

A Survey of the Health problems associated with the Production and Use of High Density Chrysotile Products

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Introduction

Chrysotile asbestos has been used in high density cement and friction products for nearly one hundred years. Since this paper is not presented as a definitive history of asbestos in all its guises and since early developments in friction products involved textiles among a variety of other materials and a quite different technology to that used today it seems reasonable to restrict the contents to those products developed and used in the last 50 years. In that time, of all the world production of chrysotile some 90% has been used in asbestos-cement products such as pipes, plates, sheets, mouldings and shingles (tiles). Some 7% is used for friction products such as brake linings and clutch facings and only 3% is used in other materials including textiles, clothing, electrical insulation materials, gaskets, paper products, vinyl sheet and vinyl floor tiles - where chrysotile has been used as a filler - and as a filler in cement, plastics, roof coatings and various mastic and caulking compounds.

Health and safety records for workers in the chrysotile industry have been kept for perhaps only the last 60 years. They show few health problems among those involved in the manufacture and use of these products even in the early days when there was little attempt at dust suppression. This has been borne out by every health study of chrysotile cement or friction product workers that has ever been carried out. Concerns about the use of chrysotile in such products have been raised by those who have used health statistics from other forms of asbestos or from mixed fibre exposures or who have failed to understand the confounding role of smoking in the epidemiology. The concerns have been compounded through reliance on questionable mathematical models, rather than actual worker health data. It is relevant at this point to note that no such risk models have been used to calculate worker health risks from the use of substitute fibres in these products. In fact regulators have, at times, deliberately avoided the issues of toxicity for substitute fibres and not questioned the claims that they are safe. This 'turning a blind eye' has strengthened the call for a ban for all asbestos products in a number of countries. Such a ban has been successfully carried in the EU, but failed in countries including the US, Canada, Brazil and India. The ban in the EU extends to all forms of asbestos regardless of type and regardless of product. 'Alternative materials', production of which is largely seated in the countries keenest on a ban, give an economic rationale. In the EU there is cynicism in the official reason that the ban was to protect the health and safety of workers. Anyone truly concerned would have taken heed of the following:

- An INSERM report [5; p. 2] on the health effects of several asbestos substitutes notes: "Because the fibre structure of asbestos is a major pathogenic factor, any new fibre proposed as an asbestos substitute (or for any other use) should automatically be suspected of being pathogenic because of its structure."
- The Scientific Committee of the European Commission's Directorate General DG XXIV stated in a February 1998 report that: "...there is no significant epidemiology base to judge

the human health risks (of substitutes)hence the conclusion that specific substitute materials pose a substantially lower risk to human health, particularly public health, than the current use of chrysotile, is not well founded.....” [12].

Misinformation about asbestos is not the prerogative of the official world but is particularly rife in the public domain. The media in their constant search for sensationalism have created a climate where the word "asbestos," now causes immediate panic verging on hysteria. As with some other environmental problems, heavy occupational exposure to asbestos is counted equal with very low environmental exposure. The level of disease, which at the worst occurs in less than 10% of those exposed occupationally is translated by many, and particularly the media, to a death sentence following the tiniest exposure in the public arena. While it is true that asbestos, particularly blue and brown asbestos, in even modest quantities, are a significant health risk for those occupationally and also para-occupationally exposed this is not the case for chrysotile (white asbestos). Dangerous levels of exposure can result from those who just live near blue asbestos mines where fine weather and mine tailings used as road metal keep high concentrations of fibres in the air. However, studies (Camus...) of the very large population who live near chrysotile mines or on chrysotile ore bodies has shown that there is no excess asbestos-related disease in these areas. Even industrially the comparatively small danger posed by the present low levels of exposure to chrysotile in the industry cannot be shown to produce a significant risk of disease.

History

Asbestos is the commercial name applied to a group of six naturally occurring fibrous minerals. Five of these belong to the mineralogical type known as amphiboles (and include blue and brown asbestos otherwise known as crocidolite and amosite respectively) and one is a serpentine called chrysotile or white asbestos which is the principal mineral of commerce (~90% of world production). These mineral fibres are highly effective insulation materials and were applied to pipes and boilers by the millions of tons each year until the 1950s. They were also used in the insulation of steel-framed building and a myriad of other products. Since chrysotile accounts for the majority of the world's production of asbestos minerals, it is very important to understand that it is chemically and crystallographically distinct from the five amphibole minerals including blue and brown asbestos. Chemically chrysotile is a magnesium silicate similar in many ways to talc with which it is sometimes found, crystallographically it is found to have soft curly fibres. The outside of the chrysotile fibres are a magnesium oxide/hydroxide structure readily degraded in the body. The amphiboles are also metal silicates but with a more complex structure and form hard needle-like fibres. Blue and brown asbestos owe their colour to the large amounts of iron they contain although other amphiboles may be iron free. The outside of amphibole fibres is similar to quartz and is not degraded appreciably in the body. One of the important consequences of the differences is that chrysotile has a very much lower toxicity than the amphiboles.

Dangers associated with the use of asbestos were not realised when it was first used commercially towards the end of the nineteenth century. A very few reports did appear in the late

nineteenth/early twentieth century but it was 1924 before lung fibrosis was associated with workers heavily exposed to asbestos (Cooke, 1924). It was nearly ten years later (Lynch & Smith, 1935) that an association was noted between asbestosis and lung cancer. The UK Factory Inspectorate in their annual report for 1947, presented in 1949, found an excess of lung cancer in those with asbestosis. It was not until 1955 that Sir Richard Doll undertook the first cohort mortality study of asbestos workers and showed an excess of lung cancer in those with asbestosis, but not in those without the disease. A few years later, in 1960, a medical connection was made between blue asbestos and mesothelioma, a malignancy (tumour) of the lining of the chest wall. That year Dr Chris Wagner and his colleagues reported 33 cases of mesothelioma in the north-west Cape of South Africa (Wagner, Sleggs, and Marchand 1960). Twenty-eight of these individuals had worked or lived in the Cape (blue) asbestos field; four had worked in other parts of the asbestos industry. Shortly after this, in 1964, Dr. Irving Selikoff published a study of 632 New York insulation (shipyard) workers, which demonstrated an increased risk of lung cancer (Selikoff, Churg, and Hammond 1964).

The inevitable consequence of the massive asbestos use of the 1940s and two or three subsequent decades accompanied by minimal occupational hygiene exposed millions of people to high fibre levels from the range of asbestos types. As a consequence, we see today massive levels of asbestos-related diseases. There are four diseases linked with asbestos, two benign and two malignant. Asbestosis (fibrosis) is the condition where asbestos has damaged the lung tissue and caused scars to form. The lungs are the organ of the body which enables oxygen to pass from the air into the blood. The scars interfere with this process, so people suffering from asbestosis become breathless. Pleural disease has two forms. One is diffuse pleural thickening, which is linked with asbestosis. The other is pleural plaques; these are also scars, but they occur outside the lungs, in the chest wall, and because of this they are quite harmless even though their appearance on chest X-ray can be quite startling. The two malignant diseases are a very different matter. They are mesothelioma, which is a cancer of the pleura, the outer covering of the lungs; this is invariably fatal, and lung cancer, similar to that caused by smoking, which even with the best treatment, has only a five-year survival of less than 10%.

Today, workers' health and safety issues are not so easily ignored - legally or socially - as they were in the past, and there is a price to pay. Since approximately 1955, tens of thousands of workers have filed both compensation claims and tort suits against asbestos manufacturers claiming that they were made ill by asbestos and were suffering from asbestosis, lung cancer, or mesothelioma. Companies and their insurers have spent hundreds of millions of dollars defending and settling these claims. Some, like Johns-Manville and Keene re-organised through bankruptcy. Others folded and went out of business.

Fear of health effects has led to the development of an abatement (removal) industry. Removal of asbestos from buildings is a lucrative occupation. In 1992 there were 1040 contractors licensed to carry out asbestos removal in the UK. In the US it is a major industry costing the public and private sector billions of dollars a year [79]. Recently, a plea went out in the US {'USA today' - leading article} saying that asbestos abatement had so far cost the country 50 billion dollars to no good effect on public health. [Some years earlier Forbes Magazine had estimated that the final cost in the USA would be about \$200 billion if the abatement program was completed.] The

industry is driven by public fear and frequently runs ahead of other industries associated with mineral wool and cellulose insulation. The best advice, and UK HSE policy, is still non-removal of asbestos when the insulation is in good condition and doesn't need to be disturbed. The USEPA and Canadian agencies give similar advice [1]. The EPA's *Purple Book* (Guidance for Controlling Asbestos Containing Materials in Buildings) [80] states: 'The presence of asbestos in a building does not mean that the health of building occupants is necessarily endangered. As long as asbestos-containing material (ACM) remains in good condition and is not disturbed, exposure is unlikely.'

Scientific studies and health issues

Decades of study have taught us a lot about the asbestos minerals and the diseases they may cause. Asbestos fibres in a certain size range can enter the respiratory system and penetrate deep into the lungs. All fibres and other particles, of whatever composition or form, that can do this are known as respirable. However, not all particles are fibres and not all fibres, by general agreement, cause disease. Many studies have shown that only asbestos fibres that are greater than 5 µm long (l), less than 3 µm in diameter (d) and with an aspect ratio (l x d) greater than 3:1 can cause disease. This is the definition of the World Health Organisation and fibres that fall within this definition are sometimes called WHO fibres.

The European Union uses this definition in its Directive:

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DIRECTIVE 2003/18/EC OF THE EUROPEAN PARLIAMENT AND OF THE COUNCIL of 27 March 2003 amending Council Directive 83/477/EEC on the protection of workers from the risks related to exposure to asbestos at work

For the purpose of measuring asbestos in the air, only fibres with a length of more than five micrometres, a breadth of less than three micrometres and a length/breadth ratio greater than 3:1 shall be taken into consideration.

Many studies have shown that the danger posed by an asbestos fibre increases with its length. Stanton (1981) suggested that an optimum of danger occurred with fibres of length greater than 8 µm and diameter less than 0.25 µm. This has become known as the Stanton hypothesis. While this work has not fully stood the test of time there is general agreement that long thin fibres do pose the greatest danger with the suggestion that fibres longer than 20 µm are the most important.

The human lungs are delicate organs and do not like respirable particulate matter. However, they are not defenceless and providing the concentration of particles is not too great they have the means to cleanse themselves. They use the 'mucociliary escalator' for the airways and at the lowest depths of the lung, the alveolar region, where the thin lining allows diffusion of oxygen in and carbon dioxide out of the blood, the primary defence is a mobile white cell, the macrophage. The airways are lined with fine hairs, cilia, down to the level of the finest bronchial tubes and

have mucus-producing glands. The mucus traps particles and the hairs sweep it back up to the throat where it is swallowed. Particles that find their way deep into the lungs meet the macrophages, otherwise known as phagocytes. These have a dual role, phagocytosis and regulation. Phagocytosis is the process of ingesting and where possible digesting particles which have passed beyond the mucociliary escalator. Regulation includes the production of chemicals called cytokines. These are chemical messengers, whose role includes summoning other cells when defences are breached, starting and stopping the processes of inflammation, and repair of tissue damage as necessary. While isomorphous particles such as diesel fume, particulates in tobacco smoke and bacteria are easily removed fibres are difficult - shape matters. Fibres may be too long for the macrophages to ingest completely. The result is that having failed in their attempt to ingest a fibre they remain in an activated state, their mobility is reduced, and their cytokine production continues uncontrolled. If there are only a few fibres, or if the fibres are soluble or breakdown, other uninvolved macrophages can neutralise the inappropriate cytokines. But if the fibre inhalation is high, too many will remain in a 'frustrated macrophage' state, until they die with too few left to regulate the system. The result is inflammation, tissue damage and need for repair. This situation is known as functional overload. If the cleansing systems cannot cope the lungs are damaged and the durable fibres may remain in the body throughout the person's life. The longer a fibre remains in the lung the more likely it is to cause disease and this residence time appears to be related to the life-time of the animal (rodents or man) rather than to any absolute measure.

A phenomenon unique to durable fibres in the lung is the 'asbestos body'. Amphibole fibres, but only very rarely chrysotile fibres, can develop nodules spaced along their length which are thought to be the remains of the 'frustrated' macrophages that have failed in an attempt to ingest and remove the fibre. Long fibres, under the microscope, have the appearance of a string of beads. The asbestos body has no pathologic significance but the number of such bodies in the lung is indicative of past level of exposure.

Although all the asbestos types and indeed, probably, all durable particles, in sufficient quantity, may cause disease it is the amphibole fibres that are of the greatest concern since they are the most durable physically and chemically little altered in the body. Chrysotile fibres, on the other hand, which are not very durable, readily break down in the lung into smaller particles and fibres that are removed through both chemical degradation and scavenging by macrophages followed by transport up the mucociliary escalator. Chrysotile is said to have a much lower biopersistence than amphiboles. Long chrysotile fibres are cleared from the body within a few weeks, amphibole fibres may remain throughout life. As a consequence the potential of chrysotile for causing disease, particularly mesothelioma, is at worst, minimal. The majority of scientific and medical opinion is that chrysotile alone does not produce mesothelioma. When there is dissension it is often revealing to read the author's affiliation.

Thresholds

The threshold of exposure is the level of cumulative exposure below which disease caused by asbestos will not be detectable. Risk of disease then increases as the exposure increases.

Exposure is not a single event, such as swallowing an aspirin, it continues for a period of time and is described as total or cumulative exposure. It is normally defined in terms of fibre/millilitre years, (f/ml years) and the definition is based on the level of exposure in the workplace, measured as the numbers of fibres found in each ml of air, in the air breathed at work, multiplied by the number of years, or fractions of a year, during which the worker is exposed.

For example: 1 year worked at a level of 5 f/ml gives a cumulative exposure of 5 f/ml years

5 years	""	""	5 f/ml	""	""	25 f/ml years
½ year	""	""	5 f/ml	""	""	2.5 f/ml years.

Note that this is really a measure of the number of fibres inhaled. We actually inhale about 2½ billion ml of air at work during one year, so that 1 f/ml year represents the inhaling of 2½ billion fibres, but the term fibre year is just a convenient way of dealing with these large numbers.

Note also that since asbestos is present as a minor pollutant in all air and is breathed by everyone we all receive an annual exposure of 0.001 f/ml years while urban dwellers receive maybe several times that.

Thresholds and Health effects

Exposure to high levels of any of the asbestos types can cause asbestosis. Although asbestosis is non-malignant it is a debilitating, restrictive lung disease that has been known since 1927. In the early decades of the twentieth century little was done to protect acutely exposed insulators and shipbuilders and bystander trades, particularly during the Second World War. More recently, with awareness of the dangers of exposure to high levels of any dust, it has been recognised that there is a threshold below which disease will not occur:

- "In our judgement, asbestosis can be deemed a disease of past high exposure levels and will not occur in workers exposed to the regulated levels of occupational exposure now in force in Ontario." (Dupré J S, Mustard J F & Uffen R J. Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. Ontario Ministry of the Attorney General, 1984:94-97.)
- The UK Health and Safety Executive in their 1996 review of fibre toxicology "... concluded that there will be a threshold level of exposure below which no radiological or clinical manifestations of pulmonary fibrosis (asbestosis) will occur. The value for the threshold, and indeed the slope of the dose-response curve, depends on the fibre type and the fibre size-distribution in the workplace."

Very good evidence of a threshold comes from the observation that there are a large number of people who have lived for extended periods of time (several generations in some cases) in regions with elevated chrysotile fibre levels in the ambient air (exposed to 200-500 times urban levels elsewhere) yet have no fibrotic disease. Recent studies of these environmentally exposed populations has confirmed there is no excess disease (Camus, Churg).

Unfortunately, asbestos also causes cancer. Two tumour types are recognised: lung cancer, more specifically bronchiogenic carcinoma, pathologically similar to that caused by smoking and mesothelioma, a cancer of the lining of the chest wall. Any agent that causes cancer is deemed not to have a threshold dose. However, as our understanding of the biochemical changes that can lead to cancer improve it is now obvious that this is not necessarily the case. Many observations and studies of several carcinogens have revealed that the only sensible explanation for their mode of action is a threshold.

Asbestos provides a good example in the case of lung cancer following asbestos exposure. There has long been argument that a tumour will only develop subsequent to the development of asbestosis. However the evidence that asbestosis is necessary before lung cancer develops is very strong (Browne, Borm (Borm, P. J. (2002). "Munich Workshop on evaluation of fibre and particle toxicity." *Inhalation Toxicology* 14: 1-4.) and others). Since it is accepted that there is a threshold for asbestosis, *ipso facto*, there must be one for asbestos-related lung cancer. The confounding influence of smoking prevents good correlations since it has been shown there is a complex interaction between the two (Liddell). Certainly, in the absence of asbestosis compensation boards are not likely to make an award for damages due to asbestos if there is a history of smoking. In the UK no award is given unless asbestosis is present (and with the minor addition that diffuse pleural thickening with lung function deficit is also present).

- "To me the available data indicate that the only scientifically established association of lung cancer and asbestos exposure is the association of asbestosis and lung cancer; thus, only the presence of asbestosis can be used to incriminate asbestos as a cause of lung cancer." (Churg A. Asbestos, Asbestosis and Lung Cancer, *Modern Pathology* 6: 1993; 6:509-511).
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- Also, in the HSE review: "There appears to be an association between pulmonary fibrosis and lung cancer in that both diseases show a similar dose-response relationship with respect to asbestos exposure, show similar latent periods for development, show a similar dependence on fibre type and size, and both diseases emanate from the same underlying chronic inflammatory condition. These observations suggest that asbestos-induced lung cancer, like fibrosis, is a threshold phenomenon. It can be concluded that exposures which are insufficient to elicit chronic inflammation/cell proliferation (manifest for example, as alveolar Type 11 cell hyperplasia or fibrosis) will not incur any increased risk of lung cancer." (HSE, Review of Fibre Toxicology, 1996).

Mesothelioma was known as a disease before the asbestos industry developed. In the absence of external causative agents it is a rare tumour with an incidence believed to be about 1-2 per million per year or 1-2 in every 10,000 deaths (McDonald & McDonald, 1987a). The tumour has been found in just about every animal species from shellfish to the higher mammals (Ilgren & Wagner). Unlike lung cancer, the risk of mesothelioma is not affected by smoking (Muscat & Wynder, 1991). The commercial exploitation of asbestos has considerably enhanced the incidence of mesothelioma to a level, in men, hundreds of times greater than background. It must not be forgotten, though, that it is not just an asbestos-related disease. Although asbestos exposure is the most frequently recognised cause approximately 20% of all mesotheliomas are not related to asbestos exposure (Baldi et al., 2002). A number of other etiologic factors that can

cause the condition include exposure to other mineral fibres, chronic inflammation/scarring - as in cases of tuberculosis and empyema, heredity, irradiation, viruses, and exposure to other non-fibrous minerals and organic chemicals. The sporadic occurrence of mesothelioma in children, particularly young boys has not been linked to asbestos exposure. A search of the medical literature shows a number of reports of idiopathic tumours (for example: Erkilic et al, 2001; other refs.) but overall mesotheliomas from the variety of pathologic and iatrogenic causes account, at the present time, for a minority of the cases.

As for lung cancer, so for mesothelioma: studies of those occupationally exposed suggest a practical threshold of exposure but several cases of non-occupational disease are known in family members of asbestos workers (para-occupational exposure) (refs.). The occasional reports of familial cases of MPM are mostly associated with documented asbestos exposure although this not always the case. One study found 17% of cases that had no documented exposure (Dawson et al. 1992). Overall, only 3%-8% of asbestos workers develop MPM (alternative source: only 2-10% of the individuals with heavy exposure to asbestos will develop mesothelioma (Bell & Testa 1997)) Inherited susceptibility to mesothelioma has been suggested by studies showing that among the family members of asbestos workers, first degree relatives are at a higher risk than spouses (Browne 1995), so the occurrence of this tumour in families suggests that genetic factors may be important. The rationale presented is to say that, theoretically, there is no threshold level of asbestos exposure below which MPM will not occur (the one fibre can kill, theory); therefore, a small percentage of tumours may develop secondary to environmental contamination or undetected occupational or environmental exposure. However, there is no data available to suggest that low-level environmental exposure such as may be found in schools, etc., poses a health hazard (Mossman et al. 1990). Cases in people who live in asbestos mining areas or who have lived in the vicinity of asbestos plants have only been found when the mineral mined was blue asbestos and mine tailings or asbestos-containing outcrop rock have been used as road metal or a related use where further comminution could release fibres and so their exposure is undoubtedly higher than might have been expected.

To support the claim that mesothelioma is almost exclusively asbestos-related it has been proposed that even when the lungs are found to have low asbestos body counts (less than 20 asbestos bodies per gram of wet lung tissue) that sub-microscopic fibres that do not readily form asbestos bodies are involved. This in spite of the wealth of good scientific evidence that points to long fibres as the causative agent. Such arguments are difficult to refute since studies involving effects of small amounts of substances upon people suffer the handicap that they cannot be experimentally tested. Although the consequences of heavy asbestos exposure are readily related to disease, lower exposure is not. It is a fact that many patients with MPM do not have a documented history of asbestos exposure (range, 0%-87% in many literature studies) but that does not mean they have never been exposed.

With regard to overall toxicity it is generally accepted that the carcinogenic potential of chrysotile, is weaker than that of the amphiboles (Mossman et al., 1990; Browne, 1995; [Churg, 1988](#); McDonald & McDonald, 1996; Stayner et al., 1996; Hodgson and Darnton, 2000). For lung cancer some clinicians even doubt that chrysotile causes lung cancer in the absence of smoking. Even when there is asbestosis. This is nearly impossible to prove because for reasons

unknown, the level of smoking is higher than average in asbestos workers. It would be difficult to find a large enough cohort of chrysotile-exposed life-long non-smokers to do a proper epidemiological study.

In the case of mesothelioma there is now a solid body of opinion that says that exposure to pure chrysotile does not cause the disease. This should be a more definite statement but the picture is confused because some chrysotile deposits are contaminated with the amphibole tremolite, a non-commercial amphibole, and it is most probable that at least some of the mesothelioma cases reported in chrysotile workers are due to this contamination (McDonald & McDonald 1996, Stayner et al. 1996). Industrial cohorts which in the past were reported as exposed to chrysotile alone, and where cases of mesothelioma have occurred have, on recent more thorough investigation, been found to have been exposed to blue asbestos for some of their working life or to have had such a varied employment record that it is impossible to determine what they may have been exposed to. Finding out whether or not amphibole exposure occurred, other than by post-mortem lung fibre analysis, is not easy. Many of the people suffering from mesothelioma today were first exposed during war-time when records of materials may not have been kept, or if kept, have not survived. Even more recent work ([Yano, 2001](#)) may suffer from lack of relevant information. (and see below)

Contentious issues

Contentions arose once the spectrum of diseases caused by asbestos types was established. They have arisen because evidence was incomplete and because the science advances slowly but mostly because of the money involved in litigation. There is good evidence that some manufacturers did not inform their workforce of the latest health concerns as they arose nor take steps to reduce their exposure to asbestos fibres. In consequence many workers were exposed to levels of fibre now known to have been damagingly high. The very high toxicity of blue asbestos and comparable toxicity of milled amosite were unsuspected but when it became known exposures could have been reduced. Implementation of good health and safety practices at that time would have reduced the legacy of asbestos-related diseases we see today. In the event any perceived economy was false since the employers were subsequently held liable for damages and the legal profession geared up for the financially crippling legal battles in the asbestos-fest that exists today. A battle in which they are the only winners.

Science has been largely pushed aside in the aftermath, even at times by scientists. There is too much money at stake to allow scientific argument with its attention to detail and presentation of probabilities to lead the discussion. As a result a number of non-scientific and even nonsensical claims have arisen and persist today, in spite of sound evidence to the contrary. The most important are that all asbestos types have equal toxicity and cause the same spectrum of diseases; that any dose, however small, can trigger an asbestos-related disease - particularly cancer; and that asbestos alone is the only causative agent for mesothelioma. All three are palpable nonsense. Most importantly note the insistence on the use of the word 'asbestos'. Legislators who have no science believe it to be the generic, lawyers revel in the obfuscation that results from its use.

Even the HSE which seems largely to have forgotten the words of wisdom in its authoritative 1996 review, accepts the first premise is wrong. Common sense denies the second. Since life in a large city may mean inhaling about 14,000 asbestos fibres a day the validity of the statement must be in doubt. Also, there are many places in the world where people are exposed to much higher levels than this every day of their life. Finally, at death we all have in our lungs, high levels of, largely, amphibole fibres, maybe as many as a million per gram lung dry-weight.

The greatest contention comes in the case of mesothelioma although it was recognised as a disease before the asbestos industry developed. However, because of the fortunes to be made through asbestos litigation, lawyers, and probably only lawyers, claim that MPM is solely an asbestos-related disease and there is no other cause. Even where there is no occupational exposure the claim is made that somewhere, at some time, there must have been.

The tremolite issue

The largest study ever carried out on asbestos workers was that on 9780 men born between 1891 and 1920 who were employed for at least a month in the Quebec chrysotile producing industry (Liddell et al., 1998). The earliest workers started in 1904. The men were followed to 1992 by which time 8000 had died, 657 from lung cancer and 33 from mesothelioma (out of 7456 (0.4%) - this was an earlier study). The results showed that, even after extremely high dust exposure, the risks of excess lung cancer were very low. Overall it was estimated as no more than 65 deaths (0.8% of 8009), while little or no excess was seen in men exposed to less than 300 mpcf x years¹ - this is a massive dose, orders of magnitude higher than any that would be allowed today. Regarding the mesotheliomas it was realised that these men and also a number of those with lung cancer had worked in the central area of Thetford mines where the chrysotile is contaminated with the amphibole tremolite. It was also found that some of those with mesothelioma had worked with blue asbestos. A consequence of this has been that the trace amounts of tremolite that may remain in chrysotile after processing (Sebastien et al., 1989) are held responsible by some for causing disease. The argument is: you may say that chrysotile is safe but what about the tremolite? Does it matter to the unfortunate person concerned what fibre the mesothelioma is due to? That is certainly true, but there is human evidence and very good animal evidence that such trace quantities do not cause mesothelioma (NIOSH study noted in Ilgren & Chatfield, 1998). There are two other reasons why tremolite is no longer a concern. It has been shown that for a miner to collect enough tremolite in his lungs to cause a mesothelioma he had to have very heavy exposure in the high tremolite areas, enough to cause asbestosis¹¹. Also, new geological research has shown that tremolite is not uniformly mixed with the chrysotile, but occurs in separate seams which can be identified and avoided¹². As a result the high tremolite-contaminated mines in the central Thetford area have closed as have the high-tremolite areas in the huge Asbest mine in Russia, so supplies from these sources now have minimal or no tremolite.

¹ mpcf x years is (million particles per cubic foot) x years. 300 mpcf.y is roughly equivalent to 1000 (fibres/ml) x years, or, say, 10 years in the 1940s at 80 fibres/ml.

Asbestos Containing Materials (ACMs)

High density cement and friction products containing asbestos are just some of a variety of products known as Asbestos Containing Materials (ACMs). They mostly contain only chrysotile although crocidolite and amosite have been used in admixture for some pipes and flat wall boards. Friction products have been almost exclusively chrysotile. Their production accounts for some 90% of all the world production of chrysotile. They are the most common asbestos products to be found in the domestic environment. Any fibre release from them is extremely low and yet they are regulated as if they were loose fibre. Regulators and so the general public regard them as a cancer risk. Why? It is difficult to imagine the sort of treatment they would require to liberate enough fibre to cause asbestosis and so, possibly, lung cancer in anyone exposed. No chrysotile products are thought ever to have produced mesothelioma.

“There has probably never been an attributable, clinically and pathologically proven case of mesothelioma in any manufacturing industry, e.g. cement, friction products, or textiles, amongst the many tens of thousands of workers where chrysotile alone has been used.” ([Ilgren & Chatfield, 1998](#)) - even though the time since studies began stretches back to the careless handling of asbestos 50 or more years ago.

Manufacture and disease in the manufacturing industries

When the first cohort studies of deaths among asbestos workers appeared in the 1960s and 70s, they nearly all related to those working with mixtures of chrysotile with crocidolite and amosite. In most cases chrysotile was the major fibre component, with only small amounts of amphibole added, so that as a result chrysotile was blamed for the deaths along with the amphiboles. However, there is a simple way to test that idea, which is to look at mortality studies of workers in industry where only chrysotile has been used.

There have been 8 cohort mortality studies of industries where chrysotile has been the only fibre used.

- A cohort mortality study is one where you start with an unselected group of fit people - say, all those who started work at a factory at a particular time - and then follow them up for a number of years to find out what they die of. As each death occurs, the cause is recorded and the final totals are compared with the causes of deaths in the general population not exposed to asbestos.

Another relevant definition is the Standardised Mortality Ratio (SMR):

- The standardised mortality ratio is the ratio of deaths observed, D , to those expected, E , on the basis of the mortality rates of some reference population. There are certain assumptions-- that D was generated by a Poisson process and that E is based on such large numbers that it can be taken as without error. It is often expressed as a percentage: % relative risk.

No risk would be represented by an SMR of 100. If working in the factory caused disease (from whatever cause) this would show as an SMR of greater than 100. It is common in worker cohorts

to find an SMR of less than 100 but this does not mean that it is healthier to work. The phenomenon is called the healthy worker effect and demonstrates that only the healthy do work. The general population, on the other hand includes healthy people and sick people. An SMR of less than 100 is taken to mean that there is no risk attached to the work of that group of workers.

There are 4 studies of asbestos cement factories¹⁻⁴, 2 of friction materials^{5,6}, and 2 relating to chrysotile mining^{7,8} although only the Italian study is noted here. The Canadian study, by far the largest mining study, gave similar results but it was such a complex study that it is difficult to summarise in a comparable way to the other 7.

Asbestos cement products

Asbestos Cement is primarily a cement-based product where about 10% to 15% w/w asbestos fibres are added to reinforce the cement. Asbestos cement is weatherproof in that although it will absorb moisture, the water does not pass through the product. Asbestos-cement products account for some 90% of all the world production of chrysotile. The products made include water cisterns, rainwater gutters, down pipes, pressure pipes, underground drainage and sewer pipes, corrugated sheets, mouldings and slates or shingles (tiles). They are the asbestos containing materials (ACMs) that most people come into contact with in a non-occupational setting. However, the greatest exposure to asbestos comes from those who make the products rather than the end-users. End-users can only ever receive a fraction of the exposure of those who manufacture the product. Although most asbestos cement products are made solely with chrysotile some products were made with a mixture of asbestos types. This is no longer the case but of course the materials containing mixed fibre may still exist.

Asbestos cement products have a cement-rich surface with the asbestos fibres encapsulated within. In products used outdoors for weatherproofing a small amount of fibres may be released during natural weathering although greater amounts of fibre can be released if the products are subject to any abrasive cleaning or working. However, chrysotile is chemically altered to a greater or lesser degree within the cement matrix and also most of the fibres breakdown with the cement as part of the weathering process. The degree to which this latter effect occurs depends largely on the acidity of the rain. Acid rain removes magnesium from the surface of the chrysotile fibres. Examined under a microscope many fibres can be seen to be coated with small crystals of calcite.

Four studies have been made in asbestos cement production plants that only used chrysotile looking at the risk of lung cancer. The results of the studies are summarised in this table where the studies are named according to the principal investigator:

Chrysotile-only Cohorts Male Mortality				
1. ASBESTOS-CEMENT				
	total deaths no.	% relative risk (SMR)	lung cancer no.	% relative risk (SMR)
Gardner	384	94	35	92
Thomas	351	102	30	91
Ohlson	220	103	11	122
Weiss	66	61	4	93
TOTAL	1021	95	80	95

The studies were all quite large and in total considered over 1,000 deaths from all causes (column 1). The next column (SMR) shows whether there was any serious risk in working in those factories by comparing the numbers of deaths in these workers with numbers you would expect in people of the same age in the general population. Clearly there wasn't any serious risk: that is, in these factories, it has been at least as safe working with chrysotile based asbestos-cement as it has been for the rest of the population. The last two columns look at the lung cancers in the group. In 3 out of the 4 studies^{1,2,4} the death rate from lung cancer was lower than for a comparable group of men not working not in these factories. The only apparent exception is the small Swedish study³, and the increase here could perhaps be due to it being a small sample.

But even for the Swedish workers, closer study shows that the evidence of risk disappears. Manual workers tend to smoke more heavily, at least in Europe and North America where these studies originated, and differences in smoking habits, as noted above, could swamp effects from other causes.

Evidence for other confounding influences such as smoking can be determined by making comparisons within the cohort. If the amount of disease increases as exposure increases, this is an important indication that the exposure is causing the disease; put another way, if chrysotile causes a lung cancer risk, the more exposure to chrysotile, the longer men have worked in the factory, the greater the risk to be expected. Examining the Swedish study, and the other 4 studies (below), it can be seen that all except one actually show a negative dose-response; in other words, those with higher exposure actually have lower mortality rates! The McDonald⁵ and Finkelstein⁶ studies are both of friction materials factories, and Piolatto⁷ looked at the

Chrysotile mine at Balangero in Italy. SMR1 gives the risk for short-term workers (the lowest exposure category) compared with the general public, and SMR2 the risk for the long-term workers with the highest exposures.

Chrysotile-only Cohorts		Male Mortality	
2. Negative dose-response			
	lung cancer no.	SMR 1	SMR 2
Ohlson **	9	278	103
McDonald **	73	167	137
Finkelstein **	11	490	110
Piolatto **	22	120	105

**SMR1: lowest duration category SMR2: all longer exposures

In each case, the longer the workforce was exposed to chrysotile, the lower their risk. This is not to suggest that chrysotile is good for your lungs, and short-term manual workers are notorious for having an unhealthy lifestyle⁹. but the figures show that other causes of lung cancer were increasing the risk, and there was no evidence that working with chrysotile had any harmful effect. A slight question mark would remain over the Connecticut friction materials workers⁵, because the difference is not very great, but the 3 remaining studies do not give any firm evidence of risk.

Six of the 8 cohorts were in manufacturing industry, and none had any evidence of a lung cancer risk. Just as important is the fact, that in these 6 cohorts there was not a single attributable case of mesothelioma among 2,288 deaths. This was not the case in the two mining cohorts. Here though, the few mesotheliomas that occurred can nearly all be attributed to amphibole exposure.

MESOTHELIOMAS IN CHRYSOTILE - ONLY COHORTS		
<u>Manufacturing:</u>	no.	
All cohorts:	0	No attributable mesotheliomas in 2288 deaths from all causes
<u>Mining and milling:</u>	no.	
Balangero	3	all exposed >300 f/ml years
Quebec	31	all exposed > 2 years

Evidence that has appeared in the past few years explains probably all of these cases. It has emerged that just after the war some processing of crocidolite was carried out at the Balangero mines and these cases may have been working there or in a nearby plant that used asbestos at the time (Health Effect of C, p.177). In the Quebec miners McDonald has shown that the small number of mesotheliomas which have occurred is almost certainly not due to chrysotile, but to the amphibole tremolite, which is known as a contaminant in some areas of the mines¹⁰ (see above).

A new study from South Africa has confirmed previous evidence that no case of mesothelioma has ever been identified among chrysotile miners there, despite large numbers employed^{13,14}. Very little tremolite is found in the chrysotile from this area. In California little individual data is available from Coalinga where a short fibre amphibole-free chrysotile was mined. Although there has been no epidemiological study of the 900 miners and millers there has been a very small study from some of the 279 men who worked in a Johns-Manville on-site plant. Of a group of 40 men who had worked for 30 years and were followed-up none had asbestos-related diseases. Similarly among 50 men who had X-rays none showed asbestos-related changes. (Ilgren, pers. comm)

There has been some dissension in a recent study from China that claimed to show that mesotheliomas could arise from work with tremolite-free chrysotile¹⁵. However, the analysis for tremolite was inadequate; the only statistically valid method of assessing a chrysotile sample for tremolite is through chemical digestion. The authors used only microscopy, which can rarely detect the small amount of tremolite present, and the chrysotile came from a mining area known to be heavily contaminated with tremolite¹⁶. Then exposures were very heavy and no post-mortem lung fibre burdens were examined. Also, of the two mesotheliomas found, one had a latent period of less than 14 years to death - less than any known verified case^{17,18}, (one case occurring after a shorter period reported by Lanphear¹⁹ had had previous domestic exposure) and the other was a peritoneal case, which many have said do not occur with chrysotile²⁰, so the paper has really no credibility.

Asbestosis is a disease that results from heavy occupational exposure. As noted earlier, it is accepted that there is a threshold at which it occurs. It is difficult now to estimate the exposure levels at which the disease begins to appear, since deaths directly from asbestosis are relatively uncommon in recent years. And while advanced asbestosis is relatively easy to diagnose, deciding the point at which it begins is as difficult as defining the moment at which dawn

begins when you are in the middle of a city with street lights on, car headlights flashing by and neon signs blazing. Since the exposures of people engaged in manufacture of high density chrysotile products are low to obtain an idea of the levels required for the onset of the disease it is necessary to study those with higher exposure. One of the best attempts to define the threshold for asbestosis was made with the Rochdale textile cohort²¹. There it was found that there was a 1% risk of asbestosis at a cumulative exposure of 72 f/ml years.

THRESHOLD FOR ASBESTOSIS		
Asbestos Textiles	Chrysotile and Crocidolite	Cumulative exposure for 1% risk
Possible asbestosis		55 fibre/ml years
Definite asbestosis		72 fibre/ml years

However, this has been judged too low to be applied to chrysotile asbestos-cement or friction materials workers for two reasons. The first reason is that this plant used an appreciable amount of crocidolite mixed in with the chrysotile, and the second is that figures derived from past textile operations may not be generally applicable. But we can get a clue from the lung cancer figures. There is occasional dispute about whether the risk of lung cancer from asbestos exposure is only present in people who already have asbestosis although the evidence is very strong that this is the case. What is generally accepted is that if you have asbestosis, your lung cancer risk rises very sharply. So it is safe to assume that where there is no evidence of extra lung cancer risk, there is no appreciable amount of asbestosis

Since the highest fibre level seen in modern asbestos-cement and friction materials production is some 0.5-1.0 f/ml asbestosis and so lung cancer, is most unlikely today. Even in the bad old days, although we do not know the fibre levels, there was no detectable lung cancer risk and therefore no appreciable asbestosis.

The calculations relating fibre levels and disease discussed so far used cumulative exposure, which is not a convenient measure of exposure for those responsible for regulating exposures to prevent disease. Regulators need a fibre level which can be permitted however long people work. Vacek and McDonald²² were able to calculate, from the mortality studies for which they had dust measurements, fibre levels in the workplace below which people can work for as long as they like without detectable risk of lung cancer and therefore of asbestosis. The point is that the lungs have their defences. They can deal with a certain level of dust, and these figures give an indication of what that level may be.

APPROXIMATE THRESHOLD EXPOSURE LEVELS	
Lifetime exposure Chrysotile only	
Lung cancer	
Mining and milling (Quebec)	30+ fibre/ml
Friction materials (Connecticut)	30+ fibre/ml
[Textiles (South Carolina)]	3 fibre/ml]

Derived from Vacek and McDonald

Friction products

Two studies of workers employed in a chrysotile only plant are noted above. There have been other studies but in plants where crocidolite was used as well. In one of these friction product plants there was excess mortality from pleural mesothelioma of 13 deaths in men. Eleven of these were known to have had contact with crocidolite, of the other two, the diagnosis was uncertain in one and the occupational history of the other was not well established [6,7]. Contact with crocidolite does not have to be heavy or prolonged before the risk of mesothelioma is raised considerably. Probably the most striking evidence for this comes from data on short-term exposures to crocidolite of workers in Canada and the UK engaged in the manufacture of military gas masks [8,9]. However, although chrysotile alone has been shown not to cause disease in manufacture there are still concerns voiced about fitters and the effect of in-use wear causing an increase in environmental levels of chrysotile.

from Asbestos in friction materials

Before the development of alternative formulations, the brake and clutch linings of automobiles contained from 10 to 70% of chrysotile with typical figures being 40 to 50%. This use has decreased considerably in recent years in Western countries. Until alternatives began to be sought chrysotile was the only fibrous mineral used in brakes or similar friction products such as clutch plates. It was used because of all the materials tested both in the development of modern brakes and more recently in the search for alternatives for asbestos, it is the best material that has ever been found. To this day no other single material combines the frictional performance, thermal stability, wear resistance, strength and cost in such an effective way. A feasibility analysis [27] of asbestos replacement pointed out that “Mandating non-asbestos friction materials for vehicles that were originally equipped with asbestos-based linings could lead to a potentially serious customer safety risk unless stringent friction material qualification specification tests are included.”

By design every application of brake or clutch is associated with a little wear of the lining [28, 29]. Not all of the material that is dislodged in this way is released to the atmosphere though; after a careful study of general test vehicles it was concluded that for cars 81.6% of the wear material was deposited on the ground, 14.4% retained in the brake housing and only 3% emitted to the atmosphere. It is most probable that this last fraction is the only contributor to public exposure to chrysotile from this use. With the increasing popularity of open disk brakes more material is liable to be emitted to the atmosphere although this will not be chrysotile since brake-pads are no longer made with this material. Studies have shown that only a tiny fraction of the material from brake shoes consists of asbestos as chrysotile [30]. In this particular study 99.7% of the chrysotile had been converted by heat into olivine and forsterite particles. It has also been shown that chrysotile asbestos not only loses its mineral properties but also its biological activity even at temperatures below the olivine transformation point. It seems that minimal degradation of the chrysotile surface structure imparts a disproportionately great effect on its biological activity ([Langer, 2003](#)). Other work [31] has found that only 0.2% of the lining chrysotile was released as fibres, most was converted to non-fibrous material. US studies [31,32] have calculated that airborne fibres from brake usage were responsible for, respectively,

only 0.0000021 and 0.0000017 fibres/ml. A minute fraction of average urban levels; even for the worst case, Los Angeles, fibres from braking contributed only 0.0000077 fibres/ml. Although these various studies show that few fibres are released it is obvious that such release from vehicular traffic will be higher near busy road junctions or other areas of high braking activity than elsewhere, albeit the total levels will still be very low. For example Bruckman [33] found levels increased in the vicinity of toll malls on US roads. Two fairly congested road junctions in Greater London area were also surveyed for airborne asbestos fibres released as a consequence of the braking of vehicular traffic [28]. All the analyses were carried out using transmission electron microscopy (TEM). The total asbestos fibre levels found at these junctions ranged from < 0.00055 to 0.0062 fibres/ml. The 'regulated fibres', i.e. fibres > 5 µm long and < 3 µm diameter, were < 0.0004 fibres/ml. Another study in the Tokyo area [34] monitored the concentration of phenolic resin in the particulate matter deposited on roads. From this the concentration of asbestos was estimated knowing the concentration of asbestos in the original friction materials. The asbestos concentrations, calculated simply as the proportion of the original mix, were 0.36 - 2.1, 0.20 - 3.0 and 1.5 - 3.1 mg/g dust on one national highway and loop lines No. 7 and No. 8, respectively. The actual concentration of asbestos dust having a 'long-fibre shape' was estimated to be about 1% of this or less. These various levels can all be regarded as very low and presenting no health risk to the public [35]. Other measurements have shown that airborne fibre levels 30 m from the nearest traffic were below the limit of detection.

Friction Plants: A mortality study (1942-1980) [6] was carried out on 13,460 workers in a factory producing friction materials (see above for further discussion of this plant). The only type of asbestos used was chrysotile, except during two well-defined periods before 1945 when crocidolite was used, and over 99% of the population was traced. Compared with national death rates, there were no detectable excesses of deaths due to lung cancer, gastrointestinal cancer, or other cancers. The exposure levels were low with only 5% of men accumulating 100 fibre-years/ml. The authors state: "The experience at this factory over a 40-year period showed that chrysotile asbestos was processed with no detectable excess mortality." The situation was unchanged when 7 years later this study was re-examined (1941-1986) [7]. The authors from this later study confirmed that there was no excess of deaths from lung cancer or from chronic respiratory disease. After 1950, hygienic control was progressively improved at this factory, and from 1970 levels of asbestos have not exceeded 0.5-1.0 fibres/ml. The authors concluded: "It is concluded that with good environmental control chrysotile asbestos may be used in manufacture without causing excess mortality."

Brake servicing

Lax practices such as the use of compressed air to clean brake drums and other parts are generally and quite rightly proscribed. This is not necessarily because of any risk from airborne chrysotile; one study in Wales (Professor RC Brown, personal communication) could not find any fibres in brake drum dust although other studies (for example: [92]) do find some chrysotile fibres, but the silica and heavy metal components of the dust could be injurious to health. Studies in vehicles with chrysotile brake shoes of actual exposure to mechanics removing brake drums and replacing the shoes has shown that airborne chrysotile fibre exposure exposures for each test remained below the currently applicable limit (in the US) of 0.1 f/ml (8-h TWA) (Blake et al., 2003).

Floor Tiles and Mastic

Another group of high density products includes floor tiles (vinyl and asphalt), roofing materials, mastics and caulking. In these products chrysotile is usually tightly bound to the matrix material and therefore not easily isolated: the materials are non-friable. Very few studies have been carried out on such materials - almost none on roofing or caulking materials.

Vinyl floor tiles and the glues (mastic) used to attach these tiles to a surface commonly had asbestos incorporated as a component ([Lange, 2004](#)). A recent investigation on the type of asbestos reported that only chrysotile was found ([Lange, 2004a](#)). Asbestos-containing floor tile and mastic materials are identified as, and regulated as - by agencies in the United States, asbestos-containing materials (ACM). They contain chrysotile at a concentration that is commonly above one-percent. The purpose of including these building materials in such a classification is claimed to be to protect the environment, occupational workers and public health from exposure and subsequently related diseases that may occur as a result of this exposure.

Asbestos-containing vinyl floor tiles and mastic are building materials that do not readily release fibres; thus, are not capable of “increasing” levels of asbestos in the air during normal use or on removal. Because of this there have been numerous discussions on the issue as to whether these materials as well as possibly others (e.g. gaskets, roofing materials) ([Lange, 2000](#); [Spence and Rocchi, 1996](#)) should be included within the regulatory frame of ACM. Some ([Lange, 2002](#)) have considered inclusion of asbestos-containing floor tile and mastic as a regulated material a form of legislating science, which is a method of expanding the precautionary principle into a regulatory framework without adequate scientific data to support cause and effect relationships ([Lange, 2002](#); [Wong, 2001](#)). This concept is best illustrated through the asbestos concerns about the debris from the World Trade Center ([Lange, 2001, 2002a, 2003, 2004b](#)). Here bulk and airborne samples comfortably exceeded the established criteria throughout much of Manhattan, yet regulatory agencies, forced to quote science and allay public concern, described these levels as not a health concern ([Lange, 2002, 2003](#)). Since the basis for regulating any material is its potential to cause disease, the nub of the argument is the level at which the material is regulated. Events at the World Trade Center showed that this is a moveable feast. The precautionary principle which had set airborne levels for chrysotile before 9/11 had been applied for political reasons rather than health protection.

Floor tiles and mastic do not, by their nature, liberate fibres when new. Whether they do so in use, through wear, is little studied and at the present time the concern regarding these products is with their removal. Even here there are only a few published studies ([Lange, 2004](#)) on exposure levels associated with abatement (removal) of asbestos-containing floor tile and mastic. Because levels of airborne fibres are low during such work measurement and analysis of results is difficult. The investigations that have been carried out used techniques that can be categorised into three groups:

- task-length average (TLA) area and personal samples that then evaluated exposure by phase contrast microscopy (PCM) and transmission electron microscopy (TEM) ([Crossman et al., 1996](#)),
- time-weighted average (TWA) with PCM personal sample measurements (Lange and Thomulka, 2000),
- and a TLA and TWA PCM area and personal samples ([Lange and Thomulka, 2000a](#); [Lange et al., 1996](#)).

TLA - the average time taken to complete the job.

TWA - the time-weighted average concentration for a normal 8-h day and 40-h week.

PCM - Phase contrast microscopy is a contrast-enhancing optical technique that can be used to produce high-contrast images of transparent specimens, such as living cells micro-organisms, fibres, glass fragments, etc.

TEM - transmission electron microscopy. Materials for TEM must be thin enough to allow electrons to pass through the sample, just as light is transmitted through materials in conventional optical microscopy. TEM is ideally suited to measuring asbestos fibres of size below the resolution of optical microscopy.

When conducting various abatement practices to remove floor tile, [Crossman et al., \(1996\)](#) reported a TLA area using PCM and TEM analysis. Because of the lower resolution of the optical microscope the TEM counts (which are given as structures per cc (s/ml) for all counts, for counts $>0.5 \mu\text{m}$ and for counts $\geq 5 \mu\text{m}$) were all generally greater than the PCM counts (f/ml). The sample concentrations reported during different types of abatement practices in removing floor tile showed by PCM that they were similar to background or baseline samples. For these baseline air samples the PCM counts were greater than the TEM counts, with a similar finding for final clearance samples. When conducting floor tile abatement using the recommended work practices of the Resilient Floor Covering Industry, similar results were obtained ([Williams and Crossman, 2003](#)). The highest concentrations in this study for TEM were obtained during removal of sheet vinyl, with the lowest associated for 12 x 12 inch vinyl tile and asphaltic tile. The highest exposure levels for mastic were associated with TEM. PCM sample results for the various types of tile and mastic were similar to the background levels reported in the earlier study by [Crossman et al. \(1996\)](#). These two studies only collected area samples.

The results by [Crossman et al \(1996\)](#) and [Williams and Crossman \(2003\)](#) showed that most of the fibres detected were $\leq 5 \mu\text{m}$ in size. These data also suggest that most asbestos fibres are missed when evaluated using PCM as compared to TEM. However, a difficulty in making this evaluation and subsequent conclusion is that f/ml and s/ml cannot be directly compared, as they are obtained by counting methods according to two different criteria ([Ilgren, 2001](#)). There are also no studies relating s/ml to health outcomes, while the OSHA regulations and epidemiological investigations ([Liddell et al., 1997](#)) have related effects to f/ml.

Studies employing area measurements do not represent the actual exposure to workers. It has been shown that area samples cannot be used to represent personal exposure (Lange & Thomulka, 2000), although there is a continuing effort to establish a “comparison” between these two measurement methods (Lange, 2003; [Lange et al., 1996](#)). Since many earlier studies and most recently that of Bernstein et al. (2003) have shown that the hazard from any asbestos type is from longer fibres the use of these data to determine and evaluate the potential for disease would greatly over exaggerate the risk (Lange, 2004b). The current mesothelioma trends in the US (Price and Ware, 2003) do not support a potential cause and effect relationship for exposure as suggested by Williams and Crossman (2003) and Crossman et al., (1996).

Studies ([Lange and Thomulka, 2000a](#); [Lange, 2004](#)) that collected and evaluated personal samples during asbestos abatement of floor tile and mastic reported that exposures do not exceed current the Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) of 0.01 f/ml-TWA. It has been shown that personal samplers generally show a higher concentration than area samplers (Lange and Thomulka, 2002a). However, depending how the samples are collected, area samples can give higher concentrations than personal measurements ([Lange et al., 1996](#)). How the sampling is conducted greatly influences the relationship between area and personal samples.

There have been other PCM studies ([Lange, 2000a](#)) that have evaluated both area and personal samples during abatement of floor tile and mastic. No distinct relationship could be shown between area and personal samples (Lange, 2000a) with both sampling methods reporting low exposure levels. Comparing studies that reported personal (Lange and Thomulka, 2000), area and personal ([Lange and Thomulka, 2000a](#)) and only area exposure measurements ([Lange et al., 1996](#)), the exposure levels were all similar. A few other studies that employed PCM for detection ([Mlynarek et al., 1996](#); [Lange and Thomulka, 2001](#)) that reported on exposure to flooring materials, along with other types of ACM bound in a matrix (e.g. roofing) ([Lange, 2000b](#)), support the low levels of exposure found in the studies ([Lange, 2002](#); [Lange and Thomulka, 2000a](#)) that evaluated floor tile and mastic abatement activities in more detail. All these data suggest that exposure levels associated with abatement of floor tile and mastic for fibres measurable by PCM are near background level. Inclusion of TEM exposure data in these and other studies would be inappropriate because this technique attempts to establish risk with short fibres (< 5 µm) as if they were equal in risk to longer fibres.

Chrysotile exposure from floor tile and mastic are much lower than from other forms of ACM. Therefore the potential for them to cause disease, is at the worst low but in practical terms non-existent ([Lange, 2004](#)). The low risk is not only because there is little or no exposure, but that exposure is only to short fibre chrysotile (< 5 µm).

Conclusion

The last 20 years or so have seen enormous advances in our knowledge and understanding of asbestos-related disease. Unfortunately, governments and regulatory agencies have largely ignored the findings. Lawyers and pressure groups vigorously resist them. Pressure groups,

once launched, do not want to have their minds changed by new evidence, particularly when they are supported by manufacturers of substitutes for asbestos who have their own interests in getting all asbestos, including chrysotile, banned. Lawyers do not want anything that makes their pleas more difficult and may reduce their earning potential. Governments and regulators are always very reluctant to admit that they have been wrong, particularly when much of the evidence comes from other countries.

In summary the facts are:

1. Chrysotile differs markedly from all other commercial asbestos: It is not acid-resistant, it is readily broken down in the lung and removed while amphiboles persist.
2. Early mortality studies which led to the regulations we have today were concerned mainly with industries using mixtures of fibre types
3. All studies of industries where only chrysotile was used show that, even at high exposures, its toxicity is relatively low.
4. Animal experiments confirm the conclusions from human studies.

Regarding thresholds:

For manufacturing industries, (excluding textiles for which a small doubt remains) there is good evidence that after exposure to chrysotile there is no epidemiologically detectable risk for mesothelioma and, incidentally, gastro-intestinal cancer. The risk of asbestosis and so lung cancer after exposure throughout a working life of 40 years only appears where the exposures were in excess of 20 f/ml. This level is 40 times greater than the permitted level in the UK and 100 times that for the USA.

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It is worth, perhaps, in the light of our new knowledge re-considering the (EU) Commission Directive 97/69/EC of 5 December 1997 adapting to technical progress for the 23rd time Council Directive 67/548/EEC on the approximation of the laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances (Text with EEA relevance)
Official Journal L 343 , 13/12/1997 P. 0019 - 0024

(c) The following Notes Q and R are added to the Foreword:

'Note Q:

The classification as a carcinogen need not apply if it can be shown that the substance fulfils one of the following conditions:

- a short-term biopersistence test by inhalation has shown that the fibres longer than 20 μ m have a weighted half life less than 10 days,
- or a short-term biopersistence test by intratracheal instillation has shown that the fibres longer than 20 μ m have a weighted half life less than 40 days, or
- an appropriate intra-peritoneal test has shown no evidence of excess carcinogenicity, or
- absence of relevant pathogenicity or neoplastic changes in a suitable long term inhalation test.

Note R:

The classification as a carcinogen need not apply to fibres with a length weighted geometric mean diameter less two standard errors greater than 6 μm .’;

which should be read together with this quote from the most recent large fibre inhalation study using rats:

(Bernstein DM, Rogers R, Smith P: The Biopersistence of Canadian Chrysotile Asbestos following Inhalation. *Inhalation Toxicology*, 2003;15:1247–1274). concluded:

“Chrysotile was found to be rapidly removed from the lung. Fibers longer than 20 μm were cleared with $T_{1/2} = 16$ days, most likely by dissolution and disintegration into shorter fibers. The shorter fibers were also rapidly cleared from the lung, with fibers 5–20 μm clearing even faster ($T_{1/2} = 29.4$ days) than those <5 μm in length. The fibers <5 μm in length cleared at a rate ($T_{1/2} = 107$ days) that is within the range of clearance for insoluble nuisance dusts.”

A very marginal situation indeed!

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