Asbestos

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CAS No. : 1332-21-4 (unspecified), 12001-29-5 (chrysotile), 12001-28-4 (crocidolite), 12172-3-5 (amosite).

Copyright: LAS Ltd. What is asbestos and how to deal with it. Steven Lea, July 21, 2016. https://las-asbestos.co.uk/

Asbestos is the generic commercial designation for a group of naturally occurring mineral silicate fibers of serpentine and amphibole series. These include the serpentine mineral chrysotile (also known as 'white asbestos'), and the five amphibole minerals – actinolite, amosite (also known as "brown asbestos"), anthophyllite, crocidolite (also known as 'blue asbestos'), and tremolite.

The silicate tetrahedron (SiO_4) is the basic chemical unit of all silicate minerals. The number of tetrahedra in the crystal structure and how they are arranged determine how a silicate mineral is classified.

Serpentine silicates are classified as 'sheet silicates' because the tetrahedra are arranged to form sheets. Amphibole silicates are classified as 'chain silicates' because the tetrahedra are arranged to form a double chain of two rows aligned side by side.

The structure of silicate minerals may be fibrous or non-fibrous. The terms 'asbestos' or 'asbestiform minerals' refer only to those silicate minerals that occur in polyfilamentous bundles, and that are composed of extremely flexible fibers with a relatively small diameter and a large length. These fiber bundles

have splaying ends, and the fibers are easily separated from one another. Asbestos minerals with crystals that grow in two or three dimensions and that cleave into fragments, rather than breaking into fibrils, are classified as silicate minerals with a 'non-asbestiform' habit [IARC].

Usage and exposure

Asbestos has been used intermittently in small amounts for thousands of years. Modern industrial use dates from about 1880, when the Quebec chrysotile fields began to be exploited. During the next 50 years gradual increases in production and use were reported with a cumulative total of somewhat less than 5000 million kg mined by 1930. By the time industrial and commercial use of asbestos peaked, more than 3000 applications or types of products were listed. Production and consumption of asbestos has declined in recent years due to the introduction of strict regulations governing exposure and/or outright bans on exposure.

Asbestos is used as a loose fibrous mixture, bonded with other materials (e.g. Portland cement, plastics and resins), or woven as a textile. The range of applications in which asbestos has been used includes: roofing, thermal and electrical insulation, cement pipe and sheets, flooring, gaskets, friction materials (e.g. brake pads and shoes), coating and compounds, plastics, textiles, paper, mastics, thread, fiber jointing, and millboard.

Since peaking in the 1970s, there has been a general decline in world production and consumption of asbestos [IARC].

Asbestos products were banned in all the countries of the European Union and in Israel.

Occupational exposure occurs in the mining and milling of asbestos (or other minerals contaminated with asbestos), the manufacturing or use of products containing asbestos, construction, automotive industry, the asbestos-abatement industry (including the transport and disposal of asbestos-containing wastes) [IARC].

Inhalation is the primary route by which the general population might be exposed to asbestos. Small quantities of asbestos fibers are ubiquitous in air, arising from natural sources (weathering of asbestoscontaining minerals), from windblown soil from hazardous waste sites or breakdown of asbestos-

containing materials such as insulation (mainly chrysotile). The results of numerous measurements indicate that average concentrations of asbestos in ambient outdoor air are within the range of 10-8– 10-4 PCM f/mL [ASTDR].

Fiber size

The size (length and diameter) of an asbestos fiber appears to be one of the most important determinants of its toxicity. Fiber size dictates respirability, deposition, and clearance from the lung. In general, only fibers less then 3 µm thick are capable of reaching lower airways. Fibers longer than approximately 5–10 µm are generally cleared more slowly than fibers shorter than 5 µm. The maximum fiber length that can be engulfed by a single macrophage is approximately 16–17 µm.

Ultimately, the size of the fiber determines its residence time in the lung. Longer fibers remain in the lung or mesothelium, whereas shorter fibers are cleared. Fibers with lengths >15–20 µm are incompletely ingested and dissolved by pulmonary macrophages, which is thought to lead to chronic and persistent inflammation and tissue damage.

Asbestos-associated diseases are attributable to fibers of different sizes. The strongest evidence for this conclusion comes from studies in animals, where chronic inhalation exposure to dust clouds rich in long fibers (those in excess of 5 µm) produces higher incidence of lung cancer than exposure to dust clouds rich in short fibers [ASTDR].

Fiber durability

Fiber biopersistance is believed to be a major mechanism of fiber-induced pathogenicity. Numerous studies have indicated that some asbestos fibers, particularly chrysotile fibers, undergo fragmentation (latitudinal breakage).

Fiber fragmentation results in shorter fibers, which are more readily cleared from the lungs by alveolar macrophages.

Differences in fiber durability may account for the differences observed in fiber potency between chrysotile and amphiboles.

Fiber type

A diversity of opinion exists regarding relative potencies of various asbestos fiber types with respect to fibrogenicity and carcinogenicity. Some investigators have proposed that amphibole fibers, such as tremolite, are

more potent than chrysotile fibers in inducing fibrotic lung disease and lung cancer. Others have suggested that the differences in the potency of chrysotile and amphibole fibers in inducing lung cancer cannot be reliably discerned from available data.

It is generally agreed that exposure to amphibole fibers can produce mesothelioma, and that the potency of amphibole fibers to produce mesothelioma is greater than that of chrysotile [ASTDR].

Asbestos Cement

Asbestos cement is usually a mixture of about 10% asbestos and 90% Portland cement. The types of asbestos used may vary, chrysotile is normally always present but crocidolite and amosite were also added to many products.

In asbestos cement most of the chrysotile fibers are encapsulated in the cement. When the cement is broken or crushed the chrysotile fibers are released from the cement.

However, the risk of exposure to airborne asbestos fibers is lower in asbestos cement products compared with some other asbestos containing products due to:

- The relatively lower amount of asbestos used in this product compared to others;
- the hard resistant nature of the cement matrix which makes it more difficult to release airborne fibers;
- the high use of chrysotile asbestos compared to amosite and crocidolite asbestos [Burgett].

Routs of exposure

Epidemiological studies of asbestos-exposed workers and supporting animal studies indicate that inhalation of asbestos is the principal route of exposure of public health concern.

Some epidemiological studies have also indicated that oral exposure may be linked to the development of gastrointestinal cancer.

Depending largely on size and shape, deposition of inhaled asbestos fibers may occur in lung tissue. Some fibers may be removed by mucociliary

clearance or macrophages while others may be retained in the lungs for extended periods. Inhalation exposure is, therefore, generally regarded as cumulative, and exposures have been expressed in terms of concentration of fibers over time or fiber-years/mL [ASTDR].

Health hazards

Studies in humans and animals indicate that inhalation exposure to asbestos fibers may lead to the development of pulmonary disease including asbestosis and/or lung cancer and mesothelioma of the pleura or peritoneum.

In general, noncancer effects in other tissues have not been detected; however, the development of cancer in other tissues (e.g., gastrointestinal tissues) in some worker populations may be related to asbestos exposure [ASTDR].

Pleural plaques

Pleural plaques represent circumscribed areas of fibrous thickening typically of the parietal pleura. In regions where plaques are not endemic, 80–90% of the plaques that are radiologically well-defined are attributable to occupational asbestos exposure [Consensus report].

The presence of pleural plaques does not imply the existence of other asbestos related diseases. On their own they do not usually cause symptoms or deficits in lung function. Minimum induction period is usually more than 10 years [Information notices].

Asbestosis

Asbestosis is defined as diffuse interstitial fibrosis of the lung as a consequence of exposure to asbestos dust. It is generally associated with relatively high exposure levels [Consensus report].

There is the evidence that the risk of developing asbestosis at cumulative exposures less than 25 fibers/ml^{-1}/year [1fiber/ml^{-1} for 25 years or equivalent] is low.

Both minimum duration of exposure and minimum induction period are evaluated as 5 years [Information notices].

Diagnostic criteria

There are no specific anatomo-pathological criteria for the diagnosis of asbestosis. The following criteria, together with a history of asbestos exposure, suggest the diagnosis of asbestosis and provide a basis for assessing its severity:

- Symptoms and signs: breathlessness; persistent bilateral late inspiratory basal crepitations; clubbing
- Chest X-ray: diffuse interstitial opacities (usually reticular or reticulonodular), mainly in the lower lung fields
- Computerized tomography: diffuse interstitial opacities mainly in the lower lung fields
- Lung function tests: restriction, reduction in gas transfer, decrease of the flow rates at low volume (flow-volume curve).

These features do not necessarily appear simultaneously, and the order in which they occur may differ from one subject to another.

At present in industrialised countries, most cases of asbestosis show up only on radiological examinations without progression to respiratory insufficiency. Early disease that is only visible on CT scanning requires expert radiological assessment [Informational notice].

Lung cancer

There is the evidence that the risk of developing lung cancer at cumulative exposures less than 25 fibers/ml^{-1}/years is low.

The risk is increased considerably by smoking.

Minimum duration of exposure is usually a few years. Minimum induction period is usually more than 15 years [Information notices].

Mesothelioma

80-90% of pleural mesotheliomas are attributable to occupational exposure to asbestos.

Smoking does not increase the risk.

Minimum duration of exposure usually a few years, but shorter exposures (as low as 3 months) have been described.

Minimum induction period is usually more than 20 years [Information notices].

There is the evidence that germline BAP1 mutation carriers are predisposed to the tumorigenic effects of asbestos and it is suggested that high penetrance of mesothelioma requires such exposure [Testa, Xu].

Carcinogenic effects – IARC evaluation

Mesothelioma: environmental and domestic exposure

An excess of mesothelioma has been observed in several studies of communities with environmental exposure to asbestos.

A large excess of mesothelioma was reported in a study of people living in villages in Turkey exposed to erionite used to whitewash their homes.

An excess in mesothelioma was reported among people living near crocidolite mining regions in South Africa and Western Australia, among people residing in areas of tremolite contamination in Cyprus and New Caledonia, and with non-occupational exposures in Europe, Italy, and California.

Mesothelioma has also been reported to occur among household members of families of asbestos workers.

Mesothelioma: fiber size

Based on findings from experimental studies, it is suspected that long and thin fibers are likely to be more potent than short and thick fibers in the induction of mesothelioma in humans.

Mesothelioma: fiber type

Although all forms of asbestos can cause mesothelioma, there is considerable evidence that the potency for the induction of mesothelioma varies by fiber type, and in particular that chrysotile asbestos is less potent than amphibole forms of asbestos.

Lung cancer: environmental exposure, fiber size and fiber type

Although a causal association between asbestos exposure and lung cancer is generally well recognized, there are still substantial controversies on how the risk might vary by exposure to different fiber types and sizes, and whether there is a risk at low levels of exposure (i.e. environmental exposures).

Particularly controversial is the question of whether chrysotile asbestos is less potent for the induction of lung cancer than the amphibole forms of asbestos (e.g. crocidolite, amosite and tremolite), which has sometimes been referred to as the “amphibole hypothesis”.

Evaluation

There is *sufficient evidence* for the carcinogenicity of all forms of asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite, and anthophyllite), all forms of asbestos are *carcinogenic to humans (Group 1)*.

There is sufficient evidence that asbestos causes mesothelioma and cancer of the lung, larynx, and ovary.

Also positive associations have been observed between exposure to all forms of asbestos and cancer of the pharynx, stomach, and colorectum [IARC].

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