed in the non-occupational setting. The use of ACMs in Australian houses was somewhat different from that in New Zealand. Mesothelioma associated with home

renovation was reported in Western Australia, [9, 70] where crocidolite asbestos was mined and used to a greater extent than in other parts of the country. These individuals are therefore more likely than their counterparts in New Zealand to have encountered crocidolite and amosite asbestos in ACMs during their renovation activities. The differences may also reflect better worker protection against asbestos exposure in earlier years in Australia, such that non-worker exposures made up a greater proportion of the Australian mesothelioma deaths.

5. Asbestos risk assessment

5.1 General concepts

Risk characterization is the integration of information on hazard, exposure, dose-response, and vulnerability to provide an estimate of the likelihood that any of the identified adverse effects will occur in exposed people. Risk assessment relates the hazard of exposure to the probability of exposures reaching certain levels. The product of risk assessment is a statement about the probability that the exposed populations or individuals will be harmed, and to what degree.

A variety of risk assessment methodologies have been developed to assess asbestos risk, integrating toxicology, epidemiology, and mathematical modelling. They involve dose-response assessment, analyzing the extent of human exposure and the incidence of adverse events (asbestosis, lung cancer, mesothelioma, etc). It is clear that with heavy occupational exposure to asbestos, the risk of these events is high. However, the capacity of epidemiological studies to measure risk becomes less reliable as exposure levels fall, in part because very low exposures are more prone to measurement error or inaccurate exposure estimation, and data are limited with regard to cohorts exposed to low doses. A degree of uncertainty in assessing the risk associated with long-term, low-level exposure therefore cannot easily be overcome, as knowledge of dose-response relationships at low exposure levels remains incomplete.

5.2 Asbestos exposure estimates

Evaluating asbestos health risks begins with exposure assessment. However asbestos sampling and measurement techniques are hampered by a number of uncertainties, and significant variability. Retrospective estimation of exposure in relation to risk has involved using job-specific questionnaires, [71, 72] or interviews [73] as well as simulation studies, [74] mathematical modelling, [75] or measurements of asbestos lung burden. [76] Accompanying uncertainties of diagnosis and death certification add to the difficulty of dose-response estimations in asbestos risk assessment.

There is a very large difference in exposure levels in occupational vs non-occupational settings. Because of this it is a common practice to express airborne asbestos fibre measurements in fibres per millilitre of air (f/mL) in the workplace and in fibres per litre of air (f/L) or fibres per cubic metre (f/m^3) for environmental exposure. [77, 78] An exposure of 1 f/mL is equivalent to 1000 f/L. These different units simply reflect different volumetric units and can be interchanged mathematically (1 m^3= 1000 L = 1,000,000 ml). For simpler comparison of non-occupational and occupational exposure levels, this report will convert all dosages to f/mL.

Time variables relating to exposure also differ greatly for occupational versus environmental exposure situations, in that environmental exposures can begin at birth and continue throughout the lifespan, whereas occupational exposures begin in adulthood, and are usually intermittent through a person's working life. Occupational exposures are generally presented as exposures averaged over an 8-hour working day (referred to as a time-weighted-average [TWA]; see section 6.1), whereas environmental exposures are considered to be continuous over a number of years. Non-occupational exposures such as those that can occur during DIY home renovation or maintenance may be intermittent.

Measuring techniques

The relationship between asbestos disease and exposure was established using fibre counts based on phase contrast microscopy (PCM) data from asbestos mines, mills, and factories, and PCM remains the primary method used for monitoring airborne asbestos concentrations and asbestos exposure. In general, asbestos fibres are defined as having a minimum length of 5µm and an aspect ratio (fibre length relative to fibre diameter) of 3:1. However, PCM cannot distinguish non-asbestos fibres of the same size and aspect ratio, and therefore many fibers counted by PCM are not asbestos. [79] In non-occupational settings where large proportions of other fibres are present (gypsum, glass etc) PCM will overestimate the asbestos fibre concentration. The minimum concentration that can be detected by PCM is around is ~0.01 f/mL, which is higher than the usual level found in non-occupational environments. [80]

Transmission electron microscopy (TEM) and scanning electron microscopy (SEM) can count smaller fibres and can differentiate fibre types, but the fibre counting accuracy is relatively low because of the small area that can be scanned at high magnification, resulting in few fibres being counted. Accuracy can be increased by increasing the number of fields counted, but this is costly. Fibre count measurements performed by TEM are at least a factor of two higher (i.e. more sensitive) than those obtained by PCM. [78] This approach is intended to complement PCM. [80]

Understanding asbestos exposure data

- Short-term asbestos exposure/concentration in air is measured in number of fibres per millilitre (f/mL) of air, detected by PCM. For occupational exposures this is expressed as a time-weighted average (TWA) to account for the average concentration over a 4- or 8hr work period. The permissible exposure limit for workers is generally 0.1 f/mL for 4hr TWA.
- Cumulative or long-term exposure is expressed in terms of the concentration of fibres over time, or fibres per mL x years (f/mL•yr)).
- Cumulative exposure can occur over a lifetime (usually estimated as 70 years), or over years of a working life (estimated as 40 years), or may have occurred through one or more intermittent, non-occupational exposures.
- Lifetime exposure can be expressed as fibres per liter (f/L) or fibres per cubic metre (f/m^3) – to calculate f/mL•yr this measure is multiplied by 70 years (assuming continuous [background] exposure to this concentration).
- To convert cumulative fibre years to lifetime exposure units, the value is divided by 70 years; so 5 fibre years equates to a lifetime exposure at an average asbestos concentration in ambient air of 71 f/L or 0.071 f/mL.

Typical asbestos concentrations in air

Asbestos exists in rock formations around the globe, and the natural processes of erosion have been releasing its fibres throughout earth's history. Asbestos is thus naturally present at low levels in ambient air and in water, including drinking water. [1] However, industrial activities have greatly increased the levels of airborne asbestos fibres in some locations and situations. The widespread use of chrysotile asbestos in the past made it a ubiquitous contaminant of ambient air, but usually at very low levels.

The concentrations of asbestos found in indoor air, outdoor air, and drinking water vary widely, and it is not possible to calculate human exposure levels accurately except on a site-by-site basis. Ambient air in rural areas in the US (remote from any special sources of asbestos) typically contains ~0.00001 f/mL of asbestos. Typical levels found in cities are about 10-fold higher. [80, 81] Outdoor air fibre concentrations in the vicinity of industrial sources such as asbestos factories can be around 0.003 f/mL and sometimes as high as 0.01 f/mL or higher near working asbestos mines. [80] Data on typical outdoor air asbestos concentrations around New Zealand are not available.

Asbestos cement products contain up to ~15% asbestos. Cement particles and asbestos fibres are released from weathering surfaces and become dispersed in the air and rainwater. A German study found the corrosion velocity for uncoated asbestos cement roofing tiles to be ~0.024 mm/year, [82], with the majority being washed out by rainwater. The large amount of existing asbestos cement products on buildings probably contributes to a significant release of asbestos fibres into the total environment each year.

Historical workplace exposure levels

Workplace airborne asbestos concentrations experienced in the 1950s were up to 200 f/mL in asbestos cement production factories (Germany), but as a result of the implementation of stricter regulations by the 1990s, typical concentrations were in the range of 0.3-0.7 f/mL in the same industries. [83] Exposures of even this magnitude are still above most current occupational standards (see section 6). This is important to bear in mind when analysing trends in asbestos disease incidence and assessing risks.

Disaster exposure

There has been concern over the potential risk to building occupants resulting from exposure to airborne asbestos released from ACMs damaged in natural disasters such as earthquakes. [84] Following the Loma Prieta earthquake on the central California coast in 1989, indoor air samples from buildings including schools, public and commercial buildings, and residences, collected between 1 and 5 days after the quake averaged around 0.0001 f/mL, with no significant difference between indoor and outdoor air. [84] The samples had been taken from building locations within buildings that were deemed to be the greatest potential source of airborne asbestos from the disruption of ACMs, so these findings offer some assurance that exposures in such situations are not substantial. However, ongoing exposure to low-level asbestos dust adds to an individual's cumulative exposure and should not be dismissed – careful clean up and removal of asbestos debris is important.

Renovation of damaged older homes has the potential to mobilise asbestos dust, allowing respiratory exposure, however data on such exposures are very limited. A study of flood-damaged homes in Cedar Rapids, Iowa, found levels of asbestos around 0.02 f/mL (range 0.010-0.06 f/mL) during remediation and 0.03 f/mL (range 0.01-0.08 f/mL) after remediation was complete.[85] The levels were all below the workplace permissible exposure limit in US (0.1 f/mL) despite the advanced age of the homes and the extensive nature of remediation.

In 1977, the IARC warned that "increasingly important exposures can be expected from building demolition and waste disposal."[45] However, demolition of small buildings containing ACMs does not necessarily result in significant release of fibres; this can be controlled if the materials are thoroughly wetted during the procedure. [86] A large-scale tragic "test" of possible exposure from building demolition occurred after the collapse of the World Trade Center towers in 2001, when the US EPA determined that asbestos had been "pulverized to ultra-fine particles" [87] Residences in the vicinity were professionally cleaned to remove possible asbestos dust. The EPA established a benchmark prevention criterion of 0.0009 f/mL of air in houses for all forms of asbestos – if levels in residences exceeded this limit, they would be re-cleaned. The benchmark level was set based on an estimated increase in cancer of 1 in 10,000 that would result from residential exposure (168 hours per week) at that level over a period of 30 years. [87]

Exposure to asbestos in buildings

The potential risk to building occupants posed by the presence of 'in place' asbestos in building materials has been the subject of much debate, but in general it is concluded that in-place ACM does not result in elevated airborne asbestos concentrations if the material is undisturbed. Airborne asbestos concentrations measured in homes, schools, and other buildings that contain asbestos range from about 0.00003 to 0.006 f/mL. [80] Even if the ACM is old, such asbestos concentrations do not generally approach regulatory threshold limits (see section 6). [88]

A study conducted in 1969-70 found that in a number of US urban schools that had visible damage to sprayed-on asbestos coatings (ceilings), the indoor air asbestos fibre concentrations were similar to ambient outdoor air. [89] A study of exposures conducted by the Health Effects Institute – Asbestos Research (HEI-AR) similarly found that indoor and outdoor air fibre concentrations were roughly comparable in both the US and the UK where the buildings contained ACMs. [90] A more recent large survey of 752 buildings in the US containing ACMs under conditions of normal occupancy (i.e. including maintenance) also found that most had indoor air asbestos concentrations that were not significantly different from outdoor levels. Maintenance worker exposures were generally well below US regulatory levels. [88]

Thus, ambient air sampling from outdoor air and air inside buildings containing ACMs shows that asbestos dust concentrations are similar, suggesting that ACMs in buildings generally pose no greater risk to occupants than would the air outside. Nonetheless, the main source of non-occupational exposure to asbestos currently, and that with the greatest potential for exposure in the future, is the release of fibres from deteriorating ACMs in public buildings or homes, or from disturbance of ACMs during building repair or renovation. [91] Data on housing-related risks to public health from asbestos exposure are currently minimal, but very low mean fibre concentrations have generally been recorded. [90] Random fibre release episodes, whether from repair/maintenance activities or from "falling or dislodging" of ACM, do not substantially increase average building concentrations, although these activities or events can potentially result in increased exposure to an individual who is undertaking such work or is present nearby. The health risk to most building occupants appears to be very low. [88, 90]

Exposures in construction and maintenance trades

Data on exposures to construction and building maintenance workers are relevant to the issue of exposures during home renovation and repair, as they provide information on activities that may occur during the renovation process. The HEI-AR survey (1991) found that in the absence of respiratory protection, construction workers removing, repairing or replacing ceiling tiles, or repairing roofing, drywall, or flooring containing asbestos, had exposure levels ranging from 0.01 to 1.4 f/mL (time-weighted average). [90]

Measurements taken by the Health and Safety Executive (HSE) UK indicate similar levels of exposure of workers removing ACMs, but note that the exposure from removal of sprayed insulation products is very high, even under controlled conditions. (see Table 2) [92] Sprayed asbestos insulation is generally not found in New Zealand houses.

Table 2. Average personal airborne concentration of asbestos fibres during removal of ACMs – modified from [92] –Health and Safety Laboratory UK

Product group	Controlled wet removal/good practice [f/mL]	Limited controls/ dry removal [f/mL]
Sprayed and other insulation products	14.4	358
Asbestos insulating board	0.41	15
Textured coatings	0.02	0.08
Asbestos cement	0.01	0.08
Flooring	0.01	0.05

The asbestos content of dry wall sheets can be up to 25-35%. Analysis of exposure to workers following US Occupational Safety and Health Administration (OSHA) and US EPA asbestos dry wall abatement procedures for the construction industry indicated asbestos exposures of 0.85 f/mL. These exposures are above the OSHA permissible exposure limit (PEL) of 0.1 f/mL and require use of a respirator (see section 6). [93] The probability of overexposure for dry wall material was considered low using half- and full-face masks, and it was not likely that workers would receive a large dose. Pre-abatement and final clearance air samples were all below 0.01 f/mL. Exposure from abatement of vinyl floor tiles was lower than for dry wall and was found to be below the OSHA PEL when proper procedures were followed. [93] The exposure levels nonetheless suggest that homeowners should be cautious about performing any work on dry wall material or vinyl floor tiles in older homes that may contain asbestos.

5.3 Asbestos risk estimates

Asbestos has long been classified by the IARC as being carcinogenic to humans, [45] and it is clear that high and long-term exposure in workplaces in the past has resulted in a large number of asbestos-related deaths. The occurrence of asbestosis and lung cancer correlates with cumulative exposure (f/mL•yr: the product of concentration [f/mL] multiplied by years of exposure). However, assigning a risk level to lower exposures encountered today is not straightforward. Accurate and meaningful exposure measurement is difficult. Because increased cancer risks have been observed in populations exposed to low occupational levels of these mineral fibres, the International Labour Organization (ILO) and the WHO have concluded that "*there is no evidence of a threshold for the carcinogenic effect of both chrysotile and amphibole forms of asbestos.*" [20]

The concept that very minimal exposure could potentially trigger tumour initiation is based partly on the potential genotoxic effect of asbestos fibres (see section 3.2). Genotoxic agents are considered to have no threshold because it is assumed that even a single molecule (or fibre) of a genotoxic carcinogen may cause a mutation that could initiate a neoplasm, although the increase in risk may be infinitesimally small. [94] However, the mechanisms of fibre genotoxicity appear to act predominantly via effects on chromosome number or indirect damage via generation of ROS and RNS during an inflammatory reaction, rather than DNA sequence changes. Thus, the applicability of the one-fibre theory to asbestos carcinogenicity is uncertain, and is not relevant to the practical assessment of health risk except to emphasise the importance of avoiding exposure as much as possible.

Cumulative exposure concept

The cumulative exposure concept suggests that the risk of cancer is directly related to the cumulative asbestos exposure received over a period of time. This concept assumes that the effect of an exposure to 100 f/mL for 1 hour is the same of that of 1 f/mL for 100 hours. However, this assumed equivalence applies only for short time periods, because of the long latency between accumulated exposure and cancer risk. [29] A given cumulative exposure accrued over a short period is expected to result in a higher risk than the same exposure accrued over a longer period if the exposures commenced at the same time. For example, exposure to 100 f/mL beginning at age 30 for 1 year carries a higher risk than exposure to 2 f/mL beginning at age 30 for 50 years, even though the cumulative exposure is the same. [90, 95] This is because a substantial portion of the longer exposure will occur at older ages and therefore contribute less risk than if all the exposure occurred earlier in life. Asbestos-related lung disease has been reported in workers occupationally exposed to 5 to 1200 f/mL•yr, which equates to 40 years of exposure to asbestos concentrations ranging from 0.125 to 30 f/mL. [80]

Using time working in an amosite asbestos factory as a measure of exposure dose, Selikoff and coworkers estimated in 1979 that workers with short, intense exposure (23 f/mL over 1 month) had an increased risk of respiratory cancer, and found that the lower the 'dose', the longer the latency and the smaller the magnitude of the effect. Their conclusion was that where it is not possible to avoid every exposure to carcinogenic agents, reducing the exposure can both delay the occurrence and lower the frequency of occurrence of adverse events. They also found that if heavy direct exposure occurred in men 'already at cancer age', the latency of the effect of exposure was much shorter. Thus, the length of the latency period depended on exposure dosage and to some extent, on the age at which exposure occurred. [96] This relates to the underlying susceptibility to cancer that increases with age. Children are no more susceptible to asbestos-induced cancer than are adults, but they have potentially a much longer lifespan to experience the cumulative and increasing risk. People exposed as children are thus at higher risk of developing asbestos-related cancer than their peers who have been exposed to the same levels later in life.

Application of the cumulative exposure concept to low-level, non-occupational exposures suggests that relatively high but short exposures, which add to the total cumulative asbestos exposure of an individual, may be significant for elevating disease risk. There is no evidence that episodic peaks in exposure at the low levels encountered during maintenance or repair of ACMs have a specific effect on disease risk, although they would add to cumulative exposure. [80]

Differences among fibre types – chrysotile vs. amphibole

Risk assessments for asbestos-related cancer often use knowledge of the type of asbestos in addition to the intensity and duration of exposure (the cumulative exposure), based on differences in the biological potential among the various asbestos fibre types. [97] Chrysotile asbestos is considered less potent than amphibole types, especially for mesothelioma, although this remains a subject of some debate. [98, 99] Studies of workers exposed mainly to chrysotile asbestos have found a high proportion of amphibole fibres in their lungs, despite amphibole fibres comprising a very low proportion of the asbestos to which they were exposed. [100, 101] This reflects the substantially faster clearance of chrysotile from the lungs, and has been taken to suggest that cancers occurring in chrysotile workers are actually caused by amphibole contamination. However, a study in China found that occupational exposure to pure chrysotile was associated with an increased risk of lung cancer and mesothelioma, [102] and more recently, a large cohort of chrysotile textile workers confirmed exposure link to lung cancer and asbestosis. [103]

There is some evidence suggesting that chrysotile asbestos is less potent than amphiboles at inducing lung cancer, although this remains a matter of debate. Exposure-response comparisons

suggest chrysotile workers are at lower risk than amphibole workers at similar exposures. Based on the exposure–response estimate of the US EPA, the lifetime risk of an asbestos-induced lung cancer in smoking male workers exposed for 20 years to 20 f/mL of air in primarily chrysotile industries was about 2%–10%, compared with 40% in smoking male workers in industries using amphiboles. The risk in nonsmoking asbestos workers was about 15 times lower in both cases. [29] A meta-analysis by Hodgson and Darnton [95] of 17 occupationally-exposed cohorts concluded that there was a difference in lung cancer risk for chrysotile vs amphibole exposure of between 1:10 to 1:50. However, there was an unexplained difference in risk between cohorts of chrysotile miners and millers in Quebec and textile workers in South Carolina of nearly 100-fold. Berman and Crump's [104] meta-analysis of 15 cohorts also found that for thin fibres (less than 0.2μm diameter) chrysotile fibres were less potent than amphiboles for risk of lung cancer. The IARC noted significant heterogeneity in these meta-analyses and determined that it was not yet possible to draw any firm conclusions concerning the relative potency for lung cancer of chrysotile vs amphibole fibres. [21]

There is clearer evidence that the potency differs for induction of mesothelioma, which is the tumour most relevant to consideration of (very low) non-occupational exposures. As previously mentioned, cohorts exposed to mainly chrysotile asbestos showed an increased risk for mesothelioma over background rates, but the chrysotile contained some amphibole fibres. [100, 101] A South African case-control study found no cases of mesothelioma in individuals exclusively exposed to chrysotile, but did find an association with exposures to crocidolite and amosite. [105] The IARC reported estimates of relative potency based on the meta-analyses of Hodgson and Darnton [95] and Berman and Crump, [104]. The first authors estimated that the ratio of the potency for mesothelioma was 1:100:500 for chrysotile, amosite, and crocidolite. The other group estimated that the relative potency of chrysotile was in a range from zero (no potency) to about 1/200th that of amphibole asbestos. [21]. The IARC Working Group commented, however, that there is a high degree of uncertainty concerning the accuracy of these relative potency estimates because of the potential for exposure misclassification in these studies.

Hodgson and Darnton [95] developed a model to determine the mathematical relationship between asbestos exposure and subsequent risk of lung cancer and mesothelioma, depending on cumulative exposure and fibre type. The model can be used to differentiate between the relative magnitudes of risk, and may allow extrapolation to other scenarios for which data are not available (see table 3). However, the results are estimates only – the numerical form may suggest more confidence in the accuracy of the estimate than is warranted. The model may be less reliable when extrapolating beyond the exposure ranges for which there are epidemiological data, due to uncertainties in the dose-response relationship at lower levels.

Table 3. Estimated lifetime (to age 80) risk of asbestos related cancer per 100,000, for cumulative asbestos exposures accrued over 5-years from age 30

Cumulative exposure (f/mL•yr)	Continuous exposure level (f/mL)	Crocidolite risk (range)	Amosite risk (range)	Chrysotile risk (range)
10	2.0	5600 (3200 - 8400)	2300 (960 - 4000)	56 (23 – 340)
1	0.2	750 (250 – 1600)	180 (35 – 570)	6 (1 – 45)
0.1	0.02	120 (24 – 360)	21 (2 – 100)	1 (0.1 – 7)

*Based on Hodgson and Darnton [95] best-slope model with maximum and minimum estimates based on the range of predictions consistent with the high-slope and low-slope models.

Assessing risks of non-occupational exposure

The assessment of risks of low-level asbestos exposure has had to rely on extrapolation from studies of more heavily exposed occupational groups. Although there is considerable uncertainty about the magnitude of risks at low doses, it is clear that the risks are very substantially lower than those at higher occupational levels. Several attempts have been made at estimating minimal risk thresholds, although it is generally accepted that there is no level of exposure that is absolutely safe with regard to carcinogenic potential. Risks differ not only on the basis of intensity and duration of exposure, but also depending on the type of asbestos to which an individual is exposed, and the possible underlying risk factors or susceptibilities of the individual (see section 1.3). The matter is further complicated by the inevitable but very low exposure to asbestos in the natural environment.

Asbestosis

Asbestosis is an outcome of very high exposure to airborne asbestos fibres. Evidence suggests a cumulative exposure threshold fibre dose of approximately 25-100 fibre years, below which asbestosis is not seen. This level is equivalent to exposure to 1 f/mL continuously for 25 years. [106] According to the US EPA's 1986 airborne asbestos health assessment update, [29] for workers exposed after 1950, the risk of developing asbestosis is less than 1% from an exposure to 0.7 f/mL for 40 years. Current non-occupational exposure levels are considered to be too low to cause asbestosis.

Lung cancer

Lung cancer incidence attributable to asbestos exposure is difficult to quantify, because lung cancer has several other contributing factors as well. The magnitude of lung cancer risk from asbestos exposure appears to be a complex function of a number of parameters, the most important of which are: (1) the level and the duration of exposure; (2) the time since exposure began; (3) the age at which exposure began; (4) the tobacco-smoking history of the exposed person; and (5) the type and size distribution of the asbestos fibres. [80]

As there is a substantial background incidence of lung cancer due to factors other than exposure to asbestos (mainly cigarette smoke), the risk attributed to asbestos exposure is often presented in terms of relative risk (RR). This is also known as a risk ratio. The RR expresses how many times more likely an exposed person is to develop the disease compared with an unexposed person. A RR of 1 means that the exposure has no effect on the risk of the outcome (in this case lung cancer). A RR >1.0 signifies an increased risk of the outcome following exposure, whereas an RR <1 would indicate a reduced risk of the outcome following exposure.

Relative Risk (or Risk Ratio):

$$RR = \frac{\text{Incidence of outcome following exposure}}{\text{Incidence of outcome without exposure}}$$

Relative Risk Increase (RRI) = |1 – RR| x 100

Example

If RR = 1.2

- The outcome is 1.2 times more likely in the exposed group
- RRI = |–0.2| x 100 = 20% increased risk in the exposed group

Final risk = baseline risk x RR

A recent mathematical modelling study of 'low-level' exposure estimated the relative risk (RR) of lung cancer to be only 1.013 for a cumulative exposure of 4 f/mL•yr – equivalent to a background (i.e continuous) exposure of 0.057 f/mL over a 70 year lifespan. [107] A cumulative exposure of 40 f/mL•yr (0.57 f/mL lifetime exposure) had an estimated RR of 1.133. The interpretation of RR = 1.013 is that persons with the stated cumulative exposure (4 f/mL•yr or 0.057 f/mL lifetime exposure) are at 1.3% greater risk of developing lung cancer than unexposed persons. Assuming that there was a lifetime risk of developing lung cancer in the population of 6.75% (675/10,000), this equates to approximately 8 to 9 additional cases of lung cancer per 10,000 lifetimes in exposed groups (675 x 0.013 = 8.8). Those with 40 f/mL•yr cumulative exposure are at 13.3% increased risk of developing lung cancer in their lifetime. In a group thus exposed, approximately 90 additional cases per 10,000 lifetimes would be expected above the background lung cancer rate. The modelling suggested that the risk of lung cancer from exposure to chrysotile asbestos was about one-third of that for amphibole asbestos. Some other studies have suggested a larger difference (see below).

The study described above considered a cumulative exposure of 4 fibre years to be a 'low-level' asbestos exposure. Cumulative lifetime exposures experienced in the non-occupational environment are usually very much lower, and in such settings, lung cancer is not generally reported as attributable to asbestos exposure. The risk of lung cancer associated with exposure to asbestos at current environmental levels in the home is expected to be extremely low.

Mesothelioma

Unlike the multiple contributing factors associated with lung cancer, the risk of mesothelioma is almost exclusively attributed to asbestos exposure. The association of mesothelioma with occupational exposure to asbestos has been clearly established, and it is generally accepted that mesothelioma can be observed at lower asbestos exposures than those that are known to increase the risks of other asbestos-related diseases. Reports of mesothelioma resulting from exposure to asbestos in the non-occupational setting continue to appear, [108] although most involve environmental exposures related to residence near asbestos mines or processing plants. [75, 109] The first reports of increased mesothelioma risk in people who did not have workplace exposure to asbestos occurred in family members of asbestos workers, often those who washed the workers' dust covered clothing. [110] Exposure estimates have not been reported in such populations, so it is difficult to relate these risks to other non-occupational exposures.

Environmental exposures in the vicinity of asbestos mines significantly increase the risk of mesothelioma. One study of women living near a Canadian asbestos mine found a 7-fold increased mortality rate from pleural cancer in the absence of any occupational exposure. [111] The estimated risk of developing asbestos-related cancer from living near a productive asbestos mine for 30 years was approximately 1:10,000 [112] Yet even these exposures are considered 100,000 times lower than past heavy industrial exposures.

Iwatsubo et al. [113] carried out a mesothelioma dose-response assessment to determine the risk associated with low (<1 f/mL) and sporadic (<5% of work time) occupational asbestos exposure. The cumulative exposures were considered low – 23% of cases were exposed to <0.5 fibre years (f/mL•yrs). Mesothelioma risk increased with frequency of exposure, but subjects with sporadic exposure were not at greater risk of mesothelioma than were controls. [113]

Risk assessment in New Zealand – the Canterbury Home Repair Programme

A number of concerns have been raised about the level of asbestos monitoring and care taken during remediation of damaged houses in Christchurch following the Canterbury earthquakes. As mentioned above, ACM removal and repair processes by their nature disturb and release asbestos fibres, potentially resulting in elevated exposure and health risks. The use of proper abatement and

cleanup procedures can reduce these risks. For example, most asbestos removal procedures involve wetting the surface to reduce the release of dust. Dry scraping or sanding of ACMs should be avoided.

To evaluate whether exposures may have been elevated to dangerous levels in Christchurch, a simulation study was conducted to determine levels of exposure generated using sub-optimal abatement procedures (i.e. dry scraping) in removing textured asbestos coatings from walls and ceilings in three damaged Christchurch homes. [114] The removal of textured coatings is representative of a significant proportion of the repair work carried out as part of the Canterbury Home Repair Programme (CHRP). The aim was to establish a range of exposure values and apply them to a published risk formula to estimate the level of risk to exposed workers.

The removal of textured coatings had previously been studied extensively in the UK as part of the Regulatory Impact Assessment for a proposed new Control of Asbestos at Work 2006. [115] In that study, the overall mean fibre concentration during simulated 'worst case' removal procedures was 0.08 f/mL, with an average sampling time of approximately 2.5 hours. The results indicated that textured asbestos coating removal was associated with a relatively low asbestos exposure risk.

The Christchurch air sampling simulation study was conducted in a similar manner to the UK study. It was carried out over three days in three separate homes where textured coating removal was conducted by specialist contractors. The simulation study was designed to reflect the nature of previous removal work (in terms of duration and dust production) that had been carried out on Christchurch houses in the first year after the earthquakes, before stricter procedures for asbestos monitoring and abatement were fully operational.

The simulations were meant to provide exposures in the 'worst-case' situation – dry scraping with no extraction and small room volume. Over a 60 minute period the PCM airborne chrysotile fibre concentration was estimated to be just below 0.1 f/mL – this was considered typical of peak exposure that would be experienced in non-test situations.

The average 10-minute exposure value was 0.76 f/mL for dry scraping, and 0.64 f/mL for cleanup activities, both of which are well below the NZ 10 minute control limit of 6 f/mL. These values were used to calculate a conservative cumulative exposure estimate for full-time removal over an entire 8 hr period, six days per week for three years. The lower end of the cumulative exposure range was calculated at 0.54 f/mL•yr and the upper end was 1.7 f/mL•yr. The increased lifetime risk of lung cancer from these exposures was estimated at 0.0006% to 0.0017%, or between 6 and 17 new cases among 1 million workers. The excess risk estimates for both lung cancer and mesothelioma were considered to be consistent with existing background risks in the everyday environment.

The calculations in this study are likely to overestimate the actual exposure and risk to workers, who would normally carry out tasks such as removal of textured ceilings over a ~2 hour period and not as a full-time job. Homeowners and housing occupants are unlikely to experience anything close to the simulated exposure scenarios during the course of their home remediation activities. Although no threshold can be robustly established, for practical purposes there is a level of exposure below which the risk from asbestos is too small to be distinguished from the background risk. It should be noted, however that the simulation study was based on sampling from only 3 houses and only involved removal of textured ceilings. It is possible that work involving other types of ACMs could generate different exposure levels, and work with power tools might result in significantly higher levels.

A summary investigation report on the CHRP procedures in relation to the repair or removal of ACMs [116] concluded that while the management of asbestos in the first year after the Canterbury

earthquakes did not fully comply with regulations, the resulting exposures were likely to be well below the workplace exposure standard even for full-time abatement work. Therefore the risk to homeowners is likely to be very low. It is, however, still important to ensure that work areas are properly cleaned after remediation work is complete, so that any possible exposures within the home are not prolonged.

A further issue that has been raised with the public was exposure to dust at the time of the earthquakes themselves. Most of that dust originated from the liquefaction and ground disturbance and not from ACMs, and even when it involved building materials, the transient exposure to asbestos, while unmeasured, was likely to have been minimal.

6. Asbestos regulation: managing the risk

In efforts to reduce or avoid the potential risk of harm from asbestos exposure to workers and the general population, asbestos is now a regulated substance and is banned completely in many countries. European legislation prohibits the use, reuse, sale, supply, and further adaptation of materials containing asbestos fibres. There have been many calls for a worldwide ban on all forms and uses of asbestos. [117-119] The WHO and the ILO set out an outline for the development of national programmes for elimination of asbestos-related diseases, [20] which is mainly concerned with countries that are still using chrysotile asbestos, but also addresses efforts to prevent asbestos-related diseases arising from exposure to the various forms of asbestos already in place, and as a result of their use in the past (see box). WHO member countries in Europe agreed in the Parma declaration of 2010 to "develop by 2015 national programmes for elimination of asbestos-related diseases in collaboration with WHO and ILO."[120]

World Health Organization outline for the development of national programmes for elimination of asbestos-related diseases

WHO, in collaboration with ILO and with other intergovernmental organizations and civil society, will work with countries towards elimination of asbestos-related diseases in the following strategic directions:

- by recognizing that the most efficient way to eliminate asbestos-related diseases is to stop the use of all types of asbestos;
- by providing information about solutions for replacing asbestos with safer substitutes and developing economic and technological mechanisms to stimulate its replacement;
- by taking measures to prevent exposure to asbestos in place and during asbestos removal (abatement);
- by improving early diagnosis, treatment, social and medical rehabilitation of asbestos-related diseases and by establishing registries of people with past and/or current exposures to asbestos.

6.1 Asbestos regulation in the occupational environment

Risk assessments such as those described in section 5.3 have been used to help set workplace exposure limits in occupational safety regulations. Standards that are set for occupational exposure to hazardous substances are designed to minimize risks, though it should be clear that exposures at

such prescribed levels still involve some element of risk. Standards are meant to be a reflection of an acceptable level of risk. They should be measurable, achievable, and enforceable. The goal is to keep exposures as low as is reasonably practicable to ensure the safety of workers.

Defining an acceptable level of risk

For known carcinogens such as asbestos, exposure levels generally regarded as acceptable by regulators are those that represent lifetime cancer risk to an individual of between 10^{-4} (1 in 10,000) and 10^{-6} (1 in 1,000,000) using information on the relationship between the dose and response. The NZ MoH defines an acceptable level of risk as 10^{-5} (1 in 100,000).

In 2010 the Health Council of the Netherlands performed a reassessment of previous asbestos risk meta-analyses in order to calculate asbestos concentrations consistent with a maximum permissible risk level (MPR; 10^{-4} lifetime risk) and a negligible risk level (NR; 10^{-6} lifetime risk) for mesothelioma and lung cancer. [78] The MPR and NR risk levels are expressions of the likelihood of death from cancer as a result of exposure to asbestos from lifetime exposure at the specified levels. The lifetime exposure in this context is defined as exposure over a period of 70 years. A lifetime exposure to the MPR concentration should result in a lifetime risk of death from cancer of no more than one in ten thousand (10^{-4}), whereas the cancer mortality risk associated with a lifetime exposure to the NR should not exceed one in a million (10^{-6}). It is also specified that a *year* of exposure to the MPR concentration should result in a risk of cancer mortality of no more than one in a million (10^{-6}), and for a *year* of exposure to the NR, the cancer mortality should be ≤1 in 100,000,000 (10^{-8}). The MPR would be equivalent to a workplace exposure standard (WES) or permissible exposure limit (PEL) used in occupational safety regulations (see below), whereas the NR represents an environmental quality objective for asbestos that is 100 times lower than workplace control level.

As part of the Netherlands study, a new meta-analysis was conducted using stricter criteria to determine the suitability of individual studies for inclusion. [78] The analyses confirmed the differences in carcinogenic potential between chrysotile asbestos and amphiboles, calculating that amphiboles were 50 times more potent than chrysotile for the combined outcomes of mesothelioma and lung cancer. The analysis used TEM measurements rather than PCM, the more common technique for measuring workplace exposure, and assumed a 2-fold higher sensitivity for TEM (i.e. values obtained with TEM are twice the values obtained by PCM). The proposed values based on the new analysis are roughly 40 times lower than existing values for chrysotile, and around 30 times lower for amphiboles. The existing MPR and NR levels and the proposed levels based on the new meta-analysis, presented in f/mL PCM values, are shown in table 4. These values represent background (continuous) exposure levels – to calculate cumulative exposure they should be multiplied by years of life (typically 70). The Dutch analysis was done for the purpose of setting public health and occupational health standards, and is informative for identifying risk levels for other populations and exposures.

Table 4. Netherlands - existing and proposed maximum permissible risk (MPR) and negligible risk (NR) values for lifetime exposure based on asbestos types (PCM measurements) [78]

	Existing values (f/mL)		Proposed values (f/mL)		
Risk level	Chrysotile	Amphibole	Chrysotile	Mixed*	Amphibole
MPR	0.05	0.005	0.0014	0.00065	0.00015
NR	0.0005	0.00005	0.000014	0.0000065	0.0000015

*Chrysotile mixed with up to 20% amphibole

Standard exposure control limits

US regulations

The US EPA regulates asbestos as an air pollutant via the National Emission Standards for Hazardous Air Pollutants (NESHAP) [121] Asbestos was identified as a hazardous pollutant in 1971 - notified in the NESHAP in 1973 and comprehensively amended in 1990. Demolition of multiple houses as part of urban renewal projects, highway projects, or for construction of industrial or shopping complexes was included as subject to the NESHAP. The rule requires that asbestos-containing waste material be sealed in leak-tight containers while wet and disposed of in a landfill qualified to receive asbestos waste (special requirements for handling and securing asbestos waste to prevent release into the air).

Standards for exposure set to ensure worker protection by the Occupational Safety and Health Administration (OSHA) in the US include the permissible exposure limit (PEL), and the short-term exposure limit (STEL). The PEL is measured as a time-weighted average (TWA) exposure over an 8 hour shift, and is set at 0.1 f/mL. The STEL is 1 f/mL as averaged over a sampling period of 30 minutes. A worker may be exposed to concentrations higher than the PEL for a short period, as long as the TWA is not exceeded and the STEL is not exceeded.

OSHA notes that all asbestos abatement activities carry risk, and has defined 'acceptable risk' to be exposure below the PEL. [122] The OSHA PEL for asbestos was designed for an exposure period of 40 h/week, 50 weeks/year and 45 years in a lifetime - so most domestic renovation exposures would be well below the limit.

UK regulations

The UK sets standards similar to the US, defining a level of asbestos fibres in air that should not be exceeded, either in the workplace or anyone's personal exposure, over a set period of time. New regulations proposed in 2005 suggested a change to the Approved Code of Practice (ACoP) for workers, lowering the Control Limit (equivalent to the US PEL) from 0.2 f/mL for amphibole and 0.3 f/mL for chrysotile to 0.1 f/mL for all types – over an 8 hr shift. [115] As in the US, a short-term exposure limit (STEL) has been set to enforce high standards of control, maintaining a limit for peak exposures and signalling a need to wear respiratory protective equipment. The STEL is 2.4 f/mL over 10 mins, which is equivalent to exposure at the control limit over 4 hours.

The ACoP also defines what types of work would be exempt from requiring a licence, based on determination that exposure would be sporadic and low-intensity. It is suggested the strict regulations don't apply if:

a) the exposure of employees to asbestos fibres is sporadic and of low intensity;
b) it is clear from the risk assessment that the Control Limit for asbestos will not be exceeded in the air of the working area; and
c) the work involves:
    - short, non-continuous maintenance activities
    - removal of materials in which the asbestos fibres are firmly linked in a matrix
    - encapsulation or sealing of asbestos-containing materials, or
    - air monitoring and control, and the collection of samples to ascertain whether a specific material contains asbestos.

UK Health and Safety Executive (HSE) considered the relative risk to be highest when working with asbestos insulation. The risk was considered to be much lower for asbestos cement/insulation board and even lower for textured coatings. [115] This is because these textured coating products have a relatively low percentage of asbestos (~1.8% chrysotile). Cement has approximately 10% asbestos fibres.

Australian regulations

Regulations for maximum permissible workplace exposures in Australia are the same as in the US and the UK, although no short-term limit (STEL) has been set. The guidelines stipulate that exposure should be eliminated if possible, and if not, should be minimized to the lowest practical level. The exposure standard for asbestos is a respirable fibre level of 0.1 f/mL of air measured in a person's breathing zone, and expressed as a TWA fibre concentration calculated over an eight-hour working day and measured over a minimum period of four hours. [123] The regulations also require that workers who are likely to be exposed to asbestos are informed of the health risks and that health monitoring is provided prior to starting work with asbestos.

Work with all forms of asbestos (both raw and in ACMs) has been prohibited since 31 December 2003, with limited exceptions; however there is still a significant amount of asbestos present in structures and equipment in workplaces.

New Zealand regulations

Asbestos regulation is fragmented across several different authorities in New Zealand. The NZ EPA oversees the HSNO legislation under which asbestos is classified as an unapproved hazardous substance, [124] and the New Zealand Customs Service manages the prohibition of imported substances that do not have approval. [125] The health effects of asbestos and asbestos in public places is the concern of the NZ MoH and local public health units, [48] while asbestos in occupational settings and asbestos-related occupational disease is regulated by WorkSafe NZ. Local territorial authorities have duties and powers to prevent or control asbestos hazards under the Health Act 1956, [126] the Building Act 2004, [127] the Resource Management Act 1991, [128] and the Waste Minimisation Act 2008. [129]

Work with asbestos in New Zealand is regulated under the Health and Safety Employment (Asbestos) Regulations of 1998. [130] The maximum permissible levels for amphibole asbestos (workplace exposure standard; WES) is the same as the US PEL (0.1 f/mL, though the TWA is over 4hours), but the allowable concentration for chrysotile is substantially higher – 1 f/mL (4 hour TWA) – the same as the US 30 minute STEL. The short-term (10 minute) chrysotile exposure limit for New Zealand is 6 f/mL. The amphibole concentration limit is 10-fold lower (0.6 f/mL) (see table 5).

Table 5. Maximum permissible concentrations of asbestos in New Zealand workplaces [130]

Asbestos types	Concentration
Chrysotile	• An average concentration over any 4-hour period of 1 f/mL of air; and • An average concentration over any 10-minute period of 6 f/mL of air.
Amosite, crocidolite, fibrous actinolite, fibrous anthophyllite, and fibrous tremolite	• An average concentration over any 4-hour period of 0.1 f/mL of air; and • An average concentration over any 10-minute period of 0.6 f/mL of air.

These exposure limits are under review by WorkSafe NZ, and the concentration limit for chrysotile is likely to be lowered by a factor of 10, such that the WES is 0.1 f/mL for all asbestos types. Under the proposed new guidance the control level for all asbestos types would therefore be the same as the US PEL. Fibre concentrations ≥0.02 f/mL (20 f/L) would signal the need to stop work and determine the cause of the increased exposure (see table 6).

Table 6. Control levels for monitored airborne asbestos fibres in New Zealand [131]	
Control Level (airborne asbestos f/mL)	**Control/Action**
<0.01	Continue with control measures
≥0.01	Review control measures
≥0.02	Stop work and find the cause

NOTE: These standards are under review to ensure alignment with international standards.

6.2 Policy responses to non-occupational asbestos risk

The health risks of heavy exposure to asbestos are not disputed. There are uncertainties, however, around the calculation of the risk of an asbestos-related disease occurring as a result of very low exposure, such as that from living and working in buildings containing potentially deteriorating ACMs. Uncertainties affect the perception of risk, and can generate fear. Asbestos has in fact become one of the most feared environmental contaminants on earth.

The US Asbestos Hazard Emergency Response Act (AHERA) example

Public policy decisions of the past, made in response to increasing awareness of real or perceived risks of asbestos exposure without thorough input from experts, have in some cases proven to be costly and have not resulted in adequate risk reduction. [132] In the USA, the Asbestos Hazard Emergency Response Act (AHERA) is a prime example. The AHERA protocol was established in 1986 as part of the US federal legislation on the management of asbestos in schools.

The realization in the 1980s that thousands of public buildings in the US, and in particular schools, contained deteriorating ACMs caused concern over the risks of exposure to such 'in–place' asbestos to occupants, workers and schoolchildren. [41] In 1985 Doll and Peto [133] estimated the lifetime risk of cancer at 10 per million for children exposed for 8 hours per day, 5 days per week, for the 10 years from age 8 to 18 in a school where asbestos fibre concentrations of 0.5 f/L were present. Misinterpretation and resulting public alarm led to promulgation of the AHERA protocol, resulting in a massive abatement effort based on limited or no information on actual exposures. [134] The lack of attention to basic toxicological principles, including the importance of dose-response, led to exaggerated public concern and misunderstanding.

"The EPA called for an exercise by school administrators involving an algorithm to determine the course of action to be taken in a particular school building. The algorithm drew on seven observable physical features of the school and involved performance of calculations to arrive at a final number which indicated whether or not action should be taken, namely removal of the asbestos-containing materials (ACM). In the vast majority of cases the result of the exercise was to call for removal. The algorithm was subsequently disproved on the grounds that it did not correlate with any measurements of asbestos-in-air." [135]

Early guidance for schools to manage asbestos suggested that removal was prudent, but later guidance suggested otherwise. Abatement was costly and essentially ineffective. EPA studies monitoring the removal or encapsulation of ACMs in US public schools found little improvement in asbestos fibre levels in air following physical removal, and in some cases exposure may have increased. As a result, the health risk and cost-benefit of asbestos removal versus encapsulation have been questioned. Widespread removal of asbestos is now not recommended; encapsulation of

potentially friable material (that which is not tightly bonded in a matrix, or which is deteriorating such that the matrix is easily crushed) is preferred. [91] In 1990 the EPA indicated that "*removal is often not a school district's best course of action to reduce asbestos exposure*" and that "*improper removal can create a dangerous situation where none previously existed.*" [136]

The AHERA abatement situation in the US resulted from public demands for action that were based on fear and misunderstanding, and provides an example of the importance of clear risk communication and well-considered policy responses to avoid remedial activities that are at best unnecessary, and at worst may increase the public health risk.

7. Summary of risks of asbestos exposure in New Zealand

7.1 What are the risks?

Household sources of exposure to asbestos include degradation, removal and repair of ACMs. There remains some scientific uncertainty regarding the danger of minimal exposure, and the exact nature of the general risk of asbestos exposure that continues to exist because of its presence in older buildings and homes. Assessment of residential exposure is difficult, since levels are generally very low and duration and frequency of exposure, and types of fibre, are usually not known precisely. The existence of a small increase in cancer risk is plausible but data are inadequate to quantify it. This does not by any means imply that workers or homeowners should be complacent when it comes to asbestos risk. As with other known carcinogens, a risk of harm can exist even at very low levels of exposure.

The asbestos found in older homes in New Zealand is mainly of the serpentine chrysotile variety, which if inhaled, has been shown to be more readily cleared from the lungs than amphibole types of asbestos. While all varieties of asbestos have the capacity to cause asbestosis, lung cancer, malignant mesothelioma, and other cancers, the potency of chrysotile fibres has been determined to be lowest, particularly for mesothelioma. However, the precautionary principle and other considerations have led public health agencies to treat serpentine and amphibole hazards as if they carried equal risk. This approach is particularly prudent in regard to encouraging health protective action in low-income countries that are still producing and using chrysotile asbestos and ACMs.

In relation to the current non-occupational exposure situation in New Zealand, assuming equal risk for all asbestos types may mean that the risks associated with exposure to amphiboles are understated, and those of chrysotile overstated in some scenarios. The established occupational exposure limit is likely to be sufficiently protective for chrysotile, but an excess risk for amphibole exposure is still present with the current standard of 0.1 f/mL of air, and both construction workers and DIY home renovators should be aware of this. While the public can be reassured that the risks they face with asbestos in their homes is very low, the possible presence of small amounts of highly potent amphibole asbestos fibres should not be ignored, and proper procedures for dealing with asbestos should continue to be promoted and followed.

The asbestos hazard in New Zealand has not been well-managed in the past. New Zealand regulations have lagged behind many other countries, and the importation of ACMs containing chrysotile asbestos has yet to be banned in this country. The devastating earthquakes in Canterbury in 2010-2011, which damaged thousands of buildings including many houses containing ACMs, potentially increased the risk of exposure to asbestos fibres in the community. Concerns were raised as to whether contractors working in the CHRP took sufficient precautions to manage the potential risks of this exposure. Although flaws were identified in the monitoring and mitigation of asbestos hazards in the CHRP, an analysis of exposure levels suggested that, even considering a 'worst-case' scenario, the errors that occurred would not result in a significant increase in risk to homeowners and occupants of damaged houses who may have been living in the houses while work was being carried out. Nonetheless, steps have been taken to correct the procedures for dealing with asbestos during remediation work, and homeowners undertaking repair and renovation work themselves should be made aware of the potential hazards if asbestos is disturbed.

In relation to asbestos management during disaster recovery, the New Zealand Ministry of Business, Innovation and Employment advises remediation workers to make pragmatic decisions based on the age and construction of the buildings or structures, and if in doubt, proceed as if the building contains asbestos. Rubble should be dampened before disturbing, a dust mask or respirator should be worn, dusty overalls should be bagged before removal of the mask, and a shower should be taken after work. [137] A similar pragmatic approach can be taken by homeowners when considering the possible exposure risks in their homes. Table 7 shows a basic flowchart table for homeowners to make an initial assessment about whether they should be concerned about asbestos exposure, based on the age of their house and the presence of certain materials that *may* contain asbestos. The materials should be assumed to be ACMs if there is uncertainty.

Table 7. Residential risk assessment based on age of home, presence of ACMs, and activities that could increase or decrease risk to bystanders/occupiers. The table should be read left to right to follow the possible presence of ACMs toward an estimation of risk. The yellow colour indicates possible presence of a hazard but probable low risk, green indicates minimized risk, and orange indicates ongoing presence of the hazard and higher risk.

Building age	Possible ACMs present	Status of ACMs if present	Activities impacting ACMs and exposure	Risk level
Pre-1940 unrenovated	None likely			None or negligible risk
Pre-1940, renovations performed 1950-1985	Exterior – corrugated cement roofing, Fibrolite or Hardiplank cladding, Fibrolite eaves	Cracks, chips or breaks in roofing or exterior cement sheeting (walls and eaves)	Materials wet during removal, not sanded or drilled, OR materials sealed/encapsulated	Extremely low risk
			Present when damaged materials were sanded or drilled	Possible short-term exposure – very low risk
		Materials undamaged and well-maintained (sealed and painted)		Extremely low risk
	Interior - textured ceilings, wall linings, vinyl flooring	Decorative ceiling crumbling or removed, vinyl flooring uplifted or old wall board crushed or drilled	Present during removal, but cleanup thorough	Possible short-term exposure – very low risk
			Home furnishings contaminated with dust, not cleaned or removed	Low risk but possible ongoing low-level exposure *
		Materials intact		Extremely low risk
1940 to 1990	Exterior – corrugated cement roofing, Fibrolite or Hardiplank cladding, Fibrolite eaves	Cracks, chips or breaks in roofing or exterior cement sheeting (walls and eaves)	Materials wet during removal, not sanded or drilled, OR materials sealed/encapsulated	Extremely low risk
			Present when damaged materials were sanded or drilled	Possible short-term exposure – very low risk
		Materials undamaged and well-maintained (sealed and painted)		Extremely low risk
	Interior - textured ceilings, wall linings, vinyl flooring	Decorative ceiling crumbling or removed, vinyl flooring uplifted or old wall board crushed or drilled	Present during removal, but cleanup thorough	Possible short-term exposure – very low risk
			Home furnishings contaminated with dust, not cleaned or removed	Low risk but possible ongoing low-level exposure *
		Materials intact		Extremely low risk
Post-1990	None likely			None or negligible risk

* Risk is dependent on amount of ACMs and extent of disturbance/works carried out. Although the risk is low in absolute terms, it will increase with time if steps are not taken to remove the asbestos fibres after work has been completed.

7.2 Risks in perspective

We are exposed to risks and vulnerabilities on a daily basis, as innovations are continually introducing new risks. Risk assessment is an imprecise exercise; it requires many assumptions to be made, since complete data on exposures are often unavailable, and uncertainty is inherent in the process. In this context, decisions are made based on both science and considered judgment. Because science can never offer 100% definitive proof, judgment is regularly employed when scientific evidence is used to make inferences about disease causation in risk assessment. The psychological acceptability of a risk is also a judgment call that is influenced by recall of past events and the ability to envisage future events, as well as by actuarial calculations. For instance, families or communities that have been adversely affected by occupational asbestos exposure in the past may overestimate the risks associated with lower exposures because they can envisage the consequences, whereas people who have never encountered asbestos-related disease may dismiss a low risk as inconsequential. Asbestos hazards in the home are judged differently from other hazards, because the home environment is a place that should be considered safe. Yet even within the home, hazards are also judged differently according to the way in which exposures occur. Involuntary exposures, no matter how inconsequential, can raise alarm, whereas voluntary exposures such as those encountered during DIY home renovation are often not given the attention they deserve. Generally people will accept much higher levels of risk from voluntary exposures than from involuntary ones, especially those viewed as being the result of mismanagement by authorities. The communication of risk needs to take such perspectives into consideration.

The concept of risk associated with hazardous substances for which there is no minimal exposure that is known to be "safe" is one that regulators face constantly. For some substances, minimal exposure is inevitable because of their naturally occurring presence in the everyday environment, even where there is no human intervention (e.g. asbestos, radiation, cadmium, lead). When human activity can increase the exposure above background, the regulator uses the approaches described in this paper to establish a statistically acceptable level of risk in order to determine maximum tolerable exposures (for example exposure to medical or airport security X rays which involve radiation). In the face of uncertainty and the need to protect public health, risk assessments are generally conservative, and usually overestimate risks. With asbestos exposure in the home, risk assessment exercises judge the risk to be very low for individuals who are not involved in renovation or repair work themselves.

The risk associated with exposure to low concentrations of asbestos fibres should therefore be seen in its proper perspective, which should reassure the public. Nevertheless, risks must neither be underestimated nor denied, and authorities such as WorkSafe NZ and NZ MoH need to be vigilant in maintaining awareness of the risks of asbestos exposure in New Zealand homes, particularly when ACMs could be disturbed during home renovation. Both of these organisations provide useful documents and web resources for businesses and the general public. [48, 131, 138-140] Despite considerable uncertainty about minimal exposure risks, the risks of higher exposures are reasonably well understood and should serve as a caution against complacency, but not as a fuel for unnecessary anxiety.

A prudent approach would be to follow the lead of many other countries that have banned the continued importation and use of *any* ACMs, and this should be brought to the Government's attention.

Abbreviations

ACM	asbestos-containing material
AHERA	Asbestos Hazard Emergency Response Act (US)
DNA	deoxyribonucleic acid
EPA	Environmental Protection Agency (US); Environmental Protection Authority (NZ)
f/L	fibres per litre
f/mL	fibres per millilitre
HEI-AR	Health Effects Institute – Asbestos Research
HSE	Health and Safety Authority (UK)
HSNO	Hazardous Substances and New Organisms Act (NZ)
IARC	International Agency for Research on Cancer
IPF	Interstitial pulmonary fibrosis
ILO	International Labour Organization
MPR	maximum permissible risk
NESHAP	National Emission Standards for Hazardous Air Pollutants (US)
NR	negligible risk
NTP	National Toxicology Program (US)
OSHA	Occupational Safety and Health Administration (US)
PCM	phase contrast microscopy
PEL	permissible exposure limit
ROS	reactive oxygen species
RNS	reactive nitrogen species
RR	relative risk (or risk ratio)
SEM	scanning electron microscopy
STEL	short-term exposure limit
TEM	transmission electron microscopy
TWA	time-weighted average
WES	workplace exposure standard
WHO	World Health Organization

References

1. Sporn, T.A., *The mineralogy of asbestos*, in *Pathology of Asbestos-Associated Diseases*, T.D. Oury, T.A. Sporn, and V.L. Roggli, Editors. 2014, Springer-Verlag: Heidelberg.
2. Henderson, D.W. and J. Leigh, *The history of asbestos utilization and recognition of asbestos-induced diseases*, in *Asbestos: risk assessment, epidemiology, and health effects, Second Edition*, R.F. Dodson and S.P. Hammar, Editors. 2011, CRC Press: Boca Raton, FL.
3. Craighead, J.E. and A.R. Gibbs, *Asbestos and its diseases*. 2008, New York: Oxford University Press.
4. Government Office for Science, *Innovation: managing risk, not avoiding it: Evidence and case studies*, 2014, UK Government: London.
5. Asveld, L. and S. Roeser, eds. *The Ethics of Technological Risk*. 2009, Taylor & Francis: Abingdont/New York.
6. Maines, R., *Asbestos and fire: technological trade-off and the body at risk*. 2005, Piscataway, NJ: Rutgers University Press.
7. Craighead, J.E. and A.R. Gibbes, eds. *Asbestos and its diseases*. 2008, Oxford University Press: New York.
8. WHO International Programme on Chemical Safety (IPCS), *Environmental Health Criteria 203. Chrysotile asbestos*, 1998, World Health Organization: Geneva.
9. Olsen, N.J., et al., *Increasing incidence of malignant mesothelioma after exposure to asbestos during home maintenance and renovation.* Med J Aust, 2011. **195**(5): p. 271-4.
10. International Labour Organization, *C162 - Asbestos Convention, 1986 (No. 162). Convention concerning Safety in the Use of Asbestos (Entry into force: 16 Jun 1989)*, 1986, ILO: Geneva.
11. Anderson, H.A., et al., *Mesothelioma among employees with likely contact with in-place asbestos-containing building materials.* Ann N Y Acad Sci, 1991. **643**: p. 550-72.
12. Lilienfeld, D.E., *Asbestos-associated pleural mesothelioma in school teachers: a discussion of four cases.* Ann N Y Acad Sci, 1991. **643**: p. 454-58.
13. Testa, J.R., et al., *Germline BAP1 mutations predispose to malignant mesothelioma.* Nat Genet, 2011. **43**(10): p. 1022-5.
14. Carbone, M., et al., *Malignant mesothelioma: facts, myths, and hypotheses.* J Cell Physiol, 2012. **227**(1): p. 44-58.
15. Coggon, D., et al., *Differences in occupational mortality from pleural cancer, peritoneal cancer, and asbestosis.* Occup Environ Med, 1995. **52**(11): p. 775-7.
16. Lemen, R.A., *Epidemiology of asbestos-related diseases and the knowledge that led to what Is known today*, in *Asbestos: Risk assessment, epidemiology, and health effects, Second edition*, R.F. Dodson and S.P. Hammar, Editors. 2011, CRC Press.
17. International Agency for Research on Cancer, *Some inorganic and organometallic compounds.* IARC Monogr Eval Carcinog Risk Chem Man, 1973. **2**(1): p. 181.
18. National Toxicology Program, Department of Health and Human Services, Public Health Service, *First Annual Report on Carcinogens*, 1980: Washington, D.C.
19. International Programme on Chemical Safety (IPCS), *Environmental Health Criteria 53. Asbestos and other natural mineral fibers* 1986, United Nations Environment Programme, International Labour Organisation, and World Health Organization: Geneva.
20. ILO/WHO, *Outline for the development of national programmes for elimination of asbestos-related diseases*, 2007, International Labour Organization and World Health Organization: Geneva.
21. International Agency for Research on Cancer, *Arsenic, metals, fibres, and dusts. A review of human carcinogens.* IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, 2012. **Volume 100C**: p. 219 - 294.
22. Chapman, S.J., et al., *Benign asbestos pleural diseases.* Curr Opin Pulm Med, 2003. **9**(4): p. 266-71.
23. O'Reilly, K.M., et al., *Asbestos-related lung disease.* Am Fam Physician, 2007. **75**(5): p. 683-8.
24. Doll, R., *Mortality from lung cancer in asbestos workers.* Br J Ind Med, 1955. **12**(2): p. 81-6.
25. Breslow, L., *Industrial aspects of bronchiogenic neoplasms.* Dis Chest, 1955. **28**(4): p. 421-30.
26. Selikoff, I.J., E.C. Hammond, and J. Churg, *Asbestos exposure, smoking, and neoplasia.* JAMA, 1968. **204**(2): p. 106-12.

27. Wagner, J.C., C.A. Sleggs, and P. Marchand, *Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province.* Br J Ind Med, 1960. **17**: p. 260-71.
28. Selikoff, I.J., J. Churg, and E.C. Hammond, *Relation between Exposure to Asbestos and Mesothelioma.* N Engl J Med, 1965. **272**: p. 560-5.
29. U.S. Environmental Protection Agency, *Airborne asbestos health assessment update*, 1986, EPA: Washington, D.C.
30. Delgermaa, V., et al., *Global mesothelioma deaths reported to the World Health Organization between 1994 and 2008.* Bull World Health Organ, 2011. **89**(10): p. 716-24, 724A-724C.
31. Peto, J., et al., *The European mesothelioma epidemic.* Br J Cancer, 1999. **79**(3-4): p. 666-72.
32. Finley, B.L., et al., *Cumulative asbestos exposure for US automobile mechanics involved in brake repair (circa 1950s-2000).* J Expo Sci Environ Epidemiol, 2007. **17**(7): p. 644-55.
33. Huncharek, M., *Changing risk groups for malignant mesothelioma.* Cancer, 1992. **69**(11): p. 2704-11.
34. Paustenbach, D.J., et al., *An evaluation of the historical exposures of mechanics to asbestos in brake dust.* Appl Occup Environ Hyg, 2003. **18**(10): p. 786-804.
35. Huncharek, M., *Non-asbestos related diffuse malignant mesothelioma.* Tumori, 2002. **88**(1): p. 1-9.
36. International Agency for Research on Cancer, *Erionite.* IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, 2012. **Volume 100C**: p. 311 - 316.
37. Carbone, M., et al., *A mesothelioma epidemic in Cappadocia: scientific developments and unexpected social outcomes.* Nat Rev Cancer, 2007. **7**(2): p. 147-54.
38. Yates, D.H., et al., *Malignant mesothelioma in south east England: clinicopathological experience of 272 cases.* Thorax, 1997. **52**(6): p. 507-12.
39. Institute of Medicine, *Asbestos: Selected Cancers*, 2006, National Academies Press: Washington, DC.
40. Reid, A., N. de Klerk, and A.W. Musk, *Does exposure to asbestos cause ovarian cancer? A systematic literature review and meta-analysis.* Cancer Epidemiol Biomarkers Prev, 2011. **20**(7): p. 1287-95.
41. Mossman, B.T., et al., *Asbestos: scientific developments and implications for public policy.* Science, 1990. **247**(4940): p. 294-301.
42. Stanton, M.F., et al., *Relation of particle dimension to carcinogenicity in amphibole asbestoses and other fibrous minerals.* J Natl Cancer Inst, 1981. **67**(5): p. 965-75.
43. Dodson, R.F., M.A. Atkinson, and J.L. Levin, *Asbestos fiber length as related to potential pathogenicity: a critical review.* Am J Ind Med, 2003. **44**(3): p. 291-7.
44. Agency for Toxic Substances and Disease Registry, Division of Health Assessment and Consultation, *Report on the Expert Panel on Health Effects of Asbestos and Synthetic Vitreous Fibers: The influence of fiber length*, 2003, ATSDR: Atlanta.
45. International Agency for Research on Cancer, *IARC monographs on the evaluation of the carcinogenic risk of chemicals in man: Asbestos*, 1977, World Health Organization: Lyon.
46. Walker, C., J. Everitt, and J.C. Barrett, *Possible cellular and molecular mechanisms for asbestos carcinogenicity.* Am J Ind Med, 1992. **21**(2): p. 253-73.
47. Kjellstrom, T.E., *The epidemic of asbestos-related diseases in New Zealand.* Int J Occup Environ Health, 2004. **10**(2): p. 212-9.
48. Ministry of Health, *The management of asbestos in the non-occupational environment: Revised edition* 2013, Ministry of Health: Wellington.
49. Graham, B., *Inventory of New Zealand imports and exports of asbestos-containing products. Report to the Ministry for the Environment*, 2014, Graham Environmental Consulting Ltd.
50. Virta, R.L., *World asbestos consumption from 2003 through 2007*, 2009, Department of the Interior, U.S. Geological Survey.
51. Australian Competition & Consumer Commission. *Urgent safety alert on stacked stone tiles.* 2010; Available from: https://http://www.accc.gov.au/media-release/urgent-safety-alert-on-stacked-stone-tiles.
52. Building Research Association of New Zealand (BRANZ): Renovate. *The technical resource for industry.* 2014 [cited 2014 Nov 30]; Available from: http://www.renovate.org.nz.
53. Capital Environmental Services. Wellington NZ; Available from: http://www.fibres.co.nz/index.html.
54. Isaacs, N. *Thermal insulation required in NZ homes - 1 April 1978.* NZ History website [cited 2014 10 Dec]; Available from: http://www.nzhistory.net.nz/page/thermal-insulation-required-nz-homes.
55. Asbestos Advisory Committee, *Report of the Asbestos Advisory Committee to the Minister of Labour* April 1991, Occupational Safety and Health Service, Department of Labour: Wellington, NZ.

56. Ministry of Business, Innovation and Employment, *Asbestos and other occupational lung diseases in New Zealand: 2012 Annual Report*, 2012, MBIE: Wellington, NZ.
57. Kjellstrom, T. and P. Smartt, *Increased mesothelioma incidence in New Zealand: the asbestos-cancer epidemic has started.* N Z Med J, 2000. **113**(1122): p. 485-90.
58. Ministry of Health. *Cancer: New Registrations and Deaths 2011*. 2014; Available from: http://www.health.govt.nz/publication/cancer-new-registrations-and-deaths-2011.
59. Smartt, P., *Mortality, morbidity, and asbestosis in New Zealand: the hidden legacy of asbestos exposure.* N Z Med J, 2004. **117**(1205): p. U1153.
60. WorkSafe New Zealand, *Asbestos and other occupational lung diseases in New Zealand: 2013 Annual Report*, 2014, New Zealand Government: Wellington.
61. McCormack, V., et al., *Estimating the asbestos-related lung cancer burden from mesothelioma mortality.* Br J Cancer, 2012. **106**(3): p. 575-84.
62. Ministry of Health, *Mortality and Demographic Data 2010: Mortality tables*, 2013, Ministry of Health: Wellington.
63. Leigh, J., et al., *Malignant mesothelioma in Australia, 1945-2000.* Am J Ind Med, 2002. **41**(3): p. 188-201.
64. Reid, A., et al., *Cancer incidence among women and girls environmentally and occupationally exposed to blue asbestos at Wittenoom, Western Australia.* Int J Cancer, 2008. **122**(10): p. 2337-44.
65. House of Representatives Standing Committee on Aboriginal Affairs, *The effects of asbestos mining on the Baryulgil community*, 1984, Parliament of the Commonwealth of Australia: Canberra.
66. Leigh, J., et al., *The incidence of malignant mesothelioma in Australia 1982-1988.* Am J Ind Med, 1991. **20**(5): p. 643-55.
67. Australian mesothelioma registry, *1st Annual Report, Mesothelioma in Australia 2011*, 2012, Safe Work Australia.
68. Australian Institute of Health and Welfare (AIHW). *Australian Cancer Incidence and Mortality (ACIM) books: Mesothelioma*. 2015; Available from: http://www.aihw.gov.au/acim-books.
69. Reid, A., et al., *The mortality of women exposed environmentally and domestically to blue asbestos at Wittenoom, Western Australia.* Occup Environ Med, 2008. **65**(11): p. 743-9.
70. Park, E.K., et al., *Asbestos exposure during home renovation in New South Wales.* Med J Aust, 2013. **199**(6): p. 410-3.
71. Ahrens, W., et al., *Retrospective assessment of asbestos exposure--I. Case-control analysis in a study of lung cancer: efficiency of job-specific questionnaires and job exposure matrices.* Int J Epidemiol, 1993. **22 Suppl 2**: p. S83-95.
72. Orlowski, E., et al., *Retrospective assessment of asbestos exposure--II. At the job level: complementarity of job-specific questionnaire and job exposure matrices.* Int J Epidemiol, 1993. **22 Suppl 2**: p. S96-105.
73. Rake, C., et al., *Occupational, domestic and environmental mesothelioma risks in the British population: a case-control study.* Br J Cancer, 2009. **100**(7): p. 1175-83.
74. Paustenbach, D.J., et al., *Chrysotile asbestos exposure associated with removal of automobile exhaust systems (ca. 1945-1975) by mechanics: results of a simulation study.* J Expo Sci Environ Epidemiol, 2006. **16**(2): p. 156-71.
75. Maule, M.M., et al., *Modeling mesothelioma risk associated with environmental asbestos exposure.* Environ Health Perspect, 2007. **115**(7): p. 1066-71.
76. Berry, G., A.J. Rogers, and F.D. Pooley, *Mesotheliomas--asbestos exposure and lung burden.* IARC Sci Publ, 1989(90): p. 486-96.
77. Goldberg, M. and D. Luce, *The health impact of nonoccupational exposure to asbestos: what do we know?* Eur J Cancer Prev, 2009. **18**(6): p. 489-503.
78. Gezondheidsraad, *Asbestos: Risks of environmental and occupational exposure*, 2010, Health Council of the Netherlands: The Hague.
79. Baron, P.A., *Measurement of airborne fibers: a review.* Ind Health, 2001. **39**(2): p. 39-50.
80. Agency for Toxic Substances & Disease Registry, *Toxicological profile for asbestos*, 2001, U.S. Department of Health and Human Services, Public Health Service: Atlanta.
81. Committee on Nonoccupational Health Risks of Asbestiform Fibers, Board on Toxicology and Environmental Health Hazards, National Research Council, *Asbestiform Fibers: Nonoccupational Health Risks*. 1984, Washington, DC: The National Academies Press.

82. Spurny, K.R., *On the release of asbestos fibers from weathered and corroded asbestos cement products.* Environ Res, 1989. **48**(1): p. 100-16.
83. Hagemeyer, O., H. Otten, and T. Kraus, *Asbestos consumption, asbestos exposure and asbestos-related occupational diseases in Germany.* Int Arch Occup Environ Health, 2006. **79**(8): p. 613-20.
84. van Orden, D.R., et al., *Evaluation of ambient asbestos concentrations in buildings following the Loma Prieta earthquake.* Regul Toxicol Pharmacol, 1995. **21**(1): p. 117-22.
85. Hoppe, K.A., et al., *Assessment of airborne exposures and health in flooded homes undergoing renovation.* Indoor Air, 2012. **22**(6): p. 446-56.
86. Perkins, R.A., J. Hargesheimer, and W. Fourie, *Asbestos release from whole-building demolition of buildings with asbestos-containing material.* J Occup Environ Hyg, 2007. **4**(12): p. 889-94.
87. Contaminants of Potential Concern (COPC) Committee, World Trade Center Indoor Air Task Force Working Group, *World Trade Center indoor Environment Assessment: selecting contaminants of potential concern and setting health-based benchmarks*, 2003, U.S. Environmental Protection Agency.
88. Lee, R.J. and D.R. Van Orden, *Airborne asbestos in buildings.* Regul Toxicol Pharmacol, 2008. **50**(2): p. 218-25.
89. Nicholson, W.J., et al., *Asbestos contamination in United States schools from use of asbestos surfacing materials.* Ann N Y Acad Sci, 1979. **330**: p. 587-96.
90. Health Effects Institute - Asbestos Research (HEI-AR), *Asbestos in public and commercial buildings: a literature review and synthesis of current knowledge*, 1991: Cambridge, MA.
91. Darcey, D.J. and C. Feltner, *Occupational and environmental exposure to asbestos*, in *Pathology of asbestos-associated diseases*, T.D. Oury, T.A. Sporn, and Roggli, Editors. 2014, Springer-Verlag: Berlin Heidelberg. p. 11 - 24.
92. Health and Safety Authority, *Asbestos-containing materials (ACMs) in workplaces - Practical guidelines on ACM management and abatement* 2013: Dublin.
93. Lange, J.H. and K.W. Thomulka, *An evaluation of personal airborne asbestos exposure measurements during abatement of dry wall and floor tile/mastic.* Int J Environ Health Res, 2000. **10**: p. 5-19.
94. Scientific Committees on Health and Environmental Risks, Consumer Products, and Emerging and Newly Identified Health Risks (SCHER/SCCP/SCENIHR). *Risk assessment methodologies and approaches for genotoxic and carcinogenic substances.* 2009; Available from: http://ec.europa.eu/health/ph_risk/committees/04_scher/docs/scher_o_113.pdf.
95. Hodgson, J.T. and A. Darnton, *The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure.* Ann Occup Hyg, 2000. **44**(8): p. 565-601.
96. Seidman, H., I.J. Selikoff, and E.C. Hammond, *Short-term asbestos work exposure and long-term observation.* Ann N Y Acad Sci, 1979. **330**: p. 61-89.
97. Bernstein, D., et al., *Health risk of chrysotile revisited.* Crit Rev Toxicol, 2013. **43**(2): p. 154-83.
98. Nicholson, W.J., *The carcinogenicity of chrysotile asbestos--a review.* Ind Health, 2001. **39**(2): p. 57-64.
99. Yarborough, C.M., *Chrysotile as a cause of mesothelioma: an assessment based on epidemiology.* Crit Rev Toxicol, 2006. **36**(2): p. 165-87.
100. Pooley, F.D., *An examination of the fibrous mineral content of asbestos lung tissue from the Canadian chrysotile mining industry.* Environ Res, 1976. **12**(3): p. 281-98.
101. Rowlands, N., G.W. Gibbs, and A.D. McDonald, *Asbestos fibres in the lungs of chrysotile miners and millers--a preliminary report.* Ann Occup Hyg, 1982. **26**(1-4): p. 411-5.
102. Yano, E., et al., *Cancer mortality among workers exposed to amphibole-free chrysotile asbestos.* Am J Epidemiol, 2001. **154**(6): p. 538-43.
103. Hein, M.J., et al., *Follow-up study of chrysotile textile workers: cohort mortality and exposure-response.* Occup Environ Med, 2007. **64**(9): p. 616-25.
104. Berman, D.W. and K.S. Crump, *A meta-analysis of asbestos-related cancer risk that addresses fiber size and mineral type.* Crit Rev Toxicol, 2008. **38 Suppl 1**: p. 49-73.
105. Rees, D., et al., *Case-control study of mesothelioma in South Africa.* Am J Ind Med, 1999. **35**(3): p. 213-22.
106. British Occupational Hygiene Society, *Report from the Committee on Asbestos. A study of the health experience in two U.K. asbestos factories.* Ann Occup Hyg, 1983. **27**(1): p. 1-55.
107. van der Bij, S., et al., *Lung cancer risk at low cumulative asbestos exposure: meta-regression of the exposure-response relationship.* Cancer Causes Control, 2013. **24**(1): p. 1-12.
108. Lacourt, A., et al., *Occupational and non-occupational attributable risk of asbestos exposure for malignant pleural mesothelioma.* Thorax, 2014. **69**(6): p. 532-9.

109. Bourdes, V., P. Boffetta, and P. Pisani, *Environmental exposure to asbestos and risk of pleural mesothelioma: review and meta-analysis.* Eur J Epidemiol, 2000. **16**(5): p. 411-7.
110. Vianna, N.J. and A.K. Polan, *Non-occupational exposure to asbestos and malignant mesothelioma in females.* Lancet, 1978. **1**(8073): p. 1061-3.
111. Camus, M., J. Siemiatycki, and B. Meek, *Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer.* N Engl J Med, 1998. **338**(22): p. 1565-71.
112. Marier, M., et al., *Exploratory sampling of asbestos in residences near Thetford Mines: the public health threat in Quebec.* Int J Occup Environ Health, 2007. **13**(4): p. 386-97.
113. Iwatsubo, Y., et al., *Pleural mesothelioma: dose-response relation at low levels of asbestos exposure in a French population-based case-control study.* Am J Epidemiol, 1998. **148**(2): p. 133-42.
114. Simpson Grierson, *Investigation of airborne asbestos exposure related to removal of textured coatings, three residential properties, CHRP New Zealand*, 2014, Noel Arnold & Associates Pty Ltd. .
115. Health and Safety Commission, *Proposals for revised Asbestos Regulations and an Approved Code of Practice: Consultative document*, T. Slater, Editor 2005, HSE: London.
116. WorkSafe New Zealand, *Investigation report: Asbestos risks in the Canterbury Home Repair Programme*, 2014, WorkSafe New Zealand.
117. LaDou, J., et al., *The case for a global ban on asbestos.* Environ Health Perspect, 2010. **118**(7): p. 897-901.
118. Collegium Ramazzini, *Asbestos is still with us: repeat call for a universal ban.* J Occup Environ Med, 2010. **52**(5): p. 469-72.
119. International Commission on Occupational Health. *ICOH Statement on Global Asbestos Ban and the Elimination of Asbestos-related Diseases.* 2013 October [cited 2014 September 10]; Available from: http://www.icohweb.org/site_new/multimedia/news/pdf/2013_ICOH Statement on global asbestos ban.pdf.
120. WHO, *Parma declaration on environment and health*, 2010, World Health Organization Regional Office for Europe: Copenhagen.
121. U.S. Environmental Protection Agency. *Asbestos National Emission Standards for Hazardous Air Pollutants (NESHAP).* 2014 [cited 2014 September 25]; Available from: http://www2.epa.gov/asbestos/asbestos-neshap.
122. Occupational Safety & Health Administration. *Asbestos. Standard 1910.1001*. Standards - 29 CFR: Toxic and Hazardous Substances 1986; Available from: https://http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=9995.
123. Safe Work Australia. *Workplace exposure standards for airborne contaminants.* 2013; Available from: http://www.safeworkaustralia.gov.au/sites/swa/about/publications/pages/workplace-exposure-standards.
124. Enviromental Protection Authority, *HSNO Enforcement Agencies. Roles and responsibilities: identifying a lead agency following a hazardous substance non-compliance or incident*, 2012, New Zealand Government: Wellington.
125. New Zealand Customs Service. *Prohibited imports.* Available from: http://www.customs.govt.nz/features/prohibited/imports/Pages/default.aspx.
126. New Zealand Government. *Health Act 1956 No 65* (as at 05 September 2014) Public Act - New Zealand Legislation; Available from: http://www.legislation.govt.nz/act/public/1956/0065/latest/DLM305840.html.
127. New Zealand Government. *Building Act 2004 No 72* (as at 01 January 2015) Public Act - New Zealand Legislation; Available from: http://www.legislation.govt.nz/act/public/2004/0072/latest/DLM306036.html.
128. New Zealand Government. *Resource Management Act 1991 No 69* (as at 03 March 2015) Public Act - New Zealand Legislation; Available from: http://www.legislation.govt.nz/act/public/1991/0069/latest/DLM230265.html.
129. New Zealand Government. *Waste Minimisation Act 2008 No 89.* (as at 01 July 2013) Public Act - New Zealand Legislation; Available from: http://www.legislation.govt.nz/act/public/2008/0089/latest/DLM999802.html.
130. Parliamentary Council Office, Government of New Zealand, *Health and Safety in Employment (Asbestos) Regulations* 1998: New Zealand.

131. New Zealand Demolition and Asbestos Association (NZDAA), *Asbestos - New Zealand guidelines for the management and removal of asbestos (3rd Edition)*, 2013, WorkSafe New Zealand.
132. Weill, H. and J.M. Hughes, *Asbestos as a public health risk: disease and policy*. Annu Rev Public Health, 1986. **7**: p. 171-92.
133. Doll, R. and J. Peto, *Effects on health of exposure to asbestos*, 1985, Health and Safety Commission: London.
134. Wilson, R., et al., *Asbestos in New York City public school buildings--public policy: is there a scientific basis?* Regul Toxicol Pharmacol, 1994. **20**(2): p. 161-9.
135. Corn, M., et al., *Airborne concentrations of asbestos in 71 school buildings.* Regul Toxicol Pharmacol, 1991. **13**(1): p. 99-114.
136. U.S. Environmental Protection Agency, *The Asbestos Informer, EPA 340/1-90-020* 1990: Washington, D.C.
137. Ministry of Business, Innovation and Employment. *Disaster Recovery - Asbestos Management*. [cited 2015 8 Jan]; Available from: http://www.dol.govt.nz/quake/asbestos-management.asp.
138. WorkSafe New Zealand. *Asbestos information for householders*. 2014; Available from: http://www.business.govt.nz/worksafe/information-guidance/guidance-by-hazard-type/asbestos/asbestos-information-for-householders.
139. Ministry of Health. *All about asbestos*. HealthEd, HE7021 2013; Available from: https://http://www.healthed.govt.nz/resource/all-about-asbestos.
140. Ministry of Health. *Removing asbestos from the home*. HealthEd, HE7022 2008; Available from: https://http://www.healthed.govt.nz/resource/removing-asbestos-home.