Mineral Dusts are Dangerous: Asbestos and Disease

Gerard Slavin

Abstract. Asbestos minerals have been used from prehistoric times because of their strength, durability and resistance to heat. Widespread use was brought about by the need for insulation in the Steam Age, coupled with the opportune discovery of large mineral deposits in Africa, Russia and America. The main asbestos minerals are chrysotile (white asbestos) and amphiboles (including crocidolite, blue asbestos), which offer the greater health hazard. Initially, environmental control of asbestos dust was poor, and workers in mining and manufacturing industries exposed to high levels of the dusts for many years developed fibrosis of the lungs - asbestosis - leading to respiratory and cardiac failure. Recognition of this sequence led to stringent environmental controls in industries where the risk of asbestos was perceived. Lesser degrees of exposure may be carcinogenic, but only after a prolonged latent period. It was only recognised after decades of use that exposure to asbestos was associated with an increase in lung carcinoma (cancer) and mesothelioma, a highly malignant tumour of the pleura. These tumours occurred in workers not in occupations where the asbestos risk was apparent (and guarded against) but in maintenance workers, plumbers and electricians exposed incidentally to asbestos in their work. It has been estimated that between 1968 and 2050 about 90,000 deaths from mesothelioma will have occurred in Britain; 65,000 of these will be after 2001. Asbestos fibres occur normally in the atmosphere in the western world, but in very small amounts, which are not dangerous. In some Mediterranean countries with ophiolite at outcrop, tremolite occurs in dust, and the cumulative retention of these fibres in the lung is comparable to that in persons occupationally exposed in N W Europe. In these populations, carcinomas and mesotheliomas occur with increased frequency. In Anatolia in three villages, 50% of deaths are attributed to mesothelioma produced by exposure to dust containing erionite, a fibrous zeolite which develops in weathered tuffs.

Asbestos in history

Asbestos is named from the Greek *asbeston*, meaning inextinguishable or unquenchable, and describes not a single mineral, but a number of silicates distinguished by their fibrous structure, flexibility, strength and high resistance to heat and chemicals. These properties ensured the minerals' use from the earliest times. Archaeologists have identified asbestos as a strengthener in earthenware from East Finland aged about 4.5ka. There is evidence of asbestos use in Cyprus during the classical period, for the manufacture of cremation cloths, lamp wicks, hats and shoes. The Egyptians used asbestos as embalming cloths, and the Romans for cremation wrappings.

In early times, there was difficulty in deciding whether asbestos was a stone or vegetable. The geographer Strabo (64BC - 21AD) described quarries on the Greek island of Euboea where *there is found in the earth a stone which is combed like wool, and woven, so that napkins, which when soiled are thrown into the fire and cleaned as in the washing of linen*, and Pliny (77AD) described it as *linus vivum* - durable linen. In post-classical times and throughout the Middle Ages, its origin provoked much speculation and alchemists claimed that asbestos was the hair of a fire resistant salamander (Ross & Nolan, 2003).

Commercial use began in the Ural region of Russia in the early eighteenth century, where the fibres were woven into cloth for gloves, aprons and hats in high-temperature metallurgical shops. In the nineteenth century, Jean Aldini, professor of physics at Bologna University, tailored asbestos cloth into a suit to protect against fire. He exhibited his suit in European capitals, including at the Royal Institution in London, and was commended by Michael Faraday (Murray, 1990).

Asbestiform mineralogy

The word *asbestos* gives rise to confusion for it is often misused as a mineralogical term, whereas it is a commercial-industrial term to describe a mineral habit - *asbestiform* - with distinctive physical properties. In this paper *asbestos* is used as a simple generic term to describe any minerals with the asbestiform habit. To add to the confusion, these belong to two completely separate groups of silicate minerals - serpentine and amphibole; there is a plethora of alternative common and proper names for the individual minerals (Table 1).

Asbestos minerals readily separate into long, thin fibres that are flexible enough to be woven (Fig. 1). They are heat-resistant, chemically inert and good electrical insulators. Properties that distinguish them from non-asbestiform polymorphs include -

- Fibre-like morphology, and dimensions with very small crystal diameter, large aspect ratio and smooth and parallel longitudinal faces:

- Enhanced strength and flexibility; the tensile strength of commercial quality asbestos is about 20-50 times greater than that of the same non-asbestiform minerals:
- Diameter-dependant strength; longitudinally, the unit strength of the fibre increases as its diameter decreases:
- Increased physical and chemical durability compared to non-asbestiform polymorphs (CNHAF, 1984).

Historically the definition of asbestos was based primarily on appearance, but is now augmented with a statement of properties to exclude fibrous minerals (such as the quartz pseudomorphs, including tiger's eye) that break into irregular fragments unrelated to the fibrous structure and which do not have the physical properties of asbestos.

The asbestos minerals

Chrysotile is a member of the serpentine group, and is typically pale green, but is normally white in its fibrous form, which provides its common name as white asbestos. It is a phyllosilicate, recognisable in its formula by the 2:5 ratio of its silicon and oxygen atoms (Table 1). This has a sheet atomic structure, in which a silica layer is joined to a brucite (MgOH) layer. There is an inherent misfit between the octahedral brucite layer and the tetrahedral silica sheet, with consequent mismatching and strain on the crystal lattice. This is partly compensated by tight concentric or spiral curling of the layers and by elongation along the crystallographic *a*-axis - hence creating a fibrous habit from a sheet atomic structure. Each individual chrysotile fibre has an external diameter of 20-40 nm (20-40 x 10^{-9} m), with a central "capillary" space about 2-4 nm in diameter (Roggli & Coin, 2003). Chrysotile fibres may exceed 100 μm in length, and have a curved and curly morphology with splayed ends because of separation of the fibrillar units (Fig. 2). It should not be confused with chrysolite, which is an old name for olivine, and is sometimes still ascribed to yellowish varieties of peridot, the gem-quality olivine.

Asbestos mineral within the serpentine group		
Chrysotile	$Mg_3[Si_2O_5](OH)_4$	***White Asbestos***
Asbestos minerals of the Amphibole group		
Crocidolite	$Na_2Fe_3Fe_2[Si_8O_{22}](OH)_2$	***Blue Asbestos***
Amosite	$(Fe,Mg)_7[Si_8O_{22}](OH)_2$	***Brown Asbestos***
Anthophyllite	$(Mg,Fe)_7[Si_8O_{22}](OH,F)_2$	white, brown
Minor asbestiform minerals in amphibole group		
Fluor-edenite	$NaCa_2Mg_5[AlSi_7O_{22}](F)_2$	white
Tremolite	$Ca_2Mg_5[Si_8O_{22}](OH)_2$	white, pale green
Actinolite	$Ca_2(Mg,Fe)_5[Si_8O_{22}](OH)_2$	dark green

Table 1. *Asbestos mineralogy*

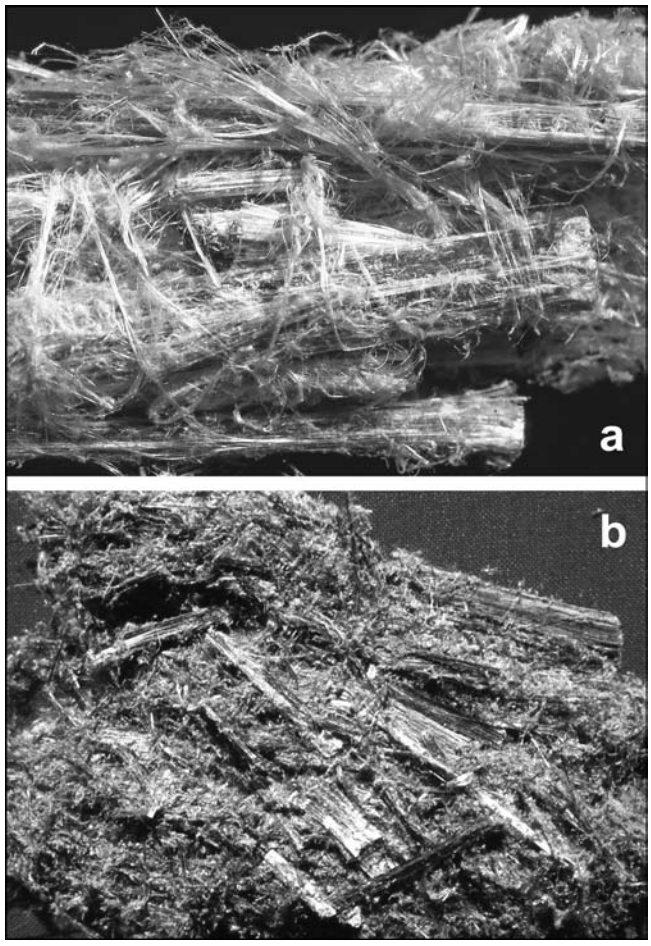

Figure 1. *Asbestos minerals in hand specimens:* ***a*** *- chrysotile:* ***b*** *- crocidolite*

Amphiboles are a large group of inosilicates, with linked double chains of silica tetrahedra, recognisable in their formulae by the ratio 4:11 of the silicon and oxygen atoms (with or without one atom of silicon substituted by aluminium). The chains are separated and bonded to each other by planes of cations. The amphibole structure permits extensive ionic replacement, to produce a wide range of chemical compositions. Most non-asbestiform amphiboles occur as elongate prismatic crystals. The asbestiform habit results from a strongly preferred mineral growth along the *c*-crystallographic axis, but only some amphiboles exhibit this habit and only three occur in commercially viable quantities (Table 1).

Crocidolite is the dominant amphibole asbestos, and its distinctive blue colour provides its common name as blue asbestos. It is the fibrous variety of riebeckite, the most widespread alkali amphibole, with sodium and both ferrous and ferric iron.

Amosite is a brown, iron-rich, fibrous variety of grunerite, a monoclinic amphibole containing neither calcium nor alkali. It was named after the acronym of the Asbestos Mines of South Africa, and is also known as brown asbestos.

Anthophyllite is one of the few orthorhombic amphiboles, and is grey or greenish in colour.

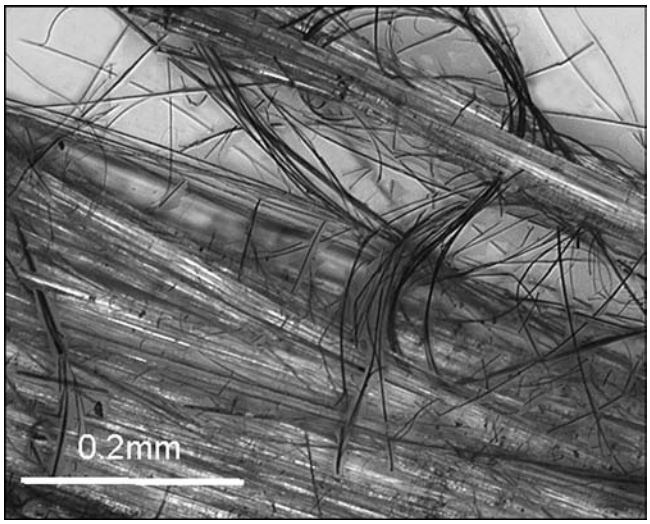

Figure 2. *Curled fibres and fibrils of chrysotile, seen under an optical microscope (Photo: Barbara Cressey)*

Fluor-edenite has only recently been found to constitute some of the amphibole asbestos. Edenite is a member of the complex hornblende group, and has one extra sodium atom to compensate for the one aluminium substitution, and the fluor-edenite has fluorine in place of its hydroxyl.

Tremolite and ***actinolite*** are members of the non-sodic, calcium amphiboles, and occur only rarely in asbestiform habit.

Amphibole asbestos fibre lengths and diameters are widely variable. Amosite fibres generally have lengths >200 μm and diameters <1 μm; crocidolite generally has the finest fibres, and the non-commercial amphiboles the coarsest. Amphibole asbestos fibres are straight, with parallel sides, and may show longitudinal grooving; they do not have the splayed ends or show the longitudinal splitting seen in chrysotile (Fig. 3).

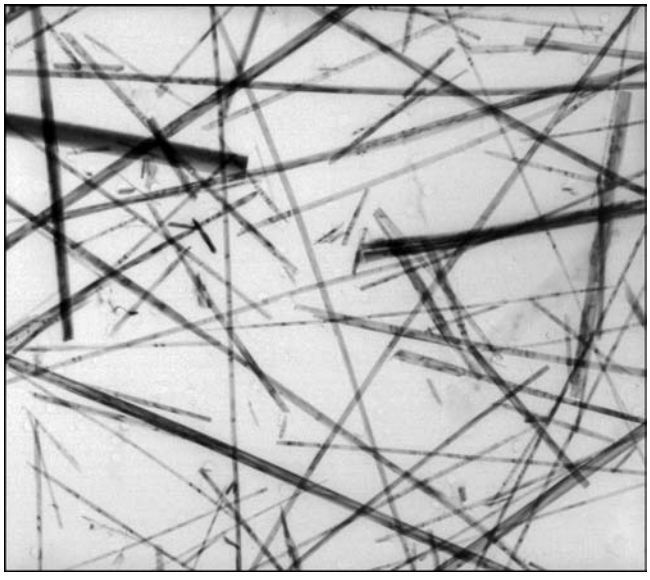

Figure 3. *Straight fibres of crocidolite, seen under scanning electron microscope (Photo: Fred Pooley)*

The commercial value of asbestos fibres depended almost wholly on their ability to be spun, with high grade minerals having long silky fibres. The heat resistance of all fibre types is about the same, but the differing chemical and physical structures give different properties. Thus, chrysotile is decomposed by hydrochloric acid, so that amosite and crocidolite were sometimes preferred for their acid resisting properties. Importantly, from a health viewpoint amphibole asbestos is more biopersistent when introduced into tissues, and the hazards differ significantly between chrysotile and amphiboles - though this is often overlooked when authorities consider asbestos risks. Crocidolite amphibole (blue asbestos) is far more dangerous than chrysotile (white asbestos).

Occurrence

Amphibole and serpentine minerals are widely distributed in the Earth's crust, and both can occur as massive, non-asbestiform deposits under conditions of moderate temperature and pressure. Subsequent asbestiform recrystallisation occurs within environments undergoing deformation by folding, faulting, shearing and dilatation, commonly accompanied by magmatic intrusion of sills and dykes (Ross & Nolan, 2003; Roggli & Coin, 2003).

Chrysotile is the least abundant of the serpentine minerals, but has accounted for more than 95% of the world asbestos trade. It occurs as large veins or masses in highly tectonised serpentinites associated with ophiolite complexes, and formed during late stage hydrothermal activity. Most reserves are found in southern Africa, Canada, China and the Ural Mountains, but deposits have been worked in more than 40 countries. Altered carbonate rocks also host serpentine minerals and chrysotile asbestos deposits that have been worked in South Africa and Arizona (Veblen & Wylie, 1993).

The major world deposits of amphibole asbestos, crocidolite and amosite, are within Precambrian banded ironstone terranes in western Australia and southern Africa. The mineral occurs as seams up to 300 mm thick, aggregated into "reefs" tens of metres thick, which may be traced over many kilometres. Deposition of the African banded ironstones was at about 2200-2500 Ma, with asbestos mineral seams growing during diagenesis under an extensional stress regime and low grade metamorphism at low pressures. Higher temperatures in the contact aureole of the Bushveldt intrusion (2100 Ma) induced the formation of amosite rather than the low-temperature crocidolite. Anthophyllite, has been extracted from an ophiolite complex in East Finland (Gibbons, 2000).

Mining and modern use of asbestos

The uses for asbestos burgeoned in the early and mid-nineteenth century with the requirement of insulation for the developing steam industry. This led to the rejuvenation of old mines in the Italian Alps. Then, in

the mid-1800s, large chrysotile deposits were discovered in Quebec and in the Urals. In South Africa, crocidolite was found in the Northern Cape in 1812, but mining development did not develop for a further 90 years. Amosite asbestos was discovered in the Transvaal in 1906, and commercial mining began in 1916 (Alleman & Mossman, 1997).

By the early 20th Century a boom was well underway, with asbestos being used in many diverse applications that reflected its inert properties. New manufacturing techniques allowed fabrication of asbestos threads, ropes, spun products and heat insulating boards. Asbestos was the basis of many building materials, including insulation, textured paints, wallboards, roofing tiles and concrete pipes. It was laminated as paper, woven into cloth, used as filter material and made into friction products such as motor car brake pads.

It has been estimated that, from the time of the first use of asbestos by Stone Age man to 1900, the total world production was around 250,000 tonnes of asbestos. By contrast the total world production between 1931 and 1999 was 166M tonnes, of which about 95% was chrysotile. About 3M tonnes each of amosite and crocidolite have been mined worldwide, together with about 350,000 tonnes of anthophyllite in east Finland. There has been little commercial production of tremolite and actinolite asbestos, but they are present as contaminants in other mined materials such as vermiculite. At present the major asbestos producing countries are Russia, China, Canada, Brazil, Zimbabwe and Kazakhstan (Ross & Nolan, 2003).

Exposure to asbestos

Ambient background

Geological weathering and erosional processes are responsible for the large amounts of natural dusts that enter the hydrosphere and atmosphere, but there are few quantitative studies of the mineralogical contents. One study from Japan reported an ambient average background level for globally distributed chrysotile fibres as 14.1 fibres/litre (Klein, 1993). This may originate from local soils and rock exposures, or by long-distance aeolian transport. Superimposed on this background is anthropogenic dust arising from industrial sources.

Evidence of exposure to asbestos in urban air can be found in the lungs of those livng in industrialised countries. Thomson *et al* (1963) reported that 25% of lungs, without evidence of pulmonary disease, examined at post mortem in Cape Town contained "asbestos bodies" - asbestos fibres coated with an iron-protein-mucopolysacharide material (Fig. 4). Subsequent quantitative studies confirmed the appreciable numbers of fibres found in the lungs. In Vancouver, Churg and Wiggs (1986) analysed the asbestos fibre content of the lungs of general members of the population coming to post mortem (Table 2), and Churg (1993) calculated that the lung of an urban

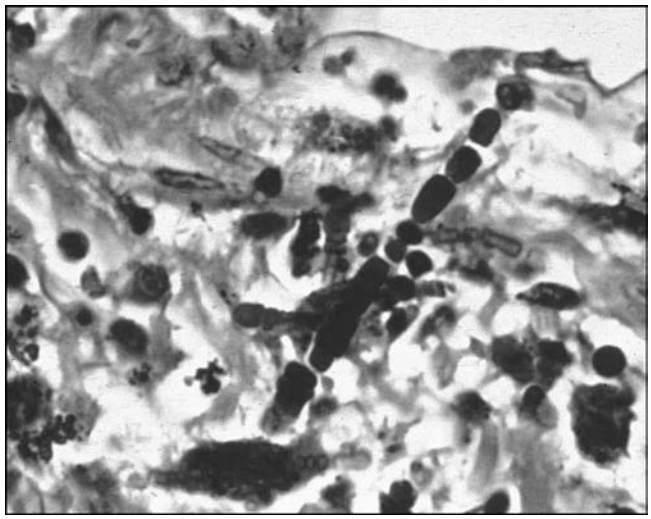

Figure 4. *An asbestos body from a human lung, with its characteristic segmented shape (and golden brown colour) ; these are generally 10 to 50 μm long. (Photo: Paolo Domizio)*

dweller there might contain as many as 40,000,000 each of chrysotile and tremolite fibres and 400,000 fibres of crocidolite and amosite. Such fibre burdens were without any evidence of associated disease.

Similar studies were made in mining towns in Quebec where the ambient fibre content of the air is several hundred times that of North American urban areas. Individuals resident for long periods in the towns (but never employed in the mining or milling processes) had lung fibre burdens five to ten times greater than dwellers in other urban areas. There is no evidence that such individuals suffer an increased frequency of mesothelioma or carcinoma (cancer) of the lung (Churg, 1986; McDonald,1985).

Excessive exposure to asbestos dust can arise in several ways. It may be by direct industrial exposure, or by peripheral exposure - including wives washing their husbands' heavily contaminated work clothes. Exposure may occur around asbestos mines, dumps and factories, or in buildings where there is a source of asbestos dust (Gibbons, 2000; Attanoos & Gibbs, 2003). Ambient non-industrial exposure arising from asbestos minerals in soil or outcrops may also occur, as in the Mediterranean region (see below).

Lung defences against dust

Inhaled particles follow the air into the lungs, and the depth to which they penetrate depends on particle size, shape, density and aerodynamic properties. When a particle comes into contact with an airway or the terminal alveolar airspace it is deposited. Large, dense

fibre type	million fibres/gram of dried lung	
	mean	*median*
Chrysotile	0.3	0.2
Tremolite	0.4	0.2
Amosite + crocidolite	0.001	0

Table 2*. Asbestos fibre contents of the lungs of the general population of Vancouver, Canada (after Churg, 1993)*

or compact particles deposited in the more proximal airways and surrounded by mucus are actively transported upwards by ciliated surface cells (the mucociliary escalator) and swallowed or expectorated. Fibres, such as asbestos, with high aspect ratios and fibre diameter <1.0 μm, may be carried axially in the airstream until they reach the smallest airways, where they collide with the walls of the respiratory and terminal bronchioles. In this way, amphibole asbestos fibres up to 200 μm long have been found in the periphery of the lungs of shipyard workers. The regularity of fibre shape also influences deposition, and the curled fibres of chrysotile are intercepted earlier in the airways than are the straight stiff fibres of amphiboles (Parkes, 1996).

Distally deposited particles are partially cleared by pulmonary macrophages (mobile scavenger cells) which phagocytose (engulf) and transport them to the mucociliary escalator. Short fibres (<5 μm) are easily engulfed, but this process fails with longer fibres. These fibres reach the interstitial tissue of the lung by direct transport across the alveolar walls, and are phagocytosed by macrophages within the tissue surrounding small airways and blood vessels. Heavy and prolonged dust burdens overload this macrophage dust clearance system, and particles then accumulate to provoke a chronic inflammatory response.

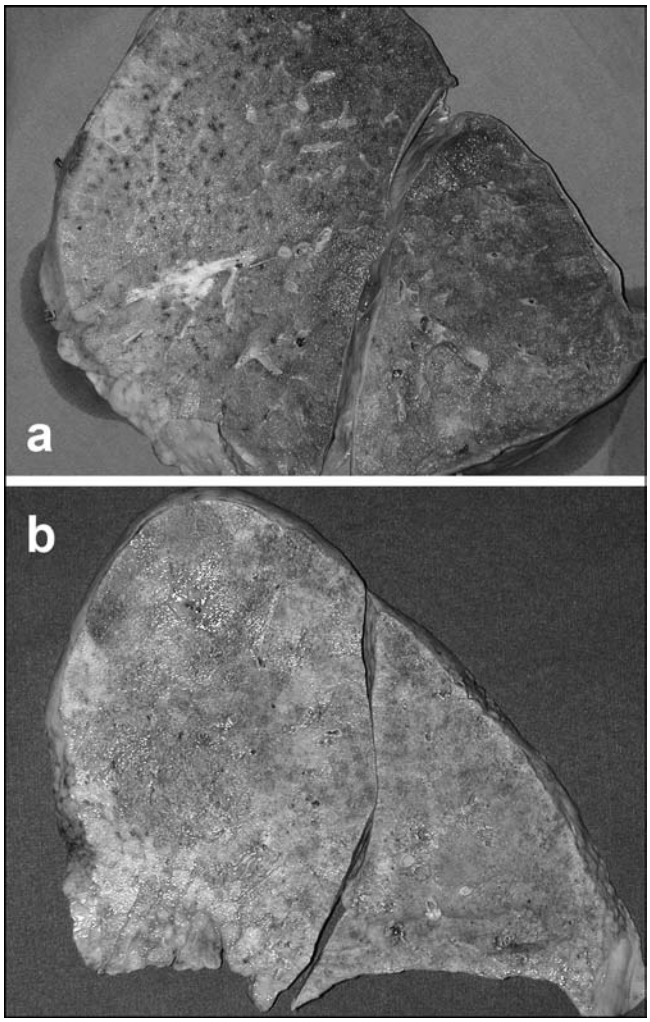

***Figure 5**. Slices through human lungs; a - a normal lung, with the thin pleural layer that covers the lung surface; b - lung with asbestosis, with extensive paler fibrotic tissue beneath the pleura. (Photos: Allen Gibbs)*

There is a marked difference between amphibole and chrysotile fibre persistence in the tissues. Continued exposure to amphiboles leads to persistent and increasing fibre levels within the lungs, with an estimated amphibole half-life measured in decades. By contrast, there is a negligible increase in the lung fibre burden with continued exposure to chrysotile. Chrysotile is rapidly cleared, and its half-life is only weeks to months.

The difference is in part due to the solublity of chrysotile in acid solutions, with leaching of magnesium from its surface. Chrysotile fibres are extremely fragile in the lung environment, and they tend to break transversely into short segments that are easily engulfed and removed, in part by acid dissolution within macrophages. Asbestiform amphiboles show no elemental loss, and their fibres do not break down, so their lengths remain too great to be engulfed by macrophages. As a consequence, persons with prolonged exposure to mixed dusts exhibit a relative increase in amphibole fibres (compared to chrysotile fibres) that remain in the lung for many years. The lungs of former Quebec chrysotile miners and millers, at post mortem, contain up to 80% tremolite fibres and only about 20% chrysotile, though the initial contamination from the chrysotile ore had only a tiny proportion of tremolite (Churg, 1993). Fibre durability in the lungs may underlie perceived differences in amphibole and chrysotile hazard.

Diseases produced by asbestos exposure

When the asbestos load is sufficient to overcome the respiratory defences, pulmonary disease follows. Various aspects of asbestos fibres are important in the cause of pulmonary disease. The geometry and dimensions of the minerals govern their deposition, clearance, biologic activity and dissolution in the lung. This gave rise to the Stanton hypothesis based on animal models (Stanton *et al*, 1977), which implied that the carcinogenicity of inorganic fibres depended principally on their size and shape. However, other factors include chemical and surface properties (absorbtion, oxidation/reduction reactions and surface charge), and these also play major and important roles in biopersistence, cellular responses and disease development (Veblen & Wylie, 1993; Hochella, 1993).

Chronic inflammation and repair

Continuing injury to tissues produces chronic inflammation with repair by fibrosis. In lungs, damaged by exposure to prolonged high concentrations of asbestos dust, this response is modulated by macrophages which in addition to their primary phagocytic function (engulfing) have a major role in the production of chemical inflammatory

mediators. These are released when asbestos fibres are phagocytosed by macrophages, and especially when phagocytosis is frustrated and ineffective on long asbestos fibres (Mossman & Churg, 1998). Fibrous scarring, as the end-result of chronic inflammation and repair, is seen in the lung and its coverings.

In the lungs, fibrous repair results in bilateral interstitial fibrosis, known as asbestosis. It occurs initially about the smallest airways, and then extends proximally to larger airways and distally to the air spaces of the alveolar ducts and alveoli. The walls of the latter are slowly and progressively thickened, with eventual obliteration of the alveolar spaces (Fig. 5). There is a loss of lung elasticity and impairment of respiratory gas exchange across the thickened alveolar walls (Fig. 6). Respiratory failure and cardiac failure, secondary to respiratory disease, eventually supervene.

Recognition of pulmonary fibrosis associated with inhalation of asbestos dust came in the 19th and early 20th centuries, in countries involved in mining and milling asbestos (Simson, 1928), as well as in importing countries (Seiler, 1928). Evidence was initially anecdotal, exemplified by a report in 1906, which described a case of pulmonary fibrosis to a government committee on compensation for industrial disease. The patient, an asbestos carder from Barking, described how he alone of ten men working in the card-room was the only one still alive. All the others died at about the age of 30 (Murray, 1990). Unfortunately, the committee did not allow compensation for any occupational pulmonary disease because of the confounding effects of tuberculosis, which was common at that time.

The association between diffuse fibrosis of the lungs and exposure to asbestos in the United Kingdom was firmly established by Merewether & Price (1930), who found pulmonary fibrosis in 25% of workers in an east London asbestos textile factory. They correlated the development of fibrosis with the intensity and duration of asbestos exposure, and described a latent period of 7-25 years between initial exposure and the development of asbestosis. Their observations led in 1933 to the introduction of the Asbestos Industry Regulations, which reduced the burden of dust exposure. These regulations progressively tightened so that exposures of >100 f/ml encountered in the 1930s were reduced to ~1f/ml in 1980.

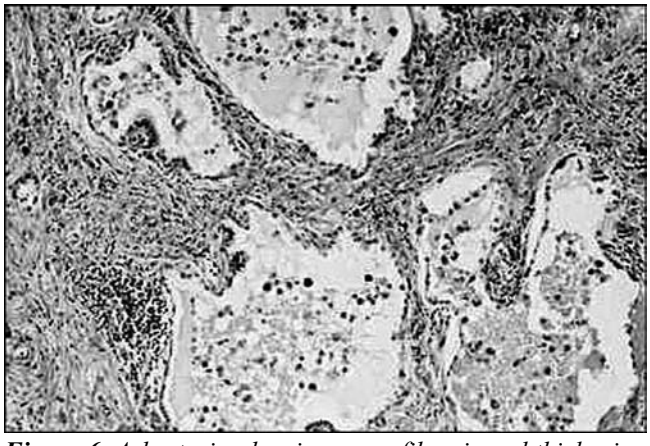

***Figure 6**. Asbestosis, showing gross fibrosis and thickening of the alveolar walls (around the pale annualar air spaces), which provides an impediment to gas exchange. (Photo: Paolo Domizio)*

period	exposure in years	
	range	*average*
1913 - 1940	1.5 - 19.0	7.0
1940 - 1950	—	10.0
1950 - 1960	3 - 32	14.5
1960 -	4 - 35	17.5

***Table 3**. Duration of exposure to asbestos prior to certification of asbestosis, in employees of an east London factory (after Smither, 1965)*

Asbestosis only develops in workers heavily exposed to asbestos dust, over and above a threshold of about 25-100 fibres/ml/year, below which asbestosis is not seen. Asbestosis was first recognised in workers with prolonged high exposure, notably miners, asbestos millers, asbestos textile workers and insulators. However, during World War II asbestosis occurred in shipyard workers exposed to very high levels for relatively short periods (Mossman & Churg, 1998), which emphasised that the time from initial exposure to first signs or symptoms of the disease, is inversely proportional to the exposure level. In the 1930s, before dust control regulations took effect, the average time of exposure before onset of certified asbestosis was seven years. More recently, the latent period has responded to stricter controls in the workplace (Table 3), and it is now uncommon to see patients with asbestosis who are very short of breath and with marked active changes on chest X-ray.

In the pleura (the thin layer of tissue lined with mesothelium that covers the surface of the lungs and lines the chest walls), repair occurs as thickened fibrous plaques that are often calcified. Pleural plaques cause no symptoms, and are frequently incidental findings on routine chest radiographs that provide a means of screening populations at risk of damage due to asbestos dust (Constantopoulos *et al*, 1987; Manda-Stachouli *et al*, 2004). The exposure levels to induce pleural plaques are an order less than those to produce asbestosis.

Malignant tumours

Malignant tumours arise with increased frequency in those exposed to asbestos, and may arise with or without asbestosis. Doll (1955) established that cancer of the lung arising from the bronchial mucosa is a hazard to asbestos workers, and that the risk among men employed in the industry for more than 20 years was ten times that of the general population. The risk lessened as the duration of employment under the old dusty conditions decreased, following the Asbestos Regulations. It is disputed whether the increase in lung cancer is due to any exposure to asbestos, to levels of exposure sufficiently high to have the potential to produce asbestosis, or only occurs when some degree

of pulmonary fibrosis has already been induced (Attanoos & Gibbs, 2003). There is synergism between asbestos exposure and tobacco; smokers who are heavily exposed to asbestos have a much greater risk of developing lung cancer than do those more lightly exposed to asbestos (Selikoff *et al*, 1964; Hammond & Selikoff, 1979).

Mesotheliomas are highly malignant tumours arising from the layer of cells which covers the lungs and lines the pleural cavities. The tumour grows rapidly and spreads to surround the lungs and heart (Fig. 7). It was regarded as rare until the 1960s, when the orthodox view was that most pleural tumours were secondary spread from undetected primary carcinomas of the lung or abdominal organs. Wagner *et al,* (1960) described 33 cases of mesothelioma in patients from the N W Cape Province of South Africa. Most had worked in asbestos mines or had been involved in asbestos transport with exposure to crocidolite, and in many of these patients their disease had been regarded as tuberculous pleurisy. They established the long latent period between initial exposure to asbestos and the diagnosis of mesothelioma, which averaged 44 years in their patients, and noted that several patients had never worked in the asbestos industry but had lived close to milling plants or waste dumps on which they had played as children. This report was criticised because only four cases came to autopsy and the pathology details were inadequate (Willis, 1967). However, it withstood the criticism, and was seminal in drawing attention to the linkage of asbestos exposure and mesothelioma. It became the most cited paper in industrial medicine (Wagner, 1991).

Increased mesothelioma in industrialised nations

With heightened awareness, it became apparent that mesothelioma was increasingly common in the industrial western nations, and in 1968 the UK Health and Safety Executive established a register of deaths due to mesothelioma. Since that time, there has been an epidemic of mesothelioma in Britain and Europe (Peto *et al*, 1995, 1999). The annual number of mesothelioma deaths in Great Britain has risen from 153 in 1968 to 1848 in 2001, and is predicted to peak at up to 2450 deaths each year between 2011 and 2015, with a rapid decline thereafter. The eventual death toll will depend on any residual asbestos exposure, but it is estimated that between 1968 and 2050 there will have been about 90,000 deaths from mesothelioma in Great Britain, 65,000 of which will occur after 2001 (Hodgson *et al*, 2005). Similar figures are predicted for Western Europe, with about 250,000 deaths over the next 30 years.

Risks of asbestos fibres as a carcinogen

Most epidemiologists agree that all forms of asbestos can cause both cancer of the lung and mesothelioma, but there is controversy on the relative contribution of chrysotile and the amphiboles to the overall incidences

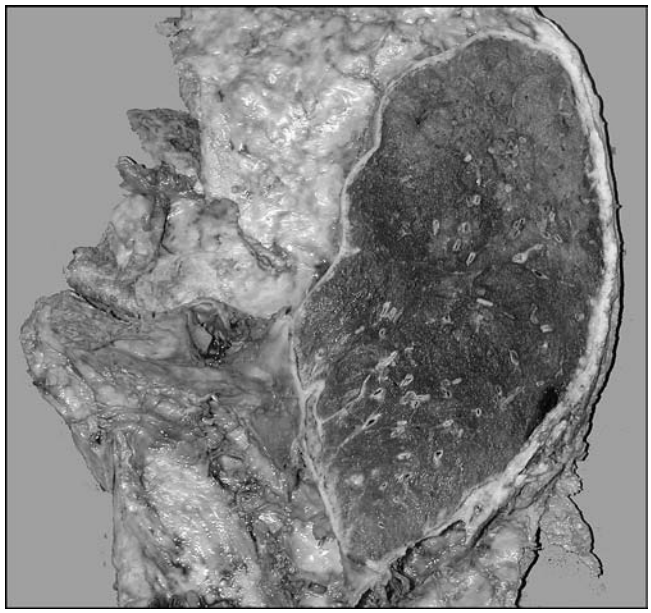

Figure 7*. White tumour tissue of mesothelioma that has encased and compressed the lung; the tumour grew from the mesothelial layer of cells that cover the lungs. (Photo: Allen Gibbs)*

of these tumours (Peto *et al*, 1998; Liddell, 1997). A recent study found that the relative risks for the development of mesothelioma for the various fibre types at occupational exposure levels were chrysotile ~1, amosite ~100 and crocidolite ~500; for lung carcinoma the risk differential between chrysotile and amphibole fibres was less, between 1:10 and 1:50 (Hodgson & Darnton, 2000). Others have claimed chrystotile as the principal cause (Smith & Wright, 1996); Cullen (1998) suggests that, although less dangerous than the amphiboles, chrysotile is used far more widely worldwide, and is probably the main cause of mesothelioma. These reports have been questioned, as they arose from potentially biased sources involved in litigation. By contrast, studies of Quebec asbestos miners and millers for more than thirty years, showed that very few mesotheliomas are caused by pure chrysotile, and most are attributed to contaminant tremolite (McDonald *et al*, 1996, 1997). The problem is not easily resolved, for few workers are exposed to chrysotile alone. Most exposure measurements in the past were of "asbestos" particles, rather than of specific fibre types. Moreover, the interpretation of lung burden studies in humans, in contrast to those on experimental animals (Elmes, 1991), is vitiated by the relatively rapid pulmonary clearance of chrysotile compared to amphibole fibres, and also to the prolonged latent period between initial exposure and tumour development.

Other tumours ascribed to asbestos exposure include carcinoma of the larynx, stomach and colon, but social class, alcohol intake and smoking act as confusing factors, and these linkages are not proven (Attanoos & Gibbs, 2003).

Failure of industrial practice and control

Human costs

Excessive exposure to asbestos has occurred, despite regulations of workers' time, in mines, mills and industrial plants, and also in the surrounding environment. In the producer countries, some of this has been due to negligence and disregard of regulations, and some has been, allegedly, due to concealment of health risks. Gross examples have occurred in South Africa, Australia and the USA.

- In 1949, a doctor at the Penge amosite mine in the Transvaal reported: "Exposures were crude and unchecked. I found young children, completely included in large shipping bags, trampling down fluffy amosite asbestos, which all day long came down cascading over their heads. They were kept stepping lively by a burly supervisor with a hefty whip. I believe these children to have had the ultimate asbestos exposure. X-ray revealed several to have radiologic asbestosis, with heart failure before the age of 12." (Meeran, 2003).
- The surrounding environment of mine and adjacent homes were not spared. Also in South Africa, 2000 miners at Prieska began a legal action against Cape plc in 1998. Their representative, whose job had been sorting and packing asbestos with bare hands, was never provided with a mask or gloves. Now suffering from asbestosis, he related that his wife, parents and brothers had been killed by asbestos-induced disease. He said: "The dust was everywhere. It lay up to an inch thick. There were no warnings, nothing. Children played in it. I lived half a kilometre from the factory but in order to drink I had to scrape a layer of asbestos off the top of my water jar." (Talbot, 1999). A cohort study of 399 whites born in Prieska between 1932 and 1936 showed that 9% of the deaths up until 1990 were due to mesothelioma (Reid *et al*, 1990). The health damage to Africans is not known because of poor records but is probably worse (Gibbons, 2000).
- In Australia, despite the introduction of Federal regulations from 1945, asbestos was widely abused in the thermal power station industry, and power stations became virtual mountains of asbestos. This was crudely swept up and spread by workers boots, or was more widely disseminated by blasting with compressed air, so that the entire area of each power station was continuously blanketed by clouds of fibres (Gibbons 2000).
- In the United States in 2005, a federal grand jury indicted current and former executives of the W R Grace Company for endangering the inhabitants of Libby, Montana, by concealing information about its vermiculite mine (Spadaro, 2005). Libby residents have an increased incidence of asbestos-related diseases, including carcinoma of the lung which is 30% higher than expected compared to other areas of Montana and the U.S. It is alleged that since 1970, W R Grace and its executives hid the fact that tremolite was present as a contaminant in the vermiculite, and allowed vermiculite to be widely distributed throughout Libby. Workers left the plant covered in dust; residents were allowed to take vermiculite for their gardens, and vermiculite tailings were given to Libby schools for use as foundations for running tracks and an outdoor ice skating rink. The mine closed in 1990, and it is alleged that W R Grace sold contaminated properties without disclosing the nature or extent of contamination.

In user countries such as the United Kingdom, initial reports of asbestos-related disease concerned workers in asbestos factories and metal plate workers (including shipyard workers, and railway carriage builders where large amounts of amphibole asbestos were used) and exposures were intense. It was in these areas that regulations were most stringently applied because of the perceived risk of asbestosis. However, the worst carcinogenic effects of asbestos have been experienced by the generation who began work in the mid-1960s, after the health hazards from asbestos were well recognised. They worked in occupations where the risk from asbestos was not appreciated - in the construction and power generating industries, and as maintenance workers such as electricians and plumbers (Attanoos & Gibbs, 2003). At that time, amosite insulation boards and tiles were widely used in buildings; chrysotile was imported for incorporation into asbestos cement and floor tiles, but monitoring and exposure control was limited.

An example in the East Midlands concerns exposure to a mixture of asbestos fibre types. Mr R D, suffering from mesothelioma, alleged at the County Court that he was exposed to asbestos dust at work. Trackside cable troughs, which run alongside the railway, contain either mechanical or electrical cabling. During the relevant period of his exposure, most troughs were made of concrete, but some were asbestos concrete, as on the main line south of Chesterfield. The asbestos content was mainly chrysotile, but small quantities of crocidolite were present in older troughs. He was involved in their removal by smashing and breaking up the troughs. He related an occasion when a supervisor, asked if it was safe to deal with asbestos troughing, replied that white asbestos was safe. He also worked in signal boxes where ceiling tiles and wall boards contained amosite. At times he was engaged in pushing cabling through holes in the ceiling with inevitable chafing on the edges resulting in the release of asbestos dust. The court found for the claimant; he died in 2001 from mesothelioma (Sheffield County Court, 2000).

Costs to industry

The Faculty and Institute of Actuaries forecasts that the total future UK cost of asbestos-related diseases is £8-20bn. The UK insurance industry is expected to face a bill of £4-10bn. More than half of this sum will be for mesothelioma claims, which will continue to rise for the next ten years. By contrast, claims for asbestosis will fall due to the declining use of asbestos in the UK since the 1970s. The future cost represents 80,000-200,000 new insurance claims over about the next 30 years (Asbestos Working Party, 2004).

Asbestos hazard in buildings?

The lung can tolerate greater burdens of chrysotile and tremolite asbestos than is found in the lungs of urban dwellers. Churg and Wiggs (1986) provided re-assurance about the innocuous effects of the general background level of asbestos, and also about the effects of low levels of chrysotile release inside public buildings such as schools, where internal levels are close to the levels of ambient air. Nevertheless, public awareness of the perceived dangers of asbestos brought about near-panic in many parts of the Western world, and billions of dollars were spent in America removing asbestos from the structure of schools and public buildings.

A cold douche of common sense and hard data was needed to curtail the asbestos panic, which was founded on fear rather than evidence. Mossman *et al* (1990) gave very low risk estimates for children attending classes in schools built with asbestos-containing material. Data from HELAR (1991) and Wilson *et al,* (1994) showed that the average concentration of asbestos measured in 219 American schools was 0.00022 f/ml. Using this figure, Ross & Nolan (2003) calculated that in the worst-case calculated prediction, the risk for residing in the classroom for six hours a day, five days a week for 14 years is one excess cancer death per million lifetimes. Further evidence is provided from Quebec mining towns, where Camus *et al*, (1998) reported that ambient asbestos levels exceeded 1f/ml before the introduction of modern dust control procedures in the 1970s. Ross & Nolan (2003) calculated that the ambient asbestos dust levels in these towns varied from 230 to 23,000 times the average levels found in schools with asbestos insulation. Yet a mortality study of women who lived in these towns at that time (but did not work in the asbestos industry) showed that their health was unaffected by the very high, lifetime exposure to asbestos dust (Siemiatycki, 1982).

Most of the asbestos imported into Britain between 1960 and 1980 is still in place in buildings, but airborne levels are very low during normal use, and the material is not an immediate health risk to the general public. The danger now lies in the work of maintenance men (carpenters, electricians and plumbers) and demolition workers who may be unknowingly exposed to asbestos in their duties; this applies especially those working in small firms where they may be inadequately monitored. Since the 1980s, asbestos removal has become a specialised industry, and this new and initially inadequately regulated industry may well have increased the future burden of occupational asbestos disease (Peto *et al*, 1995).

Continuing use of asbestos

There are calls for a worldwide ban on the use of asbestos (Cullen, 1998; Collegium Ramazzini, 2001; Guidotti, 2001). These have now achieved some governments` support, and have led to country by country bans. Such calls are opposed by asbestos-producing countries. The Canadian asbestos industry is the world's second-largest producer of chrysotile, and more than 90% is exported. In 1991, the Canadian asbestos lobby successfully challenged the U.S. Environmental Protection Agency's ban on asbestos products. Since 1991, members of the European Union have individually banned asbestos on the grounds that there is no safe exposure level. Fearing global expansion of the ban, Canada challenged France (its largest European customer) at the World Trade Organization (WTO) in 1998 to repeal its unilateral ban on asbestos. The WTO found that all levels of asbestos posed some cancer risk, that safer substitute products were available, and that there was no such thing as "controlled use". The failure of the challenge allowed other countries to ban asbestos; a European Union asbestos-ban deadline of January 1st, 2005 was met by all member countries. E U directive 1999/77/EC bans placing, marketing and use of products that contain asbestos after 2005; directive 2003/18/EC prohibits all activities in which workers are exposed to asbestos fibres in asbestos mining or in production and processing of asbestos products, with effect from 2006.

There is, however, an inherent fallacy in the proscription of all asbestos minerals as a single coherent health risk. All agree that asbestiform amphiboles should be banned because of their undoubted toxic effects and their long persistence in the human lung, but the banning of chrysotile asbestos is on much less firm ground. The Canadian challenge is based on epidemiological studies over thirty years in Quebec asbestos mines and mills, which show that with strict controls chrysotile usage is essentially innocuous, and the probability is that tremolite contamination is responsible for most cases of mesothelioma associated with heavy chrysotile exposure (McDonald *et al* 1996, 1997; Liddell, 1997).

In response to falling markets in the industrial nations, the Canadian asbestos industry has progressively transferred its commercial activities to the Third World. Country by country bans have shifted rather than eliminated the use of asbestos. Southeastern Asia has been targeted as an export market, where asbestos is used to make piping, roofing and flooring material. It is argued that to deny a developing country the use of asbestos cement is to deny it adequate housing, drainage and water, with loss of protection against catastrophic waterborne diseases (Liddell, 1997). The Canadian Government and industry promote a concept called "safe use", with good ventilation, protective equipment, embedding the fibres and wetting the material when it is being handled. In practice the reality may be far from that. The poor countries of Southeastern Asia do not have good occupational health and safety regulations or good industry controls, and statistics on asbestos diseases are lacking. In Canada, rigorous controls may work. In Third World environments, there may be no such thing as safe use, but it may be a matter of balancing public health risks.

Non-industrial exposure to fibrous minerals

Asbestos

Pulmonary diseases including pulmonary fibrosis, pleural plaques, carcinomas of the lung and mesotheliomas have been reported to occur with increased frequency in inhabitants of the Mediterranean region, and have been attributed to environmental exposure to asbestos and other fibrous minerals present in local rocks and soils (Table 4). Many cases are found in areas on or near ophiolite outcrops, and are associated with exposure to tremolite (Ross & Nolan, 2003). Such exposure appears not to be related to quarrying or industrial use, but mainly to the use of white *luto* soils, that are contaminated with the amphibole, as an interior and exterior whitewash for house walls and floors. Langer *et al* (1987) report that in some parts of Turkey the whitewash consists of almost pure asbestiform tremolite (Fig. 8). In the Metsovo area of N W Greece, where the high prevalence of mesothelioma has been recognised for more than 20 years and the whitewash has not been used since 1985, a decrease in the number of cases of pleural plaques and of mesothelioma is reported (Manda-Stachouli *et al*, 2004). It has been estimated that about 16,000,000 people living in rural Anatolia have been exposed to asbestos mineral dusts, and in these the cumulative retention of fibres in the lung is comparable to that found in patients occupationally exposed to asbestos in northwest Europe (Dumortier *et al*, 1998).

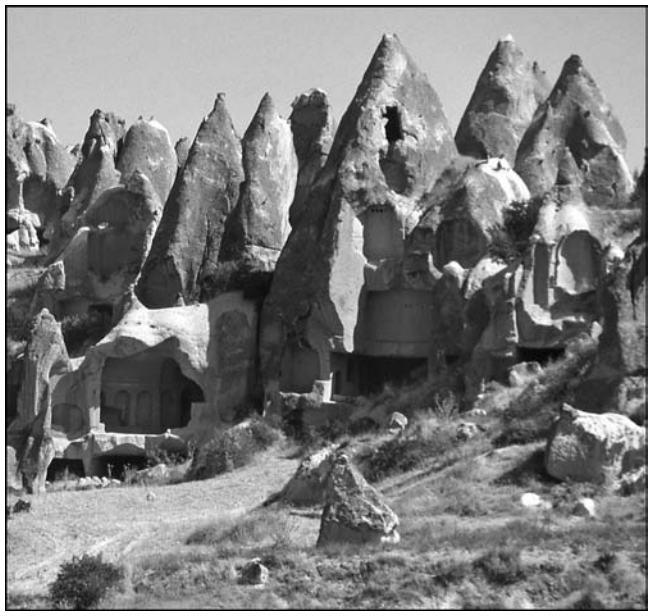

Figure 8. *Anatolian landscape where the volcanic tuffs have been gullied and weathered to leave the cones and pinnacles of Cappadocia; many cones have had artificial caves cut into their soft rock.*

Zeolites

Parts of the Anatolian plateau, in southeastern Asian Turkey, are covered with tuffs from the two volcanoes of Ercives and Hasandag. The weathered tuffs form the dramatic landscape of conical and hive-shaped landforms that are well known in Cappadoccia (Fig. 9). These tuffs are soft and easily excavated or quarried, and have provided cave dwellings or building materials for churches, houses and byres from ancient times. Weathering of the tuffs produces a variety of minerals in the clay and zeolite groups. Zeolites (from the Greek for *boiling stone*) are hydrated tectosilicates that give off water on heating. They typically occur in amygdales, cavities and pore-spaces in basic volcanic rocks, and as alteration products of feldspars, feldspathoids and volcanic glass. They have open, wide-meshed, 3-D atomic framework structures that can lose water or undergo cationic exchange while retaining their crystal shape. They have therefore been used commercially as molecular sieves and petroleum cracking catalysts (Bish & Guthrie, 1993). The zeolite group includes erionite (from the Greek for *wool*), a fibrous variety of the more widespread thomsonite, which in aggregate looks and feels like felt or wool.

Location	Mineral	Environmental factors	Pleural plaques, pulmonary fibrosis	Carcinoma of lung	Mesothelioma	Source
S E Turkey Madnen ophiolite	Tremolite	Stucco whitewash	◆	◆	◆	Yazicioglu *et al*, 1980 Senyisit *et al*, 2000
Turkey Anatolia	Tremolite Actinolite	Stucco whitewash	◆		◆	Zeren *et al*, 2000
Turkey Cappadoccia tuffs	Erionite	Cave dwelling	◆	◆	◆	Rohl *et al*,1982 Baris *et al*,1987
N W Greece Pindos ophiolite	Tremolite	Stucco whitewash	◆		◆	Sakellariou *et al*,1996 Constantopoulos *et al*, 1987 Langer *et al*, 1987 Manda-Stachouli *et al*, 2004
Cyprus Troodos ophiolite	Tremolite Chrysotile	Fibres in dust, domestic and environmental			◆	McConnachie *et al*, 1987
Corsica Cap Corse ophiolite	Tremolite Chrysotile	Asbestos outcrops	◆		◆	Rey *et al*, 1994
Eastern Sicily Etna volcanics	Flour-edenite	Contaminant of building blocks			◆	Gianfagna *et al*, 2003

Table 4. *Reports of pulmonary disease attributed to fibrous minerals in the Mediterranean area;* ◆ *indicates lesions reported.*

The incidence of mesothelioma in Anatolia has been estimated to be 45 per million inhabitants, and in some reports is linked to environmental exposure to tremolite. However, in three villages in central Anatolia the estimated incidence is even greater - 996 per 100,000 inhabitants - with more than 50% of deaths due to mesothelioma. A cohort of 162 Turkish emigrants from one of these villages to Sweden was followed: 18 deaths occurred between 1965 and 1997, and 14 of these were due to mesothelioma. Compared to the general Swedish population, the standardised incidence rates for mesothelioma were 135 times higher among men and 1336 times higher among women (Metintas *et al*, 1999). The three villages with the highest rates of pleural and peritoneal mesothelioma have raised environmental levels of erionite, compared to adjacent villages. Erionite fibres were a major part of airborne dust, and their source was identified as a poorly consolidated tuff that crops out naturally, and still forms the walls of caves used as utility rooms and animal quarters (Wagner *et al*, 1985).

Measurement of fibres taken from the Anatolian village environments showed erionite to approach the dimensions of asbestos, with fibres up to 50 µm long and 0.5-1.5 µm in diameter. Small amounts of chrysotile and tremolite were also found. In tissues from the lung and pleura of mesothelioma patients, 90% of the fibres were erionite, although there was a smaller amount of tremolite (Rohl *et al*, 1982).

Experimental studies of rats exposed to erionite, by inhalation or by injection, showed that erionite from the Turkish villages has a potential to produce mesotheliomas enhanced in comparison to crocidolite (Wagner *et al*, 1985). This may be due to its tectosilicate structure, which gives a greater internal surface area with enhanced surface effects, and its ability to facilitate ion exchange and catalyse biological reactions (Bish & Guthrie, 1993). However, the causes of mesothelioma in the villagers appear to be more than simple dust exposure; families in adjacent houses in the same village have very different incidences of the tumour, despite apparently similar exposure. In a genetic and epidemiological study of a group of 526 related persons (Hammady *et al*, 2001), susceptibility to mesothelioma appeared to be inherited.

A caution for the future

The debate over health hazards from fibrous mineral dusts is not yet over. There is still a need for substances with the industrial properties of asbestos, and substitutes are actively sought, but such replacement fibres may have a similar biological potential to that of asbestos. Animal models are of little use because of interspecies variability and because of the discrepancy between animal lifespan and the lengthy latent period in human asbestos-induced diseases. Awareness of the *possible* hazards in fibrous dusts must lead to active preventative measures by authority, employers and workers alike, from the period of initial use, if another asbestos-like industrial catastrophe is to be avoided.

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Gerard Slavin, Emeritus Professor of Histopathology,
Medical College of St Bartholomew & Royal London Hospital,
Queen Mary College, London University.
gsbms@darleyd.fsnet.co.uk