OIR: 2324/838

28 March 2024

[redacted]

Tēnā koe [redacted],

Request for Information under the Local Government Official Information and Meetings Act 1987 (the Act) (the LGOIMA)

Thank you for your email of **13 March 2024** requesting the following information:

The questions (which were tabled at the meeting) are as follows:

1. ***Does Kapiti water pipes contain asbestos?***

Yes, there is approximately 180,000 meters of water reticulation pipelines constructed of Asbestos.

   a. ***If yes, Why is this poison in our pipe that people consume?***

   Asbestos is only a poison if inhaled, although it is possible to ingest fibres, and inhalation is the only route that has been established as causing harm. Please see attached to this letter the review for 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**'.

2. ***If the pipes break, is there a risk of asbestos contamination leaking into people's drinking water and other main water pipes?***

There is a very small risk of asbestos contamination following a burst pipeline, but as noted above, ingesting asbestos fibres has not been established as causing harm. Note The World Health Organization (WHO 2003, 2017) concluded that there was little evidence suggesting that ingested asbestos is hazardous to health and therefore did not feel it necessary to establish a health-based guideline value for drinking water.

3. ***Does the Kapiti Council test the water pipes for asbestos? How often? What areas?***

Kapiti Coast District Council does not carry out routine testing of the water supply for Asbestos.

   a. ***If yes, at what cost to the ratepayer?***

   No cost incurred as Kapiti Coast District Council are not testing for Asbestos and it is not required by the agency Taumata Arowai, who are known as a drinking water regulator.

Ngā mihi,

Sean Mallon
Group Manager Infrastructure and Asset Management
Kaiwhakahaere Rōpū Anga me te Whakahaere Rawa

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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Office of the Prime Minister's
Chief Science Advisor

the ROYAL SOCIETY of NEW ZEALAND
TE APĀRANGI

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.

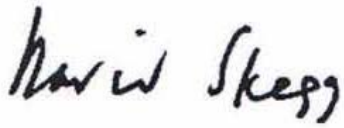
While there is no absolutely safe level of asbestos exposure, asbestos fibres in very low concentrations also exist in the natural environment, and therefore some exposure is unavoidable. The risk at very low exposure levels needs to be in put in the context of other inevitable risks, such as low-level 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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Table of Contents

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]

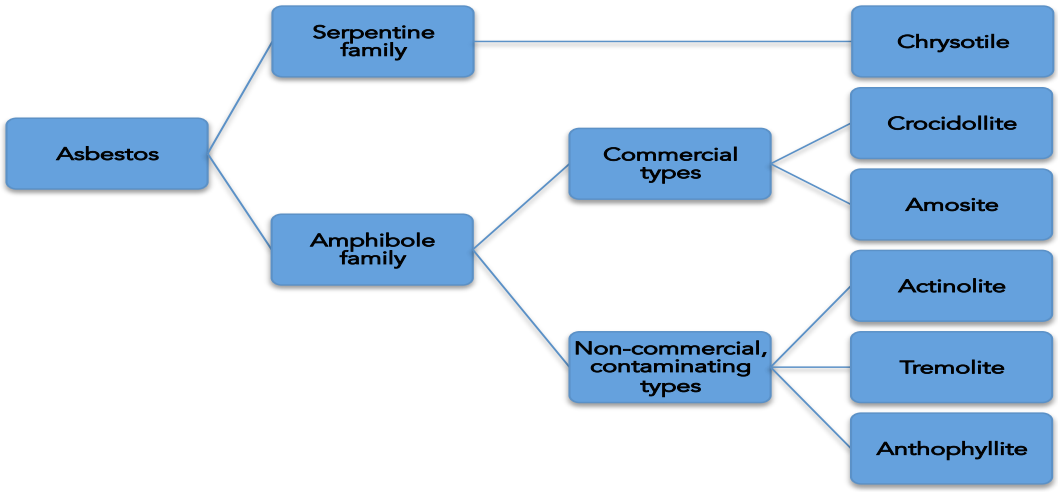
The 'asbestiform habit' refers to mineral crystals that grow in a single dimension, as opposed to random, multidimentional prismatic patterns. Asbestiform minerals form long, threadlike fibres that bend like wire rather than shattering under pressure. There are two 'families' of asbestos types; the serpentine 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.

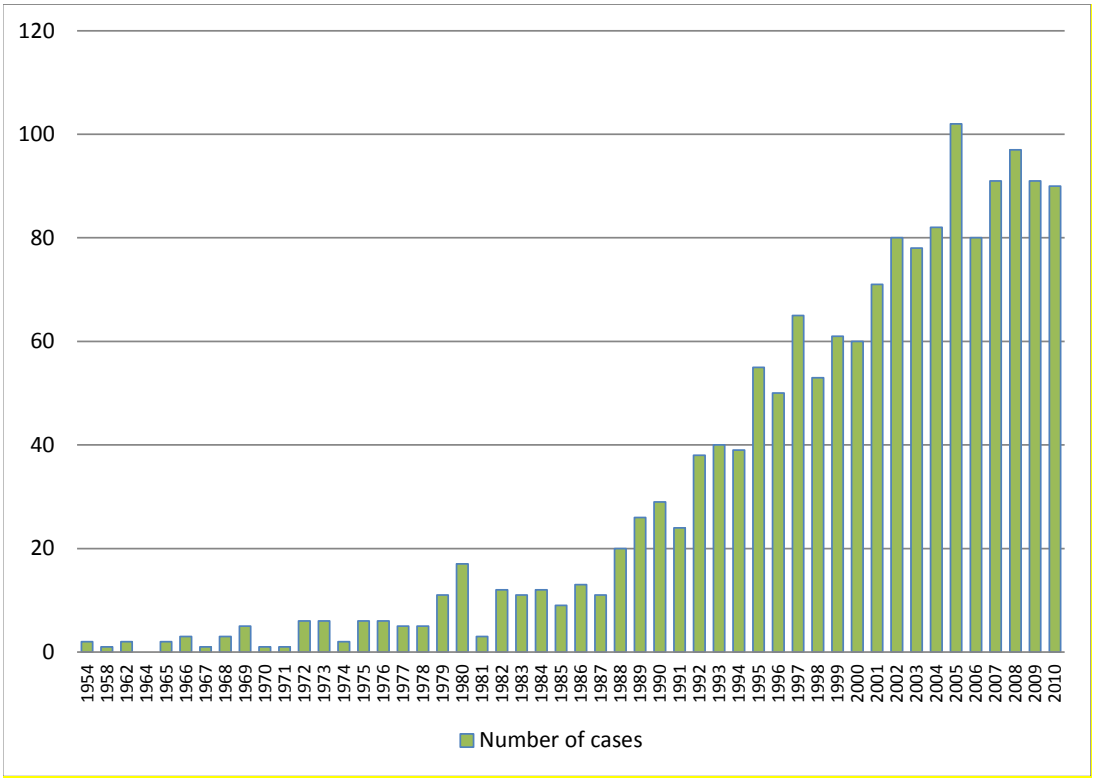
Asbestos products likely to be found in New Zealand in houses built between ~1940 and 1990:

- 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])

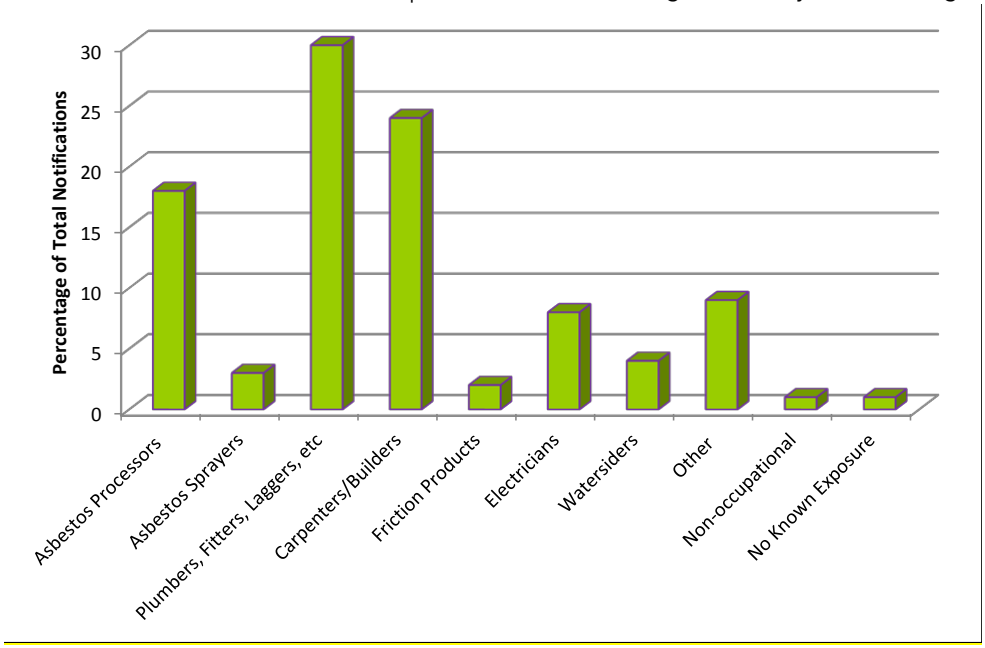


Mesothelioma is primarily a disease of older age; 49% of reported cases in New Zealand since 1994 were in people aged 70 or over. [56] NZCR data indicate that over 68% of individuals registered with mesothelioma in 2011 were in this age bracket. [58] Over 86% of cases were in men, as would be expected 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.

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Guidelines for Public Health Units

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Preface

The unique properties of asbestos have made it a valuable and, in some applications, an essential commercial material for which manufactured substitutes are still inadequate or very expensive.

Before the health risks were more completely appreciated, asbestos was regarded as a 'miracle' fibre of great versatility and usefulness.

Public concern regarding ambient levels of asbestos fibres in the air has arisen from an awareness that occupational airborne exposures to asbestos, especially in the extraction and manufacturing processes in the past, caused serious health problems, including asbestosis, lung cancer and mesothelioma.

In 1994, the Public Health Commission (PHC) released its policy advice to the Minister of Health on hazardous substances, which included the recommendation that '… the PHC, in consultation with the … [other agencies] … prepares recommendations on the control of asbestos outside the workplace for the purpose of avoiding or reducing unacceptable risks to health' (PHC 1994). The Ministry of Health published a guideline on *The Management of Asbestos in the Non-Occupational Environment* in 1997 as a result of this recommendation.

To protect the health of the public, the policy on the management of asbestos needs to be focused on the risks to individuals that asbestos may present and on sensible action that is related to the level of risk.

These guidelines are directed at non-workplace exposure to asbestos in air. The risk to health from workplace exposure is a matter for WorkSafe New Zealand (WorkSafe).

These guidelines are intended to assist public health units of district health boards address public concerns and give sensible advice. In addition to drawing together background information, it suggests a protocol for a response related to the likely level of risk to health, and considers how risks may be evaluated and communicated.

This 2017 revised edition builds on the 1997, 2007, 2013 and 2016 editions. Changes to the guidelines include:

- advice on health hazards of ingesting asbestos has been updated
- references to the two resources *All About Asbestos* and *Removing Asbestos from the Home* were updated as these underwent major revisions.

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These guidelines were derived from work undertaken by John Feltham for the Ministry of Health.

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Introduction

Background

More than a century ago the adverse effects on health caused by the inhalation of asbestos fibres were recognised in the United Kingdom. Fibrosis of the lungs amongst asbestos cloth workers was further observed and the term asbestosis coined in the 1920s. The need for regulatory action was recognised in 1931 by the United Kingdom Government, and Asbestos Industry Regulations came into full force in 1933.

In New Zealand, prior to World War II, products containing asbestos were imported but not manufactured. It was not until after the war, with the production of asbestos-cement building materials, that manufacturing involving asbestos commenced in any volume.

In 1964 a New Zealand occupational standard was set for asbestos fibres in air and, in 1984, the import of raw friable crocidolite and amosite was banned by a Custom Import Prohibition Order. In 1999 this ban was extended to chrysotile . The import of raw asbestos is now prohibited by the Hazardous Substances and New Organisms (HSNO) Act 1996 as this substance does not currently have a HSNO Act approval.

On 1 October 2016, the import of asbestos-containing products was banned by Order in Council under the Imports and Exports (Restrictions) Act 1988. In specific, limited circumstances a permit to import can be sought from the EPA. These circumstances are: no alternative product is available or the alternative is disproportionately expensive compared to the risk of asbestos exposure; and that any risk of asbestos exposure is able to be safely managed. If granted, a permit will be valid for a year. The number of permits is likely to be low, and for asbestos-containing products associated with older machinery and the restoration of vintage planes and ships.

Until the 1980s concerns in developed countries about asbestos were primarily related to the gross occupational exposures that had formerly taken place and the legacy of asbestos-related occupational disease that was still emerging after long latency periods.

However, asbestos exposure may also occur to a limited degree through para-occupational exposure, such as living in the vicinity of asbestos-related industries or bringing home contaminated clothes, tools, etc. The general population may also be exposed if they live close to asbestos-related industries or an asbestos-containing waste site, or may be exposed from a variety of asbestos-containing products, from poorly performed asbestos removal, or from living with deteriorating asbestos material.

Public agencies found they needed to respond to the new public interest in asbestos, particularly where it occurred in such places as schools. For example, in the USA, Australia and New Zealand, programmes to manage asbestos risks in schools and hospitals began in the early 1980s. In New Zealand, a report by the Asbestos Advisory Committee was made to the Minister of Labour in April 1991 (OSH 1991).

Some public concern continues to be expressed about asbestos-cement products, which were used widely in New Zealand buildings until the mid 1980s. These products normally provide a matrix that binds asbestos fibres, preventing their release, but drilling or sawing, especially with power tools that disturbs or damages the material can lead to fibres being released into the air. Airborne asbestos fibres released as a result of such mechanical work are a hazard to health. Super 6 corrugated sheets were commonly used up until the 1970s as a roofing material in both domestic and industrial buildings. Deteriorated sheets will shed fibre over time with age and weather creating potential public health risk.

Purpose of the guidelines

The guidelines provide guidance to public health units (PHUs) that contribute to the management of risks to health from asbestos in non-occupational settings. Also, technical information is provided to assist PHUs in their risk assessments.

People may be exposed to asbestos in non-occupational settings, primarily in and around the home.

Properly applied, the guidelines will assist with determining:

- the risk arising from an asbestos hazard
- appropriate advice on managing the risk, including risk communication.

In the non-occupational setting, asbestos exposure is unlikely to present a high level of risk. If exposure is encountered the affected person should be encouraged to inform their general practitioner. People exposed or potentially exposed to asbestos in an occupational or para-occupational setting may have heightened concerns about asbestos and require health counselling or other support.

Exclusions

These guidelines exclude the following settings and activities:

- places of work
- ambient (outside) air
- drinking-water
- manufactured mineral fibres, such as wool, glass.

Management of enquiries concerning asbestos

The number of agencies that are potentially involved when members of the public make enquiries concerning asbestos often leads to confusion and frustration. The usual agencies involved are public health units, regional councils, WorkSafe New Zealand (WorkSafe) and territorial authorities.

This guideline provides guidance to PHUs on how to approach asbestos enquiries and how to manage interagency involvement. These measures will require cooperation and coordination at a local level by each agency and should involve formal agreements on how to proceed. Identifying a lead agency in any given set of circumstances may be required. The following issues need to be addressed.

- Is the issue about public health?
- Is the issue about worker safety?
- What is the lead agency?
- What role do other agencies have?

Is the issue about public health?

Public health is defined in the New Zealand Public Health and Disability Act 2000 (NZPHD) as the health of all of:

- the people of New Zealand, or
- a community or section of people.

The public health role is managed by the public health units of the district health boards (DHBs) as contracted by the Ministry of Health and defined in the NZPHD.

Asbestos issues or hazards have a general and a specific component derived from sections 22 and 23 of the NZPHD.

1. The **general component** is derived from section 22 of the NZPHD, which sets out the objectives of DHBs.

   22(1) Every DHB has the following objectives. (Amongst others)

   (a) to improve, promote and protect the health of people and communities.

   For asbestos this obligation will be met by:

   - responding to public (non-occupational) enquiries
   - providing technical information and advice on asbestos-related matters
   - directing enquiries/complaints to an appropriate lead agency
   - investigating asbestos situations that may have public health implications
   - investigating non-occupational asbestos disease notifications
   - responding to emergencies involving asbestos in a non-occupational setting.

2. The **specific component** is derived from section 23 of the NZPHD – Functions of DHBs:

   (h) to promote the reduction of adverse social and environmental effects on the health of people and communities.

   This specific public health role relates to the definition of public health as 'a community or section of such people'. These people are the people not covered by statutory responsibilities of other agencies in relation to asbestos and public health.

   The other agencies that have health responsibilities relating to asbestos are:

   - WorkSafe (Health and Safety at Work Act 2015 ; the HSW Act)
   - regional councils (Resource Management Act 1991; the RM Act)
   - territorial authorities (Health Act 1956, Building Act 2004).

   Each of these agencies is the lead agency under its legislation. PHU staff need to be careful to avoid taking the lead role in situations that are properly the responsibility of the affected person or of other regulatory agencies.

Is the issue about worker safety?

The Department of Health and the Department of Labour jointly administered the Asbestos Regulations 1978 through to 1983. In 1983 new regulations were then prepared to take notice of changes in attitudes to asbestos. With the advent of the HSW Act 2015 and changes to how workplace health and safety were to be managed, the Health and Safety at Work (Asbestos) Regulations 2016 became the sole responsibility of WorkSafe. In effect, all activities involved with asbestos were to be managed under the all-inclusive workplace regime. The only area not covered concerns the private person in their own home and these guidelines deal with this area.

WorkSafe has prepared guidelines for working with asbestos. Readers are encouraged to read them to understand the procedures for safe working with asbestos in any environment (see www.business.govt.nz/Worksafe).

Places of work are covered by the HSW Act WorkSafe is responsible for enforcing the HSW Act. The home, public buildings and schools may be places of work if contractors are doing work in them. Section 45 of the HSW Act covers duties of workers, for example:

> *While at work, a worker must—*
>
> *(b) take reasonable care that his or her acts or omissions do not adversely affect the health and safety of other persons.*

Identify the lead agency in any particular instance

Ambient (outside) air is covered by the Resource Management Act 1991. The Ministry for the Environment administers the RM Act and regional councils implement the RM Act in so far as it relates to the discharge of contaminants to air. Asbestos also occurs in ambient air from natural sources and sources like vehicle brakes. The management of such diffuse sources of exposure is not considered in these guidelines but the PHU should be aware of them. Air inside dwellings and point source release of asbestos around dwellings would, however, be covered by these guidelines. Drinking-water is covered by the *Drinking-water Standards for New Zealand* (Ministry of Health 2008).

Risk analysis

Most asbestos situations will involve personal health issues and will be related to a single person or a family. Public health advice can be given in these cases by the PHU if workers are not involved. PHU staff need to take care to avoid becoming involved in situations that are properly the responsibility of the person affected or other agencies.

If it is considered that the PHU should be involved, a risk analysis may assist in the decisions to be made.

A public health risk analysis model is outlined in *A Guide to Health Impact Assessment* and forms the basis for these guidelines (Ministry of Health 1998). There are three sequential steps in the process of decision-making regarding risk:

1. risk assessment
2. risk communication
3. risk management.

Risk assessment asks: 'What are the risks?' and 'Who will be affected, how, and to what extent?' It includes hazard identification, dose–response assessments, exposure assessment, and risk characterisation.

At the first step in the risk assessment process, hazards have to be identified. If the assessment of the hazard suggests that the likelihood of a risk is small, or that control is straightforward and safe, it may not be necessary to proceed to the quantification of risk. In general, as exposures experienced by the public will normally be considerably lower and less frequent than those experienced in the workplace environment, the expected exposure to asbestos will be likely lower than those by workers.

The second step in risk assessment is the consideration of dose–response of the health effects from exposure to the identified hazards. Dose–response models are developed from occupational data and extrapolated to low levels of exposure. The dose–response models that are used are subject to considerable debate about the validity of the assumptions made.

The next step in risk assessment considers who might be exposed and their characteristics, the routes of exposure and the extent, duration and frequency of the exposure to the hazards identified.

The information from these three steps is used in risk characterisation, the final step of risk assessment.

The acceptability of risk is a decision for either individuals or society as a whole. Without societal judgements about acceptable risk, no decisions can be reached on proposals that carry both benefits and risks. On the other hand, individuals expect to suffer no more than negligible harm from environmental hazards, unless they are taking voluntary risks in the pursuit of some activity in which they see benefits. Various scientific and regulatory bodies have set levels of what they consider to be acceptable risks, but it is uncertain whether these levels will be understood or accepted by individuals.

Although risk management and risk communication are discussed separately (see Chapters 3 and 4), these two other steps in risk analysis need to be integrated in the delivery of services. During any communication of risk, there must be adequate consultation on the risks, and public concerns must be taken into account. Risk management seeks to address the following questions: 'How can risks be avoided or reduced?', 'What are the options?', 'Are contingency and emergency plans adequate?', 'How can differing perceptions of risk be mediated?' and 'Can future health risks be predicted?'

Chapter 1: Risk assessment Part 1: Hazard identification

Asbestos

Asbestos is a common term describing a variety of naturally occurring hydrated silicate minerals which exhibit properties rendering them useful in manufactured products. Asbestos is composed of silicate chains bonded with magnesium, iron, calcium, aluminium, and sodium or trace elements to form long, thin, separable fibrils. These fibrils are arranged in parallel and a single microscopically-observed asbestos fibre can represent multiple fibrils that have not separated.

The morphology of the asbestos fibres differs between the groups. The most commonly mined forms of fibrous asbestos are:

- serpentine: chrysotile, an iron–magnesium silicate white in colour
- amphiboles: crocidolite, an iron–sodium silicate blue in colour; and amosite, an iron and magnesium silicate grey-brown in colour. Actinolite, tremolite and anthophyllite occur in both fibrous and non-fibrous forms and have rarely been mined as commercial asbestos. Amphiboles are distinguished readily only on basis of variation in chemical composition.

Both groups are naturally fibrous but the sizes (length and width) and the shapes of industrial fibres may differ. Figure 1 illustrates the different types of asbestos fibres and their theoretical formulae.

Chrysotile fibres are a bundle of thousands of agglomerated fibrils, which in section appear like a scroll of paper resulting in a vast surface area and possess elasticity, flexibility and good tensile strength. It is based on an infinite silica sheet (Si_2O_5) in which all the silica tetrahedra point one way. On one side of the sheet structure, and joining the silica tetrahedra, is a layer of brucite, $Mg(OH)_2$. Chrysotile fibres have a width of 0.1–1.0 μm (fibrils are less than 0.020 μm) and are thinner and curlier compared with those in the amphibole group (see Figure 2).

The amphibole group are straight, hard, sharp, needle-like structures with a double chain of silica tetrahedral which makes it very strong and durable. The external surface of the crystal structures of the amphiboles is quartz-like, and has the chemical resistance of quartz. The fibres may break longitudinally to form very fine fibrils. Crocidolite retains good flexibility and has high tensile strength. Fibres are 1–2 μm in width (fibrils 0.080μm) and up to 70 mm long. Amosite fibres are quite flexible and are weaker than the other forms. Fibre width is 1–2 μm (fibrils 0.100 μm) and length is up to 70 mm (see Figure 3).

Mechanisms of action

The exact mechanism responsible for the carcinogenicity of asbestos fibres is not known although available data indicates that both interaction between fibres and cellular components and cellular mediated pathways may be involved (ATSDR 2001a). For a given concentration of asbestos in air, the degree of exposure (magnitude or intensity, frequency and duration), fibre dimension (length and diameter), fibre durability or persistence in the lung and iron content are important determinants of asbestos toxicity (ATSDR 2001a). These fibre properties are briefly summarised below.

Fibre dimension (length and diameter)

Fibre dimension affects respirability (respiratory zone falls off above aerodynamic diameters of 5 μm) and clearance by alveolar macrophages (Donaldson and Tran 2004). These properties of asbestos fibres make them accessible to lung and other tissues through inhalation. It is believed that the dimensions of the asbestos fibre determines how far into the lungs it is likely to be deposited and how quickly it is cleared. Longer fibres induce a more vigorous acute and chronic inflammatory response than shorter fibres. Longer fibres are also more fibrogenic and carcinogenic than shorter fibres. The exact basis for these size-dependent differences is unclear (Goodglick and Kane 1990). Short fibres are cleared more efficiently than longer ones. Fibres >100 μm long are not respirable and hence do not pose a risk, unless they are first broken down into shorter fibres (enHEALTH 2005). Literature reviews of fibre sizes have concluded that the shorter fibres present low or no risk to human health (ATSDR 2003). However several studies (Dodson et al 2003; Dodson et al 2005; Suzuki et al 2005; Dodson et al 2007) report the presence of very short fibres in lung and pleural tissue from patients with malignant mesothelioma. Furthermore, some animal studies found that short, thin chrysotile fibres were contributing to the induction of malignant mesothelioma and concluded that asbestos fibres of all lengths induced pathological responses including the induction of experimental mesothelioma (Suzuki et al 2005). Wide fibres (diameter greater than 2 to 5 μm) tend to be deposited in the upper respiratory tract and cleared.

Fibre durability

Fibre durability is believed to be a major determinant of fibre-induced pathogenicity. Fibres whose chemical structure renders them wholly or partially soluble once deposited in the lung are likely to either dissolve completely, or dissolve until they are sufficiently weakened focally to undergo breakage into shorter fibres. The biopersistence of fibres may explain their ability to produce diseases with long latency periods (Broaddus 2001). The remaining short fibres can then be removed through phagocytosis and clearance. The largest size asbestos particles tend to deposit on the nasal mucosa or the oropharynx and are sneezed out or swallowed and never reach the lungs (NIOSH 2011). Durability seems to be greatest for amphiboles and less for chrysotile (Lippman 1984). For example acidic conditions (eg, in the stomach) and high temperatures will cause chrysotile fibres to dissolve rapidly while the amphibole fibres are degraded more slowly than serpentine fibres of the same dimensions (Schreir 1989).

Fibre type

There has long been considerable debate about the health risks associated with specific types of asbestos (McDonald and McDonald 1997). Although all forms of asbestos are considered hazardous, different types of asbestos fibres may be associated with different health risks. Results of several studies suggest that amphibole forms of asbestos may be more harmful than chrysotile, particularly for mesothelioma risk, because they tend to stay in the lungs for a longer period of time (ATSDR 2001a; IARC 2012a). Scientific reviews support that the difference in mesothelioma potency, that is, the estimated risk of mesothelioma associated with a unit increase (in fibre-years) in exposure to amphibole versus chrysotile asbestos fibres is considerable. However, estimates vary. For example, Hodgson and Darnton (2000) estimated that the potency differential between chrysotile and amphibole asbestos for lung cancer was between 1:10 and 1:50. Bardsley (2015) refers to one estimate suggesting the risk for mesothelioma according to fibre type was 1:100:500 for chrysotile, amosite, and crocidolite, respectively. In a 2010 analysis, which included more mesothelioma cases from updated cohorts, Hodgson and Darnton (2010) estimated that the ratio of potency for mesothelioma was smaller:1: 14 for chrysotile versus amosite and 1:54 for chrysotile versus crocidolite.

The risk of lung cancer associated with exposure to chrysotile compared with amphibole fibres is still highly contested. Hodgson and Darnton (2000) estimated the potency differential between chrysotile and amphiboles for lung cancer to be between 1:10 and 1:50. Berman and Crump (2008) reported similar findings — they estimated that chrysotile was less potent than amphiboles by a factor ranging between 6 and 60, depending on the fibre dimensions considered.

Iron content

The presence of iron in the fibres or the ability of the fibres to absorb and accumulate iron is a suggested mechanism for explaining the toxic and particularly carcinogenic effects of asbestos (Fubini and Mollo 1995). The presence of iron in the fibres (which may contain up to 30% of iron w/w) can act as a catalyst for the Fenton reaction that generates highly toxic hydroxyl radicals from hydrogen peroxide (Broaddus 2001). This seems to be also a key factor for asbestos toxicity and for the formation in the lung of the asbestos bodies that are the hallmarks of asbestos exposure (Ghio et al 2004). All types of asbestos contain iron cations, either as part of their crystalline lattice structure (crocidolite and amosite, and amounting to as much as 27% by weight in crocidolite) or as a surface impurity (chrysotile). Reactive oxygen species may be generated at the surface of asbestos fibres by chemical reactions that are catalysed by the iron component of the fibres. Or they may be the result of frustrated phagocytosis of asbestos fibres by alveolar macrophages or neutrophils (Shukla et al 2003).

Figure 1: Types of asbestos fibres and their theoretical formulae

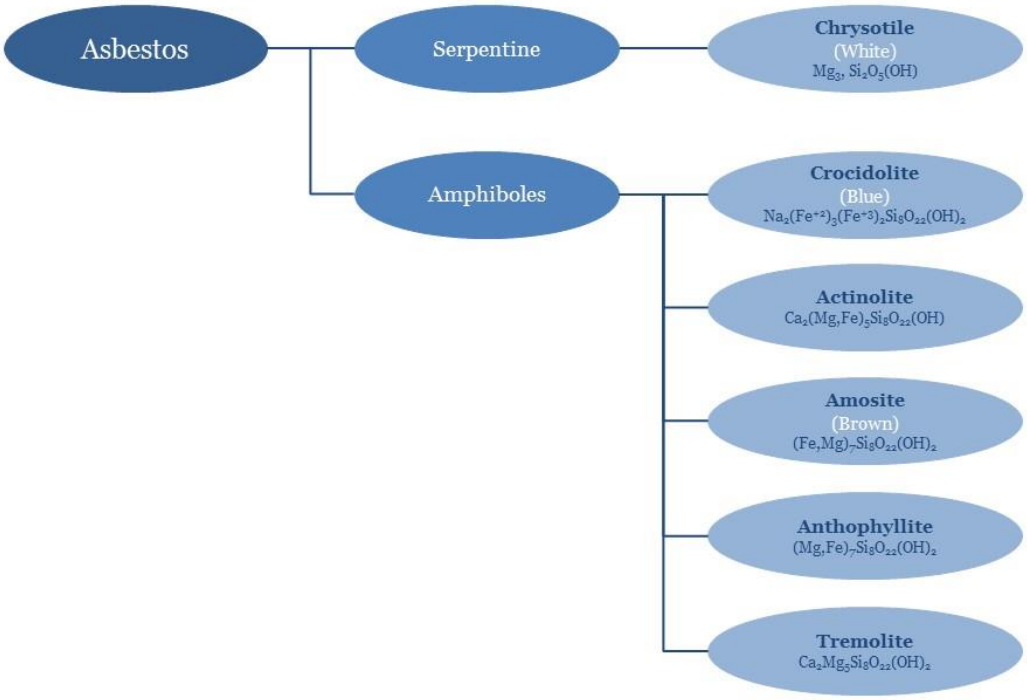

Figure 2: Serpentine asbestos

Source: www.atsdr.cdc.gov

Figure 3: Amphibole asbestos

Source: www.atsdr.cdc.gov

Kinetics

Health effects due to asbestos exposure have been clearly associated with inhalation where inhaled asbestos fibres are deposited in the upper and lower respiratory tracts. The relevance of the oral intake of asbestos fibres for human health is unclear. For this reason, only the deposition, retention and clearance of fibres from the human lungs are described.

Deposition

The degree of penetration in the lungs is determined by the fibre dimension (length and diameter), with thin fibres having the greatest potential for deep lung deposition (NTP 2014). In addition to the degree of exposure , the carcinogenic potential of asbestos fibres is associated with fibre durability in the lung and capacity to translocate across membranes (ATSDR 2001a). It is believed that the dimensions of the asbestos fibre determines how far into the lungs it is likely to be deposited and how quickly it is cleared. Wide fibres (diameter greater than 2 to 5 μm) tend to be deposited in the upper respiratory tract and cleared.

Clearance and retention

Fibres whose chemical structure renders them wholly or partially soluble once deposited in the lungs are likely to either dissolve completely, or dissolve until they are sufficiently weakened to undergo breakage into shorter fibres. Some of the smaller inhaled asbestos fibres are deposited on the surface of the larger airways where some of them are cleared by mucociliary transport and swallowing (Broaddus 2001). The remaining short fibres can then be removed through phagocytosis and clearance. Short fibres are cleared more efficiently than longer ones (enHEALTH 2005).

Several mechanisms are involved in the clearing of fibres from their site of deposition, ie mucociliary clearance, translocation of alveolar macrophages containing small fibres, and uptake by epithelial cells lining the airways. The most important physiological clearance mechanism is by alveolar macrophages with respect to phagocytosis. Short fibres are easily phagocytised, while the clearance for fibres longer than 20 μm is prolonged. Inflammatory conditions in the lung (for example, smokers) also contribute to impairment of alveolar macrophage-mediated mechanical clearance. One of the most effective clearance mechanisms (mucociliary clearance) is impaired by smoking. Co-exposure to tobacco smoke and asbestos fibres substantially increases the risk for lung cancer and the effect is at least additive and the heavier the smoking, the greater the risk (WHO 2014). Overall, the clearing mechanisms are very effective (95–98%) in non-smokers, although some fibres can remain in the alveolar regions.

Mechanisms of toxicity

The exact mechanisms by which asbestos causes disease are not fully clear (ATSDR 2001a; Barile 2010). In the IARC report, Kane (1999) proposed five mechanistic hypotheses involved for pathogenicity of asbestos:

1. Fibres generate free radicals that damage DNA (eg, see iron content above).
2. Fibres interfere physically with mitosis.
3. Fibres stimulate proliferation of target cells.
4. Fibres provoke a chronic inflammatory reaction leading to prolonged release of reactive oxygen/nitrogen species from macrophages.
5. Fibres act as co-carcinogens or carriers of chemical carcinogens to the target tissue.

Further research is needed to clarify evidence in favour of or against any of these proposed mechanisms.

Health effects

Asbestos is a proven human carcinogen (IARC Group 1). All forms of asbestos can cause cancer (WHO 2010). The inhalation of airborne asbestos in significant quantities causes mesothelioma (a cancer of the pleural and peritoneal linings), asbestosis (fibrosis of the lungs) (WHO 2014) and cancer of the lung, larynx and ovary (IARC 2012a; WHO 2014). Some scientists believe that the amphibole type is more potent in causing mesotheliomas than the serpentine type (chrysotile) (IARC 2012a).

Asbestosis

Asbestosis refers to diffuse or multi-focal fibrosis (scarring) in the lungs caused by the inhalation of asbestos fibres. It is considered that this disease only occurs in those who have been exposed to considerable concentrations of asbestos over a long time. The symptoms do not usually appear until about 20 to 30 years after the first exposure to asbestos. The person develops an insidious onset of shortness of breath and dull chest pains. Fibres penetrating to the peripheral air spaces initiate alveolitis, which, if chronic, results in scarring and fibrosis. The extent of lung inflammation and destruction is related to the amount of asbestos retained in the lungs, the fibre type and length, and individual susceptibility. Some evidence suggests that more retained asbestos is required to produce asbestosis than to produce asbestos pleural plaques.

The risk of asbestosis is insignificant for those who do not work with asbestos. The disease is rarely caused by neighbourhood or family exposure (US EPA 2007). High fibre doses (25–100 f/mL/yr) are generally required to produce clinically significant asbestosis within an individual's lifetime, with milder fibrosis at lower dose levels (Smartt 2004). Asbestosis is a marker of high asbestos exposure in individuals and its prevalence is a potential indication of high exposure in populations.

Non-malignant pleural conditions

Pleural plaques, and the less common diffuse pleural fibrosis, have been correlated with higher lung burdens of amosite, crocidolite and probably chrysotile than those of the general population. Cases are asymptomatic. They tend to develop after long latency periods, usually more than 20 years after exposure. Prevalence is related to duration of exposure and possibly to peaks of exposure. Although pleural plaques are not precursors to lung cancers, evidence suggests that people with pleural disease caused by asbestos exposure may be at increased risk of lung cancer (ATSDR 2001a).

Benign diseases of the pleura may be the only manifestations of exposure to asbestos (occupationally and even non-occupationally). They are considered to be important as they are likely to be the most common way in which those who may be affected by asbestos exposure can be identified (HEI – Asbestos Research 1991).

Carcinoma of the lung

The most common form of cancer caused by asbestos is bronchogenic carcinoma or lung cancer. Epidemiological studies in the occupational setting have confirmed an association between asbestos exposure and lung cancer, even in non-smokers. This association is considered causal, although the rates in various studies have differed. The latency period is measured in years and appears to be directly related to cumulative exposure. Exposure to asbestos in combination with cigarette smoke results in more than additive risk of lung cancer. Asbestos workers who smoke are about 90 times more likely to develop lung cancer than people who neither smoke nor have been exposed to asbestos (US EPA 2007).

Mesothelioma

Mesothelioma is a rare cancer of the cells lining body cavities. It is the classic tumour associated with asbestos exposure, and is unrelated to smoking (Muscat and Wynder 1991). Epidemiological evidence shows that approximately 70 to 90 percent of mesothelioma cases can be related to asbestos exposure (Youakim 2005). It is reasonable to assume that virtually all cases of mesothelioma are linked with asbestos exposure (US EPA 2007).

Cases of death from mesothelioma have been reported in studies of workers or of people exposed environmentally to each of the main types of asbestos, predominantly chrysotile, amosite, crocidolite and tremolite. There are several studies that suggest that amphibole asbestos (tremolite, amosite and crocidolite) may be more potent than chrysotile (IARC 2012a). The latency period is usually 35 to 40 years or more from the time of first exposure, although shorter periods have been recorded. The initial symptom is likely to be chest pain. Most cases have been associated with occupational exposure to asbestos or contact with contaminated clothing of asbestos workers (IARC 1987).

Asbestos-containing materials

Because of their exceptional insulating, fire-resistant and reinforcing properties, asbestos-containing materials have been utilised widely: in buildings in surface-applied finishes (for acoustical, decorative and fire-retardant purposes); in asbestos-cement products in sheet and other moulded forms; as thermal insulation in the construction of buildings; as well as in equipment used in buildings.

After about 1960, chrysotile was predominantly used in asbestos-containing materials in buildings such as asbestos-cement products, decorative coatings, vinyl sheet floor covering (Lino) and tile flooring. Crocidolite and amosite are also likely to be present, though in smaller amounts, in products prior to about 1960.

Over the last 50 years the removal of sprayed-on asbestos insulation materials from public buildings has increased and now few buildings still have this material. Decorative coatings in buildings and homes have also been largely removed as redecorating becomes necessary. Much asbestos-backed floor covering has been removed as well but, due to the difficult nature of this activity, some floor coverings have been overlaid with new coverings. By far the largest quantity of asbestos-containing materials around today is textured ceilings and wall cladding followed by cement sheet. These materials usually do not present a high risk unless they are disturbed. To a considerable extent, they are also gradually being removed as upgrading or demolition takes place.

The presence of asbestos in materials cannot be determined definitively by visual inspection. Actual determinations can only be made by instrumental analysis (polarised light microscopy (PLM), scanning electron microscopy (SEM), or transmission electron microscopy (TEM)). It is best to assume that the product contains asbestos until laboratory analysis proves otherwise.

In the home the primary asbestos-containing materials are:

- surfacing materials (such as asbestos-cement products used for cladding and roofing, and decorative/textured internal coatings on ceilings)
- thermal and fire insulation
- moulded materials (such as asbestos-cement products for gutters and down-pipes)
- electrical backboards
- backings to vinyl sheet floor coverings, and in the matrix of vinyl tiles.

Much less common asbestos-containing material may occur in the home as:

- lagging or insulation in old heating appliances, or around pipes and older hot water cylinders
- some external textured coatings
- plastic products, caulkings and other composites
- a woven sheath around old vulcanised India rubber insulated wiring
- built-up roofing felts.

Old household items with asbestos-containing material may include: asbestos simmer mats for stoves, oven gloves, mats on ironing boards, fire blankets and other asbestos textiles, electric heaters, hair dryers and older model toasters. All these items are unlikely to be in common use today.

Other sources of asbestos dust in the home may include dust from automotive friction materials or take-home dust from occupational exposure to asbestos.

In public buildings used as workplaces, asbestos-containing material may occur as:

- surfacing materials (eg, sprayed or trowelled asbestos-containing material on surfaces such as decorative finishes on ceilings, fireproofing materials on structural members, acoustical asbestos-containing material on the underside of concrete slabs or decking, and fire- and heat-resistant linings to boiler rooms)
- thermal system insulation (lagging and moulded insulation of heating and cooling service pipes, ducts, boilers and tanks to prevent heat loss, gain or condensation, and thermal insulation coatings or layers in the structure)
- miscellaneous asbestos-containing material such as asbestos-cement panels, cladding, roofing, pipes and other mouldings, asbestos-containing ceiling or floor coverings, and incidental uses in packings, friction materials, textiles, plastics reinforcement, gaskets and filters.

Asbestos-cement products

Composite materials containing Portland cement, sand and some form of fibrous reinforcement may generically be called ‘fibre-cement’ products. These products occur in housing and public buildings.

Chrysotile, in particular, is resistant to alkaline cement, which gives it an advantage over other reinforcing fibres. From 1982 to the present day, cellulose fibres have replaced asbestos in the make-up of a proprietary product called *New Hardiflex*.

In asbestos-cement products, the asbestos fibres are bound in the cement/sand matrix, often in small bundles just visible to the naked eye (usually about 10–12 percent asbestos). The material tends to become more brittle with age. Surface deterioration can occur due to acid rain, abrasion, or persistent damp conditions aided by organic growths.

Asbestos containing products typically used in housing included *Fibrolite* (from 1972 to 1982), *Durock* (up to 1974), *Coverline* or *Highline* profiled sheets (1972 to 1982), *Hardiflex* or *Hardiplank* (up to 1982), *Harditherm* (1972 to 1982) and *Durotherm* (up to 1974).

The *Fibrolite*, *Durock and Polite* mix was similar and contained chrysotile and a small amount of amosite. The mix was mouldable into corrugated and other forms such as gutters. Early products, up to the 1950s, probably contained crocidolite and the percentage of asbestos was higher, reputedly up to 50 percent. *Hardiflex* was not a mouldable material but was more flexible. (Fletchers produced an equivalent product, some of which was in the form of siding.) Sheet material may be found internally as linings in wet areas such as bathrooms, or in storage areas, occasionally as bench tops. *Harditherm* and *Durotherm* (22 percent asbestos) were used for fire protection and insulation. They were softer and easily nailed; they may become friable at the edges.

Summary of uses

1. Roofing: bold roll corrugated sheet (super 6); narrow roll corrugated sheet, shingles.
2. Walls: shingles, flat sheet (generally 9 mm), profiled sheet (eg, Coverline and Hiline).
3. Ancillary: guttering and down-pipes in various sizes, other moulded items (eg, garden troughs), roofing components (eg, verge and ridge trim).

Decorative coatings

Decorative internal coatings produced between 1964 and 1983 generally contained 5–9 percent chrysotile asbestos, and were applied as textured ceilings in housing and public buildings. The coating was able to mask imperfections in a substrate otherwise unsuitable for paint finishes. In public buildings, similar coatings were applied for decorative and/or acoustic purposes on ceilings and other surfaces out of reach. Also textured external/internal paint finishes and fake brick overlays were introduced. Use of product containing asbestos probably ceased around 1984.

The asbestos fibres and other fillers (such as expanded vermiculite and polystyrene) were bound together with adhesives to form the product. Portland cement was not included. Trade names of decorative coatings include *Glamortex* and *Whispar*. Licensed contractors were generally used by the producers of the products. Nuplex Industries, for example, are able to identify from their records particular jobs, their dates and the contractors used.

The products up to the late 1970s may be beige in colour (those containing vermiculite or perlite); those from 1980 to 1983 may be white (containing expanded polystyrene granules). The coatings are rather soft, because of the expanded materials in them, and could be damaged by impact or abrasion. Despite the soft nature of the coating, the fibres are generally well bound in a matrix of adhesive and filler-binders unless damaged or disturbed, when the material can become friable. The coating may be softened by water, and any areas that have become damp could suffer deterioration and poor adhesion to substrates; after drying out the material may be friable.

External decorative textured coatings were also made, using resin binders. Use is likely to have ceased around 1984.

Vinyl tile and sheet

There are two categories of resilient floor coverings containing asbestos: sheet material, consisting of a polyvinyl chloride layer with a chrysotile paper backing, and floor tiles, in which chrysotile is uniformly dispersed throughout the material. Vinyl-asbestos floor tile is made of 15 percent polyvinyl chloride (and sometimes asphalt) as the thermoplastic binder, with 10 to 20 percent asbestos and other mineral additives and pigments. The products may have been installed up to 1989. When sheet material is removed, the backing tends to remain adhered to the floor by the glue layer, presenting problems for safe removal. Vinyl sheet floor covering may be referred to as *Lino*.

The vinyl-asbestos floor tile must be regarded as a special type of asbestos-containing material in that abrasion in normal use can release dust if not properly maintained. Properly waxed, these floor coverings can be considered as encapsulated. However, buffing, wax stripping and other abrasive treatments can cause the release of fibres. Unique analytical problems arise in examining dust from such floors, and most fibres are less than 3 μm in length (HEI – Asbestos Research 1991).

Asbestos-containing material generally only in larger buildings

The following are types of asbestos-containing material that are largely found only in larger buildings.

- **Acoustic plaster soundproofing** is a firm, open-pored, plaster-like material, applied by trowel. The soundproofing material is usually exposed and not painted.
- Asbestos-containing material used in **insulation used for thermal system insulation** (TSI) – air-conditioning ducts, hot and cold water pipes, hot water reservoirs, pressure tanks, and boilers – is generally covered with a fabric or metal jacket. Fire doors often contain laminates of asbestos materials covered by wood or metal. The asbestos-containing material enclosed by the outer coverings is likely to be friable. These are old techniques and unlikely to be found today.
- A number of methods of **lagging** have been used on boilers, condensate tanks and steam headers; and pipes carrying steam, hot and chilled water and condensate, including:
  - raw asbestos/water mixture (or pre-formed asbestos blocks attached to the underlying surface) with an outer layer of wire-netting reinforcement finished with a cement of fine clay/asbestos
  - pre-formed pipe lagging of asbestos-containing material, usually in two halves wrapped in calico and traditionally painted red or white
  - asbestos paste to finish lagging around valves and bends, and as repair to damaged areas.

These are older techniques that are not likely to be used today.

Typical concentrations of fibres in various environments

The outdoor environment

Typical concentrations of asbestos fibres in the outdoor environment provide a useful yardstick for comparison with indoor environments. Examples of reported values are 0.1 f/L (fibres per litre), with more than 0.1 f/L downwind of local sources, such as vehicle braking (ATSDR 2001a). Fibre concentrations (fibres >5 μm in length) in outdoor air ranged between 0.1 and about 10 f/L, levels in most samples being less than 1 f/L based on surveys conducted in Austria, Canada, Germany, South Africa and the USA before 1986 (WHO 1998). A later survey carried out in Canada, Italy, Japan, the Slovak Republic, Switzerland, the United Kingdom and the USA showed means and medians of between 0.05 and 20 f/L (WHO 1998). The usual concentrations are 0.01 f/L in rural areas and up to 0.1 f/L in urban areas (ATSDR 2001a).

Public and residential buildings

Airborne asbestos concentrations in residences containing asbestos-containing material (ACM) are often reported combined with those for schools and other buildings as the number of sampled residences reported in the literature is small.

Reported concentrations in residential buildings all relate to building with asbestos-containing materials, which were, or were suspected of being, friable or were being damaged. The concentrations ranged from 'not detected' to 2.0 f/L but were generally in the range of 0.2 f/L to 0.4 f/L (Bignon et al 1989; CPSC 1983; HEI – Asbestos Research 1991; WHO 1986). ATSDR (2001a) suggests that the background indoor air levels average around 0.20 f/L.

Typical concentrations of asbestos fibres in public buildings, even those with friable asbestos-containing materials, are within the range of those measured in ambient air. Fibre concentrations (fibres >5 μm in length) in buildings in Germany and Canada reported before 1986 were generally less than 2.0 f/L. Mean values reported in surveys in Belgium, Canada, the Slovak Republic, the United Kingdom and the USA were between 0.05 and 4.5 f/L. Only 0.67 percent of chrysotile fibres were longer than 5 μm (WHO 1998).

Campopiano et al (2004) found a maximum level of 2.2 f/L in 59 Italian schools with ACM. The concentration exceeded the acceptable level of 2 f/L for re-occupancy following removal only in areas in which the ACM was undergoing continuing disturbance or seriously damaged.

Lee and Van Orden (2008) evaluated the results from 752 US buildings (only five were residences) with ACM sampled for defendants involved in asbestos in buildings litigation. Airborne asbestos was not found in 64 percent of indoor samples and no airborne asbestos ≥5 μm long was found in 97 percent. The mean indoor concentration of fibres ≥5 μm was 0.12 f/L.

The ATSDR's toxicological profile on asbestos reports indoor concentrations ranging from about 0.03 to 6 f/L. However concentrations depend on the amount, type and condition of ACM used in the building, eg, asbestos in floor tiles is less friable than that in sprayed coatings and release of fibres from ACM is sporadic and episodic (ATSDR 2001a).

Caretaker, maintenance, and renovation personnel may disturb or damage ACM during their work resulting in brief, relatively high exposure episodes. Such episodes have been poorly characterized. However there is no evidence that episodic peaks in exposure affect disease risk other than affecting cumulative exposure (ATSDR 2001a).

Release of asbestos fibres from asbestos-containing material

Intact asbestos-containing material is not a risk merely by its presence. Potential health problems only occur if asbestos fibres become airborne. Fibres are released when physical actions (deliberate or accidental) disturb the surface. Asbestos-cement materials will release fibres when sawed, drilled or otherwise worked or damaged. Materials such as asbestos-cement pipe can release asbestos fibres if broken or crushed when buildings are demolished. Other asbestos-containing material, such as decorative coatings, acoustic insulation and thermal system insulation, is vulnerable to damage during building maintenance operations, from vandalism and accidental damage. The use of power tools to drill and cut through asbestos-cement material can generate a significant number of airborne fibres.

In a small study undertaken for claimants in litigation against the manufacturer of amphibole asbestos-contaminated attic insulation[1] three US homes were sampled over three to four days during various activities. Background air samples were below the limit of detection or very low. Activities included cleaning in the attic, removing the insulation, and cutting a hole in the ceiling of a living space below insulation. The average depth of insulation above the ceiling was 10 cm and the hole was of a size that might be needed to install a recessed light (Ewing et al 2010).

Release of asbestos fibres during removal

During the disturbance and removal of friable spray-on asbestos in multi-storey buildings, high concentrations of asbestos fibres have been reported outside the enclosures where work was taking place (IPCS 1989). In the examples given, before work commenced, the background average concentration was less than 0.2 f/L. During the removal phase, concentrations outside the enclosures increased to between 14 and 290 f/L (generally around 70 f/L) then they declined over 16 to 35 weeks to between 1.0 f/L and 0.4 f/L. During a simulated maintenance activity on sprayed asbestos, a local concentration of around 30 f/L was observed. Such levels are unlikely to be reached during careful removal of asbestos-cement materials from the home. However, using electrical sanders to remove the backing material of vinyl sheet covering or Lino will produce significant airborne fibres.

Release of asbestos fibres during normal wear and weathering

Fibres can also be released naturally through corrosion and weathering of asbestos-cement products. The measurement of fibres released from corroded and weathered asbestos-cement products has been attempted by Spurney (Bignon et al 1989). Investigations measured the release of fibres in simulated wind speeds between 1 and 5 metres/sec. The results showed that:

- asbestos-cement surfaces corrode and weather as a result of aggressive atmospheric pollution (eg, sulphur dioxide, aerosols and acid rain) in proportion to the acidity of the rain and concentration of pollutants
- the surface cement matrix is destroyed and a layer approximately 0.1–0.3 mm of free fibres is built up and bundles of fibres are visible to the naked eye
- wind can disperse the fibres into the ambient air with emissions in the range 10^6 to 10^9 fibres per square metre per hour (with rates affected by pollution intensity and weather)
- about 20 percent of fibres are dispersed in the air and 80 percent washed out by rain

[1] Vermiculite attic insulation is contaminated with amphibole asbestos (mainly tremolite, winchite and richterite) at concentrations of less than 1 percent.

- there were crystallographic changes in the corroded chrysotile fibres, and pollutants (metal and organic substances) were deposited on the free fibres
- fibre concentrations in the vicinity of buildings with corroded and weathered asbestos-cement products (fibres greater than 5 μm) were from 0.2 to 1.2 f/L.

A study on asbestos-cement products in Western Australia found that deteriorating asbestos-cement roofs were common and that asbestos was present in the gutters and run-off water. The highest concentration of asbestos was from roofs 10–17 years old; younger and older roofs produced lower concentrations. Air monitoring at nine sites suggested that the air concentrations are likely to be less than 2.0 f/L and more likely to be less than 0.2 f/L (Western Australian Advisory Committee on Hazardous Substances 1990).

Elevated levels of fibres have been detected following the use of high pressure jets for cleaning asbestos-cement roof surfaces. These fibres may be deposited on soil and other surfaces around the home and create an increased risk of airborne fibres when dry.

Release of asbestos fibres from a fire

Asbestos was widely used because of its fire resistance properties, however it is not thermally stable when exposed to high temperatures. Chrysotile decomposes at 800–850°C and the amphiboles at 800–1000°C. Asbestos fibres will readily be converted to dust at prolonged exposure to such temperatures.

In sheet form asbestos does not offer any fire resistance and it cracks in building fires. In a fire, asbestos cement sheeting will disintegrate and can explode, releasing fibres over a wide area, mostly in the direction of prevailing wind. Further information is provided in Appendix 2.

Drinking-water and aerosolisation

There is a theoretical possibility of exposure to airborne asbestos from drinking-water aerosols and dried asbestos deposits. Where significant fibre concentrations were found in water supplies, the fibre length median was generally between 0.5 and 1.0 μm, although fibres greater than 5 μm were present in the distributions (HEI – Asbestos Research 1991). Hence, the aerosolisation of water from faucets and showers or secondary resuspension of deposits remaining after evaporation of water, may give rise to indoor air concentration of asbestos fibres. In another study (Webber et al 1988) air measurements were made in some homes using water containing 24 million f/L and in other homes containing 1.1 million f/L. Mean values from a combination of background, showering and vacuuming activities showed that homes with the more polluted supply gave fibre and mass concentrations about four times higher than the ones with the less polluted supply. No data on exposure from this source have been found.

Most asbestos fibres in water are chrysotile and are <5 μm in length (ATSDR 2001a). Available data on effects of exposure to chrysotile asbestos specifically in the general environment, including data from ecological studies of populations in Connecticut, Florida, California, Utah and Quebec and from a case-control study in Puget Sound, Washington State, are restricted to those in populations exposed to relatively high concentrations of chrysotile asbestos in drinking-water, particularly from serpentine deposits or asbestos-cement pipe. Limited data indicates that exposure to airborne asbestos released from tap water during showers or humidification is negligible (WHO 2013, 2017).

Chapter 2: Risk assessment

Part 2: Dose response, exposure assessment and risk characterisation

Dose–response

Dose refers to the amount of material potentially available for deposition in the respiratory tract (ie, the number of fibres in the air inhaled by the exposed person), while response is defined as the cumulative risk of developing an abnormality. Asbestos causes cancer in a dose-dependent manner (WHO 2000a). The greater the exposure, and the longer the time of exposure, the greater the risk of contracting an asbestos-related disease. The relative risk of lung cancer increases with cumulative dose of asbestos (Gustavsson et al 2002). Although no increased incidence of cancer has been observed in some exposed populations, no threshold has been identified below which no carcinogenic effect will occur (WHO 2014; IARC 2012). Exposure should therefore be kept as low as possible (WHO 2000a).

The International Agency for Research on Cancer (IARC) reviewed available data on the carcinogenicity of asbestos (IARC 1987). Overall, there was sufficient evidence for carcinogenicity and asbestos was classified as group 1, namely carcinogenic to humans. Although not entirely established, asbestos may be considered a genotoxic carcinogen hence is thought not to exhibit a threshold under which adverse effects are not seen. There is evidence that chrysotile is less potent than the amphiboles, but as a precaution chrysotile has been attributed the same risk estimates.

The total burden of residual fibres in the lungs depends not only on the size of the fibres but the amount of fibres inhaled from the environment (Dodson et al 2003). A clear dose-response relationship between cumulative exposure to asbestos and pleural mesothelioma in a population-based case control study in five regions in France with retrospective assessment of exposure was reported by Iwasatubo et al (1998). Current non-occupational exposure levels are considered to be too low to cause asbestosis. Mesothelioma (a highly specific outcome of asbestos exposure) occurs at lower exposure levels than asbestosis or lung cancer. It is the disease most likely to occur in relation to non-occupational exposures (Bardsley 2015). 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 (ie cumulative amount breathed in) (Bardsley 2015).

Potential for non-occupational human exposure

The primary route by which the general population might be exposed is inhalation of air that contains asbestos fibres. Asbestos can also enter the body through ingestion but is a less common exposure pathway. Significant skin contact is unusual, but asbestos fibres may penetrate into the skin and can lead to calluses or corns (NTP 2014). However, asbestos fibres that penetrate the skin do not appear to pass through the skin into the blood (ATSDR 2001a).

Small quantities of asbestos fibres are ubiquitous in air, arising from natural sources (weathering of building asbestos-containing, wind-blown soil from hazardous waste sites, deterioration of automobile clutches and brakes, or breakdown of asbestos-containing materials). The levels of asbestos in dust and wind-blown soil may be higher for those living close to a site for mining or processing asbestos and certain other ores, or a building containing asbestos products that is being demolished or renovated, or a waste site where asbestos is not properly covered. In studies of asbestos concentrations in outdoor air, chrysotile is the predominant fibre detected (IARC 2012b).

In non-occupational exposure, the typical exposure is a low or very low, almost unmeasurable, background concentration, but occasional high exposure when there is a disturbance of some kind (Hillerdal 1999). A simulation study carried out by Goswani et al (2013) showed that the results for domestic exposures are lower than workers' exposures and are proportionate to background concentrations. The highest risk of exposure to asbestos in the home is through home maintenance, renovating, repair, and remodeling, where home occupants can potentially be exposed to higher levels of airborne asbestos than levels in general ambient air (ATSDR 2001b), for instance by cutting or drilling through asbestos-cement sheeting or sanding down asbestos-containing Lino or tiles. Left undisturbed, such materials pose a negligible risk; therefore it is recommended that asbestos-containing material in good condition be left alone.

There is an ongoing, although low, risk of exposure to asbestos fibres in a home from damaged or deteriorating asbestos-containing insulation, walls, ceiling or floor tiles. Friable asbestos (which would crumble easily if handled) is more likely to generate airborne fibre, hence increasing the risk of exposure to asbestos. The risk of generating airborne asbestos fibres can be reduced by appropriate management measures (eg, removing the friable material or sealing the surface).

There remains the possibility that individuals engaged in asbestos-related activities – such as renovating or demolishing buildings with damaged or deteriorating asbestos – could bring asbestos into the home. Workers' families and other household contacts may be exposed by breathing asbestos dust: from workers' skin, hair and clothing, and during laundering of contaminated clothes (Goswani et al 2013).

Exposure assessment

A knowledge of exposure is essential for environmental epidemiology and hazard control. Asbestos exposure affects not only asbestos workers but also their families, users of asbestos products, and members of the public who are exposed to building materials and asbestos in heating and ventilating systems (LaDou 2004).

Routes of exposure

Inhalation

Inhalation is the primary route by which the general population might be exposed to asbestos. Small quantities of asbestos fibres are ubiquitous in air, arising from natural sources (weathering of asbestos-containing materials), wind-blown soil from hazardous waste sites, deterioration of automobile clutches and brakes, or breakdown of building asbestos-containing materials. Indoor air in buildings with asbestos-containing materials can be a major source for non-occupational asbestos exposure (HEI – Asbestos Research 1991).

Non-occupational exposures may also occur by way of para-occupational exposure. In some of these cases, workers' families may inhale asbestos fibres released by clothes that have been in contact with asbestos-containing material. People who live or work near asbestos-related activities may also inhale asbestos fibres that have been released into the air by the activities.

Oral

Drinking-water

The general population can be exposed to asbestos in drinking-water. Asbestos can enter potable water supplies through the erosion of natural deposits or the leaching from waste asbestos in landfills, from the deterioration of asbestos-containing cement pipes used to carry drinking-water or from the filtering of water supplies through asbestos-containing filters (IARC 2012b).

The adverse effects following ingestion of asbestos have not been clearly documented. ATSDR (2001a) considers few fibres are able to penetrate the gastrointestinal tract. This means non-gastrointestinal effects from oral exposure to asbestos are unlikely. There is considerable controversy as to whether ingested asbestos fibres can penetrate and pass through the walls of the gastrointestinal tract in sufficient numbers to cause adverse effects. There is inconsistent evidence of carcinogenicity of ingested asbestos in epidemiological studies of populations with drinking-water supplies containing high concentrations of asbestos. Moreover, in extensive studies in experimental animal species, asbestos has not consistently increased the incidence of tumours of the gastrointestinal tract. There is therefore no consistent evidence that ingested asbestos is hazardous to health. The primary issue surrounding asbestos-cement pipes is for people working on the outside of the pipes (eg cutting pipes) because of the risk of inhalation of asbestos dust (WHO 2003, 2017).

The World Health Organization (WHO 2003, 2017) concluded that there was little evidence that ingested asbestos is hazardous to health and therefore did not feel it necessary to establish a health-based guideline value for drinking water.

Soil

Soil may be contaminated with asbestos by the weathering of natural asbestos deposits, or by land-based disposal of waste asbestos materials.

Food and other sources

The use of asbestos filters in food or pharmaceutical preparations has been discontinued in the US since 1976, and intake of asbestos through foods or drugs is now unlikely. However, asbestos has been found in art supplies such as crayons, probably as a contaminant of the talc used to strengthen the crayons (IARC 1977). Asbestos was detected in crayons in New Zealand in 2015 (http://www.health.govt.nz/news-media/media-releases/asbestos-crayons).

Dermal exposure

Asbestos fibres can penetrate into the skin, producing asbestos warts. However, asbestos fibres that penetrate the skin do not appear to pass through the skin into the blood (ATSDR 2001a).

Measurement of exposure

Exposure to asbestos cannot be measured by absorbed dose or other biological measurement (at least not until after death) – unlike, for example, lead exposure. The options available, therefore, may be ranked as:

- direct estimation by personal air sampling (in the breathing zone)
- indirect estimation by stationary air sampling of personal environments
- qualitative exposure categorisation on the basis of questionnaires, interviews, inspections, historical records and/or exposure simulations
- categorisation into 'exposed' and 'unexposed' populations.

For all measures of exposures there are ethical, practical, and cost limitations. Logistical issues, quality control, sampling methods, sensitivity and specificity all need to be considered, and expert laboratory advice is generally needed before exposure measurement is undertaken. Appendix 1 details the procedures and issues around asbestos sampling and analysis.

Risk characterisation

Underlying asbestos risk assessment, and hence its health impact, are assumptions that are difficult to make. Extrapolation from observations of asbestos workers to predict the cancer risk caused by exposure in non-occupational situations involves estimating exposure and establishing a formula for the relationship between exposure and risk. While there may be difficulty in estimating the excess risk, provided that the excess is substantial and suitable comparison rates are available for the local population, measuring exposure and choosing an appropriate dose–response model are substantially more difficult.

Risk characterisation involves integrating the outcomes of the previous steps in the risk assessment: hazard identification, dose response assessment and exposure assessment. Achieving this integration requires making a number of assumptions in cases where empirical information is not available. These assumptions result in a number of uncertainties associated with the risk assessment, which need to be acknowledged and discussed.

In assessing the risks of asbestos in the non-occupational environment, it is necessary to consider a number of uncertainties, ranging from indirect estimates of exposure to the use of mathematical equations derived by the application of mathematical models to observations in workers. The following are some of these uncertainties:

- It can be difficult to identify and characterise the hazards.
- Risk estimation for non-occupational exposure relies on extrapolation from much higher levels of exposure in industry.
- Assessment of risk at low concentrations of asbestos fibres can be only indirect.
- The concentrations of asbestos fibres to which people may be exposed are far below the levels at which adverse effects have been reported for workers in the past.
- Reported cases of mesothelioma from non-occupational exposure to asbestos have been associated with para-occupational exposure, domestic exposure, and/or neighbourhood exposure near asbestos mines or asbestos-using industries.

- For lung cancer, there are data to support increased incidence related to cumulative dose at high and moderate levels, but there are no real grounds that a linear relationship for lung cancer can be extrapolated back to the dose in non-occupational settings.
- There may also have been a low incidence of lung cancer as a consequence of para-occupational exposure to asbestos but it has not been possible to demonstrate this epidemiologically (because of the high background incidence of this disease). Alternatively, the inability to measure an effect of low levels of asbestos on lung cancer may be because this disease is not caused by low levels of exposure to asbestos.

Table 1 illustrates 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 ACMs. The materials should be assumed to be ACMs if there is uncertainty.

Table 1: 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 minimised 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 clean-up 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 clean-up 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.

Source: Bardsley 2015

Public health risks from non-occupational exposure to asbestos-containing materials

There is little epidemiological evidence for health effects from inhalation exposure to ACM in buildings, and what evidence we have is weak (Goldberg and Luce 2012). This is not surprising given the difficulties of carrying out such an epidemiological study.

Despite several decades since public concerns about health risks from asbestos in buildings surfaced uncertainty about the cancer risk estimates remains high. Reasons include lack of satisfactory statistical power to detect effects at very low levels of exposure, issues relating to fibre measurement,[2] the representativeness of sampled buildings, possible insufficient latency period for pleural mesothelioma as use of ACM in buildings started in the 1960s, difficulty in evaluating individual cumulative exposure, and the lack of a truly unexposed population.

As the mechanism of action for asbestos-associated disease is unknown risk assessment models typically extrapolate from historical high occupational levels to low non-occupational levels using a linear no-threshold approach. The estimates are generally regarded as the upper limits of risk based on worst case assumptions such as amphibole or mixed amphibole and chrysotile exposure.

For 752 US buildings involved in litigation, using the US Environmental Protection Agency Integrated Risk Information System model, cancer risk estimates ranged from 2.1 per million for people working in schools to 1.1 per million for people working in public/commercial buildings. The cancer risk estimate for background outdoor exposure was 0.4 per million (Lee and Van Orden 2008). This compares to a previous lifetime asbestos-related cancer[3] risk estimate for building occupants based on results from buildings not involved in litigation by the US Health Effects Institute (HEI) of about 4 per million (HEI – Asbestos Research 1991).[4]

To better address the significant uncertainties more recent risk assessment often includes a range of exposure (or dose) – response models leading to a range of possible outcomes. For example, ATSDR derived risk estimates based on an exposure-response model for lifetime estimated risk of mesothelioma and lung cancer combined for exposure to naturally occurring asbestos in El Dorado County, California which ranged from 0.1 per 10,000[5] to 22 per 10,000 (Case et al 2011).

Typically epidemiological studies of non-occupational asbestos exposure and health effects include para-occupational exposure in the definition of 'non-occupational' and often do not include results without this exposure. For example, Bourdes et al (2000) carried out a meta-analysis of studies on domestic or neighbourhood exposure and pleural mesothelioma published from 1966 to 1998.[6] Only eight studies were identified. There was a range of study types and sources of asbestos exposure but not ACM in buildings. Domestic exposure included para-occupational exposure in all but one study.[7] Due to the inclusion of para-occupational exposure and study location generally in areas with predominant or concomitant amphibole exposure the

[2] Issues include fibre size and type, analytical methods, and lack of or little measurement.

[3] Mesothelioma and lung cancer.

[4] The mean concentrations of the 198 US schools, residences and public and commercial buildings containing ACM on which the HEI's risk estimate was based ranged from 0.04 to 2.43 f/L (95th percentile 1.4 f/L, n of air samples=1377). The mean concentration for 96 included residences (n of air samples=215) was 0.19 f/L (HEI – Asbestos Research 1991).

[5] A risk of 1 in 100,000 (0.1 per 10,000) is defined as an acceptable risk by the Ministry of Health.

[6] The text says 1988, but the references indicate this is a typographical error.

[7] In this study, domestic exposure was from natural asbestos-based materials used to whitewash floors and walls.

exposure characteristics, and hence summary relative risk, are not relevant to the New Zealand domestic context.

Some reports, published prior to 2000, of cases of mesothelioma were only due to known exposure to ACM in buildings. There have also been several earlier US studies of the prevalence of pleural and/or parenchymal radiological abnormalities in caretaker and maintenance staff in asbestos-insulated schools but none included a control group and confounding factors were not or only partially taken into account (Goldberg and Luce 2012).

Five mesothelioma cases (expected 0.2-0.7) were reported in 2001–02 among staff of a French university campus with asbestos-insulated buildings and no other known exposure. It was unclear whether the principal exposure was from proximity to construction of asbestos-insulated buildings or working in them (Buisson et al 2006[8] cited in Goldberg and Luce 2012).

A small European multi-centre case control study of mesothelioma suggests domestic exposure from handling ACM for maintenance or the presence of ACM susceptible to damage increases mesothelioma risk. Thirty-two[9] (32/215) cases in this study had domestic and/or environmental exposure only but for eight the only known source of exposure was some form of ACM in the home, in particular an asbestos roof (n=6). All six of these cases came from one city (Magnani et al 2000).

An Italian population-based case control study defined domestic exposure as ACM in the garden, courtyard, roof or inside the house. The estimated excess mesothelioma risk due to domestic exposure was 1.3 (95% CI 0.6–2.7).[10] This was similar with adjustment for residential distance from an asbestos cement plant (RR 1.3; 95% CI 0.8–2.3) (Maule et al 2007).

Analysis of cases from the Western Australian Mesothelioma register supports the need for health and safety guidelines in any renovation where asbestos exposure is possible. From 1960 to 1988 5.3 percent of cases were attributed to home renovation/maintenance or exposure as a bystander during these activities (n=87/1631). This compares to 4.6 percent of cases with no known exposure source. Western Australian incidence rates from non-occupational exposure have been increasing since the mid-1980s. Among developed countries Australia had the highest per capita use of asbestos-cement products and more than 60 percent of crocidolite mined in Australia was used in asbestos-cement product manufacture (Olsen et al 2011).

A small US study found pleural radiological abnormalities from non-occupational community exposure to amphibole asbestos from the processing of vermiculite. The association was stronger for long-term lower exposure from background exposure to fugitive plant emissions than intermittent high exposure from activities such as playing on vermiculite waste piles (Alexander et al 2012).

Several studies suggest environmental exposure from a natural or industrial source increases mesothelioma risk even at a distance where exposure is likely to be very low. A case control California cancer registry-based study of several thousand mesothelioma cases found the odds of mesothelioma, adjusted for occupational exposure, age and gender, decreased 6.3 percent with every 10 km of residential distance from the nearest source of naturally occurring asbestos (95% CI 1.8–10.5%; p=0.006). Study limitations included use of residence at time of diagnosis (when causative exposure occurred decades earlier), the assumption that ultramafic rock

8 This paper has not been seen, but the authors of the review (Goldberg and Luce 2012) were study co-authors.

9 Of the 32 cases, 14 had domestic and environmental exposure, seven had environmental exposure only and 11 had domestic exposure only. Three of the cases with domestic exposure were para-occupational exposure and eight were ACM in the home.

10 Adjusted for age, gender and occupational exposure.

location is a marker of asbestos exposure, incomplete information on asbestos exposure in occupations, lack of lifetime residential and occupational histories and no data on domestic asbestos exposure (Pan et al 2005).

A population-based case control study around Casale Monferrato, Italy, site of an asbestos cement plant found mesothelioma risk decreased rapidly with residential distance but at 10 km was still high (OR 5.8 95% CI 1.7–19.3 adjusted for age, gender, occupation in the asbestos cement industry, domestic exposure to ACM, and occupation in the asbestos cement industry of any relative). Lifetime residential and occupational histories were available in this study (Maule et al 2007).

A review of the health risks of non-occupational asbestos exposure by the Royal Society of New Zealand and the Office of the Prime Minister's Chief Science Advisor found asbestos exposure during the rebuild following the Canterbury 2010–11 earthquakes was unlikely to cause a significant increase in risk among residents, unless they were carrying out the work themselves, without taking proper precautions. Overall the risk was considered to be low if proper precautions were followed (Bardsley 2015).

Chapter 3: Risk communication

The general public does not base their perception of risk on technical risk assessment alone. Public recognition of risks, in contrast to risk assessment based on probabilities prepared by experts, includes intuitive risk perception. The characteristics of such perception appear to be related to concepts of fairness, familiarity, future and present 'catastrophic potential', and outrage at involuntary exposure to hazards not of one's own making. When communicating risks it is important to show commitment, empathy and sound knowledge.

Asbestos hazards at home, where people expect to be safe, are among the hazards that the public will judge based on more than a scientific risk assessment. Comparisons of the level of risk with common risks, such as road traffic crashes, will generally not convince a person who feels that they – or their child – is at risk. Involuntary exposures that could cause a dreadful disease at some unknown future time, in a way that is still not understood, and for which there is little hope of cure, are particularly alarming. The level of alarm is compounded for asbestos due to its legacy of a high incidence of disease in the occupational setting and allegations of mismanagement by regulators in the mid-20th century.

Effective risk communication is more likely to be achieved if:

- a careful and sensitive explanation is given to assist and improve the level of understanding of the risk
- the feelings of dread towards asbestos-related disease are recognised, and efforts are made to assist a person to come to terms with those feelings before decisions are made
- the response to hazards that may affect a large number of people (especially children) is made with urgency and at an appropriate level.

Bear in mind that in general:

- younger adults and better educated individuals tend to have more technical, scientific and medical knowledge about hazards
- the most concern about risks tends to be expressed by women, particularly those with young children, and by older people
- people tend to simplify complex and uncertain information into 'rules of thumb' (which, in the case of asbestos, may relate to the perception of occupational risk)
- people attempt to impose patterns on patternless events
- people overestimate the frequency of rare events and underestimate the frequency of common events
- individuals taking voluntary risks tend to be overconfident and believe they are not subject to the same risk as other individuals
- individuals forced to take involuntary risks overestimate the risk, and are unwilling to agree to 'acceptable risk' criteria set out by national and international agencies
- people tend to use past life experiences to relate to new situations, affecting their perception of the new situations (Health and Welfare Canada 1990).

Risk communication needs to be a two-way process, as described in some detail in *A Guide to Health Impact Assessment* (Ministry of Health 1998). It needs to be done in such a way that people are well informed and guided in the actions they can take, while knowing that the experts are taking account of, and acting on, their concerns.

To be effective communicators of the risks associated with asbestos in the non-occupational environment, PHU staff need to build credibility and trust with the affected individual or communities. Thus in any interaction, they need to:

- show that they are professionals committed to helping the affected people
- be open and receptive to the concerns expressed by the affected people
- establish their credentials to advise on the effects of asbestos
- be empathic.

In many cases, difficulties in managing environmental issues or communicating risks arise because the regulator's expectations differ from those of the affected people. Thus it is important to establish early in the process what the issues are, who is affected and what can be done about it and by whom. That is, the scope of the issue needs to be defined tightly (refer to Ministry of Health 1998).

For example, home owners who have put themselves at risk from asbestos eg, DIY renovation need to recognise that they are responsible for the problem and that they will need to manage it even though they can get advice from agencies. If contractors have caused the problem, then the issue should be referred to WorkSafe. This referral should be made in a way that cannot be interpreted as 'passing the buck'.

Chapter 4:
Risk management

Risk management

Priorities for managing risk should be based on the risk assessment, but should also consider public perception of risk. The range of risk reduction alternatives must be evaluated, including in relation to their social, economic and cultural implications.

This evaluation could be undertaken along two lines:

1. control of actions and events that can translate an asbestos hazard into an asbestos risk
2. the removal or near-permanent containment of the asbestos hazard.

Asbestos exposures in non-occupational settings may vary greatly. A protocol for the management of such exposures should aim to provide a response that is graded according to the likely harm (exposures are likely to be very low).

PHU staff investigating complaints should assess the issue and proceed according to a graded response protocol, identifying and assigning responsibility for the issue to the appropriate agency.

Summary of the *Graded Response Protocol*

Note: This guide is essentially an operational document for PHU staff to refer to when investigating complaints. The question 'Is it a public health issue – that is, a non-workplace issue?' needs to be spelt out first. If a member of the public has decided to engage a contractor then it becomes a workplace issue for the employer to manage, with WorkSafe enforcing the standards.

Step 1: Initial response and preliminary assessment

1.1 Gather and record information.

1.2 Identify and assess the hazard.

1.3 Decide whether to proceed to Step 2.

1.4 Identify and inform the agency most appropriate to take any further action.

1.5 If not proceeding to Step 2, provide support and precautionary advice.

Step 2: Inspection and hazard evaluation

2.1 Co-ordinate action/enforcement with the regulatory agency as appropriate and seek to carry out joint inspections.

2.2 Confirm initial information.

2.3 Obtain and record additional information to enable an adequate hazard evaluation.

2.4 Identify and characterise hazards.

2.5 Decide whether to proceed to Step 3.

2.6 Provide advice to manage hazards and potential exposure, and ensure action is taken.

Step 3: Exposure measurement, risk estimation and assessment

3.1 Co-ordinate action/enforcement with the regulatory agency as appropriate and encourage joint inspection visits.

3.2 Sample and analyse to determine presence and type of asbestos.

3.3 Measure the concentration of respirable asbestos fibres in air.

3.4 Estimate the exposure under normal conditions.

3.5 Estimate the excess risk from the exposure and assess its significance.

3.6 Communicate the risk.

3.7 Recommend actions to manage risk and ensure action is taken.

Background to the *Graded Response Protocol*

The guidelines and protocol aim to assist the PHU to identify asbestos hazards and risks using a combination of interviews, observations and laboratory testing. Risk communication and recommendations for the management of hazards and risk may then proceed.

The mere presence of asbestos does not always create a risk. The risk of developing asbestos-related disease depends on exposure to airborne fibres of respirable size. A graded response is based on the following three elements.

Hazard
|
Linking event or action causing exposure
|
Risk, and who is affected and in what way

More specifically, these elements are:

1. the nature and scale of the asbestos hazard and the corresponding potential to be a risk to human health
2. mechanisms that may open pathways of exposure to create risk
3. the nature of the risk in terms of probability, likely consequences, people affected and the degree of risk each may face. The existing state of health of each person will influence likely consequences for that individual.

Approaches to assessing hazards and risks

A graded response requires some way of assessing likely or actual human exposure to airborne asbestos. There are two complementary approaches.

1. Inspect, identify and assess deterioration of the material and the potential for fibre release (ie, a hazard)

This approach identifies the **hazard**. It should be used in every situation before exposure measurement.

A close inspection and assessment of the material alleged to contain asbestos will allow future action to be identified. If the condition of the material could result in the release of asbestos fibres into the air, then corrective action is justified. In making this assessment, the following questions should be answered.

- What type of asbestos-containing material is involved?
- Where is the material located?
- Is the material friable and likely to release fibres?
- Is there a potential for future disturbances, which may release fibres?
- Are individuals likely to be exposed to airborne fibres?

The nature and scale of the hazard should be estimated.

Settled surface dust sampling and analysis indicate the presence of asbestos contamination and the hazard, but cannot reliably indicate airborne dust concentrations.

At the end of the inspection and assessment process, a judgement may be made as to whether asbestos is being released and thus causing some unquantified exposure. It can be said that a risk has been established (ie, hazard + exposure) but the significance of the risk is still uncertain and unquantified.

2. Measure the actual or potential exposure (respirable fibres in air) from which health risk may be estimated

Measurement of exposure over time is important in quantifying **risk**. Exposure measurement by air sampling provides a measure of exposure over the time of sampling. However, air sampling should not be carried out in isolation and it is important that consideration should be given to the circumstances that created the need for air sampling. The type and extent of damage to the ACM involved needs to be taken into account to enable a full assessment to be taken. This adds to the uncertainty of risk assessment and underscores the importance of inspection and assessment.

Measuring airborne fibres in a non-occupational environment is difficult due to the low levels of fibre likely to be found. An assessment of the level of risk will need to be made before sampling is undertaken. Sampling is warranted if public health risk is likely, eg when the asbestos-containing material is friable and the likelihood of exposure is high.

Graded Response Protocol

How to use the *Graded Response Protocol* and *Report Sheets*

The *Report Sheets* at the end of the guidelines have spaces for information and decisions corresponding to the *Graded Response Protocol*. They repeat the information required but, once users are familiar with the guidelines, the *Report Sheets* may be used in the field without the whole document.

The principle is to grade the response to the level of hazard

In practice, Step 1 will always be completed. If the situation involves a place of work then the person conducting a business or undertaking (PCBU)[11] has duties to manage the risk, including through identifying hazards and ensuring a safe place of work. Steps 2 and 3 will only apply if a private home is involved and no contractor was involved – that is, not a place of work.

Step 1: Initial response and preliminary assessment

The aims of Step 1 are to:

- provide an initial response and support for the concerned person
- identify the agency most appropriate for further action
- identify the procedure to be followed for corrective action.

1.1 Gather and record information

Using the *Report Sheets* (Appendix 3), collect initial information from the informant by personal or telephone interview or possibly by a site visit.

Record informant details

- Contact person, their address and telephone number.
- Nature of concern.

Determine the location of potential asbestos hazard, type of building and building use

- Location (street address) of the suspected asbestos-containing material.
- Type of building(s) (eg, dwelling, school, public building).
- Building use(s).
- Other type of location (eg, a landfill, building or demolition site).

Assess the nature, condition, quantity and accessibility of potential asbestos hazard

- Description of the suspected asbestos-containing material.
- Information on the date that the suspected asbestos-containing material was installed (if known).
- Likelihood of being an asbestos-containing material (judging by the description and age).
- Likelihood of being a friable asbestos-containing material.
- Whether it is inside or outside the home or building.
- Level of accessibility of the suspected asbestos-containing material.

[11] While a PCBU may be an individual person or an organisation, in most cases the PCBU will be an organisation (eg, a business entity such as a company).

- Potential for future damage, disturbance or erosion of the suspected asbestos-containing material.
- Quantity of the suspected asbestos-containing material.
- Condition of the suspected asbestos-containing material (eg, deteriorating asbestos-cement product, damaged insulation).
- Whether the hazard is airborne (eg, visible dust or other assessment).

Identify actions that may translate the hazard into a risk to health

- Who is taking, or is proposing to take, or has taken the action.
- The nature of the action or disturbance affecting the suspected asbestos-containing material.
- Whether it appears likely that asbestos will be (or has been) released by the action.
- When the action or disturbance of asbestos-containing material is proposed, or when it happened.
- Whether a change in building use is proposed that may require interior modification and disturbance of surfaces that may use asbestos-containing material.
- Whether renovation or remodelling is proposed that may disturb asbestos-containing material.

Identify people at risk

- Who is at risk of exposure?
- How the people at risk of exposure could be exposed?
- The period over which the people may have been or will be at risk of exposure.
- The state of health of the people or population at risk of exposure.
- Who may be at greatest risk (eg, children, smokers)?

1.2 Identify and assess the hazard

Identify and assess:

- the possible presence of a friable asbestos-containing material,[12] its condition and accessibility
- actions that may release asbestos fibres.

1.3 Decide whether to proceed to Step 2: Inspection and hazard evaluation

Take into consideration the need to:

- recommend action where it appears that work on asbestos-containing material may have released asbestos fibres already
- support the informant by explaining the nature of the hazard and its management.

[12] Friable in relation to asbestos is defined in the HSW (Asbestos) Regulations 2016 as 'in a powder form or able to be crumbled, pulverised, or reduced to a powder by hand pressure when dry'.

1.4 Identify the most appropriate action to take

Consider whether the identified asbestos hazard requires the attention of WorkSafe, the territorial authority or (less likely) the regional council.

- Asbestos contamination within a private home may be the responsibility of the PHU.
- Asbestos contamination arising in workplaces, including through the actions of contractors at private homes, may require the attention of WorkSafe.
- Nuisances and/or conditions injurious to health should be acted on by the territorial authority.
- The discharge of asbestos as a contaminant involves the regional council.

> Note: Under the HSW Act, the person conducting a business or undertaking (PCBU) is responsible for managing the place of work and ensuring that it is safe for workers and others affected by the work it carries out. This responsibility applies to all work activities and all places of work. This self-management approach does not require WorkSafe to be involved unless there is concern that safety is being compromised. The HSW Act does not apply to private homes unless a contractor is engaged to work in the home.

1.5 If not proceeding to Step 2, provide support and precautionary advice

Common sense actions to avoid unnecessary exposures or hazards are required to minimise risk.

- Provide copies of the most recent resource *All About Asbestos* (HP 6710) and, if appropriate, *Removing Asbestos from the Home* (HP 6711) published by the Ministry of Health (2017a, 2017b).
- Emphasise the importance of seeking expert assistance if work with asbestos-containing materials is being considered.
- Confirm information given and remove all ambiguity. Make a site visit if you have not done so already. Be certain that information given is understood.

In all cases the capacity of the concerned person to understand the advice and take sensible action should be taken into account. Record the advice you have given in the *Report Sheets*.

> Note: The goal is to reduce the potential for exposure to airborne asbestos due to the release of fibres. A principle, therefore, is that exposure should be reduced as a result of the action recommended.

Step 2: Inspection and hazard evaluation

The aims of Step 2 are to:

- identify and characterise hazard
- achieve actions to manage hazard.

2.1 Co-ordinate action/enforcement with the regulatory agency as appropriate and seek to carry out joint inspections

2.2 Confirm initial information

Visit the site and, using the *Report Sheets*, clarify, confirm or amend the initial information.

- Clarify the nature and underlying factors of the concern, as an understanding of the way the risk is perceived is essential in framing advice.
- Inspect conditions at the site.
- Complete a new *Report Sheet* only if absolutely necessary. (Keep the first copy as a record of the initial step.)

2.3 Obtain and record additional information to enable an adequate hazard evaluation

Collect information to enable a proper assessment and characterisation of the hazard. Records of buildings held by the owners or the local authority may give some indication of asbestos-containing materials used in the construction of the building (and what asbestos-containing material may have been removed), but should not be relied on.

- Identify any further **suspected asbestos-containing material**.
- Determine **composition**, type and approximate amount of asbestos present in the suspected asbestos-containing material by sampling and analysis (see Appendix 1). Complete the asbestos sample record sheet(s), which also provides a space for results.
- Examine the **condition** of the suspected asbestos-containing material for deterioration.
- Note the **accessibility** of the suspected asbestos-containing material (eg, potential for damage, vandalism).
- Assess the **quantity** of asbestos-containing material.
- Obtain a clear description of the **actions** that could disturb asbestos-containing material and lead to the release of fibres (eg, drilling and cutting, removal, reuse, renovation, repair or redecoration).
- Identify **people at risk of exposure**.
- **Document** the information in the *Report Sheets*.

2.4 Identify and characterise hazards

From the complete information obtained, including the results of sample analysis, identify each opportunity for the release of asbestos fibres (including the likely potential release from further damage) and characterise each hazard with information about the:

- type of material – friable/not friable
- type of asbestos – chrysotile/amphibole
- degree to which asbestos may be released – significant/not significant.

Also rank the significance of the hazard (high/low), assuming that there is an open pathway for exposure to vulnerable people.

At this point of the inspection and assessment process a judgement may be made as to whether asbestos is being released and so whether a risk has been established (ie, a hazard and a pathway of exposure to a receptor exist). However, the significance of the risk may be still uncertain and unquantified.

2.5 Decide whether to proceed to Step 3: Exposure measurement, risk estimation and assessment

A decision on whether to proceed to Step 3 will depend on:

- the significance of the hazard identified in Step 2.4
- the likely pathways for exposure
- the presence of vulnerable people
- the number of people who may be exposed
- cost versus benefit of undertaking Step 3
- the degree of importance of quantifying the risk, as opposed to identifying the hazard, and whether such risk assessment will lead to a better decision on priorities and action.

In particular, consider the need to:

- show adequate support and understanding for serious concerns
- assist understanding and reduce uncertainty and/or suspicion
- provide 'hard' evidence to support the need for action and/or enforcement
- add to an understanding of environmental conditions and so assist in future situations.

> Note: The PCBU has responsibilities under the HSW Act if the site under consideration is a place of work. If a place of work is involved, they must make their own decisions about corrective action, in which they can be assisted by the information given above. A decision to proceed to Step 3 is not exclusive to providing advice as in Step 2.6 below. Advice should be offered at this stage regardless of whether exposure measurements and risk assessment will follow.

2.6 Provide advice to manage hazards and potential exposure, and ensure action is taken

Common sense actions to avoid unnecessary exposures or hazards are required to minimise risk.

- Provide copies of the most recent resource *All About Asbestos* (HP 6710) and, if appropriate, *Removing Asbestos from the Home* (HP 6711) (Ministry of Health 2017a, 2017b).
- Emphasise the importance of seeking expert assistance if work with asbestos-containing materials is being considered.
- Confirm information given and remove all ambiguity. Make a site visit if you have not done so already. Be certain that information given is understood.

In all cases the capacity of the concerned person to understand the advice and take sensible action should be taken into account. Record the advice you have given in the *Report Sheets*.

Step 3: Exposure measurement, risk estimation and assessment

The aims of Step 3 are to:

- confirm the presence and type of asbestos in air
- estimate the exposure to respirable asbestos fibres
- communicate the risk
- recommend actions to manage risk.

3.1 Co-ordinate action/enforcement with the regulatory agency as appropriate and encourage joint inspection visits

3.2 Sample and analyse to determine presence and type of asbestos

Steps 1 and 2 cannot confirm the presence of asbestos fibres in the air, their type or their concentration. Sampling and qualitative analysis of suspected asbestos-containing materials will be relatively simple and quick as no quantification is sought. Air may be sampled or, following discussion with the laboratory, settled surface dust or bulk material may also be sampled.

Take into account the:

- person(s) and location(s) that are of interest
- factors considered in Appendix 1
- limitations of the information which will do little more than assist in the characterising of the hazards as considered in Step 2.4.

3.3 Measure the concentration of respirable asbestos fibres in air

Sampling and analysis of the air can indicate the number of fibres in air. This information can then be assessed for risk against recognised standards. Sampling should be undertaken only following consultation with the examining laboratory, using its methods and equipment. The laboratory will interpret the results.

3.4 Estimate the exposure

Sampling should reflect the spaces most used so that an integrated exposure may be derived. A sufficient number of samples should be undertaken so that variations are reflected. The duration of sampling airflow through the filter membrane will be a factor in the sensitivity of the results and needs to be discussed with the laboratory.

When reliable results have been obtained, the concentration will be expressed in f/mL of air.

3.5 Estimate the risk from the exposure and assess its significance

The risks may be estimated in the following way.

- Compare the fibre concentration to which a person may have been exposed with the typical exposures and lifetime risk estimates.
- Compare the significance of the estimated risk against criteria of acceptable risk, taking into account the person at risk.
- The estimate achieved by undertaking this risk assessment should then be categorised as 'high', 'moderate' or 'low' rather than provided to the exposed person as a number. A number may imply a degree of scientific accuracy that is not possible to achieve in risk assessment.
- Note that the acceptability of the risk to the individual exposed is for them to decide; the expert assists by providing information and improved understanding.

3.6 Communicate the risk

Risk estimates are **estimates** only, and may vary by several orders of magnitude. They are useful as a tool for putting the risk in context, making comparisons with risks estimated using similar methods and prioritising management options. The public perception and judgement of risk is based on many other factors, as discussed in Chapter 3.

Individual susceptibility, tobacco smoking, other exposures to asbestos, and exposures to other hazardous substances will impact on an individual's risk of adverse health effects from any given exposure to asbestos in the home.

Further guidance on risk communication is provided in:

- *A Guide to Health Impact Assessment* (Ministry of Health 1998)
- *Communicating in a Crisis: Risk Communication Guidelines for Public Officials* (US Department of Health and Human Services 2002)
- *Risk Communication in Action: The risk communication workbook* (Reckelhoff-Dangel and Petersen 2007).

3.7 Recommend actions to manage risk and ensure action is taken

Common sense actions to avoid unnecessary exposures or hazards are required to minimise risk. The assessment of risk (rather than hazard) may provide a sharper focus on what needs to be done.

- Provide copies of the most recent health education resource *All About Asbestos* (HP 6710) and, if appropriate, *Removing Asbestos from the Home* (HP 6711) published by the Ministry of Health (2017a, 2017b).
- Emphasise the importance of seeking expert assistance if work with asbestos-containing materials is being considered.
- Confirm information given and remove all ambiguity. Be certain that information given is understood.

In all cases the capacity of the concerned person to understand the advice and take sensible action should be taken into account. Record the advice you have given in the *Report Sheets*.

Chapter 5:
Roles and responsibilities

Individuals and agencies with roles and responsibilities in preventing or managing asbestos hazards in non-occupational settings include:

- public health units
- territorial authorities (city and district councils)
- property owners, property managers and property occupiers.

Roles and responsibilities must be considered in three contexts:

1. the regulatory agency with statutory authority to bring about remedial action
2. the person or organisation responsible for taking remedial action
3. agencies with statutory functions to ensure that the facts are established and the best advice is made available.

> Asbestos hazards need to be managed collaboratively to avoid duplicated effort, wasted resources and the perception of 'buck passing', and to ensure the most effective statutory response. Thus it is important to determine who has jurisdictional responsibility as a first step, then to ensure the issues are being addressed.

Role of the public health unit

The PHU may often be the first to be made aware of a concern about asbestos. Preliminary investigations (as set out in the *Protocol* following) should establish the responsible person(s) and any need to pass on this information to the others. Particular roles for the PHU include:

- providing specialist advice in epidemiology and toxicology where risk assessment is complex
- preparing statements or advice about the risks to individuals or groups
- providing scientific advice on whether sampling is likely to be useful
- undertaking measurement and identification of asbestos
- communicating risk to the public and the media
- providing advice to other agencies on how to communicate statements about risk to the public and the media effectively
- providing advice to lead agencies with statutory authority to effect remedies.

Role of the health protection officer

The skills of the health protection officer are necessary for the following activities.

1. Initial response and preliminary assessment

- Receive, record and interpret queries and concerns.
- Identify the cause of concern or complaint, the location and the associated parties.
- Provide initial response and support to concerned people.

2. Inspection, hazard evaluation and risk assessment

- Identify person(s)/groups at risk.
- Identify confounding factors (eg, smoking, occupational exposure to asbestos).
- Identify sources of asbestos, asbestos hazards, and open pathways of exposure.
- Collect environmental samples for laboratory analysis.
- Interpret laboratory results.
- Seek advice from the Medical Officer of Health and others if necessary (eg, epidemiologists, toxicologists).
- Assess the likely health risk from the information collected.

3. Information and risk communication

- Explain how risk should be managed to the lead regulatory agency.
- Consult with building owners, building managers and occupiers as necessary.
- Refer information to the lead regulatory agency to bring about remedial action.

4. Management plans

- As a first step, determine who has jurisdiction, engage the lead regulatory agency and make sure that the issues are addressed.
- Assist the lead regulatory agency to determine appropriate action including, if necessary, the design of abatement and exposure control strategies.
- Maintain communication and cooperation with the regulatory agency and other parties (recognising privacy).
- Evaluate the effectiveness of the management plan.

5. Health protection

Here the primary role of the PHU is to support enforcement by the lead regulatory agency by providing information and advice.

The PHU may also consider health promotion initiatives aimed at increasing awareness of potential asbestos-containing materials and hazards associated with them. Health education resources are available from the Ministry of Health to support such initiatives. A general information booklet, *All About Asbestos* (HP 6710), has been prepared targeting the general public. Although obtaining expert assistance should always be recommended if work with asbestos-containing materials is being considered, a further information booklet, *Removing Asbestos from the Home* (HP 6711), is available if required.

Role of territorial authorities

In non-occupational settings, territorial authorities will normally be the lead regulatory agency with statutory authority to bring about a remedy. Territorial authority enforcement officers may collaborate with the other agencies, and the PHU should provide the territorial authority with information and advice. Since most issues to do with asbestos are likely to involve a workplace, territorial authorities should always co-ordinate action with WorkSafe to prevent duplication or confusion of roles.

Territorial authorities have duties and powers to prevent or control asbestos hazards under the following legislation.

Health Act 1956

The Health Act 1956 includes provision for territorial authorities to:

- improve, promote and protect the public health
- cause steps to be taken to abate nuisances or to remove conditions likely to be injurious to health or likely to be offensive
- enforce regulations under the Act
- make bylaws for the protection of public health
- issue cleansing orders or obtain closing orders.

Section 29 of the Act defines health 'nuisances' and generally includes matters 'likely to be injurious to health'. Particularly relevant are references to:

- accumulations or deposits
- situation or state of premises
- conduct of any trade, business, manufacture or other undertaking.

Enforcement is determined by the District Court if a nuisance is not abated voluntarily except where immediate action is necessary. Works undertaken by a territorial authority to abate a nuisance may result in costs being recovered from the owner or occupier. It should be noted, however, that any person can lay information regarding a nuisance. A nuisance has to exist before any action can be taken and, accordingly, is not an effective means of preventive action.

Under section 41 of the Act, the territorial authority may serve a Cleansing Order on the owner or occupier, specifying the work to be carried out and the time in which to complete it. A Closing Order made under section 42 or 44 can be issued as a last resort to protect the occupants, but such action will not, of course, resolve any external release of asbestos.

Building Act 2004

The Building Act 2004 includes provision for territorial authorities to:

- require work to be done to prevent buildings from remaining or becoming dangerous or insanitary
- take measures to avert danger or rectify insanitary conditions
- issue project and land information memoranda revealing (inter alia) known hazardous contaminants.

A building consent will be required in most cases where demolition or structural alteration works are to occur. The ability to impose conditions on building consents appears to be limited to inspections or entering premises (section 222). Nevertheless, territorial authorities could, at their discretion, include a 'Hazardous Building Material Warning' on relevant consent documents.

Project Information Memoranda (PIMs) issued by territorial authorities must include information identifying special features of the land relating to the likely presence of hazardous contaminants where it is:

- relevant to the design and construction or alteration
- known to the territorial authority
- not apparent from the operative district plan.

Section 44A of the Local Government Official Information and Meetings Act 1987 allows for an application for a LIM (Land Information Memorandum). Section 44A(2) states that the LIM must include information concerning the 'likely presence of hazardous contaminants'.

A PIM is required for a Demolition (Building) Consent. The PIM will advise if any restriction on demolition, for example a Heritage listing, exists in the city or district plan.

Sections 121 to 124 and 129 to 130 of the Building Act 2004 deal with dangerous or insanitary buildings. It is possible that the presence of asbestos could lead to a building being considered 'dangerous' or 'insanitary' for the purpose of the Act. 'Insanitary' buildings include those of such construction that they are likely to be injurious to health. In determining whether a building is insanitary, consideration must be given to:

- size of the building
- complexity of the building
- location of the building in relation to other buildings, public places and natural hazards
- intended use of the building, including any special traditional and cultural aspects of the intended use
- expected useful life of the building and any prolongation of that life
- reasonable practicality of any work concerned
- in the case of an existing building, any special historical or cultural value of that building
- any matter that the territorial authority considers to be relevant
- provisions of the building code.

Enforcement action is taken by way of formal notice requiring a remedy. An application for a Court Order authorising the council to do required work at the owner's expense may be made on default.

An offence is committed if a building is used for a purpose for which it is not safe or sanitary.

Cases relating to sections 64 and 65 of the earlier Building Act 1991 may be useful references. These cases include *Hyslop v Dunedin City Council* (21.6.93) AP 35/93 (J Doogue, HC, Dunedin), which deals with asbestos on a building site, and *Marlborough District Council v Chaytor* (1995) DCR 382.

Resource Management Act 1991

In the Resource Management Act 1991:

- section 15 prohibits the discharge of contaminants into the environment except where some form of authority or consent exists
- section 17 requires every person to avoid, remedy or mitigate adverse effects on the environment.

Enforcement orders (Environment Court) or abatement notices (enforcement officer) may be issued requiring a person to cease, or prohibiting a person from commencing, anything that is already or is likely to be:

- noxious
- dangerous
- offensive
- objectionable.

Similar action may require a person to take certain actions to avoid, remedy or mitigate adverse environmental effects.

The Resource Management Act 1991 also includes provision for territorial authorities to make plans and rules that deal with hazardous substances. The health protection officer should be aware of the appropriate provisions of plans, as advice given without such knowledge could create difficulties.

Waste Minimisation Act 2008

The Waste Minimisation Act 2008 includes the following provisions.

- Part 4 provides for territorial authority refuse collection and disposal of waste services.
- Disposal must be undertaken so as not to be a nuisance or injurious to health. Work generally must be to the satisfaction of the territorial authority but a Health Protection Officer may serve notice on a territorial authority for causing a nuisance.
- The Medical Officer of Health may collect and dispose of the waste concerned, and may recover the reasonable costs of doing so from a territorial authority if the territorial authority, or any person collecting the waste on its behalf, failed to comply with the notice.
- Bylaws may also be made prohibiting or regulating the deposit of refuse of any specified kind.

Demolition material containing asbestos will almost certainly arrive at council disposal sites. Service managers will need to determine strategies to deal with this issue to ensure environmental risk and council liability are minimised. Asbestos waste may only be disposed of at a place approved for the purposes by the territorial authority under the Resource Management Act 1991.

Role of property owners

Property owners and their agents should inform occupiers of the presence of asbestos-containing materials and must also act to remedy any asbestos hazards. Property owners should

seek expert advice from recognised and certified contractors if work with asbestos-containing materials is required.

Statutory obligations

Property owners must:

- meet statutory obligations (eg, under the Health Act 1956, the Building Act 2004)
- obtain necessary building consents and any other necessary consents including those for the disposal of asbestos waste.

Role of WorkSafe New Zealand

WorkSafe is responsible for the administration and enforcement of provisions under the Health and Safety at Work Act 2015 (HSWA). A guiding principle of HSWA is that workers and other persons should be given the highest reasonable level of protection against harm to their health, safety, and welfare from work risks.

WorkSafe may conduct investigations to ascertain whether the HSWA has been complied with. WorkSafe staff have considerable experience and expertise in investigation of hazards or incidents arising from incorrect or negligent use of hazardous substances in the workplace. Under provisions in the HSWA, WorkSafe may be required to investigate an asbestos incident. This may be at the request of the public health unit or a member of the public.

In general, WorkSafe will take the lead in corrective action involving asbestos contamination if contractors are involved in the home. Most circumstances where the release of asbestos fibres would be an issue will involve public buildings or a place of work. However, complaints about asbestos are likely to be received through the Ministry of Health and the PHU. Thus the PHU will have an important role in responding and co-ordinating subsequent action. It is advisable that the public health unit establish a procedure to cover the roles and responsibilities between PHUs and WorkSafe through, eg, a memorandum of understanding.

Health and Safety at Work Act 2015

The HSWA is administered by WorkSafe. Section 3(1a)of the HSWA provides for the protection of:

> *workers and other persons against harm to their health, safety, and welfare by eliminating or minimising risks arising from work or from prescribed high-risk plant.*

Section 3(2) requires that in furthering subsection 3(1a):

> *workers and other persons should be given the highest level of protection against harm to their health, safety, and welfare from hazards and risks arising from work or from specified types of plant as is reasonably practicable.*

Section 45 requires that workers, while at work, must:

> *(a) take reasonable care for his or her own health and safety; and*
>
> *(b) take reasonable care that his or her acts or omissions do not adversely affect the health and safety of other persons.*

Section 46 says:

a person at a workplace (whether or not the person has another duty under this Part) must—

(a) take reasonable care for his or her own health and safety; and take reasonable care that his or her acts or omissions do not adversely affect the health and safety of other persons.

Health and Safety at Work (Asbestos) Regulations 2016

The Health and Safety at Work (Asbestos) Regulations 2016:

- imposes general duties on persons conducting a business or undertaking (PCBUs) to ensure, so far as is reasonably practicable, that the workplace is without risks to the health and safety of any person
- prohibits a PCBU from carrying out, or directing or allowing a worker to carry out, work involving asbestos, other than in circumstances expressly permitted
- imposes a general duty on PCBUs at a workplace to eliminate exposure to airborne asbestos at the workplace
- requires asbestos removal work to be licensed, and requires notification of that work to WorkSafe and other persons by the PCBU with management or control of the workplace and by licensed removalists
- requires licensed asbestos removalists to ensure asbestos removal workers have appropriate training, and to provide information about the health risks of exposure to asbestos and the need for health monitoring.

Implementation of management plans

Responsible property owners will:

- administer and fund abatement work, environmental sampling and analysis
- engage licensed contractors for abatement work
- monitor the performance of contractors
- ensure that routine maintenance work practices do not generate asbestos hazards
- monitor the condition of the property and abatement work to ensure that asbestos hazards do not recur
- inform contractors, occupiers and other building users of any asbestos hazards
- advise occupiers on how to manage risks
- inform purchasers of known or suspected asbestos hazards.

Role of property occupiers

Tenants should advise their landlord of the development of any asbestos hazard, minimise damage to asbestos-containing material, cooperate with the landlord in facilitating abatement work and act on advice from the health protection team regarding the avoidance of asbestos hazards.

References

Alexander BH, Raleigh KK, Johnson J, et al. 2012. Radiographic evidence of non-occupational asbestos exposure from processing Libby vermiculite in Minneapolis, Minnesota. *Environmental Health Perspectives* 120(1): 44–9. ATSDR. 2001a. *Toxicological Profile for Asbestos*. Atlanta: Agency for Toxic Substances and Disease Registry.

ATSDR. 2001b. *Chemical-specific Health Consultation: Tremolite asbestos and other related types of asbestos*. Atlanta: Agency for Toxic Substances and Disease Registry.

ATSDR. 2003. *Report on the Expert Panel on Health Effects of Asbestos and Synthetic Vitreous Fibres: The influence of fiber length*. Atlanta: Agency for Toxic Substances and Disease Registry.

Bardsley A. 2015. *Asbestos Exposure in New Zealand: Review of the scientific evidence of non-occupational risks*. 43 pages. URL: http://www.pmcsa.org.nz/wp-content/uploads/Asbestos-exposure-in-New-Zealand_9April15.pdf (accessed 21 August 2015).

Barile F. 2010. *Clinical Toxicology: Principles and mechanism* (2nd edition). Informa Health Care USA, Inc, New York. In book: Clinical Toxicology: Principles and Mechanisms, Edition: 2nd, Publisher: Informa HealthCare Publishers, Editors: Frank A. Barile

Berman DW, Crump KS. 2008a. A meta-analysis of asbestos-related cancer risk that addresses fiber size and mineral type. *Crit Rev Toxicol* 38(suppl 1):49–73.

Berry G, Rogers AJ, Pooley FD. 1989. Mesotheliomas–asbestos exposure and lung burden. In: J Bignon, J Peto, R Saracci (eds). *Non-Occupational Exposure to Mineral Fibres*. IARC Scientific Publications No. 90. Lyon: IARC, pp. 486–96.

Bignon J, Peto J, Saracci R (eds). 1989. *Non-Occupational Exposure to Mineral Fibres.* IARC Scientific Publications No. 90. Lyon: World Health Organization International Agency for Research on Cancer.

Bourdes V, Boffetta P, Pisani P. 2000. Environmental exposure to asbestos and risk of pleural mesothelioma: review and meta-analysis. *European Journal of Epidemiology* 16(5): 411–17.

Broaddus VC. 2001. Apoptosis and asbestos-induced disease: is there a connection? *Laboratory and Clinical Medicine* 137(5): 314–5.

Campopiano A, Casciardi S, Fioravanti F, et al. 2004. Airborne asbestos levels in school buildings in Italy. *Journal of Occupational and Environmental Hygiene* 1(4): 256–61.

Case BW, Abraham JL, Meeker G, et al. 2011. Applying definitions of 'asbestos' to environmental and 'low-dose' exposure levels and health effects, particularly malignant mesothelioma. *Journal of Toxicology and Environmental Health* 14: 3–39.

CPSC. 1983. *Report to the Consumer Product Safety Commission by the Chronic Hazard Advisory Panel on Asbestos*. Washington: Directorate for Health Sciences, US Consumer Product Safety Commission.

Bernstein D, Dunnigan J, Hesterberg T, et al. 2013. Health risk of chrysotile revisited. *Crit Rev Toxicol* 43(2): 154–83 DOI: 10.3109/10408444.2012.756454.

Department of Health and Ageing. 2005. *Management of Asbestos in the Non-Occupational Environment*. Canberra: Department of Health and Ageing.

Ding M, Dong Z, Chen F, et al. 1999. Asbestos induces activator protein-1 transactivation in transgenic mice. *Cancer Res* 59(8): 1884–9.

Dodson RF, Atkinson MA, Levin JL. 2003. Asbestos fibre length as related to potential pathogenicity: a critical review. *Am J Ind Med* 44: 291–7. doi:10.1002/ajim.10263 PMID:12929149.

Dodson RF, Graef R, Shepherd S, et al. 2005. Asbestos burden in cases of mesothelioma from individuals from various regions of the United States. *Ultrastruct Pathol* 29: 415–33. doi:10.1080/019131290945682PMID:16257868.

Dodson RF, Shepherd S, Levin J, et al. 2007. Characteristics of asbestos concentration in lung as compared to asbestos concentration in various levels of lymph nodes that collect drainage from the lung. *Ultrastruct Pathol* 31: 95–133. doi:10.1080/01913120701423907 PMID:17613992.

Donaldson K, Tran CL. 2004. An introduction to the short-term toxicology of respirable industrial fibres. *Mutat Res* 553: 5–9. PMID:15288528.

enHEALTH. 2005. *Management of Asbestos in the Non-occupational Environment*. Canberra: Commonwealth of Australia.

Ewing WM, Hays SM, Hatfield R, et al. 2010. Zonolite attic insulation exposure studies. *International Journal of Occupational and Environmental Health* 16(3): 279–90.

Fubini B, Mollo L. 1995. Role of iron in the reactivity of mineral fibers. *Toxicol Lett* 82–83: 951–60.

Ghio AJ, Churg A, Roggli VL. 2004. Ferruginous bodies: implications in the mechanism of fiber and particle toxicity. *Toxicol Pathol* 32: 643–9.

Goldberg M, Luce D. 2012. The health impact of non-occupational exposure to asbestos: what do we know? *European Journal of Cancer Prevention* 18(6): 489–503.

Goodglick LA, Kane AB. 1990. Cytotoxicity of long and short crocidolite asbestos fibers in vitro and in vivo. *Cancer Res* 50: 5153–63.

Goswani E, Craven V, et al. 2013. Asbestos exposure: a review of epidemiologic and exposure data. *Int J Environ Res Public Health* 10: 5629–70; doi:10.3390/ijerph10115629.

Gustavsson, et al. 2002. Low-dose exposure to asbestos and lung cancer: dose-response relations and interaction with smoking in a population-based case-referent study in Stockholm, Sweden. *Am J Epidemiol* 155(11): 1016–22.

HEI – Asbestos Research. 1991. *Asbestos in Public and Commercial Buildings: A literature review and synthesis of current knowledge*. Cambridge, Massachusetts: Health Effects Institute.

Hillerdal G. 1999. Mesothelioma: cases associated with non-occupational and low dose exposures. *Occupational Environmental Medicine* 56: 505–13.

Hodgson JT, Darnton A. 2000. The quantitative risks of meso-thelioma and lung cancer in relation to asbestos exposure. *Am Occup Hyg* 44: 565–601.

Hodgson JT, Darnton A. 2010. Mesothelioma risk from chrysotile [Letter]. *Occup Env Med* 67: 432.

IARC. 1977. Asbestos. IARC Monographs, Volume 14. Lyon, France: World Health Organization, International Agency for Research on Cancer. URL: http://monographs.iarc.fr/ENG/Monographs/vol1-42/mono14.pdf

IARC. 1987. Asbestos and certain asbestos compounds. In: *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Chemicals, industrial processes and industries associated with cancer in humans*. IARC monographs, Vol 1–42. IARC monographs supplement 7. Lyon, France: World Health Organization, International Agency for Research on Cancer, 29–33, 56–58.

IARC. 2012a. Asbestos (chrysotile, amosite, crocidolite, tremolite, actinolite, and anthophyllite). IARC Monographs, Volume 100C. Lyon, France: World Health Organization, International Agency

for Research on Cancer. URL: http://monographs.iarc.fr/ENG/Monographs/vol100C/mono100C-11.pdf

IARC. 2012b. Metals, arsenic, dusts and fibres. *IARC Monogr Eval Carcinog Risks Hum* 100C: 219–309.

IPCS. 1989. *Reduction of Asbestos in the Environment*. Working Group Report ICS/89.34. Geneva: International Programme on Chemical Safety.

Iwatsubo Y, et al. 1998. Pleural mesothelioma: dose-response relation at low levels of asbestos exposure in a French population-based case-control study. *Am J Epidemiol* 148(2).

Jaurand MC. 1997. Mechanisms of fibre-induced genotoxicity. *Environ Health Perspect* 05(Suppl 5): 1073–84.

Johnson AM, Jones AD, Vincent JH. 1982. The influence of external aerodynamic factors on the measurement of the airborne concentration of asbestos fibres by the membrane filter method. *Annals of Occupational Hygiene* 25(3): 309–16.

Kamp DW, Panduri V, Weitzman SA, et al. 2002. Asbestos-induced alveolar epithelial cell apoptosis: role of mitochondrial dysfunction caused by iron-derived free radicals. *Molecular and Cellular Biochemistry* 234/235: 153–60.

Kane AB. 1996. Mechanisms of mineral fiber carcinogenesis. *IARC Scientific Publications* 140: 11–34. World Health Organization, International Agency for Research on Cancer.

LaDou J. 2004. The asbestos cancer epidemic. *Environmental Health Perspectives* 112(31): 285–90.

Larson T, Antao V, Bove F. 2010a. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. *JOEM* 52(5): 555–60.

Lee RJ, Van Orden DR. 2008. Airborne asbestos in buildings. *Regulatory Toxicology and Pharmacology* 50(2): 218–25.

Lippmann M. 1984. Peer review: inhalation and elimination of MMMF aids to the understanding of the effects of MMMF. In: *Biological Effects of Man-made Mineral Fibres*. Copenhagen, WHO Regional Office for Europe 2: 355–66.

Lippmann M. 1990. Effects of fiber characteristics on lung deposition, retention, and disease. *Environ Health Perspect* 88: 311–7.

Magnani C, Agudo A, Jonzalez CA, et al. 2000. Multicentric study on malignant plueral mesothelioma and non-occupational exposure to asbestos. *British Journal of Cancer* 83(1): 104–11.

Maule MM, Magnani C, Dalmasso P, et al. 2007. Modeling mesothelioma risk associated with environmental asbestos exposure. *Environmental Health Perspectives* 115(7): 1066–71.

McClellan RO, Miller FJ, Hesterberg TW, et al. 1992. Approaches to evaluating the toxicity and carcinogenicity of man-made fibers: summary of a workshop held November 11–13, 1991, Durham, North Carolina. *Regul Toxicol Pharmacol* 16: 321–64.

McDonald JC, McDonald AD. 1997. Chrysotile, tremolite, and carcinogenicity. *Ann Occup Hyg* 41: 699–705.

Ministry of Health. 1998. *A Guide to Health Impact Assessment: Guidelines for public health services*. Wellington: Ministry of Health.

Ministry of Health. 2008. *Drinking-water Standards for New Zealand*. Wellington: Ministry of Health.

Ministry of Health. 2017a. *All About Asbestos*. Wellington: Ministry of Health.

Ministry of Health. 2017b. *Removing Asbestos from the Home*. Wellington: Ministry of Health.

Mossman BT, Bignon M, Corn M, et al. 1990. Asbestos: Scientific developments and implications for public policy. *Science* 247(4940): 294–301.

Muscat JE, Wynder EL. 1991. Cigarette smoking, asbestos exposure, and malignant mesothelioma. *Cancer Research* 51: 2263–7.

National Academy of Sciences. 2006. *Asbestos: Selected Cancers*. Washington DC: The National Academies Press, p 394.

NIOSH. 2011a. Asbestos fibers and other elongate mineral particles: state of the science and roadmap for research. *Current Intelligence Bulletin* 62: 2011–159. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute of Occupational Safety and Health.

NTP (National Toxicology Program). 2014. *Report on Carcinogens* (13th edition). Research Triangle Park, NC: US Department of Health and Human Services, Public Health Service. http://ntp.niehs.nih.gov/pubhealth/roc/roc13/

Olsen N, Franklin PJ, Reid A. et al. 2011. Increasing incidence of malignant mesothelioma after exposure to asbestos during home maintenance and renovation. *MJA* 195 (5): 271–4.

OSH. 1991. *Report of the Asbestos Advisory Committee to the Minister of Labour*. Wellington: Occupational Safety and Health Service, Department of Labour.

Ozo C, Solt K. 2010. Biodurability of chrysotile and tremolite asbestos in simulated lung and gastric fluids. *Am Mineral* 95: 825–31.

Pan X-L, Day HW, Wang W, et al. 2005. Residential proximity to naturally occurring asbestos and mesothelioma risk in California. *American Journal of Respiratory and Critical Care Medicine* 172: 1019–25.

PHC. 1994. *Hazardous Substances: The Public Health Commission's advice to the Minister of Health 1993–1994*. Wellington: Public Health Commission.

Rao ST, Ku J, Rao KS. 1991. Analysis of toxic air contaminant data containing concentrations below the limit of detection. *Journal of the Air and Waste Management Association* 41(4): 442–8.

Reckelhoff-Dangel C, Petersen D. 2007. *Risk Communication in Action: The risk communication workbook*. Cincinnati: Office of Research and Development, US Environmental Protection Agency. URL: http://nepis.epa.gov/Adobe/PDF/60000I2U.pdf.

Schreir H. 1989. *Asbestos in the Natural Environment*. New York: Elsevier.

Shukla A, et al. 2003. Multiple roles of oxidants in the pathogenesis of asbestos-induced diseases. *Free Radic Biol Med* 34(9): 1117–29.

Smartt P. 2004. Mortality, morbidity, and asbestosis in New Zealand: the hidden legacy of asbestos exposure. *New Zealand Medical Journal* 117(1205): 1–15.

Suzuki Y, Yuen SR, Ashley R. 2005. Short, thin asbestos fibres contribute to the development of human malignant mesothelioma: pathological evidence. *Int J Hyg Environ Health* 208: 201–10. doi:10.1016/j.ijheh.2005.01.015 PMID:15971859.

US Department of Health and Human Services. 2002. Communicating in a Crisis: Risk communication Guideline for Public Officials. Washington DC: Department of Health and Human Services.

US EPA. 2007. *The Asbestos Informer*. URL: www.epa.gov/region4/air/asbestos/inform.htm (accessed 21 August 2013).

Webber S, Syrotynski S, King MV. 1988. Asbestos-contaminated drinking water. Its impact on household air. *Environ Res* 46(2): 153–67.

Weill H, Hughes JM, Chung AM. 2004. Changing trends in US mesothelioma incidence. *Occup Environ Med* 61(5): 438–41.

Western Australian Advisory Committee on Hazardous Substances. 1990. *Report on Asbestos Cement Products*. Perth: Western Australian Advisory Committee on Hazardous Substances.

WHO. 1986. *Asbestos and Other Natural Mineral Fibres*. Environmental Health Criteria 53. Geneva: World Health Organization.

WHO. 1988. Environmental health criteria 77: man-made mineral fibres. Vol. 77. Geneva: WHO.

WHO. 1994. *Asbestos in Drinking Water: No hazard*. WHO Press Release WHO/17 35. Geneva: World Health Organization.

WHO. 1998. *Chrysotile Asbestos*. Environmental Health Criteria 203. Geneva: World Health Organization.

WHO. 2000a. *Air Quality Guidelines for Europe* (2nd edition). Copenhagen: World Health Organization.

WHO. 2000b. *Asbestos and Health* (2nd edition). Copenhagen: World Health Organization.

WHO. 2003. Asbestos in drinking-water. Geneva: World Health Organization.

WHO. 2010. *Asbestos: Elimination of asbestos-related diseases*. Factsheet 343. World Health Organization.

WHO. 2014. *Chrysotile Asbestos*. Geneva: World Health Organization.

WHO. 2017. *Guidelines for Drinking-Water Quality* (Fourth edition incorporating the first addendum). World Health Organization. Youakim S. 2005. Understanding malignant mesothelioma. *BC Medical Journal* 47(2): 82–3.

Appendix 1: Asbestos sampling and analysis

Main points

Air sampling

- Decide on the objective of sampling; generally the sample will be of ambient conditions in areas occupied by people at risk.
- Discuss the sampling objective and methodology with the laboratory.
- Schedule sampling to represent the cycle of activity considering the circumstances that created the need for sampling.
- Keep full records of the sample and of the methods used in its examination.
- Agree measurement criteria with the laboratory, including size and types of fibres to be measured, and sensitivity required.
- Agree sampling protocol with the laboratory, including sampling rate and duration.
- If you need to compare the results with other or earlier samples, make sure you are comparing samples using similar protocols.

Sampling asbestos-containing material

- If in doubt that a material contains asbestos, have it examined by the laboratory.
- Follow the advice on how to take a sample.
- Be clear what you need to know from the laboratory examination.
- Do not expose yourself to asbestos fibres.

Safety when handling asbestos-containing material

These guidelines do not cover the safety of the PHU staff when handling asbestos-containing materials as this matter should be covered by their health and safety practices and they need to consult WorkSafe.

Introduction

The aims of sampling and analysis of asbestos-containing materials are to:

- identify asbestos and its types in an asbestos-containing material or
- in air to measure the concentration of asbestos fibres in air.

Identifying asbestos fibres – involving either suspected asbestos in a material, or the presence of asbestos in a dust – requires sophisticated technology. The measurement of exposure to particles that are invisible to the naked eye and a hazard to health requires microscopic examination that identifies the size and types of asbestos fibres. The sampling strategies and methods of examination need to be selected for each particular circumstance, requiring close cooperation between the examining laboratory and the health protection team.

Air sampling

Air sampling strategies

Aspects of an air sampling strategy to consider are:

1. objectives of air sampling
2. sampler configuration and design
3. personal versus area sampling
4. scheduling of sample collection
5. statistical design
6. record keeping and quality assurance
7. air sampling and measurement of asbestos.

1. Objectives

Once the presence of asbestos fibres in air has been established, several different objectives can be addressed in evaluating exposures to airborne asbestos, including the following.

1. Measure personal exposures of individuals at particular risk, or of those who serve as sentinels for groups having similar exposures.
2. Measure ambient concentrations in areas occupied by people at potential risk. These concentrations should be measured depending on the conditions of occupancy and activity that created the need for sampling. More elaborately, time-weighted average exposures could be calculated by combining time-activity patterns of individuals or groups and the asbestos concentrations in the areas in which they spend their time.
3. Make source-related measurements. These measurements can indicate the potential for human exposure and may range from the measurement of actual fibre release from asbestos-containing material (whether disturbed or not) to estimates of the potential fibre release under specific simulated circumstances.

2. Sampler configuration and design

The aim of configuration and design is to obtain a uniform deposition of a representative sample of airborne asbestos onto the filter surface. The laboratory should be informed of the exact situation and objectives and be asked to advise on appropriate steps.

3. Personal versus area sampling

Building employees who disturb asbestos-containing material in the course of their work will be exposed to highly variable air concentrations of fibres. These are occupational situations (the responsibility of WorkSafe) requiring personal monitors drawing from the breathing zone. It is conceivable, however, that a home owner who insists on removing, or working on, asbestos-containing material in the home may require similar personal monitoring.

For building occupants not in contact with asbestos-containing material, samples collected from representative fixed locations should provide adequate estimates of personal exposure. Compared with personal sampling, area sampling is more practical and efficient and higher sampling air flow rates are possible.

The addition and removal of asbestos in air may be viewed in terms of sources and sinks. The primary source will be the asbestos-containing material that is releasing fibres; the secondary sources will be the re-suspension of fibres that have settled within the space. Sinks will be the removal and settling of fibres. The concentration of fibres measured will be the equilibrium concentration over the time of sampling. Activity and ventilation will therefore influence the concentration measured and should remain as close to the norm as possible.

4. Scheduling of sample collection

For general building occupants, air concentrations of asbestos fibres should be measured over relatively long time periods corresponding to occupancy cycles – that is, at least one full day or long enough to capture typical building activity patterns.

In buildings with air conditioning or ventilation systems, the pattern of exposure may vary with the seasons or even with individual days. Besides the indoor sampling, outdoor air samples should be collected near ventilation inlets to determine what the outdoor air may contribute to indoor fibre concentrations.

In the home, the variation of activity between weekdays and weekends should be reflected, as should changing activity patterns (particularly of children) due to the weather.

5. Statistical design

A statistical design should be discussed and agreed with the laboratory, taking into account:

1. the purpose of the study
2. the definition of the population under study
3. a statistical sampling strategy to obtain a representative sample of that population
4. the need for multiple (spatial) or repeated (temporal) sampling
5. sample size, for example to estimate the mean exposure to a specified degree of confidence
6. the expected temporal and spatial variability in measurements.

Because of the analytical limitations for a single sample analysis when evaluating the concentration of fibres, many of the samples are generally below the analytical sensitivity. An appropriate statistical strategy may need to be considered by the examining laboratory when interpreting such data (Rao et al 1991).

6. Record keeping and quality assurance

Proper interpretation of air sampling data depends on full consideration of all data relevant to the sample. In addition to the objectives above, information will be needed to verify whether concentrations exceed some acceptable value; show a trend; correlate with building activities, maintenance or asbestos removal; or correlate with use of ventilation or air conditioning systems.

Accordingly, all sampling data should be related to factors that may influence the results or be of value in interpretation. Sample record sheets are provided in Appendix 3.

7. Air sampling and measurement of asbestos

Purpose of measurement

Measuring airborne asbestos evaluates the potential or extent of human exposure to airborne fibres. The measurement strategy needs to recognise the following (Johnson et al 1982).

1. Fibres within certain size ranges, if respired into the lung, can cause lung fibrosis, lung cancer and mesothelioma.
2. Health effects depend on where fibres are deposited (or migrate) and their physical–chemical properties. Important variables are length, width, composition, surface chemistry, and durability. (At high exposures, which are exceedingly unlikely in the context of these guidelines, consider the rate at which inhaled particles of all types accumulate, and whether this alters normal particle clearance rates. In such circumstances, additional sampling to detect peaks is required.)
3. Other particles and fibres coexist in air, often in much greater concentration than asbestos, so the appropriate method should be used.
4. Methods to identify and count asbestos fibres need to reflect the very different conditions presented by environmental concentrations, where concentrations are generally very low, compared with occupational situations.
5. The sensitivity of measurement methods needs to satisfy either: (a) typical ambient air concentrations; or (b) levels commensurate with lifetime risks of the order acceptable to the public.

Sampling of suspected asbestos-containing materials

Objectives of sampling

The objective of sampling a suspected asbestos-containing material is to verify, or otherwise, the presence of asbestos and provide other information that will help in a risk assessment. Identification of asbestos can only be achieved by scientific examination. Any suspect material should always be sent for examination as asbestos has been found to occur when not expected. Fibre type and the condition of the asbestos-containing material are also important to determine as they may influence the risk assessment.

How to sample asbestos-containing material

- Discuss the purpose and approach to sampling with the laboratory.
- Note that asbestos in some materials may not be uniformly distributed and composite samples may be needed. Laboratory advice should always be obtained **prior** to sampling.
- Ensure your safety, from both asbestos fibres and accident, while taking a sample. Friable material, easily damaged by sampling, may release significant numbers of fibres. Wear at least a half-face respirator.
- Wet the material to be sampled with water.
- Take a representative quantity of about 10 grams, say the size of a 10-cent coin or a teaspoonful, disturbing the sample as little as possible. (A core sample may be required for sprayed or trowelled insulation. Preferably use a single-use sampler that also acts as a container – such as an acrylic tube, about 12 mm wide and 100 mm long, bevelled to a cutting edge at one end fitted with caps.)

- Label with a unique number and place in a clean plastic bag; seal and protect from physical damage by packing.
- Clean debris with wet cloth and discard in a plastic bag; seal material with paint or core hole with a sealant.
- Complete relevant information in the sample record using a unique sample number.
- Send to the laboratory.

Information to be sought

The laboratory should provide information on the:

- presence of asbestos
- types of asbestos
- methods used in examination.

The laboratory may also be able to offer an opinion as to the approximate portions of asbestos in the sample if requested. If asbestos is 1 percent or greater by mass, consider the material to be asbestos-containing material. This determination is likely to be necessary only if there is some doubt about the type of asbestos-containing material.

Appendix 2: Public health aspects arising from a fire involving asbestos containing materials: fact sheet for public health units

The development of this fact sheet was prompted by the Taranaki Patea Freezing Works fire in 2008. Although the local public health unit managed the situation very well it became evident that very limited information is available with respect to the potential public health consequences when dealing with large scale fires involving asbestos containing materials (ACM).

Thermal stability of asbestos

Asbestos was widely used because of its fire resistance properties, however it is not thermally stable when exposed to high temperatures. Chrysotile decomposes at 800–850°C and the amphiboles at 800–1000°C. Asbestos fibres will readily be converted to dust at prolonged exposure to such temperatures.

In sheet form asbestos does not offer any fire resistance and it cracks in building fires. In a fire, asbestos cement sheeting will disintegrate and can explode, releasing fibres over a wide area, mostly in the direction of prevailing wind.

Effect of fire on asbestos fibre contamination

Fire can change the mineral structure and mechanical strength of asbestos, fixing the fibres and transforming it to a less hazardous state. The process will generally affect only the outer layers leaving most fibres intact within the material. Internal fibres in a fibre bundle will be unaffected due to the insulating quality of the mineral.

A study commissioned by the Victorian Department of Human Services (2006) examined the concentrations of respirable fibres away from the incident site, ie, fire location using a computational fluid dynamics programme designed to simulate fires of varying sizes. Fires within buildings comprising substantial quantities of ACM did not result in hazardous conditions with respect to respirable asbestos fibres either close to the building or away from the building. This was true of fires involving asbestos cement sheet only.

Friable asbestos within a fire does give off respirable sized fibres, such as the Broadcasting House fire.[13]

[13] Broadcasting House, a multi-purpose broadcasting centre on Bowen Street, Wellington, New Zealand, was caught by fire in 1997.

Sampling of the ash residue after a building test fire by the Centre for Environmental Safety and Risk Engineering in Australia did not find respirable asbestos fibres in the ash, however asbestos fibre bundles were present. These fibre bundles, while in their bundled form, are not respirable, however they could become respirable through the clean-up process if the bundles are exposed to further mechanical degradation. Therefore after a fire, the asbestos fibre bundles in the ash debris should be treated in the same manner as ACM during the clean-up process. Respirable fibre concentrations emitted by the fire were very low and appear to be lower than average background levels. Plume modelling was used in determining the dispersion of respirable asbestos fibres away from a fire location which demonstrated that in this particular test, respirable fibre concentrations close to the fire were extremely small. Concentrations reduce further away from the fire being, theoretically, orders of magnitude lower.

Exposure of the general population

People resident in the area, may be exposed following a fire involving materials containing asbestos. Sources of exposure include:

- direct inhalation of asbestos in the original plume
- inhalation of asbestos fibres resuspended into the air (eg, wind driven or a result of mechanical processes) following deposition on the ground or other surfaces
- ingestion of local produce.

The degree of exposure of the general public will depend upon the concentration of asbestos in the air (directly from the plume during the fire or as a result of re-suspension following fire) and subsequent actions of the public and authorities. For example, rapid removal of significant fallout will reduce the potential for significant re-suspension exposures of the general public although it may result in exposures to staff involved in clean-up.

Mitigating factors against significant exposures of the general public following a fire involving ACM

- Not all the ACM present may be involved in the fire.
- Fibres may be entrapped in large pieces of material, etc.
- During a fire, most asbestos cement sheeting will be deposited as large pieces.
- Respirable fibres will be a fraction of the total released.
- Small proportion of fibres may be 'denatured' at prolonged exposure to high temperatures in large scale fires.
- Atmospheric dispersion and deposition (particularly as a result of rain) will reduce concentrations.[14]
- Duration of exposure will be short dependent on the type of ACM present.

[14] A survey was conducted by the Capital Environmental Services Ltd in Wellington on cement sheet roofing and fibre run-off with rainwater. This was conducted over a 12-month period using encapsulated asbestos roofs, a blank and un-encapsulated roof. Using Scanning Electron Microscope, the fibre run-off with rain water was found to be in the region of 13 million fibres per litre of water with little observed difference to the results between roof type. One blank of 12 indicated more fibre when compared with the samples after a particular month of exposure. The increase was found to be due to an asbestos cement roof further down the road being removed and replaced that particular month.

Acute adverse health effects

Thermal injury or smoke inhalation is generally the most likely potential acute effect from large scale fires. Asbestos may produce irritation of the skin, eyes and respiratory tract due to mechanical action of the fibres. However this only occurs at very high air concentration levels well beyond those that members of the public would likely encounter from a fire.

Respiratory symptoms were reported by people who have been exposed in asbestos fire. However there is no hard evidence to suggest a severe acute health impact consequent on a fire incident associated with asbestos-containing fallout. Despite the lack of hard evidence of an acute health impact, it is important that health professionals are aware of the potential for patients to associate symptoms with such incidents.

Long-term adverse health effects

There is no direct evidence of long-term health risks from fires involving ACM, although the literature in this area is limited. Considering the available evidence on asbestos exposures from fires involving ACM in the context of the results of epidemiological studies of occupational and environmental asbestos exposures, it is concluded that the risk of long-term health effects (mesothelioma and lung cancer) is low if appropriate clean-up procedures occur.

Evacuation

The usual first course of action is to 'shelter in place' unless directly threatened by fire in which case fire officers will direct evacuation. If evacuation has taken place for health reasons, the Medical Officer of Health and/or Health Protection Officer will determine when to advise residents that it is safe to return home.

Laboratory analysis

The presence of asbestos in materials cannot be determined definitively by visual inspection. Actual determination can only be made by instrumental analysis, eg, polarised light microscopy, transmission electron microscopy or scanning electron microscopy. It is best to assume that the material contains asbestos until laboratory analysis proves otherwise.

Collect and send samples to Capital Environmental Services Ltd (2–4 Bell Road South, Gracefield, Lower Hutt, phone 04-566 3311, fax 04-566 3312) for asbestos analyses to confirm the presence and type of asbestos. Laboratory staff are able to provide advice on how many samples should be collected for testing and how these should be collected. If necessary, a scientist from the laboratory may be sent to the affected area to provide assistance.

In general, air sampling carried out following asbestos fires will not reveal significant levels of asbestos fibres. Therefore in many cases it will not be necessary to carry out such monitoring. Monitoring may however be appropriate after large incidents for public reassurance purposes. This is a decision that should be made on a case by case basis.

At the earliest opportunity after results are known, they should be made public so that members of the public are fully aware of the situation and can make an informed decision.

Effect of watering

Dependent on water pressure, it is important to note that the addition of water will not result in the further degradation of any asbestos fibre bundle. In particular it has been shown that the application of water is very effective in reducing the likelihood of asbestos fibres from becoming respirable in soils and sands. Land contamination issues are possible as a result of water washing asbestos fibre bundles or pooling water in an area (as a result of a fire in the area). In case of asbestos cement products, it is unlikely that the asbestos bundles would be sufficient in terms of fibre size and form to generate respirable dust cloud particles, when the water has evaporated. It could be an issue for lagging and friable material as there can be incidences of rainwater puddles from asbestos cement roof leaks that contain significant amounts of friable asbestos (L Dwyer, personal communication, 29 July 2018).

Clean-up operations within the building should be performed in accordance with WorkSafe requirements. The application of water will further reduce any exposure risk to nearby personnel working in the area, since wetting down the debris after a fire reduces the risk of respirable asbestos becoming airborne. However it should be borne in mind that amosite repels water. So if large amounts of friable amosite are present watering will have little effect (L Dwyer, personal communication, 29 July 2008).

Handling asbestos materials is a specialist task requiring appropriate training and equipment, including personal protective equipment (PPE) as there is the potential for the workers involved to be exposed during the process.

Conclusions

The mere presence of asbestos in buildings or in ash/rubble does not necessarily pose a health risk to building occupants or the public. Asbestos fibres of respirable size must become airborne in sufficient concentration to pose a risk from inhalation.

Exposure of members of the public during and in the aftermath of a fire involving ACM is expected to be minimal if appropriate clean-up operations are undertaken. Thus the potential for long-term environmental exposure and the associated risk is likely to be low.

Some members of the public perceive a greater risk from large scale fires involving asbestos than is actually the case, and this should be taken into consideration when devising and issuing public warnings.

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Bibliography

Bridgman SA. 1999. Lessons learnt from a fire associated with asbestos-containing fallout. *Journal of Public Health Medicine* 21(2): 158–65.

Bridgman SA. 2000. Acute health effects of a fire associated with asbestos-containing fallout. *Journal of Public Health Medicine* 22(3): 400–5.

Bridgman SA. 2001. Community health risk assessment after a fire with asbestos containing fallout. *J Epidemiol Community Health* 55: 921–7.

Smith KR, Saunders PJ. 2007. *The Public Health Significance of Asbestos Exposures from Large Scale Fires*. Chilton: Health Protection Agency, UK. 77 pp.

Victorian State Government, Department of Human Services. 2006. *Australia Report on the Investigation of the Effect of Fire on Asbestos Fibre Contamination*. Noel Arnold & Associates Pty Ltd (URL: www.health.vic.gov.au/environment/community/asbestos.htm).

Appendix 3: Report sheets

Copying and adapting the report sheets for your own use

Users may find it useful to copy parts of the text from the *Graded Response Protocol* and other material into the *Report Sheets*.

Step 1: Initial response and preliminary assessment

Reference number for this investigation:

Your name:

1.1 Gather and record information

Informant details

Date:

Contact person:

Address:

Phone:

Location of potential asbestos hazard, type of building and building use

The location (street address) of the suspected asbestos-containing material:

Person responsible at the site that is the subject of concern:

Address of this person:

Nature of concern:

Type of building – for example, dwelling:

Building use:

Other type of location – for example, a landfill, building or demolition site:

Spatial relationship to the site that is the subject of the concern – how far away, upwind, overlooking, etc. Use description from concerned person at Step 1.

Provide sketch and add information at Step 2. Consider photograph or video recording.

Nature, condition, quantity and accessibility of potential asbestos hazard

Description of the suspected material:

Information on the date that the asbestos-containing material was installed – may be known by the concerned person for their own home:

Is it likely to be an asbestos-containing material from the description in the last two items?

Yes **No** **Maybe**

Is it likely to be a friable asbestos-containing material?

Yes **No** **Maybe**

If 'Yes' or 'Maybe' proceed to Step 2.

Is asbestos-containing material inside the home or building?

Yes **No**

If 'Yes' proceed to Step 2 unless very minor and a simple recommendation can be made; if 'No' consider in conjunction with later questions.

Note location and other information.

Is the suspect material easily accessible to children? Note that accessibility is a measure of future damage, not exposure.

Yes **No** **Maybe**

If 'Yes' proceed to Step 2 unless very minor and a simple recommendation can be made.

What is the potential for future damage, disturbance or erosion of the suspect asbestos-containing material?

High **Low**

See table below which combines this factor with condition.

Quantity of the material: does it exceed about 15 square metres of surfacing material?

Yes **No**

If 'Yes' proceed to Step 2 unless very minor and a simple recommendation can be made.

Condition: is the suspect asbestos-containing material in good condition, a state of minor damage or deterioration, or poor condition?

Good **Minor damage/deterioration** **Poor**

See table below for recommendations on proceeding to Step 2.

Is the potential hazard airborne? (eg, visible dust or other assessment of the concerned person. May be from asbestos-containing material in the home or building, or from adjacent activity.)

Yes **No**

If 'Yes' evidence that it is asbestos needs to be confirmed. Proceed to Step 2.

Actions that may translate the potential hazard into a risk to health

Who is taking, proposing to take or has taken the action? This may be the home owner or a neighbour (residential or commercial), or it may be work being done or proposed in a public building or school.

Name (add description, eg, owner, neighbour):

Address:

What is the action or disturbance? Is it affecting the suspected asbestos-containing material? Add comment on scale, violence and duration.

Does it appear likely that asbestos will be (or has been) released by the actions?

Yes **No**

If 'Yes' proceed to Step 2 unless release can be managed by simple advice and person has good understanding of the hazard and precautions.

When is the action or disturbance of asbestos-containing material proposed, or when did it happen?

Date:

Time if relevant:

Is a change in building use proposed? Changes in use often require interior modification and disturbance of services and surfaces that may use asbestos-containing material. This would normally apply only to public or commercial buildings.

Yes **No**

If 'Yes' describe the change. Consider the need to proceed to Step 2 so that preventive action can be taken and make a note.

Is renovation or remodelling proposed? Alterations to the home of the concerned person, or of a neighbour, may require the disturbance/removal of asbestos-containing material.

Yes **No**

If 'Yes' describe the change. Consider the need to proceed to Step 2 so that preventive action can be taken and make a note.

Are there actions to do with an adjacent industry or business activity? There could be many other actions to asbestos-containing material, either in buildings or to do with processes or work. Note any other actions.

Yes **No**

People at risk

Who may be at greatest risk?

Name(s):

Relationship(s) to concerned person or other description:

How could the people at risk be exposed? For example, where are children's bedrooms or play areas relative to the site of disturbance of probable asbestos-containing material?

Over what period may the people have been, or will the people be at risk?

Who is clearly not at significant risk of exposure?

What is the state of health of the people or population at risk of exposure?

1.2 Identify and assess the potential hazard

Report Table 1.2: Actions or potential for damage, disturbance or erosion

Current condition of asbestos-containing material	Low	High
Good	Unnecessary to proceed to Step 2 unless other factors are significant	Proceed to Step 2
Minor damage or deterioration	Proceed to Step 2 unless simple advice can be safely given	Proceed to Step 2
Poor	Proceed to Step 2	Proceed to Step 2

1.3 Decide whether to proceed to Step 2: Inspection and hazard evaluation

Enter your decision and date here. Note the other factors referred to in the *Graded Response Protocol* for Step 1.3 before deciding.

Decision:

Date:

1.4 Identify and inform the agency most appropriate to take any further action

Does the asbestos hazard identified require the attention of WorkSafe, the territorial authority or (less likely) the regional council?

Yes **No** **Maybe**

Names of agencies:

Date approached:

Outcome:

Should the health protection team continue to be involved with the regulatory agency?

Yes **No** **Maybe**

Roles agreed for each:

What further inspections, assessments and action plans (which may follow a similar pattern to this Graded Response Protocol) would you recommend to any other authority?

1.5 If not proceeding to Step 2, provide support and precautionary advice

Advice based on a preliminary assessment of the hazard should be simple: take nothing for granted, and be precautionary.[15] Discourage do-it-yourself asbestos removal, and recommend that specialist firms are called in (WorkSafe has names and details).

Enter the advice given.

Date:

Advice given to:

Advice should be based on simple precautions, for example:

- Do not attempt to do anything to, or handle, friable asbestos.
- Avoid do-it-yourself asbestos removal. Call in specialist firms (WorkSafe has names and details).

Provide copy of Ministry of Health asbestos resource *All About Asbestos* (HP 6711) and, if appropriate, *Removing Asbestos from the Home* (HP 6711).

Other advice given (include information on other agencies to be involved):

Who else needs to be informed/involved (eg, landlord, property owners, other)?

Follow up on Step 1 if required

Date:

Result:

Follow-up, and/or action/enforcement by other agencies (including dates and action):

In all cases the capacity of the concerned person to understand the advice and take sensible action should be taken into account.

Step 2: Inspection and hazard evaluation

2.1 Co-ordinate action/enforcement with the regulatory agency as appropriate

Record dates and nature of contact and consultation with WorkSafe, territorial authority or other (eg, regional council).

[15] Air tests taken, in windy conditions, for a few weeks after the Broadcasting House fire in 1997 showed large amounts of airborne asbestos fibre. The fibre had been distributed over a very large area by the smoke plume. This included inside buildings with open windows and on far sides of high rise in the vicinity of the fire. Some of this fibre had been heat altered, but this was only a very small portion of what was collected by dust wipes.

2.2 Confirm initial information

Visit the site and confirm or amend all the initial information by working through all the points in Step 1.1 above. Complete a new *Report Sheet* only if absolutely necessary. (Keep the first copy as a record of the initial step.)

2.3 Obtain and record additional information to enable an adequate hazard evaluation

Identify any further **suspect asbestos-containing material** (see Step 2.3 in the Graded Response Protocol for further information).

Examine the **condition** of the suspect asbestos-containing material for deterioration (seeking comment from the laboratory if the sample includes deteriorated material).

Note main findings in words here.

Sample for composition of the suspected asbestos-containing material. Test for friable asbestos-containing material (if not evident from a visual inspection) on site by rubbing and observe production of dust and particles; wear at least a half-face respirator. Complete sample record sheet (over) which also provides a space for results. Note: discuss with laboratory prior to sampling (refer Appendix 1).

2.3.1 Asbestos sample record

Sample unique number:

Reference number of investigation:

Reference of sample:

Relationship to other samples and their unique numbers:

Examining laboratory and contact name:

Where collected: address

When collected: date

Type of premises and use:

Owner:

Occupier:

Purpose of sample:

Type of sample: air, or asbestos-containing material (describe)

Location: description and sketch in plan and elevation of sampling position – use following sheet.

Site plan: show other potential sources of asbestos fibres (eg, adjoining structures, roads where vehicles brake) – use following sheet. Mark north point.

Sampler configuration and detail (eg, size selective inlet, membrane type, pore size (μm), area (cm^2))

Sampling duration: hours (from – to, using the 24-hour clock)

Air flow rate in l/minute:

Relevant activity at time of sample collection:

Describe ventilation in area sampled: natural (what) or mechanical or air conditioning.

Describe weather during sampling (especially for outdoor sample):

- wind direction
- approximate speed
- temperature
- precipitation
- comment (eg, fine, gusty, still).

2.3.2 Blank sheet for sketch of sample location and site plan

2.3.3 Results

Fibres per litre or presence of fibres in asbestos-containing material:

Type of fibres and proportions:

Method used in examination:

Sensitivity or detection limits:

Other results, comments or queries:

(Attach copy of laboratory report)

2.3.4 Sketch of building and location of asbestos-containing material

Make a sketch on the following sheet of locations, and note materials found and their condition. In the case of large buildings, try to obtain a copy of layout drawing from the territorial authority.

Note accessibility of the suspected asbestos-containing material on sketch on next page; assess as 'easy' or 'difficult' with children in mind. Note that accessibility is a measure of future damage, not exposure.

Assess the quantity of asbestos-containing material (see Step 2.2). Note areas of surfacing asbestos-containing material on sketch on next page and lengths of pipe or duct insulation in public areas.

2.3.5 Blank sheet for sketch of building and location of asbestos-containing material

2.3.6 Describe any disturbance of asbestos-containing material

Describe the **actions** proposed (or that have taken place) that could disturb asbestos-containing material and lead to the release of fibres. Write in words below and enter on sketch if helpful.

2.4 Identify and characterise hazards

From the complete information obtained, including the results of sample analysis, identify each opportunity for the release of asbestos fibres (including the likely potential release from further damage). It may be useful to mark the sketch with the main potential sources in colour. Describe below:

Report Table 2.4: Characterisation

Where	Friable (F) or Not Friable (NF)	Asbestos type: C, A or M*	Releasability: Significant (S) or Not Significant (NS)	Hazard: High (H) or Low (L)

* C = chrysotile; A = amphibole; M = mixed

Rank hazards as 'high' or 'low', assuming that there is an open pathway for exposure to vulnerable persons.

2.5 Decide whether to proceed to Step 3: Exposure measurement, risk estimation and assessment

Enter decision: proceed to Step 3

Yes **No**

Date:

Reasons for decision:

2.6 Provide advice to manage hazards and potential exposure, and ensure action is taken

Report Table 2.6: Potential for future damage, disturbance or erosion and suggested action

Current condition of asbestos-containing material	Low	High
Good	Take no further action now beyond operations and maintenance.	Undertake operations and maintenance measures to prevent damage. May require remediation to prevent further damage or deterioration.
Minor damage or deterioration	Operations and maintenance and local remediation are required. Prevent further damage.	Remediation is required as soon as possible to prevent further damage/deterioration. Operations and maintenance cleaning is required.
Poor	Remediation is required as soon as possible. Prevent access to minimise further damage. Operations and maintenance cleaning is required.	Remediation is required urgently. Evacuate people and isolate affected space from rest of building. Operations and maintenance cleaning is required.

Discourage do-it-yourself asbestos removal and recommend that specialist firms are called in (WorkSafe has names and details).

Enter the advice given.

Date:

Advice given to:

Provide copy of the most recent Ministry of Health asbestos resource *All About Asbestos* (HP 6710) and, if appropriate, *Removing Asbestos from the Home* (HP 6711).

Other advice given (include information on other agencies to be involved):

Who else needs to be informed/involved? (eg, landlord, property owners, other)

Enter action/enforcement co-ordinated with other agencies from Step 2.1.

Note if asbestos waste generated and, if so, how, when and where waste was disposed of.

Follow up on Step 2

Date:

Result:

Follow-up, and/or action/enforcement by other agencies (including dates and action):

Step 3: Exposure measurement and risk assessment

3.1 Coordinate action/enforcement with the regulatory agency as appropriate

Record dates and nature of contact and consultation with WorkSafe, territorial authority or other (eg, regional council):

3.2 Sample and analyse to determine presence and type of asbestos

Discussed with laboratory:

Date:

With whom:

Decision:

Yes **No**

If 'Yes' record method proposed.

Enter sample details and results on the sample record sheet(s) (see Step 2.3).

3.3 Measure the concentration of respirable asbestos fibres in air

Discussed with laboratory:

Date:

With whom:

Decision:

Yes **No**

If 'Yes' record ...

Strategy:

Personal (special circumstances) **Ambient (usually)** **Source (rarely)**

Why will the results be useful?

Enter sample details and results on the sample record sheet(s) (see Step 2.3).

3.4 Estimate the exposure under normal conditions

(See *Graded Response Protocol* Steps 3.3 and 3.4 for note on adjustments.)

Report Table 3.4: Exposure estimation

Name of exposed	Age	Date(s) of exposure	f/mL	Adjusted exposure f/mL if appropriate

Note: Tables 5.1 and 5.2 make their own allowance for durations of exposure.

3.5 Estimate the risk

Consider the estimation of excess risk in Step 3.5 of the Graded Response Protocol.

Low **Moderate** **High**

3.6 Communicate the risk

Consider the factors in risk communication in Chapter 1 (and in the other references) and summarise below the key points you will make:

To whom:

Date:

What further response is required?

3.7 Recommend actions to manage risk and ensure action is taken

Reconsider the advice entered at Step 2.6 in the Report Sheets, taking into account the risk estimates now available.

Recommend actions to manage risk:

Date:

To whom:

Provide a copy of the most recent Ministry of Health asbestos resource *All About Asbestos* (HP 6710) and, if appropriate, *Removing Asbestos from the Home* (HP 6711).

Other advice given (include information on other agencies to be involved):

Who else needs to be informed/involved (eg, landlord, property owners, other)?

Note if asbestos waste generated and, if so, how, when and where waste was disposed of.

Follow up on Step 3

Visit to confirm advice:

Date:

Comments:

Enter action/enforcement coordinated with other agencies from Step 3.1.

Glossary

ATSDR	Agency for Toxic Substance and Disease Registry (USA)
µm	Micron (ie, one millionth of a metre – ie, 10^{-6} metres)
abatement	The removal or significant reduction of a source of hazard, and intervention to reduce exposure to a hazard
CPSC	Consumer Product Safety Commission (USA)
DHB	District health board
domestic	In or of the home environment
epidemiology	The study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to the control of health problems
exposure	A measure of a factor to which a population is exposed
f/L	Fibres per litre
f/mL	Fibres per millilitre
fibre.year/mL	The product of fibres per millilitre multiplied by years of exposure
friable	Defined in the Health and Safety at Work (Asbestos) Regulations 2016 as " in relation to asbestos or ACM, means in a powder form or able to be crumbled, pulverised, or reduced to a powder by hand pressure when dry"
hazard	A source or situation of potential harm
HEI	Health Effects Institute (USA)
IARC	International Agency for Research on Cancer
incidence	The number of new cases or deaths that occur in a given period in a specified population
IPCS	International Programme on Chemical Safety
ISO	International Standards Organisation
L	litre, sometimes also written as *l*
LIM	Land Information Memorandum
mean	The sum of all the values in a set of data divided by the number of values
median	The central value in a set of data
MF/L	Million fibres per litre of water
NAS	National Academy of Sciences
neighbourhood	Vicinity

NIOSH	National Institute of Occupational Safety and Health
NZPHD	New Zealand Public Health and Disability Act 2000
OSH	Occupational Safety and Health Service of the Department of Labour then the Ministry of Business Innovation and Employment (Health and Safety Group) and now called WorkSafe New Zealand (WorkSafe)
para-occupational exposure	Indirect exposure to a hazardous substance brought from the workplace to another place
PHC	Public Health Commission
PHU	Public health unit
PIM	Project Information Memorandum
PLM	Polarised light microscopy
public building	Any building that the public may enter
remediation	All measures to remedy the potential harm from a hazard, including abatement and operation and maintenance
risk	The probability of harmful consequences arising from a hazard together with a measure of the scale or severity of the harmful consequence. In qualitative terms the risk may be said to have a probability that is 'high', 'moderate' or 'low' or another chosen term. In quantitative terms, the probability can range from zero (no possible harm) to unity (certainty that harm will occur). The scale and severity of the harm may be characterised by the number of people affected and the sort of harm (eg, death or serious injury).
risk assessment	The systematic acquisition and evaluation of information that enables the probability, scale and severity of the risk to be described
risk management	All actions of a management nature that are designed to minimise risk to levels acceptable to the person(s) exposed to the risk
RM Act	Resource Management Act 1991
SEM	Scanning electron microscopy
TEM	Transmission electron microscopy
TSI	Thermal system insulation (eg, lagging around boilers, pipes and ducts) to improve (hot or cold) thermal insulation
US EPA	United States Environmental Protection Agency
WES	Workplace Exposure Standards
WHO	World Health Organization

[redacted]

From: Nick Urlich
Sent: Friday, 15 March 2024 10:35 am
To: Ramesh Pillai
Subject: FW: Asbestos in Drinking Water
Attachments: management-asbestos-non-occupational-environment-5th-edn_dec17-v2.docx; Asbestos-exposure-in-New-Zealand-April-2015.pdf

Fyi,

Some generic information, Asbestos exposure in the non-occupational environment (royalsociety.org.nz)

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

Oral

Drinking-water

The general population can be exposed to asbestos in drinking-water. Asbestos can enter potable water supplies through the erosion of natural deposits or the leaching from waste asbestos in landfills, from the deterioration of asbestos-containing cement pipes used to carry drinking-water or from the filtering of water supplies through asbestos-containing filters (IARC 2012b).

The adverse effects following ingestion of asbestos have not been clearly documented. ATSDR (2001a) considers few fibres are able to penetrate the gastrointestinal tract. This means non-gastrointestinal effects from oral exposure to asbestos are unlikely. There is considerable controversy as to whether ingested asbestos fibres can penetrate and pass through the walls of the gastrointestinal tract in sufficient numbers to cause adverse effects. There is inconsistent evidence of carcinogenicity of ingested asbestos in epidemiological studies of populations with drinking-water supplies containing high concentrations of asbestos. Moreover, in extensive studies in experimental animal species, asbestos has not consistently increased the incidence of tumours of the gastrointestinal tract. There is therefore no consistent evidence that ingested asbestos is hazardous to health. The primary issue surrounding asbestos-cement pipes is for people working on the outside of the pipes (eg cutting pipes) because of the risk of inhalation of asbestos dust (WHO 2003, 2017).

The World Health Organization (WHO 2003, 2017) concluded that there was little evidence that ingested asbestos is hazardous to health and therefore did not feel it necessary to establish a health-based guideline value for drinking water.

Nicholas Urlich
Senior Asset Planning Engineer

Tel [redacted]

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