5. Asbestos: from 'magic' to malevolent mineral

David Gee and Morris Greenberg

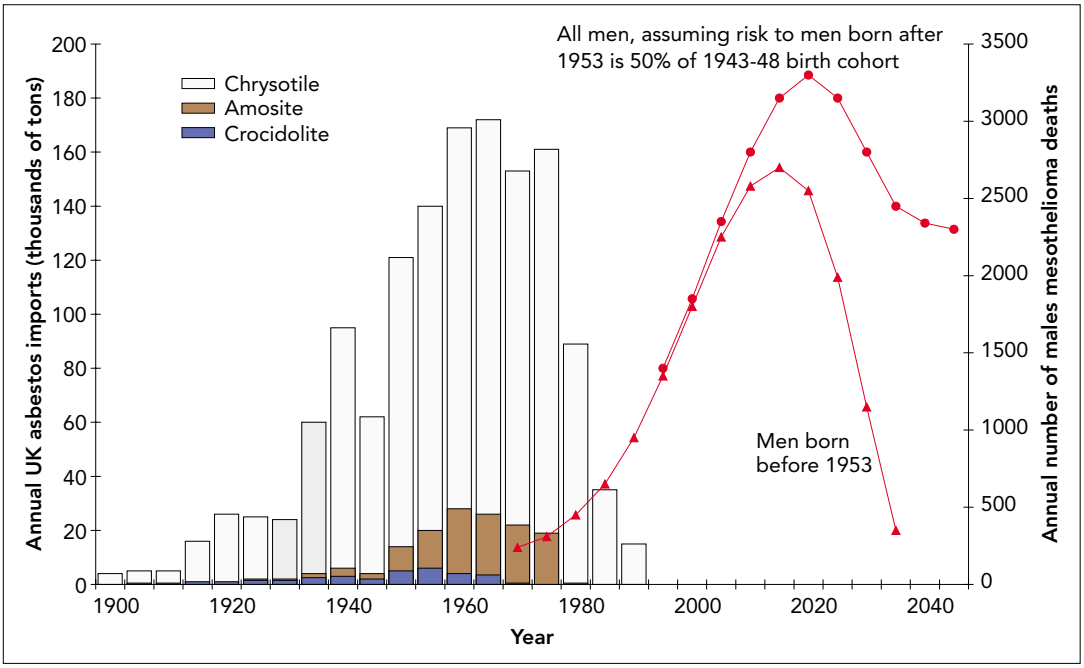
'Looking back in the light of present knowledge, it is impossible not to feel that opportunities for discovery and prevention of asbestos disease were badly missed.' Thomas Legge, ex Chief Medical Inspector of Factories, in *Industrial maladies*, 1934.

5.1. Introduction

On 20 May 2000, the family of a senior UK hospital surgeon was awarded GBP 1.15 million in compensation for his death, at 47, from the asbestos cancer, mesothelioma. The disease was caused by exposure to 'blue' asbestos dust from damaged pipe insulation which was present in the communication tunnels under Middlesex Hospital, London, where the surgeon worked for four years as a student and trainee, during the period 1966–73 (*British Medical Journal*, 2000). The main cause of mesothelioma is asbestos. It is now estimated that some 250 000 cases of mesothelioma, which is normally fatal within one year, will occur in the European Union (EU) over the next 35 years (Peto, 1999). As asbestos is also a cause of lung cancer, the total disease burden could be around 250 000–400 000 deaths, including cases of the lung disease, asbestosis, which was the first disease to be associated with asbestos exposure. Figure 5.1. shows the peak of asbestos imports into the United Kingdom being followed some 50–60 years later by the estimated peak of mesotheliomas.

Figure 5.1. **UK asbestos imports and predicted mesothelioma deaths**

Source: Peto, 1999



Ninety years before this environmental exposure in the London hospital occurred, a new global public health hazard was born when mining for chrysotile ('white') asbestos began in Thetford, Canada, in 1879. Some years later, two other types of asbestos, 'blue' (crocidolite) and 'brown' (amosite) came to be mined in Australia, Russia, South Africa and other countries, and the annual production of all types of asbestos worldwide grew to 2 million tonnes in 1998. Imports into the EU peaked in the mid-1970s and remained above 800 000 tonnes a year until 1980, falling to 100 000 tonnes in 1993.

Today, a substantial legacy of health and contamination costs has been left for both mining and user countries, and asbestos use is continuing, now largely in developing countries.

The focus of this chapter is primarily on the United Kingdom but the histories of asbestos have been similar in France, Germany, Italy, Scandinavia and the United States (Castleman, 1996), as well as in the main mining countries of Australia, Canada, Russia and South Africa. These histories are now being repeated, albeit with some differences, in Asia, Africa and South America.

5.2. The first 'early warnings' of asbestosis and some responses

Within 20 years of the start-up of asbestos mining, over 100 products made from the 'magic mineral' had been developed, but reports of serious disease had also begun to appear.

The earliest account of the health hazard of working with asbestos was provided by Lucy Deane, one of the first Women Inspectors of Factories in the UK. Writing in 1898, Deane included asbestos work as one of the four dusty occupations which came under observation that year, 'on account of their easily demonstrated danger to the health of workers and because of ascertained cases of injury to bronchial tubes and lungs medically attributed to the employment of the sufferer'.

She went on to observe that: 'the evil effects of asbestos dust have also instigated a microscopic examination of the mineral dust by HM Medical Inspector. Clearly revealed was the sharp glass-like jagged nature of the particles, and where they are allowed to rise and to remain suspended in the air of the room in any quantity, the effects have been found to be injurious as might have been expected.' (Deane, 1898)

Two similar observations by Women Inspectors followed in 1909 and 1910. They appeared in the annual reports of HM Chief Inspector of Factories, which were widely circulated amongst policy-makers and politicians.

The observations of these laywomen might not have been categorised as 'expert opinion' but they were competent observers whose discussion of occupational disease would have done credit to a medical scientist. Their reports were not refuted but simply ignored.

One year after Lucy Deane's report, Dr Montague Murray of Charing Cross Hospital, London, saw the first reported case of lung disease attributed to inhaled asbestos dust in a 33-year-old man. In Murray's words: 'He had been at work some fourteen years, the first ten of which he was in what was called the carding room, which he said was the most risky part of the work. He volunteered the statement that of the ten people who were working in the room when he went into it, he was the only survivor. I have no evidence except his word for that. He said they all died somewhere about thirty years of age.' (Murray, 1906)

This observation was brought to the attention of the UK government inquiry into compensation for industrial diseases in 1906. In the same year, a French Factory Inspector reported some 50 deaths amongst female asbestos textile workers (Auribault, 1906). This report dealt with the nature of asbestos, its processing and uses, safety and health hazards in the spinning and weaving processes, and designs for apparatus to capture dust at source. It too was largely ignored, but it was the French ban on asbestos, some 90 years later, which led to the high-profile case at the World Trade Organization (WTO) in 1999, discussed below.

The French report provided confirmation of the earlier observations of the British Women Inspectors. However, the 1906 British government inquiry did not include asbestos as a cause of industrial disease. Dr Murray had stated in evidence: 'one hears, generally speaking, that considerable trouble is now taken to prevent the inhalation of the dust, so that the disease is not so likely to occur as heretofore.' (Murray, 1906)

This may have influenced the committee. However, no attempt was made to check on the truth of Dr Murray's patient's claim about the deaths of nine fellow workers. Nor were the surviving workers at that factory investigated, despite the proposals from Lucy Deane about the kinds of mortality statistics that would be helpful.

Dr Murray's view that 'no evidence of harm' is the same as, 'there is evidence of no harm', is an early example of a common fallacy that has inhibited the identification of many dangerous substances which were initially considered to be harmless ('false negatives').

Other evidence about the hazards of asbestos was noted in workers in 1910 (Collis, 1911)

and in pioneering dust experiments with rats in 1911 (Merewether and Price, 1930), and this was later considered to have been 'reasonable grounds for suspicion that the inhalation of much asbestos dust was to some extent harmful', such that the Factory Department pressed for the installation of exhaust ventilation in the dusty processes (Merewether, 1933). However, subsequent Factory Department inquiries in 1912 and 1917 found insufficient evidence to justify further action. Meanwhile in the United States and Canada, insurance companies had seen enough proof of asbestos disease by 1918 to decline insurance cover for asbestos workers 'due to the assumed injurious conditions in the industry' (Hoffman, 1918). Unfortunately, this early precautionary action was later forgotten, such that asbestos costs to US insurers became hugely damaging in the 1990s.

In 1924 in Rochdale, home of the Turner Brothers asbestos factory since 1880, was the first inquest and pathological examination of an asbestos worker. Nellie Kershaw was diagnosed as having died of asbestos poisoning by her local doctor, Dr Joss, who observed that he saw 10–12 such cases a year. His view was corroborated by pathologist Dr W. Cooke, who wrote the case up in the medical literature (Cooke, 1924 and 1927). In Leeds, where another Turner Brothers factory was situated, a local doctor had found enough asbestos cases to produce a doctoral thesis (Grieve, 1927). By 1930 there had been at least 12 deaths amongst workers from these two factories with asbestosis cited as the cause or partial cause (Tweedale, 2000). In some cases tuberculosis, heart failure and pneumonia complicated the diagnosis, as they did for the next few decades.

However, the combination of at least some of this evidence with two other reports in the medical literature in 1928 (Simpson, 1928; Seiler, 1928), including four cases from South Africa, was sufficient to prompt a major government inquiry into the effects of asbestos dust by Dr Merewether, Medical Inspector of Factories, and C. W. Price, a Factory Inspector and pioneer of dust monitoring and control. It included the first health study of asbestos workers and found that 66 % of those employed for 20 years or more suffered from asbestosis, compared to none of those employed for less than four years, with an average of 25 % for the 363 workers studied (Merewether and Price, 1930). This was probably an underestimate, as only current workers were examined, excluding those who had left employment through ill health. However, these results led, in 1931, to the first asbestos dust control regulations, medical surveillance and compensation arrangements in the world. These remained largely unaltered (but also unenforced) until 1969, when new asbestos regulations were introduced in the United Kingdom.

5.3. Early warnings on asbestos cancers

In 1932 in a report to the Trades Union Congress (TUC), a freelance investigator, Ronald Tage, drew attention to three asbestosis cases from the Cape Asbestos Company in Barking, London, that were complicated by cancer (Greenberg, 1993). Reports of lung cancers being associated with asbestos appeared in the US, German and UK medical literature in the 1930s and 1940s (Lynch and Smith, 1935; Gloyne, 1935; Wedler, 1943; Heuper, 1942) including the 1938 Report of the Chief Inspector of Factories. In 1938, when lung cancer was generally much less prevalent, the German authorities were persuaded that the association was causal, and asbestos lung cancer was made a compensatable industrial disease in 1943. (Decades later the complication of smoking-induced lung cancer made the link with asbestos that much harder to prove.)

A high rate of lung cancer found at autopsy in asbestosis cases was reported in the Chief Inspector of Factories' Annual Report in 1949 and industry had two unpublished US reports of an excess of respiratory cancers in mice (Scheper, 1995). Three in-house investigations of cancer mortality in the Rochdale district had failed to find evidence of lung cancer in asbestos workers (Knox, 1952 and 1964), but the company doctor admitted that his knowledge of statistics was 'nil' (Tweedale, 2000, p. 148). In 1953, Turner Brothers asked Richard Doll, an independent epidemiologist, to study the mortality of Rochdale asbestos workers. He found a lung cancer risk in those who had been exposed to asbestos for 20 years or more which was 10 times that expected in the general population. Despite attempts by the Turner directors to suppress these findings they were published in the medical literature (Doll, 1955). However, it was to be another 30 years before the government accepted lung cancer from asbestos as a

compensatable industrial disease, and then only if it was accompanied by asbestosis. This was partly because future studies were to be increasingly complicated by the rising trends of lung cancer caused by smoking, which Doll had also discovered in a study of British doctors in 1955.

Later studies of asbestos workers showed that the combination of the two carcinogens, cigarette smoke and asbestos, multiplied the risks of lung cancer. Asbestos alone increased the lung cancer risk 5-fold, and smoking alone increased the risk 10-fold, but the two together produced not 15 times the risk (an additive effect) but over 50 times the risk, a multiplicative or synergistic effect. (Hammond, 1979). Smoking and radiation from uranium and other mining has a similarly synergistic effect in radiation exposed workers (Archer, 1973).

As with all other human studies of asbestos exposure, there was only a relatively small number of workers who had been working in the 'new conditions' of improved dust control for the 20–25 years before lung cancer could appear, so it was not possible to say what the risks were in 1955 until many more years had passed, when again, in conditions of improving dust control, it was impossible to know what the risks could be. This problem, which might be called the 'latency lacuna', characteristic of all long-latent-period hazards under conditions of technological change, is a major reason why preventative action is often too late.

5.4. Early, devastating warnings about mesothelioma cancer

Cases of mesothelioma, a normally very rare cancer of the lining of the chest or abdomen, had been observed in association with asbestos exposure in the 1940s and 1950s, but it was not until 1955 that Dr Sleggs, a local doctor in South Africa, noted a number of these unusual cancers at the centre of the asbestos mining areas and sent some to a pathologist, Dr Wagner. The association with asbestos was made and they toured the mining areas trying to reconstruct the history of asbestos exposure of those who had died by talking to colleagues and families. Out of 47 cases of mesothelioma, they found earlier asbestos exposure in all but two, and many of them were environmental cases including children exposed when playing on waste dumps. They published their findings in 1960 (Wagner *et al.*, 1960).

This was devastating news because the exposure needed to cause mesothelioma seemed to be a matter of months only. In contrast, most lung cancer and asbestosis cases seem to need 10 or more years of exposure to asbestos dust. The average latent period between first exposure and the mesothelioma cancer appearing was about 40 years, in contrast to the 20–25 years for lung cancer.

Wagner's paper provided evidence of a very strong association between asbestos and mesothelioma, but by 1964 most experts accepted that the relationship was causal, based mainly on the studies of Dr Selikoff in the United States and Dr Newhouse in the United Kingdom. Both worked independently of the industry, using case data from unions and hospital records respectively.

Selikoff had observed that 15 out of 17 patients from the same asbestos manufacturing plant had asbestos diseases, but as he was refused access to company records he used trade union records to show that the users of asbestos, such as insulation workers, were at even greater risk than manufacturing workers: of the 392 workers examined with 20 years or more asbestos exposure, 339 had asbestosis. The lung cancer rate was seven times normal, and a number had mesothelioma (Selikoff *et al.*, 1964). The excess of lung cancer only became statistically clear after 25 years of follow-up of workers, illustrating one of the serious limitations of so called 'negative' cancer studies, which is that the power of such studies to detect long-latent-period cancers can be very low unless some 20–30 years of follow-up has been possible.

Selikoff was to be described as a 'disturbing sore thumb' by an industry representative from the Asbestos Textile Institute (Tweedale, 2000, p. 183, footnote 17). This was a similar sentiment to that expressed by the ex Chief Medical Inspector of Factories, Dr Legge, writing in his then capacity as Medical Adviser to the TUC in 1932 about Ronald Tage, whom he said the TUC could be 'quit of' by paying him a small fee (Greenberg, 1993).

The practice of attacking the purveyors of news about hazards had been well illustrated by Ibsen in his play, *An enemy of the people* (1882), in which the local doctor notices a health hazard which, if fully recognised,

would threaten the economy of the local town. He descends from public hero to public enemy as the economic implications of his observations come to be realised by the mayor, the media and most of the citizens.

Newhouse used the long-term pathology records collected by the London Hospital between 1917 and 1964 to show that, of 76 mesothelioma cases, over 50 % had occupational or domestic exposure (lived in the house of an asbestos worker), whilst of the others, one third lived within half a mile of the Cape asbestos factory (Newhouse and Thompson, 1965). It was to be 30 years later that children exposed to asbestos in the neighbourhood of factories and who later developed mesothelioma became the first successful environmental exposure cases against Turner Brothers in the United Kingdom (Tweedale, 2000, p. 272).

Both Newhouse and Selikoff presented their findings at a conference in New York in October 1964, organised by the New York Academy of Sciences. A study of Doll's group of workers from the regulated areas of the Rochdale factory was presented as supporting the view that 'it is possible that the specific occupational hazards to life have been completely eliminated', perhaps another example of the 'latency lacuna' (Knox *et al.*, 1965) But neither Selikoff nor the UK Factory Department found such evidence of falling disease rates, mainly because they were including severe cases of dust exposure amongst the users, not just the manufacturers of asbestos, for whom dust conditions were relatively better, at least in the regulated factory production areas.

This failure to appreciate the 'worst case' asbestos exposure scenarios was part of the reason for the delayed and inadequate responses to asbestos. Julian Peto, asbestos cancer researcher, has described the focus of asbestos cancer studies on factories, rather than users, as a 'stupid mistake' (Peto, 1998).

This view was shared by a former director of the world's biggest asbestos company, Johns Manville, when reviewing why, although still profitable, it filed for bankruptcy in 1982 as a means of dealing with asbestos pollution claims. He argued that medical research, assiduous communication, insistent warnings and a rigorous dust reduction programme 'could have saved lives and would probably have saved the stockholders, the industry and, for that matter, the product' (Sells, 1994).

5.5. Actions and inactions by regulatory authorities and others

The asbestos regulations of 1931 were only partially enforced, there being only two prosecutions between 1931 and 1968 (Dalton, 1979). Their focus on just parts of the manufacturing process meant that the riskier user activities were neglected. However, the issue of dangerous asbestos was not neglected.

From 1964 to 1975 the media in both the United States and the United Kingdom kept asbestos high on the political agenda (*Sunday Times*, 1965). The ITV programme *The World in Action* in 1971, and the BBC *Horizon* in 1975, about conditions at Cape's Acre Mill asbestos plant in Yorkshire, United Kingdom, helped to initiate action by authorities, such as a Parliamentary Ombudsman Report into asbestos regulation enforcement at the factory. This report was initiated by local MP Max Madden, who lodged an official complaint against non-enforcement of the asbestos regulation of 1931. The report was very critical of the Factory Inspectors, and the government responded by appointing a government inquiry, the Simpson Committee, in 1976. Meanwhile, the 1931 asbestos regulations had been updated in 1969, and a limit for factory asbestos dust exposure of 2 million fibres per cubic metre (m^3) of air was to be gradually introduced.

Unfortunately this 'hygiene standard' did not include consideration of the lung or mesothelioma cancer hazards. It was later to be strongly criticised and associated with high asbestosis levels (one worker in 10 would get the disease) by Julian Peto in evidence to the Simpson inquiry (Peto, 1978).

The Simpson Report came out in 1979 with the following recommendations: a ban on 'blue' asbestos, which had already been withdrawn by the industry; a ban on insulation spraying, which was also largely defunct by then; contractors were to be licensed for the removal of asbestos; and the asbestos limit was to be reduced to 1 million fibres/m^3 (or 1 fibre per millilitre) by 1980 for 'white' asbestos, with a target of 0.5 million fibres/m^3 (0.5 f/ml) for 'brown' asbestos, which was considered to be more hazardous than white. An asbestos fibre that

is visible is about the diameter of a human hair, 40 microns, but it is composed of a bundle of about 2 million fibrils that can be released by abrasion or physiological processes in the body (Selikoff and Lee, 1978). Electron microscopy is needed to accurately monitor the presence of such fibrils in air or tissues.

There was, and still is, scientific controversy about the relative cancer and asbestosis potencies of the three types of asbestos, with white often being regarded as less hazardous than blue or brown. By 1986 the World Health Organization's International Agency for Research on Cancer (IARC) had concluded that all three types were carcinogenic and, as with other carcinogens, there was no known safe level of exposure to any of them.

There was no similar hygiene standard for the public's protection from airborne asbestos dust until the late 1980s, when the lowest limit detectable by the prevailing dust monitoring method, optical microscopy, was recommended by the UK Health and Safety Executive (100 000 fibres/m^3 or 0.1 f/ml).

In 1982, Yorkshire TV screened a two-hour documentary at prime viewing time, featuring Alice Jefferson, a 47-year-old who had contracted mesothelioma when working for a few months at Cape's Acre Mill asbestos plant. *Alice, a Fight for Life* had an immediate impact, even though some, like Sir Richard Doll, criticised the programme for being unscientific and emotional. The government responded to the programme by implementing the Simpson inquiry recommendations and, in 1984, by introducing the asbestos licensing regulations and a further lowering of the exposure limits to 0.5 million fibres/m^3 (0.5 f/ml) for white and 0.2 million fibres/m^3 (0.2 f/ml) for brown asbestos. A voluntary labelling scheme was introduced for some uses.

Pressure for further improvements continued to come from local MPs, some trade unions and people representing victims, such as asbestos widow, Nancy Tait. She helped reveal that the Turner Brothers' asbestos compensation arrangements, such as GBP 1 a week for the widows of workers with asbestosis, had been largely unchanged since the 1930s. Her work helped to force Turner Brothers to improve their compensation awards.

New regulations were introduced in 1987 and tightened further in 1989. In 1998 the government adopted a ban on all forms of asbestos, which was implemented the following year, along with an EU ban, which is to be implemented by Member States by 2005. Canada filed a trade barriers complaint against the French and EU ban at WTO, but this was rejected by the WTO Disputes Panel. Canada appealed against this ruling to the WTO Appellate Body, which found in favour of France and the EU (see Box 5.1.)

Box 5.1.
WTO upholds French and EU ban on asbestos

In 1997 France banned all forms of asbestos fibres and products in order to protect the health of workers and consumers. Existing 'white' asbestos products could be exempt on an exceptional, temporary and annually reviewed basis, if no effective substitute materials were available that posed a lower health risk to workers handling them. Canada objected to this ban at the WTO but the WTO found in favour of France in September 2000 (WTO, 2000). Canada appealed to the Appellate Body the WTO and the EU cross-appealed to uphold the main findings of the panel and to seek correction of some 'errors' of the panel's interpretations and conclusions. The US cross-appealed against the panel's judgement that glass fibres were as carcinogenic as asbestos. The Apellate Body issued a report in early 2001 (WTO, 2001), out of which a number of main points arose, which also have implications for other hazardous agents:

- all forms of asbestos ('white', 'brown' and 'blue') are carcinogenic;
- there is no known threshold of safety for this carcinogen;
- the risk from 'white' asbestos in products is based on evidence which 'tends to show' a risk rather than not;
- workers handling asbestos products, such as building and brake lining workers, are at risk from asbestos exposure;
- there is no WTO requirement for countries to provide quantitative risk assessment data: qualitative evidence is sufficient;
- countries can base their health/ environment/ animal welfare measures on qualified and respected scientific opinions held by only a minority of scientists: 'a Member is not obliged, in setting health policy, automatically to follow what, at a given time, may constitute a majority scientific opinion' (p. 64). This means that a WTO Panel need not necessarily reach its decision, on the scientific evidence, based on a 'preponderant weight of the evidence', but on a lower level of proof;
- the efficiency of the 'controlled use' of asbestos products was not demonstrated and the residual risk to the workers would still be significant; this risk management option could not be relied on to protect workers' health, and therefore was not a reasonable 'alternative' measure to the asbestos ban;
- in determining whether asbestos substitutes such as glass fibre were 'like' products, four criteria have been developed by WTO, including the properties and end uses of a substance, and the tastes and habits of consumers. Based on these criteria, the Appellate Body found that the panel had erred in finding that glass fibre products were 'like' products: they were not, principally because they were not as carcinogenic.

The WTO procedures for dealing with the kind of scientific and technological complexity involved in asbestos and other health and environmental hazards has been criticised by one of the scientific advisers involved in the WTO asbestos case (Castleman, 2001).

Meanwhile the annual UK cancer death rate from mesothelioma and lung cancer from asbestos is estimated by the Health and Safety Commission (Health and Safety Commission, 1994–95) to be around 3 000 deaths per year and rising (see Figure 5.1.). Despite huge amounts of research, many issues of biological mechanisms and dose-response relationships remain unclear, illustrating the limited relevance of more research to disease.

5.6. The costs and benefits of actions and inactions

It is beyond the scope of this case study to provide a detailed evaluation of the full costs and benefits of the asbestos story (see Castleman, 1996, p. 8–9). However, a few illustrative figures will indicate the dimensions of such an evaluation. At a company level, Turner Brothers made arrangements in 1994 to pay up to GBP 1 billion in asbestos claims. The insurance underwriters Lloyd's of London faced near collapse in the early 1990s from US pollution claims, many of which were for asbestos health compensation and clean-up costs.

If lives are valued at EUR 1 million each, which is common in transport studies, then the costs of the estimated 400 000 European asbestos cancer deaths expected over the next few decades is EUR 400 billion. The human costs in terms of suffering are not calculable. Removing asbestos from buildings safely at the end of their life will cost further billions. Earlier actions to reduce asbestos exposure could have saved many of these costs.

A Dutch illustration of some of the potential savings from earlier risk reduction actions has estimated that a ban in 1965, after the mesothelioma evidence had been widely accepted, instead of in 1993, would have saved the country some 34 000 victims and NLG 41 billion in building and compensation costs. This is compared to the 52 600 victims and NLG 67 billion guilders in costs expected over the period 1969– 2030, estimated by the Dutch Ministry of Health and Social Security (Heerings, 1999). In the United States, asbestos compensation settlements reached USD 2 billion, with Lloyds syndicates paying around half of that.

On the other hand, asbestos has brought some benefits, including employment. In 1919 it was estimated that fires in the world's theatres in the 1870s and 1880s caused 2 216 deaths, 95 % of which could have been saved by asbestos fire insulation, it was claimed (Summers, 1919). Asbestos boiler insulation saved energy, and asbestos brake linings saved lives, though the extra vehicle speeds it allowed complicates the picture. The *Lancet*, a UK medical journal, argued in 1967 that 'it would be ludicrous to outlaw this valuable and often irreplaceable material in all circumstances (as) asbestos can save more lives than it can possibly endanger' (*Lancet*, 1967). Apart from their gross underestimate of the health impacts of asbestos, for which their expertise was at least relevant, the replaceability of asbestos is a technological and economic question which doctors are not well qualified to judge. Little evidence was presented to substantiate their argument that asbestos was 'irreplaceable'.

Substitutes for most uses of asbestos were available by the 1970s and, in some cases, much earlier – many US oil refineries were insulated with mineral wool in the 1940s and 1950s (Castleman, 1996, pp. 456–457). The slow spread of asbestos substitutes was partly because asbestos industry cartels worked to inhibit their spread (Castleman, 1996, pp. 34– 38), and partly because the market price of asbestos was very low compared to its full production, health and environmental costs. This failure of market prices to reflect full environmental and health costs is the common cause of delay in replacing hazardous materials.

Many jobs, much profit and high dividends were generated by asbestos. Turner Brothers' profits rose strongly after 1947 and peaked at almost GBP 9 million a year in 1965 (Tweedale, 2000, p. 9). These profits suffered little from the ill health and contamination costs of asbestos, which were 'externalised' onto workers with disease, their families, the health service, insurance carriers and building owners.

An often ignored but significant non-financial benefit of asbestos compensation trials is that they frequently uncover many of the contradictions between company words

on asbestos and their actions to reduce hazards (Castleman, 1996).

5.7. What are the lessons of the asbestos story?

Asbestos offers many lessons that are relevant to numerous other agents or activities that have long-term hazardous impacts.

1. The experiences of victims, lay people and 'competent observers', such as factory inspectors and family doctors, should be taken seriously by governmental and other authorities, and followed up by appropriate investigations. They can anticipate the views of scientific experts, sometimes by many years.

2. The early warnings of 1898–1906 in the United Kingdom and France were not followed up by the kind of long-term medical and dust exposure surveys of workers that would have been possible at the time, and which would have helped strengthen the case for tighter controls on dust levels. Even now, leading asbestos epidemiologists can conclude: 'It is unfortunate that the evolution of the epidemic of asbestos-induced mesothelioma, which far exceeds the combined effects of all other known occupational industrial carcinogens, cannot be adequately monitored.' (Peto, 1999)

   Long-term environmental and health monitoring rarely meets the short-term needs of anyone, thus requiring particular institutional arrangements if it is to meet society's long-term needs.

3. The laws on prevention and compensation introduced in the United Kingdom in 1931-32 were not well implemented, and the sanctions were trivial, a pattern that was repeated down the long history of asbestos.

4. If early warnings had been heeded, and better control measures adopted, either before 1930 as Dr Legge, Chief Medical Inspector of Factories, and others (Greenberg, 1994; Bartrip, 1931) have noted, or in the 1950s and 1960s, when new cancer hazards emerged and economic circumstances were good, then much tragic loss would have been avoided. Action to curb asbestosis prior to the discovery of the cancers would at least have minimised the impact of these later 'surprises'.

   More strategically, tighter regulation of asbestos would have raised its market price to capture more of its costs of production and use, thereby stimulating the innovation that belatedly led to better and often cheaper substitutes, as well as to improved engine and building designs that generate, at source, less waste heat.

5. Economic factors played a key role as in other cases of worker, public and environmental hazards. These include the employers' need for profits and the workers' need for jobs, which can together produce an alliance which may not be in the long-term interests of workers or society. The greater the size of the 'external' cost of harm (damage costs not borne by the companies), the greater the chance that these diverging private and social costs will inhibit preventative action. Only when full damage costs, including health, building maintenance and site contamination costs, are borne by the polluters via the 'polluter pays' principle, and through liability provisions, regulations, taxes, etc., can the private and social costs of economic activity be brought closer together, thus allowing the market place to operate more efficiently. Penalties on wrongdoing by employers also need to be commensurate with the costs they inflict on others, if private and social costs and benefits are to be more closely aligned. But this is not easy. It is very difficult for governments to overcome powerful economic interests that usually operate on the same short timescales as most politicians, and to implement decisions that are in the best, longer-term interests of society, if they are perceived as imposing short-term costs on powerful groups. Again, appropriate institutional arrangements are needed to help meet society's long-term interests: a 'governance' issue that is taken up in the final chapters of this report.

6. One of the main reasons for the failure to implement control measures was the view that 'current exposures to asbestos dust are so much lower than past exposures and should therefore be safe', a view offered to the UK committee of inquiry into compensation for industrial diseases

by Dr Murray in 1906, and repeated by many others ever since. As there is a 10–40-year latent period between asbestos exposure and the diseases it causes, by the time that evidence of 'today's' exposure risks becomes available, many years of generally reducing dust levels have ensued, making it once again possible to say that risks in the new 'today' are much less than in the past, or non-existent. The point cannot be proved decisively one way or the other until another 20–40 years have passed. This 'latency lacuna', which is common to all long-latent-period hazards, is an illustration of the common error of assuming that 'absence of evidence of harm' means 'evidence of absence of harm'. It does not.

In the absence of good evidence that today's exposures to carcinogens are safe, it is wiser to apply the precautionary principle, and assume they are unsafe, especially if the disease (or ecological impacts) from higher exposures have no known threshold of exposure below which there are no effects.

This is a key lesson that is relevant to all long-latent-period hazards. The particular preventative measures that would then be required would depend on the proportionality principle — the expected benefits from prevention, including any 'secondary benefits', would need to be significant in relation to the costs of achieving such prevention.

This more precautionary approach to uncertainty and ignorance would also involve switching the current bias within normal scientific methods away from avoiding 'false positives' (with its associated bias of producing 'false negatives' such as asbestos) towards producing a better balance between false positives and false negatives. This would increase the chances of generating the costs of restricting a substance or activity that might later turn out to be safe. However, the asbestos case strongly suggests that society would gain overall from a more ethically acceptable and economically efficient balance between generating false positives and false negatives.

7. Implementation of preventative measures was also inhibited by the healthy survivors fallacy. This needs to be widely communicated and avoided as it gives rise to a general but false reassurance of safety, as it does with the general hazard of smoking. It was first described in relation to asbestos by Lucy Deane in 1898:

'Even when the evil reaches such grave proportions as to be capable of easy and tragic proof... there is always a certain proportion of 'old workers' — the survivors of their mates — who are found in every unhealthy industry and who... appear to thrive on their unhealthy calling. In less obvious unhealthy conditions the only convincing proof of actual injury, *viz.*, reliable comparative statistics of mortality, or of health standards, is practically unattainable in the case of any given factory, or at any rate with the time and opportunity at present at our disposal.' (Deane, 1898).

This argument has been used throughout the history of asbestos. For example, Dr Knox, the Turner Brothers' UK company doctor, on visiting the Canadian asbestos mines in 1952, said: 'I am assured that many workers over 70 years of age are still employed and are active and vigorous.' (Greenberg, 2000) This view was also presented to one of the authors of this case study (DG) when he visited UK asbestos plants in the 1980s as the union health and safety adviser. Workers pointed to the retired workers who had worked more than 20–30 years or so in the factory without much harm, and who were able to turn up to the annual pensioners' party. Such pensioners were cited as proof of the low or absent risks of asbestos. This could be called the 'pensioners' party fallacy', as it was the workers who did not make it to the party who provided the proof of harm, and their deaths, or illnesses, made them relatively invisible to current workers. As Deane observed, healthy survivors needed to be related to non-survivors via appropriately analysed mortality statistics.

8. It seems necessary to establish speedy, affordable and transparent compensation arrangements, based on agreed liabilities, as soon as any harmful effects become known, so as to both increase the incentives to prevent further harm and to improve the chances of recording accurate exposure histories.

Elements of such anticipatory compensation arrangements were established in the early days of the nuclear industry, when the state in many countries took on future liabilities for nuclear accidents, at least up to certain limits (for example the UK Nuclear Installations Act, 1965). A unique example seems to be the radiation-induced cancer compensation scheme for workers at British Nuclear Fuels (see the chapter on Radiation).

9. Views should be taken from a wide range of all relevant disciplines and the 'ignorant expert' should be curbed. Specialists in one discipline, for example medicine, provided 'expert' opinions about other disciplines such as dust monitoring and control (occupational hygiene and ventilation engineering) or asbestos substitutes availability. These opinions were often mistaken but went largely unchallenged, and this contributed to misplaced complacency (Greenberg, 2000).

10. It is necessary to anticipate 'surprises' and take care with substitutes. If asbestos substitutes reproduce the same physical form as asbestos – long, respirable (< 3 microns in diameter) and durable fibres — it is likely that they too will be carcinogenic (Roller and Pott, 1998), as was predicted by the UK Health and Safety Executive in 1979, and later confirmed by the IARC for some forms of synthetic mineral fibres. However, mineral wool and glass fibre appear to be much less hazardous than asbestos, and they can be manufactured to be good enough for insulation but not as thin, or durable enough in human tissue, to be carcinogenic. 'Clean' production and user techniques that minimise exposures to atmosphere, whether occupational or environmental, via 'closed loop' and eco-efficient systems, are therefore essential with whatever materials are being used. This then minimises the size of any future 'surprise' impacts from substitutes, which is an important benefit of applying the precautionary principle.

Asbestos: early warnings and actions Table 5.1.

1898	UK Factory Inspector Lucy Deane warns of harmful and 'evil' effects of asbestos dust
1906	French factory report of 50 deaths in female asbestos textile workers and recommendation of controls
1911	'Reasonable grounds' for suspicion, from experiments with rats, that asbestos dust is harmful
1911 and 1917	UK Factory Department finds insufficient evidence to justify further actions
1918	US insurers refuse cover to asbestos workers due to assumptions about injurious conditions in the industry
1930	UK Merewether Report finds 66 % of long-term workers in Rochdale factory with asbestosis
1931	UK Asbestos Regulations specify dust control in manufacturing only and compensation for asbestosis, but this is poorly implemented
1935–49	Lung cancer cases reported in asbestos manufacturing workers
1955	Doll establishes high lung cancer risk in Rochdale asbestos workers
1959–60	Mesothelioma cancer in workers and public identified in South Africa
1962/64	Mesothelioma cancer identified in asbestos workers, in neighbourhood 'bystanders' and in relatives, in the United Kingdom and the United States, amongst others
1969	UK Asbestos Regulations improve controls, but ignore users and cancers
1982-9	UK media, trade union and other pressure provokes tightening of asbestos controls on users and producers, and stimulates substitutes.
1998–99	EU and France ban all forms of asbestos
2000–01	WTO upholds EU/French bans against Canadian appeal

Source: EEA

5.8. References

Acheson, E.D., and Gardner, M.J., 1983 'Asbestos: The Control Limit for Asbestos', Health & Safety Executive, HMSO, London.

Archer, V.E., et al, 1973, 'Uranium Mining and Cigarette Smoking Effects in Man', *J.Occ.Med.,* 15, 204.

Auribault, M., 1906. 'Sur l'hygiene et la securite des ouvriers dans la filature et tissage d'amiante', in *Annual report of the French Labour Inspectorate for 1906.*

Bartrip, P., 1931. 'Too little, too late? The Home Office and the asbestos industry regulations 1931', *Medical History* Vol. 42, October, pp. 421–438.

British Medical Journal, 2000. Vol. 320, 20 May, p. 1358, at http://bmj.com/cgi/full/320/7246/1358/a

Castleman, B., 2001. Draft paper to asbestos conference at London School of Hygiene and Tropical Medicine, 5 June.

Castleman, B. I., 1996. *Asbestos: Medical and legal aspects*, 4th ed., Aspen Law & Business, Englewood Cliffs, NJ.

Collis, E., 1911. *Annual Report of HM Chief Inspector of Factories for 1910*, HMSO, London.

Cooke, W. E., 1924. 'Fibrosis of the lungs due to the inhalation of asbestos dust', *British Medical Journal* Vol. 2, 26 July, p. 147.

Cooke, W. E., 1927. 'Pulmonary asbestosis', *British Medical Journal* Vol. 2, 3 December, pp. 1024–1025.

Dalton, A., 1979. *Asbestos: Killer dust*, British Society for Social Responsibility in Science, London.

Deane, Lucy, 1898. 'Report on the health of workers in asbestos and other dusty trades', in HM Chief Inspector of Factories and Workshops, 1899, *Annual Report for 1898*, pp. 171–172, HMSO London (see also the Annual Reports for 1899 and 1900, p502).

Doll, R., 1955. 'Mortality from lung cancer in asbestos workers', *Brit. J. Industr. Med.* Vol. 12, pp. 81–86.

Gloyne, S. R., 1935. 'Two cases of squamos carcinoma of the lung occurring in asbestosis', *Tubercle* Vol. 17, pp. 5–10.

Greenberg, M., 1993. 'Reginald Tage — a UK prophet: A postscript', *Am. J. Ind. Med.* Vol. 24, pp. 521–524.

Greenberg, M., 1994. 'Knowledge of the health hazard of asbestos prior to the Merewether and Price Report of 1930', *Social History of Medicine*, 07/03/, pp. 493–516.

Greenberg, M., 2000. 'Re call for an international ban on asbestos: Trust me, I'm a doctor', Letter to the editor, *Am. J. Ind. Med.* Vol. 37, pp. 232–234.

Grieve, I. M. D., 1927. 'Asbestosis', MD thesis, University of Edinburgh.

Hammond, E.C., Selikoff, I.J., Seidman, H.,'Asbestos Exposure,Cigarrette smoking and Death Rates,' Annals of New York Academy of Sciences, p 473–490.

Health and Safety Commission, 1994–95, *Health and Safety Statistics* Vol. 55, pp. 148–151.

Heerings, H., 1999. 'Asbestos — deep in the very fibres of society', Contrast Advise study for Greenpeace Netherlands, September, Amersfoort.

Heuper, W. C., 1942 , 'Occupational Tumours and Allied Diseases', Charles C. Thomas, Springfield, Illinois.

Hoffman, F. L., 1918. 'Mortality from respiratory diseases in dusty trades', *Bulletin of the US Bureau of Labor Statistics* Vol. 231, pp. 176–180.

Knox, J. F., 1952. 'Visits to the Thetford Mines, Asbestos, Atlas Works, Keasbey & Mattison Works, Raybestos-Manhattan Works', Report to the management of Turner Brothers Asbestos, Frames 0000 0070 1950–54 in the Chase Manhattan microfilms.

Knox, J. F., 1964. 'Report of a visit to the Thetford Mines, Asbestos and Montreal', Report to the management at Turner Brothers Asbestos, Discovered documents marked 015039-015041.

Knox J. F. *et al.*, (1965) 'Cohort analysis of changes in incidence of bronchial carcinoma in a textile asbestos factory', *Annals of the NY Acad. of Sciences* Vol. 132, December, pp. 527–35.

Lancet, 1967. 17 June, pp. 1311–1312.

Legge, T., 1934, *Industrial Maladies*, Oxford University press, Oxford.

Lynch, K. M. and Smith, W. A., 1935. 'Pulmonary asbestosis 111: Carcinoma of lung in asbestosis-silicosis', *Am. J. Cancer* Vol. 24, pp. 56–64.

Merewether, E. R. A., 1933. 'A memorandum on asbestosis', *Tubercle* Vol. 15, pp. 69–81.

Merewether, E. R. A. and Price, C. W., 1930. *Report on effects of asbestos dust on the lungs and*

dust suppression in the asbestos industry, HMSO, London.

Murray, H. M., 1906. In Departmental Committee on Compensation for Industrial Diseases, 1907, *Minutes of evidence*, p. 127, paras 4076-4104, Cd 3496, HMSO, London.

Newhouse, M. and Thompson, H., 1965. 'Mesothelioma of pleara and pertitoneum following exposure to asbestos in the London area', *Brit. J. Industr. Med.* pp. 261–269.

Peto, J., 1978. 'The hygiene standard for chrysotile asbestos', *Lancet* 4 March, pp. 484–489.

Peto, J., 1998. 'Too little, too late', Interview with John Waite, BBC Radio 4, 15 October, London.

Peto, J., 1999. 'The European mesothelioma epidemic', *B. J. Cancer* Vol. 79, February, pp. 666–672.

Roller, M. and Pott, F., 1998. 'Carcinogenicity of man-made fibres in experimental animals and its relevance for classification of insulation wools', *Eur. J. Oncol.* Vol. 3, No 3, pp. 231–239.

Scheper, G. W. H., 1995. 'Chronology of asbestos cancer discoveries: experimental studies at the Saranac Laboratory', *Am. J. Ind. Med.* Vol. 27, pp. 593–606.

Seiler, H. E., 1928. 'A case of pneumoconiosis', *British Medical Journal* Vol. 2, p. 982.

Selikoff, I. J. *et al.*, 1964. 'Asbestos exposure and neoplasia', *J. Am. Med. Ass.* Vol. 188, pp. 22–26.

Selikoff, I. and Lee, D. H. K., 1978. *Asbestos and disease*, Academic Press, New York.

Sells, B., 1994. 'What asbestos taught me about managing risk', *Harvard Business Review* March/April, pp. 76–89.

Simpson, F. W., 1928. 'Pulmonary asbestosis in South Africa', *British Medical Journal* 1 May, pp. 885–887.

Summers, A. L., 1919. *Asbestos and the asbestos industry.* Cited in Tweedale, P5, fn 10.

Tweedale, G., 2000. *Magic mineral to killer dust: Turner and Newall and the asbestos hazard*, Oxford University Press, Oxford.

Sunday Times, 1965. 'Urgent probe into 'new' killer dust disease', 31 October, London.

Wagner, J. C., Sleggs, C. A. and Marchand, P., 1960. 'Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province', *Brit. J. Indust. Med.* Vol. 17, pp. 260–271.

Wedler, H. W., 1943. 'Über den Lungenkrebs bei Asbesttos', *Dtsch. Arch. Klin. Med.* Vol. 191, pp. 189–209.

WTO, 2000. WT/DST35/R, 18 September.

WTO, 2001. WT/DS135/AB/R, 12 March.