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Asbestos-related diseases of the lungs and pleura: uses, trends and management over the last century

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SUMMARY

Asbestos is a descriptive term for a group of naturally occurring minerals known to mankind since ancient times. The main types of asbestos (chrysotile, and the amphiboles crocidolite and amosite) differ in chemical structure, biopersistence in human tissue and toxicity. Commercial exploitation, with little thought for environmental controls, increased over the twentieth century, particularly after World War II, to accommodate globalisation and the demands of the world's burgeoning cities. As its ill-health effects, both non-malignant (fibrosis of the lungs or asbestosis; pleural effusion, plaques and thickening) and malignant (mesothelioma, lung and other cancers), became evident, public pressure rose to control its use. The last decades of the last century saw decreases in exposure and rates of asbestosis in industrialised and in some less-industrialised countries, where pleural plaques and malignant mesothelioma are currently the most frequent manifestations of asbestos exposure. Longer

follow-up of asbestos-exposed cohorts in mining and manufacturing has also strengthened the evidence of a fibre gradient in toxicity, with chrysotile exhibiting lower toxicity than the amphiboles, and amosite lower toxicity than crocidolite. The last decades of the twentieth century saw stabilisation and/or declines in mesothelioma rates in several industrialised countries. In less-industrialised countries, data on disease are sparse, exposure generally high and rates may peak in the future. Management of asbestos-related disease in the workplace requires collaboration between workers and unions (responsible for monitoring workplace dust levels, to which they must have access) and companies (responsible for engineering controls), reinforced by appropriate government regulations and by community support.

KEY WORDS: asbestos; diseases of the lung and pleura; trends in disease; industrialised and less-industrialised countries

ASBESTOS is a descriptive term for a group of naturally occurring minerals of fibrous habit known to and used by mankind since ancient times. Table 1 lists the mineral silicates that have been found in human lung tissue, the location of the major deposits worldwide, and main commercial uses and/or other sources of human exposure.^{1–5}

Figure 1 shows the main fibre types,⁶ which differ in their physical and chemical properties and in the consequences of exposure. These include non-malignant conditions (pleural plaques, effusions, diffuse pleural thickening, rounded atelectasis and asbestosis) and malignant conditions (mesothelioma, lung and other cancers).

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THE FATE OF INHALED FIBRES

The accumulation of fibres in the lungs results from exposure, deposition, clearance and retention, processes that depend on exposure intensity, duration and profile.⁷ Long fibres clear more rapidly than short fibres, and chrysotile fibres more rapidly than amphi-

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Table 1 Mineral silicates that have been found in human lung tissue*

Mineral: group and form	Location of major deposits, commercial and/or other	Main commercial uses and/or other sources of human exposure
Asbestos minerals		
<i>Serpentine</i>		
Chrysotile [†] (white asbestos)	Canada (Québec, [‡] British Columbia, Yukon, Newfoundland) China (Szechuan Province) Russia Brazil Mediterranean countries (Cyprus, Corsica, Greece, Italy) Southern Africa (South Africa, Swaziland, Zimbabwe)	Brake lining, ship building /repair; polishing precious stones; stone and whetstone cutting; foundry operations (mainly insulation); asbestos cement products (pipes, gutters, tiles, roofing); insulation; fireproofing; reinforced plastics (fan blades, electric switching gear); textiles; friction materials; paper and spray-on products
<i>Amphiboles</i>		
Crocidolite (blue asbestos)	South Africa (North West Cape [§]) Western Australia (Wittenoom [§])	Used in combination, mostly in cement products, also in some products listed below
Amosite (brown asbestos)	South Africa (Northern Province—former Transvaal) [§]	Filler in rubber and plastics
Anthophyllite	Finland [§]	May be removed in processing; has rural domestic use (e.g., stucco)
Tremolite	Contaminates ore in some asbestos, talc, iron and vermiculate mines; also in some agricultural soils	No commercial use
Cummington-Grunerite	Contaminates ore in some iron mines (often not fibrous)	
Non-asbestos mineral silicates		
Clay minerals (usually fine-grained, powder-like) such as kaolin and bentonite	40 countries including China, Czechoslovakia, Germany, Egypt, Japan, US (Georgia, North Carolina), UK (Cornwall)	Functional filler in paper, plastics, bricks, cement, etc; fire clays, refractories, ceramics, lubricants
Micas (usually flaky)		
Vermiculite (expands when heated)	Australia, Kenya, South Africa, USA (Montana, Virginia)	Absorbents (horticulture); plasters; boards; insulation; fire resistance
Zeolite (fibrous) (e.g., erionite [¶])	Turkey (Anatolia, Cappadocia regions); non-commercial deposits	Houses constructed in erionite rock; soil containing fibres mixed with tremolite; sepiolite used in stucco and plaster
Man-made mineral fibres		
Glass wool and filament	Produced in factories around the world	Many uses previously reserved for asbestos
Rock and slag wool	As above	Used in insulation in buildings and in car and naval construction
Ceramic fibre	As above	Used in reinforced cloth, brakes, etc; high-performance ceramic fibres used in jet engines, spacecraft

Note: As of 2006, 23 countries have prohibited the use of asbestos or severely restricted its use, but in most countries exceptions are allowed for some uses. A prohibition decree was adopted by the European Union in 2005.

* Adapted from Table 61.3 in Cowie et al.,⁵ and reproduced with permission from Elsevier Saunders. This list is not exhaustive; reader should consult other sources for further information.¹⁻⁵

[†] Accounts for over 90% of the world's production.

[‡] Currently the Jeffrey mine in Québec, Canada, suspends its operations intermittently, depending on demand.

[§] Mining no longer in operation.

[¶] In one of three epidemics of mesothelioma in Turkish villages, erionite was implicated; in the other two, tremolite or chrysotile were implicated.

bole fibres.⁵ Fibres of less than 3 μ are phagocytosed by activated macrophages, which then drain via lymphatics to the pleural surface and eventually into the pleural space.⁵ How fibres transmigrate to the parietal pleura to produce plaques is not known. Longer fibres are partly phagocytosed, usually by several macrophages, and become the core of asbestos bodies, so called because of their association with asbestos exposure and their ready identification on light microscopy. In the lungs of exposed individuals, the number of uncoated (bare) fibres exceeds the number of coated fibres (asbestos bodies) by from 5000 to 10 000 times.⁵ Most, but not all, coated fibres contain asbestos, usually an amphibole. The presence of more than one coated fibre has been accepted for many years⁸ and challenged⁹ as

necessary for the pathological diagnosis of asbestosis, even in a subject with an appropriate exposure history. Asbestos bodies found in sputum or bronchoalveolar lavage (BAL) reflect high tissue levels.¹⁰ They are more commonly found after recent exposure than after remote exposure, and after exposure to amphiboles than after exposure to chrysotile.¹¹

DIAGNOSTIC EVALUATION OF ASBESTOS-RELATED DISEASES

Clinical features

Breathlessness, the most common respiratory symptom associated with asbestosis, is usually of insidious onset and first noted on exertion.^{12,13} Symptoms should

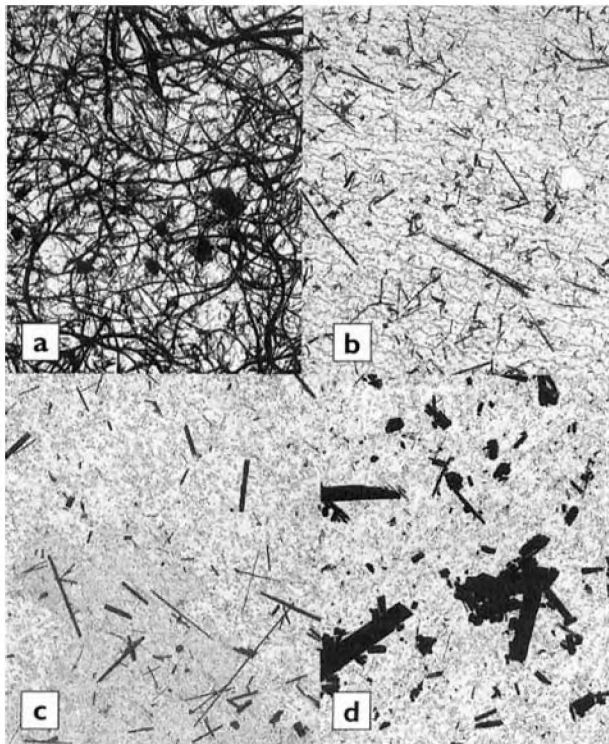


Figure 1 Different types of asbestos fibres (a, chrysotile; b, crocidolite; c, amosite; d, anthophyllite) as seen by transmission electron microscopy (UICC reference material). Reproduced from De Vuyst and Gevenois, Figure 9.2, page 144, with permission from Elsevier.⁶ UICC = International Union Against Cancer.

thus be objectively quantified using a validated questionnaire. Other features include non-productive cough and mid-to-late inspiratory crackles that persist after cough.¹⁴ Haemoptysis should prompt further assessment for malignancy or concurrent infection by tuberculosis, using sputum cytology and bronchoscopy. Oppressive chest pain, especially on exertion, was reported in up to 70% of heavily exposed workers in an Indian asbestos mill,¹⁵ and in 43% of a Western Australian cohort of 1280 subjects who had worked on or lived near a crocidolite mine.¹⁶ Extensive crackles, clubbing and/or cyanosis are associated with increased risk for asbestos-related mortality.¹⁴

Imaging

Conventional chest X-ray

A standardised system for obtaining and classifying postero-anterior chest X-rays for the presence and profusion of parenchymal opacities and pleural changes was published in 1980 (International Classification of Radiographs of Pneumoconiosis, International Labour Office [ILO]),¹ and updated in 2003.¹⁷ The original films and reading sheets for the presence and profusion of parenchymal opacities were retained, and those for pleural and other disease (chest wall, vascular, etc.) considerably expanded. Profusion scores derived from epidemiological surveys have been found

to correlate with mortality and functional impairment in asbestosis.^{18–20}

High-resolution computed tomography

High-resolution computed tomography (HRCT) has displaced conventional chest X-rays for the clinical evaluation of asbestos-exposed subjects because it is more sensitive for detecting asbestosis.^{21–24} Abnormalities that characterise asbestosis on HRCT include thickened intra- and inter-lobular lines, subpleural curvilinear lines, subpleural ‘dotlike’ opacities, parenchymal bands, small cystic spaces, patchy areas of ground-glass change and honeycombing in advanced disease (Figures 2–5).²⁵ By contrast, conventional chest X-rays are appropriate for screening asbestos-exposed populations for the extent of pleural lung abnormalities.²¹

Other imaging methods

Digital enhancement of chest radiographs improves their sensitivity and specificity in identifying pleural involvement in a clinical context.²⁶ Ultrasound may be useful in locating pleural fluid. Magnetic resonance imaging (MRI) can be of value in identifying rounded atelectasis and in distinguishing parietal lesions (chest wall and/or pleural) from fluid accumulation.²⁶ Positron-emission tomography (PET), especially when used in association with HRCT, is useful for differentiating benign from malignant effusions and for staging patients with known pleural mesothelioma (Figure 5).²⁶

Sputum analysis and bronchoalveolar lavage

Identification of asbestos bodies may help in assessing exposed individuals for asbestosis. However, sputum analyses for asbestos bodies miss almost half the occupationally exposed individuals in whom asbestos bodies are found on BAL.¹⁰ Asbestos bodies recovered on BAL are also useful in the clinical diagnosis of parenchymal involvement: the amphibole burden in the lungs correlates better with amphibole recovery on BAL than chrysotile burden with chrysotile recovery.²⁷

Pulmonary function tests

Established asbestosis may be, but is not invariably, associated with a restrictive lung function profile.⁵ With less advanced radiological disease, forced vital capacity (FVC) and diffusing capacity are usually reduced, and flow at low lung volumes may also be reduced, consistent with asbestos-related small airway disease.²⁸ A substantial proportion of workers (up to 50% in some studies) exhibit a mixed or obstructive lung function profile,²⁹ consistent with the parallel development of airways and parenchymal effects of working in dusty occupations contaminated by mineral dusts containing asbestos.⁵ Pulmonary gas exchange disturbances, reflected by reduced diffusing capacity of the lung for carbon monoxide (DL_{CO}) and widening alveolar-arterial oxygen difference at rest and exercise, are more sensitive indices of dysfunction than ventilatory impair-

ment.³⁰ Cardiopulmonary exercise testing may be useful in disability evaluations.

Many subjects with circumscribed pleural plaques have normal lung function at rest but develop shortness of breath during exercise.³¹ Controversy exists as to whether pleural plaques are associated with ventilatory dysfunction,^{22,32} while diffuse pleural thickening usually, but not always, leads to significantly reduced vital capacity and diffusing capacity.³³

BENIGN ASBESTOS-RELATED DISEASE

Pleural plaques

Pleural plaques are circumscribed, discrete areas of fibrous tissue and have long been considered markers of asbestos exposure (Figure 2). Histologically, they are

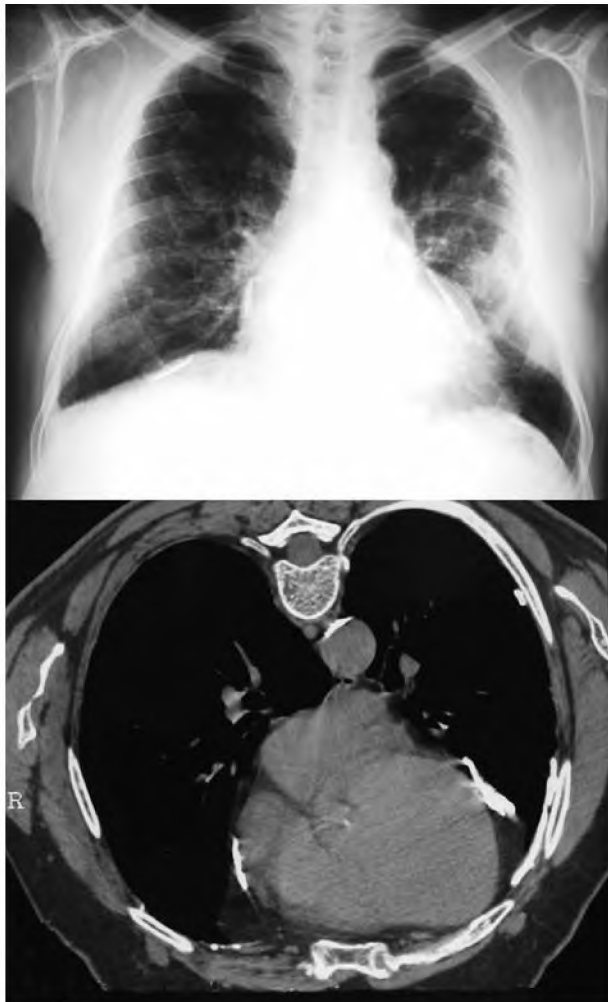


Figure 2 A 55-year-old non-smoking asymptomatic male who worked for 10 years (1968–1978) in the asbestos cement industry. Chest radiography (*top*) on the right shows calcified pleural plaques en face and on the diaphragm, and blunting of the costophrenic angle. On the left it also shows calcified pleural plaques en face and on the diaphragm. The HRCT scan (*bottom*) shows a calcified pericardial plaque on the right and calcified parietal and pericardial plaques on the left. HRCT = high-resolution computed tomography.

characterised by acellular deposits of collagen on the parietal pleura.²⁸ With the improvement of environmental controls in workplaces, an increasing number of workers are seen in whom pleural plaques are the only manifestation of asbestos exposure. In one study of 110 construction insulators, all currently working, over half exhibited isolated pleural plaques, reported shortness of breath on exertion and had an average decrease in forced expiratory volume in one second (FEV₁) and FVC of 200 and 300 ml, respectively.³⁴ These reductions could not be attributed to radiographic and/or subradiographic fibrosis, evidence that isolated pleural plaques may reduce ventilatory function significantly.³⁴

Diffuse pleural thickening

Diffuse pleural thickening is characterised by fibrosis of the parietal pleura which frequently extends to visceral pleura. The 1980 ILO Classification for Chest Radiographs for Pneumoconiosis states that pleural thickening should be considered as diffuse 'only in the



Figure 3 A 44-year-old non-smoking asymptomatic male with an 18-year history of exposure to asbestos in the asbestos cement industry. *Top*: Chest radiograph revealed ill-defined, small patchy opacifications on the right lower lobe: a diaphragmatic plaque was also suspected. *Bottom*: A sagittal reconstruction of the thorax CT scan (with contrast) shows a rounded atelectasis in the left lower lobe and a partly calcified diaphragmatic pleural plaque on the right. CT = computed tomography.

presence of and in continuity with, an obliterated costophrenic angle'.^{1,17} Thickness of at least 3 mm is required in the 2003 ILO classification for reporting pleural thickening.¹⁷ Involvement of the visceral pleura is suggested by the presence of fibrous strands extending into the parenchyma ('crow's feet').³²

Benign pleural effusion

Pleural effusions may occur early or late after the onset of asbestos exposure. They may last several months, be exudative and are often haemorrhagic. While most are asymptomatic, some present with fever and severe pleuritic pain. The clinical course is variable, and recurrence can occur on the same or the opposite side.^{24,32} A blunted costophrenic angle or diffuse pleural thickening are often the residua of acute pleural effusions. Diagnosis is usually by exclusion: fluid cytology is useful for distinguishing benign from malignant effusions.

Rounded atelectasis

Rounded atelectasis (shrinking pleuritis, Blesovsky's syndrome or folded lung) develops from infolding of thickened visceral pleura with collapse of the intervening lung

parenchyma (Figure 3). These lesions may be multiple and frequently resemble masses.^{28,32} A classic 'comet sign' can be seen on HRCT or plain films. Histological examination shows folded and fibrotic visceral pleura with atelectasis and variable amounts of chronic lung inflammation. The sudden appearance of rounded atelectasis may follow acute pleuritis with effusion.

Asbestosis

Asbestosis is characterised by pulmonary parenchymal fibrosis, in the presence of an exposure history of appropriate duration, latency (usually two decades) and intensity. Histologically, the term asbestosis refers to interstitial fibrosis caused by the deposition of asbestos fibres in the lung.²⁴ Clinical features include breathlessness, inspiratory crackles and pulmonary function abnormalities (a restrictive or mixed restrictive-obstructive pattern, decreased DL_{CO} and, in advanced cases, pulmonary gas exchange disturbances during exertion). The ILO classification remains the preferred tool to identify subjects with asbestosis:^{1,17} a profusion of irregular opacities at the level of 1/0 is commonly used as the threshold to indicate disease.²⁴

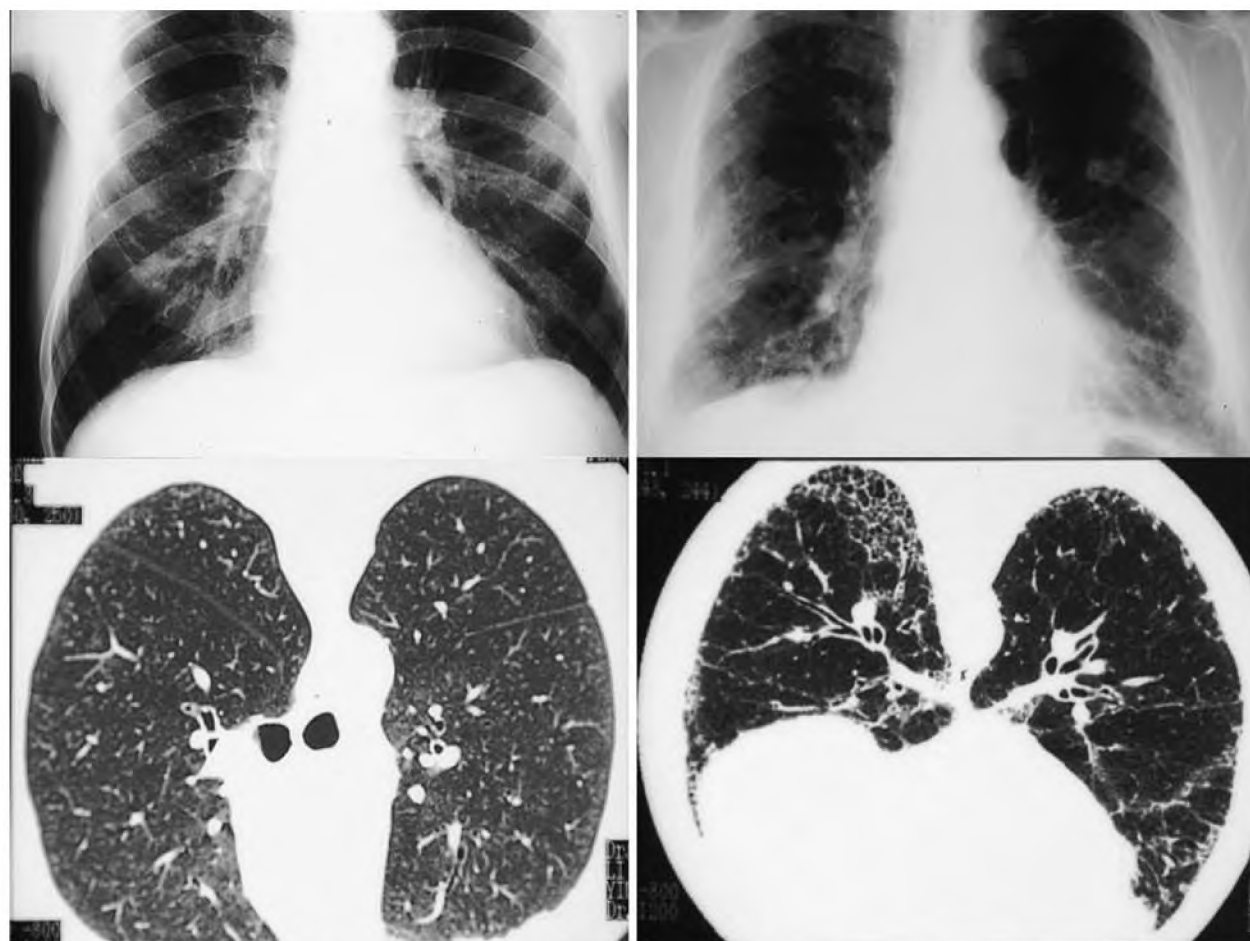


Figure 4 Chest radiograph and HRCT of two ex-workers from the asbestos cement industry with mild and advanced asbestosis (*left* and *right*, respectively). More extensive changes are seen on the HRCT scan (*bottom*) than on the chest X-ray (*top*), including parenchymal bands, honeycombing and traction bronchiectasis. Note that the opacities are mostly nodular, suggestive of silicosis but compatible with exposure in the asbestos cement industry. HRCT = high resolution computed tomography.

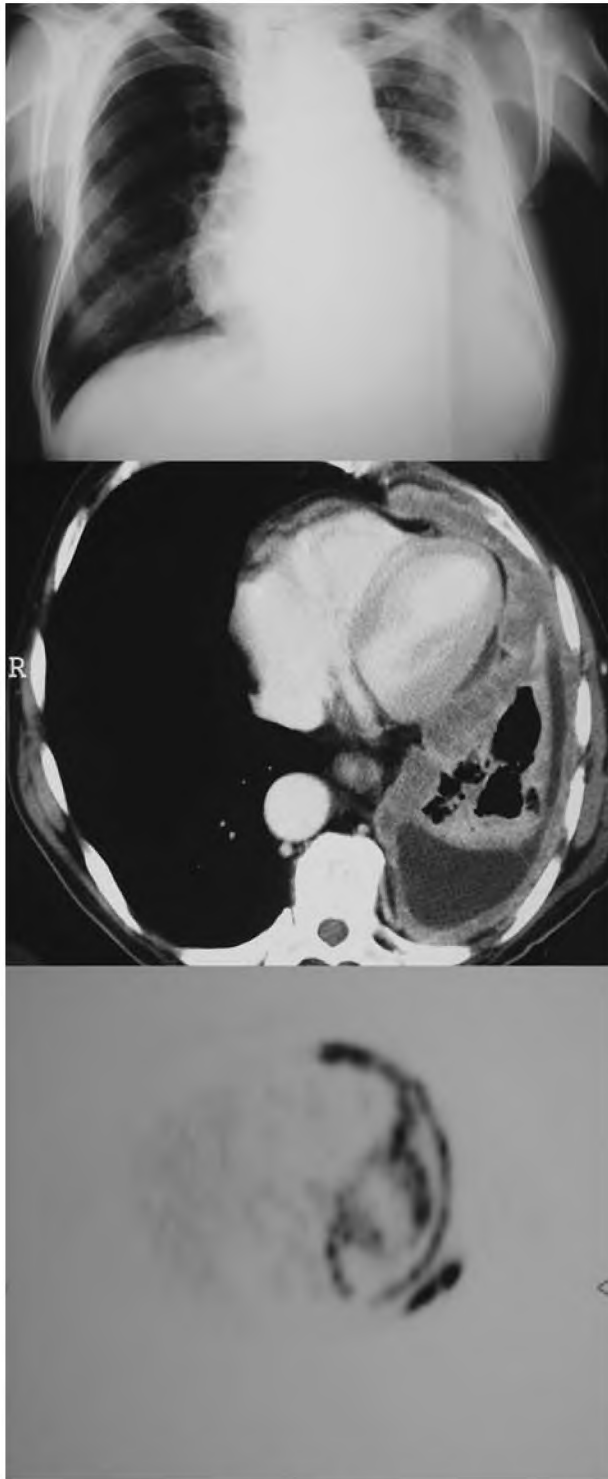


Figure 5 A 63-year-old non-smoking male who worked for 5 years in the asbestos cement industry (1960–1965) presented with a 3-month history of breathlessness and chest pain. **A.** Chest radiograph showed extensive opacification of the lower left lung fields. **B.** HRCT scan revealed a pleural effusion with increased pleural thickness. **C.** A combined F-18 fluorodeoxyglucose (FDG) PET/CT scan showed increased local soft tissue retention of the tracer, which is highly suggestive of malignancy. An open biopsy confirmed the diagnosis of epithelial mesothelioma. (Courtesy of Prof M Terra-Filho, Division of Respiratory Diseases, University of São Paulo, Brazil.) HRCT = high-resolution computed tomography; PET = positron emission tomography; CT = computed tomography.

HRCT is often useful in clarifying the exact nature of uncharacteristic or complex abnormalities seen on conventional chest X-ray (Figure 4).

MALIGNANT ASBESTOS-RELATED DISEASE

Malignant mesothelioma

Malignant mesothelioma is a hard white tumour that characteristically encases the lung.³⁵ In its early stages it may appear as white nodules studding the pleura and diaphragm. There is some evidence that these tumours start on the parietal surface. Histologically, there are three cell types: epithelial (Figure 5), mixed cell and sarcomatous, distinguished by histochemical reaction.³⁵ The median survival time for these cell types is 18, 11 and 8 months, respectively.

Lung cancer

Asbestos-related lung cancers were first considered to be scar cancers.²⁴ This changed as more data became available on the fibre burden in workers with other types of asbestos-related disease. For example, fibre concentrations in lungs of Quebec chrysotile miners and millers dying of asbestosis were shown to be on average over two-fold higher than in those dying of lung cancer (30 vs. 13 million chrysotile fibres per gram of dry lung tissue).⁵ All lung cancer cell types have been associated with asbestos exposure, further supporting the concept of exposure playing a direct role in their genesis, independent of smoking.³⁶

Other asbestos-related cancers

Other asbestos-related cancers include laryngeal cancer, for which evidence of a causal association is considered reasonably strong,³⁵ and oesophageal cancer, for which the evidence is suggestive. For other cancers, such as ovarian cancer, renal cancer and lymphoma, the evidence is less conclusive, although the possibility that these rare tumours may be caused by asbestos should not be dismissed.³⁶

EPIDEMIOLOGY OF ASBESTOS-RELATED DISEASES

Recent trends in industrialised countries

Trends in mesothelioma mortality in men have been analysed in a number of industrialised countries, to assess the status of the current mesothelioma epidemic and predict its future course³⁷ (Table 2).

In Britain, death certified as malignant mesothelioma rose steeply in men by birth cohort from 1893 to 1948, then fell.³⁸ Despite the fall, the authors of the study predict that the rate will continue to rise until about 2020, based on the assumption that the profile of deaths will not change over the next two decades.³⁸ This assumption does not account for improved exposure controls in workplaces in the last two decades

Table 2 Mesothelioma incidence in men: recent trends in selected studies carried out in industrialised countries (by year)

First author, year, reference	Source	Periods compared	Rates	Authors' comments
Peto J, 1995 ³⁸	UK Health & Safety Register	1943–1948 1948–1953 1953–1958 (age cohorts)	RR 1.00 RR 0.79 RR 0.46	Despite falling rates, annual deaths are unlikely to peak before 2010 and more likely to peak in the 2020s
Peto J, 1999 ³⁹	WHO database (1992)	1998 2018 (birth cohort 1945–1950)	5000/year ~9000/year (6 countries)	Highest risks for men born around 1945–1950. Asbestos use in Europe peaked in 1970s, remained high till the 1980s
Kjaergaard J, 2000 ⁴⁰	Danish Cancer Register	1943–1947 1983–1987 1988–1992	0.10/100 000 py 1.30/100 000 py 1.31/100 000 py	Rates peaked in the 1940–1944 age cohort. Effect of exposure regulation unlikely to occur before 2010–2015
Hemminki K, 2003 ⁴¹	Swedish Cancer Registry	1991–1995 1996–2000	1.82/100 000 1.79/100 000	Incidence stable, flattened earlier in younger cohorts
Ulvestad B, 2003 ⁴²	Norway Cancer Register	1965–1999	Rates rose to 16.6/million py	Cohort-specific rates for men born up to ~1935 increased, then seemed to stabilise
Montanaro F, 2003 ⁴³	European Cancer Registries	1978–1987 1988–2007 (21 countries)	Increase (significant) Deceleration started	Incidence still rising in some countries, deceleration has started in others.
Leigh J, 2003 ⁴⁴	Australian Registry, 1945–2002	1999 (data for age ≥20 years)	53.3/million/year	Time trend increase restricted to age ≥50 years, suggesting exposure controls in 1970s have been effective
Pelluchi C, 2004 ⁴⁵	WHO database (2003)	1970–1974 1985–1989	~8750 deaths ~9550 deaths	Data from France, Italy and Germany suggest mesothelioma deaths are levelling off
Weill H, 2004 ⁴⁶	SEER (NCI) USA	1973–1990 1990–1997	Increase (significant) Down slope (non-significant)	Outcome was age-adjusted incidence. Peak exposure was in the 1960s

UK = United Kingdom; RR = risk ratio; WHO = World Health Organization; py = person-years; SEER = Surveillance, Epidemiology and End Results Programme of the National Cancer Institute, USA.

of the twentieth century, nor for differences in mesothelioma risk associated with the different fibre types (chrysotile less than amphiboles, and amosite less than crocidolite).

A 1999 analysis of the European mesothelioma epidemic among men suggests that the rate will double between 1995 and 2018, then decline, with the greatest risk being seen in the cohort of men born between 1945 and 1950.³⁹ Asbestos use in Western Europe was high until 1980. These projections are based on a simple age-birth cohort model from 1970 to 1989 for six countries (Britain, France, Germany, Italy, Netherlands and Switzerland), which together include about three-quarters of Western Europe.³⁹

In Denmark, incidence rates of malignant mesothelioma in men peaked in the 1940–1944 cohort, then decrease in the 1950–1954 cohort.⁴⁰ The authors of this study expect a further increase to occur and that the effect of banning imports in 1979 will only become evident between 2010 and 2015.

A 2002 report based on an analysis of the Swedish Cancer Registry suggests that mesothelioma incidence in men has levelled off in that country.⁴¹ Age-standardised incidence rates fell from 1.82 per 100 000 population in 1991–1995 to 1.79/100 000 in 1996–2000; this occurred some 17 years after a drastic reduction in asbestos imports to Sweden.⁴¹ Sweden is

thus among the first countries to show a levelling-off in mesothelioma incidence.

An age-period-cohort model was used to analyse time trends for pleural mesothelioma in men in Norway.⁴² From 1965 to 1999, the annual number rose to 16.6 million person-years (py), after which rates seemed to stabilise. Importation of asbestos was banned in 1982, although imports fell for several years prior to that date. The authors consider that the delayed period effect of the ban is likely to have its greatest effect around 2010.⁴²

Geographic and temporal variations in mesothelioma incidence in men in Europe were examined in a 2003 report.⁴³ Data were extracted from 118 general cancer registries in 25 countries. Annual rates varied widely between countries, from about 8% in Britain and the Netherlands to less than 1% in Spain. Temporal rates also varied widely between countries, increasing significantly for all countries (except Denmark) between 1978 and 1987, and decelerating in the following 10 years in all countries except England and France. The authors expect the decline to continue over the next decade, given the decrease in asbestos exposure and the recent European Union ban on its use.⁴³

Time trends in malignant mesothelioma for the period 1945–2002 were the focus of a study in Australia.⁴⁴ Incidence rates for men aged ≥20 years were

53.3/million—according to the author, the highest reported in the world. In 90% of cases with no history of exposure, lung fibre counts longer than $2\ \mu$ exceeded 200 000/g of dry lung tissue, implying unrecognised exposure. The author attributes Australia's high incidence of mesothelioma to high past usage of all fibre types in many settings, and expects rates to rise until 2020.⁴⁴

In response to reports that incidence rates of mesothelioma in several European countries were levelling off, an update on the mesothelioma epidemic in Western Europe was carried out using the 2003 World Health Organization (WHO) mortality database.⁴⁵ The authors conclude that all data for the most recent period for France, Germany and Italy were lower than predicted, suggesting that mesothelioma deaths may be levelling off in much of Europe.⁴⁵

Trends in mesothelioma incidence in the United States were the focus of a 2004 publication, based on data gathered by the Surveillance, Epidemiology and End Results (SEER) programme of the National Cancer Institute of the National Institutes of Health.⁴⁶ For rates in men, a highly significant change in the upward trend was observed in the early 1990s. The authors note that the high risk for mesothelioma is prominently influenced by amphibole asbestos, usage of which peaked in the 1960s and then declined. A gradual decline in US mesothelioma cases can be expected, thus reducing the ill-health and economic burden of this lethal asbestos-related tumour.⁴⁶

Recent trends in less-industrialised countries

Asbestos production and consumption is growing in many less-industrialised countries.⁴⁷ Of the six leading producers, five are considered less industrialised (Table 3). However, the literature on the epidemiology of asbestos-related diseases in these countries is sparse.

Russia and Kazakhstan

These two republics from the former Soviet Union are today the leading producers of asbestos (Table 3). A 1999 Finnish-American-Russian project set out to develop a comprehensive health and exposure surveillance plan for Siberian asbestos miners. In a prelimi-

nary report, the authors found that pulmonary chrysotile concentrations in 47 unselected autopsies (24 chrysotile workers and 23 long-term residents of Asbest Town) were about the same as those reported previously in Canadian chrysotile miners and millers.⁴⁸ However, the mean concentration of tremolite fibres was less by at least one order of magnitude.

More recently, an update on trends in the Russian Federation was presented at the International Conference on Chrysotile, held in Montreal, Canada, in 2006.⁴⁹ Russia is currently the world's largest producer and consumer of chrysotile asbestos, and Sverdlovsk province is the most industrialised region of the Russian Federation and the site of the Bazhenovskoye mine, the world's largest chrysotile asbestos deposit.⁴⁹ Mesotheliomas have been diagnosed in 32 of 66 municipal locations of Sverdlovsk province. In areas of asbestos industrial facilities, mesothelioma incidence ranges from 4.3 cases/million (chrysotile asbestos), to 7.1/million in the Sysert area (anthophyllite-asbestos province of Urals), to 27.1/million for crocidolite asbestos. Mesothelioma-related mortality in Asbest Town, excluding cases with occupational exposure, was 1.4/million. In Sukhoy Log City and Beloyarskiy village, where factories producing asbestos cement and cardboard are located, there is no excess mesothelioma incidence.⁴⁹

China

China is the world's third leading producer of asbestos (Table 3): 95% of the Chinese production is chrysotile. In many mines and manufacturing sites, air contamination exceeds the national standard of 2 mg/m³ of total dust. Amphibole fibres (mainly tremolite) were also found in all of 10 bulk samples from six chrysotile mines: this finding may explain the excess rate of lung cancer and mesothelioma among Chinese chrysotile workers.⁵⁰

Much of the recent Chinese literature relates to environmental exposure to crocidolite in surface soil of a rural county, Da-Yao (south-western region). Since 1988, three large epidemiological studies have been conducted in this area (Table 4).^{51–53} These studies, summarised in a 2003 report,⁵⁴ showed a progressive increase in the annual mortality rate for mesothelioma, from 85/million (1977–1983) to 178/million (1987–1993) to 365/million (up to 2003). No mesotheliomas were found in the populations living in areas where crocidolite was not known to exist in the environment. However, as commercial use of crocidolite was banned only in 1984, the incidence of mesothelioma is expected to peak in this area and other areas of China in the next few years.⁵⁴

Brazil

Brazil is the world's fifth leading producer of asbestos (Table 3). Large-scale mining and milling activities

Table 3 Asbestos production by country (per 1000 tons)

	2000	2004	2005*
Russia	752	875	875
China	350	355	360
Kazakhstan	179	347	350
Canada	320	200	240
Brazil	209	195	195
Zimbabwe	152	150	100
Other countries	88	110	80
World total	2050	2232	2200

Source: US Geological Survey, Mineral Commodity Summaries.

* Estimated.

Table 4 Mesothelioma incidence in men: recent trends in selected studies carried out in less-industrialised countries (by country and most often by year)

First author, year, reference	Country	Asbestos type/ exposure site	Mesothelioma rates, fibre level in lung	Authors' comments
Tossavainen A, 2000 ⁴⁸	Russia (Asbest Town)	Autopsy material on 24 chrysotile workers and 23 Town residents	Chrysotile workers: chrysotile, 0.8–50.6 f/g amphibole, 0.1–1.9 f/g Town residents: chrysotile, 0.1–14.6 f/g amphibole, 0.1–0.7 f/g	Lung chrysotile levels in Russia about the same as in Canadian miners, but tremolite levels less by an order of magnitude
Kashansky S V, 2006 ⁴⁹	Russia (Sverdlovsk Region)	Chrysotile mine Bashenofskuye Amphibole mine Crocidolite mine Asbest Town	4.3/million 7.1/million 27.1/million 1.4/million	In Sverdlovsk province, trend towards reduction in mesothelioma incidence
Tossavainen A, 2001 ⁵⁰	China	Chrysotile mine dust (10 bulk samples from 6 mines)	Lung fibre levels in 7 deceased miners were: anthophyllite, 71 % chrysotile, 10 % tremolite, 9 %	All 10 bulk samples contained tremolite, one contained anthophyllite. Lung fibre levels show faster clearance of chrysotile vs anthophyllite
Liu X, 1990 ⁵¹	China (South West)	Crocidolite ubiquitous in soil in Da-Yao county	Of 2175 local residents studied, 16 had asbestosis and 232 pleural plaques	Crocidolite had been widely used for road pavements, stoves and wall paint
Luo S O, 1997 ^{52*}	China (Da-Yao Region)	Crocidolite	178/million/year	Cohort study ($n = 4598$) of villagers; lung cancer RR = 2.14
Zhou Y, 1998 ^{53*}	China (Da-Yao Region)	Crocidolite	1987–1995 = 79 cases 1996–1999 = 87 cases Estimated mesothelioma rate for Da-Yao county, 24/million/year	Case series from a single hospital
Luo S, 2003 ⁵⁴	China (Da-Yao Region)	Crocidolite in the soil of Da-Yao county, South West China	Mesothelioma mortality: 1984–1995 85/million/year 1996–1999 178/million/year to 2003 365/million/year in a population of 68 000	Commercial use of crocidolite banned since 1984. As mesothelioma latency is ~30–40 years, the ban had little effect in the 1990s
Zwi A B, 1989 ⁶⁰	South Africa (1976–1984)	Case registry by medical practitioners, institutions	Mesothelioma incidence 32.9, 95% CI 22.7–46.4, standardised rates/million population aged ≥ 15 years/year	Rates among the highest for a national population; likely an underestimate; legislated controls fewer in South Africa than in many countries
Sluis-Cremer G K, 1992 ⁶¹	South Africa	Crocidolite and amosite mines (1945–1955)	Mesothelioma incidence crocidolite, 44.6/100 000 py amosite, 7.8/100 000 py	Crocidolite more dangerous than amosite. Crocidolite-induced mesothelioma seen after only 1–15 years exposure
Kielkowski D, 2000 ⁶²	South Africa	Crocidolite mining/milling. Birth cohort of white residents (1916–1936)	Cause-specific mortality, mesothelioma, 32.9/million py	Exposure environmental as well as occupational. Given the long latency, mesothelioma rates likely to increase over next 10 years until 2010
Mutetwa B, 2006 ⁶³	Zimbabwe	Two chrysotile mines: Shibanie, Gaths	Between 1970 and 2001, 36 mesothelioma cases identified; exposure information scanty or non-existent	Mines use an action level of 0.5 f/cm ³ vs. a WHO level of 1.0 f/cm ³ . In 2004, 94.5% of Shibanie samples were <0.3 f/cm ³
Szeszenia-Dabrowska N, 1998 ⁶⁴	Poland (rural south- east region)	Factory using chrysotile from 1985 (for roofing, siding), and crocidolite from 1987 (for pressure pipes)	In a cohort of 1526 asbestos cement workers followed 1959–1996: SMR for mesothelioma: 80-fold excess SMR for colon cancer: 3-fold excess	The very high risk for mesothelioma exhibited by this cohort attributed to non-occupational exposure (use of asbestos cement waste for surfacing farmyards, roads, paths, sports fields, buildings)
Gaafar R M, 2005 ⁶⁵	Egypt	14 asbestos factories; two surrounded by residential areas	1989–1999, 148 cases 2000–2003, 635 cases (an increase of cases diagnosed in 3 Cairo hospitals from 0.47 % to 1.3 %)	Residential exposure experienced by 64.7% (mainly in Shobra El Khaymah, and in Helwan, El-Maasara and surrounding areas)
Emri S, 2004 ⁶⁶	Turkey (south-east region)	Tremolite, chrysotile and anthophyllite; also erionite in Cappadocia	Estimated incidence of mesothelioma in the SE region 43/million, and 996/100 000 in erionite villages	Environmental exposure from stucco, paint and whitewash on walls, floors, ceilings, etc., comparable to occupational exposure

(continued)

Table 4 (Continued)

First author, year, reference	Country	Asbestos type/ exposure site	Mesothelioma rates, fibre level in lung	Authors' comments
Dutta M, 2003 ⁶⁷	India, West Jharkhand	Abandoned chrysotile mine dump in Roro hills	Survey of 252 subjects (45% ex-miners) from 14 villages showed: 17.5% had breathing difficulties, 7.1% had haemoptysis.	Careless closure of the mine resulted in waste slipping down into paddy fields. In each monsoon waste runs off into villages and paddy fields
Murlidhar V, 2005 ⁶⁸	India (Mumbai)	Factory making textiles, brake/clutch linings, asbestos fibre	Of 181 workers examined, asbestosis diagnosed in 22%.	Prevalence of asbestosis less than expected, suggesting a 'healthy' worker effect
Joshi T K, 2004 ⁶⁹	India	Exposure to asbestos occurs in mining, milling and manufacturing sectors	Except in the mining sector, mesothelioma is not a notifiable occupational disease in India. Asbestos use not regulated in factories or construction	In India, most manufactured products are consumed domestically. Local production has decreased due to large-scale imports from Canada

* Article in Chinese but with English abstract.

f/g = fibre per gram; RR = risk ratio; CI = confidence interval; py = person-years; f/cm³ = fibres per cm³; WHO = World Health Organization; SMR = standardised mortality rates.

started in the early 1940s. A single company has been responsible for asbestos exploration (primarily chrysotile) in Brazil: from 1940 to 1967 in the north-east state of Bahia, and thereafter in the central state of Goias. In a retrospective study of 3634 past and present workers, radiographic abnormalities and lung function impairment declined significantly (three to five fold) over time as exposure was progressively reduced from between 16 and 19 fibres/cm³ to approximately 1 fibre/cm³ (in asbestos grinding, drilling, mixing and sacking operations).⁵⁵

Another important source of occupational exposure to asbestos in Brazil is the fibre-cement industry. In a cross-sectional study of 828 compensation claimants with long-term exposure, the authors found a prevalence of 8.9% for asbestosis and 29.7% for pleural thickening.⁵⁶ Pleural thickening was also independently associated with chronic bronchitis and shortness of breath.

Less is known about the malignant effects of asbestos exposure. In a retrospective study conducted in the state of Rio de Janeiro, 83 cases of pleural mesothelioma were identified among death certificates coded as pleural tumours.⁵⁷ However, their asbestos exposure could not be retrospectively ascertained.

South Africa

South Africa has a long history of asbestos mining; chrysotile, amosite and crocidolite deposits have all been exploited. Blue asbestos (crocidolite) was identified in the Northern Cape by explorers in the early 1800s.⁵⁸ The link between asbestos and mesothelioma was first reported in a case-series from the North-western Cape.⁵⁹ Mining peaked in the late 1970s, with more than 20 000 miners being then employed in the industry.⁵⁸ A study in the 1980s suggested that there had been a steady increase in annual mesothelioma incidence in white males aged ≥ 15 years from 27.6/million in 1976 to 40.5/million in 1984 (Table 4).⁶⁰ A

1992 study showed a high incidence (44.6/100 000 py) in a cohort of 1800 white men exposed only to crocidolite at work, compared to a much lower incidence (7.8/100 000 py) in a cohort of 1810 white men exposed only to amosite at work.⁶¹ Respective proportional mortality rates for mesothelioma for the two cohorts were 11.9% and 1.7%. The incidence of mesothelioma in black workers is likely to have been much higher. In a birth cohort study of white men, residents of Prieska, a small town close to a crocidolite mine productive from 1893 to the late 1960s, cause-specific mortality for mesothelioma was 32.9 (95% confidence interval 22.7–74.6) per million py.⁶² The authors point out that, given its long latency, the mesothelioma-related mortality can be expected to increase throughout the current decade.

Zimbabwe

Zimbabwe ranks sixth in the order of world production, and produced 152 000 tons of chrysotile asbestos in 2004 (Table 3), of which 83% was exported. Chrysotile is mined at the Shibanie and Gaths mines, which employ approximately 5000 persons and support more than 20 000 persons in the mining towns of Zvishawe and Moshawa.⁶³ Chrysotile manufacturing also supports over 1500 persons; over 60 000 families depend on downstream industries, as do 300 000 in the construction industry, which uses asbestos cement products in housing, water reticulation, sanitation and irrigation. The mines use an action level of 0.5 fibres/cm³, against a WHO level of 1 fibre/cm³. In 2000, 94% of samples from Shibanie mine were < 0.3 fibres/cm³ (Table 4). Thirty-six mesothelioma cases were identified between 1970 and 2001; however, exposure information was scanty or non-existent in the case files reviewed.⁶³ The author of this study concludes that chrysotile is a low-cost product, as safe as any of the substitutes available, and can be mined safely using today's technology.⁶³

Other less-industrialised countries

In a 1998 Polish report on 1526 workers in an asbestos cement factory, standardised mortality ratio (SMR) analysed using the man-year, method identified 16 cases of pleural mesothelioma; four cases had a short exposure/latency period (Table 4).⁶⁴ Additional investigations revealed that available asbestos-cement waste had been used for road surfacing.

Asbestos was used in Egypt from 3000 to 2000 BC to embalm the bodies of the Pharaohs.⁶⁵ Of the 14 factories in the country today, one began operation in 1927 before people lived in the area but is now surrounded by residential areas exhibiting a high rate of mesothelioma. Case series from two Cairo hospitals have documented an increase in mesothelioma rates from 148 cases in 1989–1999 to 635 cases in 2000–2003 (Table 4).⁶⁵

A 2004 study from Turkey estimated the incidence of malignant pleural mesothelioma at 43/million in the south-east part of the country.⁶⁶ Also, 996/100 000 inhabitants of villages in Cappadocia use erionite in their homes as a whitewash or stucco for walls, floors and roofs, and as a substitute for baby powder. A survival advantage was shown for asbestos-induced versus erionite-induced mesothelioma (Table 4).⁶⁶

In India, exposure to asbestos occurs in mining/milling, the asbestos-cement industries, civil construction and electro-mechanical appliances.^{67–69} Domestic production has decreased due to large-scale importation from Canada. India is also a high consumer of asbestos, and most products manufactured are used in the country.⁶⁹ A recent (2005) study found a prevalence of 22% for asbestosis among 181 workers of an asbestos composite mill in Mumbai.⁶⁸ The authors note that fewer than 30 cases in the country have received compensation for asbestosis. In an observational study in the small town of Roro, Eastern India, the impact on the environment and the community of careless closure of an asbestos mine in 1983 was examined.⁶⁷ With each monsoon, the waste from the dump runs off into fields, villages and streams. The older age groups who worked in the mines in the past reported shortness of breath and haemoptysis (Table 4).

CLINICAL MANAGEMENT, EXPOSURE CONTROL AND PREVENTION

Clinical management of asbestos-related disease is no different from clinical management of these conditions in the absence of a history of asbestos exposure.⁵ However, because asbestos-related disease is work-related or potentially work-related, physicians should make the appropriate notification of disease according to the jurisdiction in their local practice area. Advice concerning continuing employment in a job with continuing exposure should be based on our knowledge of its impact on the natural history of asbestos-related disease and the risk of developing new disease after

leaving a job that involved exposure,⁵ including chronic airflow limitation (chronic obstructive pulmonary disease).²⁹ No treatment (medical or surgical) has been shown to influence the clinical course of asbestosis, which appears to be more resistant than other forms of pulmonary fibrosis, perhaps because the agent (particularly if it is an amphibole) persists in the lung after exposure ceases.⁵

For individuals with a past exposure history, there is encouraging news.⁵ Only a small minority of those exposed are likely to show changes on X-ray, and only a small minority of those with X-ray changes are likely to develop clinical evidence of disease.⁵ Smoking cessation is likely to benefit an individual with past exposure more than one without because of the multiplicative interaction between smoking and asbestos.

For non-malignant pleural disorders, the evidence is now reasonably strong that subjects with pleural fibrosis (pleural effusion seems to be a step in the development of pleural fibrosis) are at greater risk for developing asbestosis.⁵ Advice against continuing in a job involving asbestos exposure (even in workplaces that respect the exposure limits) is probably sound, although it may be difficult for workers to accept, especially if it comes late in their working careers when retraining is not easy to find or undertake.

Exposure control is key to prevention, and is important in the management of asbestos-related disease. The last decades of the twentieth century saw workplace dust levels decline and work practices improve (e.g., laundering of work clothes on-site) in many industrialised countries and some less-industrialised countries.⁵ There was also a decline in rates of non-malignant and malignant asbestos-related disease in many industrialised and some less-industrialised countries: the study from Brazil⁶³ is an example of how, with decreasing exposure, radiological markers decreased and lung function levels improved.

A ban on asbestos was introduced to control the worldwide epidemic of asbestos-related disease,⁵ mainly in industrialised countries without asbestos mining and milling activities.⁴⁷ By contrast, in less-industrialised countries, which account for the bulk of the world's use of asbestos, there is concern about the lack of affordable and equally effective substitutes. It has been argued that chrysotile has made it possible for many around the world, particularly those in low-income countries, to have clean, safe water supplies and affordable housing.⁶³ This could change with the further development of waste plant-fibres, coupled with evidence that they are as effective as chrysotile (particularly in cement, friction and insulation products).⁷⁰

Clearly, wherever chrysotile is used, management of this workplace hazard requires collaboration between workers, unions (responsible for monitoring dust levels in the workplaces, to which they must have access) and companies (responsible for engineering controls within its operation and in the surrounding environ-

ment), that is supported by appropriate government regulation, including laws on cleaning up dumps following the closure of mines and/or factories, and always with community support.

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R É S U M É

L’amiante est un terme descriptif concernant un groupe de minéraux existant dans la nature et connus par l’humanité depuis l’antiquité. Les principaux types d’amiante (chrysotile, et les amphiboles crocidolite et amosite) sont différents en ce qui concerne la structure chimique, la biopersistance dans les tissus humains et la toxicité. Une exploitation commerciale, ne prenant guère les problèmes environnementaux en considération, a augmenté au cours du dernier siècle, particulièrement après la deuxième guerre mondiale, pour répondre à la globalisation et aux besoins des cités en expansion dans le monde. Au fur et à mesure que ses effets de santé, qu’ils soient non-malins (fibrose pulmonaire ou asbestose, épanchement,

plaques ou épaississement de la plèvre) ou malins (mésotéliome, cancer du poumon et autres cancers), sont devenus évidents, la pression du public pour le contrôle de son utilisation s’est accrue. Les dernières décennies du siècle dernier ont vu une décroissance de l’exposition et des taux d’asbestose dans les pays industrialisés et dans quelques pays moins industrialisés où les plaques pleurales et les mésotéliomes pleuraux sont actuellement les manifestations les plus fréquentes de l’exposition à l’amiante. Un suivi plus prolongé des cohortes exposées à l’amiante dans les mines ou les manufactures a également renforcé les preuves d’un gradient dans la toxicité des fibres, les chrysotiles montrant une toxicité plus faible que

les amphiboles et l'amosite plus faible que la crocidolite. Dans les dernières décennies du siècle dernier, on a observé une stabilisation et/ou une décroissance des taux de mésothéliome dans plusieurs pays industrialisés. Dans les pays moins industrialisés, les données sur la maladie sont médiocres, l'exposition généralement élevée et les taux pourraient connaître leur sommet à l'avenir. La prise en charge des maladies liées à l'amiante au niveau

des lieux de travail exige une collaboration entre les travailleurs, les syndicats (responsables du suivi des taux de poussières dans les lieux de travail, auxquels ils doivent avoir accès), et les sociétés (responsables des contrôles de la technique) ; ces actions doivent être renforcées par des règlements gouvernementaux appropriés et par le soutien de la collectivité.

RESUMEN

Amianto es un término descriptivo que agrupa minerales que se encuentran naturalmente y que el hombre conoce desde tiempos muy antiguos. Los principales tipos de fibras de amianto (el crisólito y las formas anfibólicas crocidolita y amosita) difieren con respecto a su estructura química, su persistencia en los tejidos humanos y su toxicidad. La explotación comercial, con escasa consideración de las regulaciones medioambientales, aumentó durante el siglo pasado, en particular después de la Segunda Guerra Mundial, con el propósito de adaptar la globalización a la demanda de las ciudades en expansión a través del mundo. Cuando fueron evidentes sus efectos perjudiciales sobre la salud, tanto benignos (fibrosis pulmonar o asbestosis ; derrame, placas y engrosamiento pleurales) como malignos (mesotelioma, cáncer de pulmón y otros tipos de cáncer), se acrecentó la presión pública por una regulación de la utilización del amianto. En los últimos decenios del siglo pasado tuvo lugar una disminución de la exposición y de la incidencia de asbestosis en los países industrializados y en algunos menos industrializados, donde las placas pleurales y el mesotelioma maligno constituyeron las manifestaciones más fre-

cuentes de la exposición al amianto. Asimismo, el seguimiento a más largo plazo de cohortes expuestas al amianto en la industria minera y manufacturera ha consolidado las pruebas sobre un gradiente de toxicidad de las fibras, según el cual la toxicidad del amianto crisólito es inferior a la de las formas anfibólicas y entre ellas, la amosita es menos tóxica que la crocidolita. En los últimos decenios del siglo pasado se observó una estabilización o una disminución de la incidencia de mesotelioma en varios países industrializados. En países menos industrializados, los datos sobre esta enfermedad son escasos, la exposición suele ser alta y su incidencia puede alcanzar puntos máximos en el futuro. El control de las enfermedades causadas por el amianto en el puesto de trabajo precisa la colaboración entre los trabajadores, los sindicatos (encargados de supervisar los datos sobre la concentración de polvo en los lugares de trabajo, a los cuales tienen que tener acceso) y las empresas (que tienen a su cargo los controles técnicos) reforzada por reglamentaciones gubernamentales apropiadas y por el apoyo de la comunidad.