

Asbestos

Asbestos refers to a number of naturally occurring mineral silicates. They are long thin flaxy fibres. There are two groups of asbestos: serpentine and amphiboles. Serpentine asbestos, such as chrysotile, has curly shaped fibres that can shear into smaller fibrils. Amphiboles crystallise in straight double chains resulting in needle-like structures. They tend to be more brittle. Six common forms of asbestos are known as actinolite, amosite, anthophyllite, chrysotile, crocidolite and tremolite. Although they are insoluble they can occur in suspension in both air and water. Asbestos is found in the environment due to natural weathering of rocks, mining (including mining for metals, gravel or stone) and the use of asbestos products. There have been many uses of asbestos, such as insulation, cement, fireproof clothing, and brake linings. However, its use is declining.

1. Physico-Chemical Properties

Concentrations of asbestos are usually reported in fibres per millilitre (f/mL) or million fibres per litre (Mf/L). The standard method of measuring asbestos in air is phase contrast microscopy (PCM). This method is not specific to asbestos. A particle is a fibre when it is longer than 5 microns and the ratio of length to thickness is greater than 3:1. The concentration of asbestos in water is generally determined by transmission electron microscopy (TEM). The physical and chemical properties of asbestos are summarized in Table-1.

Table-1. The physical and chemical properties of asbestos. (Adapted from ATSDR 1995)

| Property | Asbestos | Amosite |
|--|-----------|--|
| Synonyms | n/a | mysorite; brown asbestos; fibrous cummingtonite/ grunerite |
| Chemical Formula | n/a | $[(Mg,Fe)_7Si_8O_{22}(OH)_2]_n$ |
| CAS No. | 1332-21-4 | 12172-73-5 |
| Molecular Weight | n/a | n/a |
| Colour | n/a | light gray to pale brown |
| Physical State | solid | solid |
| Melting Point/Decomposition Temperature (°C) | n/a | 600-900 |

| | | |
|--------------------|-----------------------------------|-----------|
| Boiling Point (°C) | n/a | n/a |
| Specific gravity | n/a | 3.43 |
| Solubility | | |
| Water | insoluble | insoluble |
| Organic Solvents | insoluble | insoluble |
| Conversion Factors | 33 PCM f/mL = 1 mg/m ³ | |

| Property | Chrysotile | Tremolite |
|---|--|---|
| Synonyms | serpentine asbestos; white asbestos | Silicic acid, calcium magnesium salt (8:4) |
| Chemical Formula | $[Mg_6(Si_4O_{10})(OH)_8]_n$ | $[Ca_2Mg_5(Si_8O_{22})(OH)_2]_n$ |
| CAS No. | 12001-29-5 | 14567-73-8 |
| Molecular Weight | n/a | n/a |
| Colour | white to pale green yellow, pink | white to grey |
| Physical State | solid | solid |
| Melting Point/Decomposition Temperature (°C) | 800-850 | 1040 |
| Boiling Point | n/a | n/a |
| Specific Gravity | 2.55 | n/a |
| Solubility | | |
| Water | insoluble | insoluble |
| Organic Solvents | insoluble | insoluble |

| Property | Actinolite | Anthophyllite | Crocidolite |
|------------------|---------------------------------------|--|--|
| Synonyms | n/a | ferroanthophyllite azbolen asbestos | blue asbestos |
| Chemical Formula | $[Ca_2(Mg,Fe)_5(Si_8O_{22})(OH)_2]_n$ | $[(Mg,Fe)_7(Si_8O_{22})(OH)_2]_n$ | $[Na_2Fe_3^{2+}Fe_2^{3+}(Si_8O_{22})(OH)_2]_n$ |

| | | | |
|---|--------------------|-----------------------------|------------|
| CAS No. | 13768-00-0 | 17068-78-9 | 12001-28-4 |
| Molecular Weight | n/a | n/a | n/a |
| Colour | pale to dark green | white to grey pale brown | blue |
| Physical State | solid | solid | solid |
| Melting Point/ Decomposition Temperature (°C) | n/a | 950 | 800 |
| Boiling Point | n/a | n/a | n/a |
| Specific Gravity | n/a | 2.85-3.1 | 3.37 |
| Solubility | | | |
| Water | insoluble | insoluble | insoluble |
| Organic Solvents | insoluble | insoluble | insoluble |

2. Environmental Fate

Asbestos is neither volatile nor soluble, but small fibres occur in suspension in both air and water. Large fibres are removed from air and water by gravitational settling at a rate which depends on their size. Small fibres can remain suspended for long periods of time. Interaction with natural organic matters may increase precipitation. Fibres with aerodynamic diameters of 0.1 mm can be carried thousands of kilometers in air. In the water of Lake Superior, transport of fibres over 125 kilometers has been reported. Asbestos is removed from air or water through deposition and burial in soil and sediment. Asbestos fibres are very stable and do not undergo significant degradation and may remain in the environment for decades. There is little data on levels of asbestos in animal tissues. Data does not suggest that asbestos will bioaccumulate or biomagnify in the food chain.

3. Toxicokinetics

Absorption

Asbestos fibres are insoluble and are not absorbed in the usual sense after inhalation, oral, or dermal exposure. Asbestos fibres may enter the body after inhalation or oral exposures. The main route of uptake is through the lung. In the lungs, the shape and size of fibres will influence the deposition and fate of the fibre. Mucociliary clearance or macrophage removal will clear some fibres from the lung. The influence the location of deposition in the lung or the cellular response to the fibres adsorbed onto other dust

particles is not known. Most asbestos fibres which are ingested are not absorbed across the gastrointestinal tract. Short fibres seem to pass through the gastrointestinal epithelium more easily than longer ones. Asbestos fibres can penetrate into the skin, producing asbestos warts.

Distribution

Longer fibres that are retained in the lung may undergo a number of processes including translocation, dissolution, fragmentation, splitting or protein encapsulation. A few fibres may move through the lungs, stomach or intestine to be distributed to other tissues such as the blood, lymph, urine or various other organs (kidney, heart, liver, spleen, adrenals, pancreas, brain, prostate, or thyroid tissues). Asbestos fibres do not readily pass through the skin into the blood.

Metabolism

Asbestos fibres are not metabolized in the normal sense of the word. Amphibole fibres that are retained in the lung do not appear to undergo any major changes. Chrysotile fibres, however, appear to undergo some type of breakdown or alteration in the lung. Short chrysotile fibres appear to be cleared more easily than long ones. Long fibres that reside in the lung can form asbestos bodies, which might be due to an attempt by macrophages to digest these fibres. No data is available on the changes in asbestos fibres in the gastrointestinal tract, but it is thought that chrysotile fibres undergo some metal ion exchange and alterations in gross structure in biological fluids after oral exposure. Data on metabolism after dermal exposure are not available.

Excretion

Most of the asbestos fibres that enter the lung are removed by mucociliary action. Long fibres are cleared more slowly than short fibres. Fibres of less than 1 micron in length are cleared from the lung with a half-life of less than 10 days whereas fibres longer than 16 mm are cleared with a half-life of greater than 100 days. In the lung, fibres that are coated by mucus move upward to the throat, after which they are swallowed and then excreted in the feces. Fibres that are absorbed into the body are transported by the blood to the kidney, where they may be excreted in the urine. Fibres that are not cleared from the lung accumulate gradually with time. Chrysotile asbestos is removed faster than amphiboles since it fragments. Fragmentation results in the formation of shorter fibres which are then more readily engulfed and moved by a single macrophage. Nearly all asbestos fibres which are ingested are excreted in the faeces within 48 hours. Data is not available on excretion of asbestos fibres after dermal exposure.

4. Human Health Effects

Inhalation to asbestos poses a higher risk of health effects than either oral or dermal exposures do. Studies in humans and animals indicate that inhalation of asbestos fibres may lead to lung disease (asbestosis) hardening of lung tissue, and cancer of the lung. It may also increase the risk of other cancers.

Dermal Effects

Asbestos fibres that penetrate the skin may produce warts or corns. These are of no pathological concern.

Respiratory Effects

Numerous studies in humans have shown that inhalation exposure to asbestos fibres can lead to lung disease. In severe cases, this may ultimately result in death. Asbestosis is the result of inflammation triggered by the deposition of asbestos fibres in the lungs. Data suggests that an exposure of 10 f-yr/mL poses a minimal risk of developing asbestosis. This is approximately equivalent an average workplace exposure of approximately 0.2f/mL over 50 years. There are reports of asbestos-linked laryngitis in workers indicating that the upper airways may also be affected by asbestos.

Cardiovascular Effects

An increase in mortality from cardiovascular disease in workers exposed to asbestos has been reported. Fibrosis of the lung can result in higher resistance to blood flow in the lung with effects on the heart.

Immunological Effects

There is evidence that exposure to asbestos can depress the immune system. Although the biological significance of immunological changes observed in people with asbestos related disease is not known, depressed immune function could be a factor in the development of asbestos-induced cancer.

Genotoxic Effects and Cancer

Studies of exposed asbestos workers and mesothelioma patients suggest that asbestos is genotoxic, causing damage to chromosomes. Direct interactions between asbestos fibres and key cellular molecules, such as DNA, may be responsible in part for asbestos-related health effects. Inhalation exposure to asbestos can lead to lung cancer, mesothelioma, and cancer at other sites. Gastro-intestinal cancers have been associated with both inhalation and oral exposures to asbestos. Fibre size appears to be the most important factor that determines the potency of different fibres – long fibres are more carcinogenic than short ones. Other factors that influence the risk of cancer include: 1) the level and duration of exposure, the time since the exposure, 2) the age at which the exposure occurred, 3) the smoking habits of the person, and 4) the composition of the fibres. However, the data is insufficient at this time to characterise the risk for specific types of asbestos. Both the U.S. Environmental Protection Agency and the International Agency for Research on Cancer (IARC) have classified asbestos as a known human carcinogen by inhalation.

5. Potency

Oral-non-cancer

The US EPA has not derived an oral reference dose for asbestos. The World Health Organisation and Health Canada have concluded that levels of asbestos at concentrations normally found in drinking water do not pose a health risk and so have not established drinking water guidelines for this compound.

Inhalation-non-cancer

The US EPA has not derived an inhalation reference concentration for the non-cancer effects of asbestos.

Oral-cancer

The World Health Organisation and Health Canada have concluded that levels of asbestos at concentrations normally found in drinking water do not pose a health risk and so have not established drinking water guidelines for this compound. Based on a 1985 National Toxicology Program rat feeding study, the US EPA estimated a 1 in 1,000,000 risk of cancer through drinking water of 7.1 Mf/L. The US National Primary Drinking Water Regulations have set a Maximum Contaminant Level of 7 Mf/L, effective 1992. The 1980 Ambient Water Quality Criteria for Asbestos estimated a lifetime cancer risk of 1 in 1,000,000 at concentrations of 30,000 f/L. Based on 6 studies, the estimated risk per $\mu\text{g}/\text{asbestos}$ was calculated as 8.6×10^{-5} , which corresponds to 0.012 μg for a 1 in 1,000,000 lifetime risk. Under Proposition 65, California has set a no significant risk level (1 in 100,000 excess lifetime cancer risk) for asbestos of 100 fibres/day based on inhalation data.

Inhalation -cancer

The US EPA has derived an inhalation lifetime cancer risk to asbestos. This is based on increased mortality and incidence of lung cancer, mesotheliomas, and gastro-intestinal cancer in workers. Although there may be differences in potency between various types and length of fibres, there is insufficient data to quantify these differences. The unit inhalation risk was estimated 0.23 per f/mL using a relative risk model for lung cancer and an absolute risk model for mesothelioma. This is equivalent to a lifetime risk of 1 in 1,000,000 at concentrations of 4 E-6 f/mL. The unit risk is based on fibre counts by phase contrast microscopy (PCM) and should not be used if the air concentration exceeds 4 E-2 f/mL, since above this concentration the slope factor may differ.

The California Dept. of Health Services has estimated a lifetime exposure excess risk of 11-110 cases per million per 100 PCM f/m³ for lung cancer, and 38-190 cases per million for mesothelioma. In calculating this risk, California used a non-threshold linear model and

assumed that both long (greater than 5 microns) and short fibres (less than 5 microns) have cancer effects and that chrysotile fibres are as potent as amphibole ones.

6. References

ATSDR, 1995. Toxicological Profile for Asbestos. And References Cited. Agency for Toxic Substances and Disease Registry, U.S. Department of Health & Human Services, Atlanta, GA.

CalARB, 1986. Staff Report: Initial Statement of Reasons for Proposed Rule Making. Public Hearing to Consider the Adoption of a Regulatory Amendment Identifying Asbestos as a Toxic Air Contaminant. California Air Resources Board, Berkeley, CA.

CalEPA, 1994. Status Report: No Significant Risk Levels for Carcinogens and Acceptable Intake Levels for Reproductive Toxicants. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Berkeley, CA.

Health and Welfare Canada. 1986. Asbestos. Supporting documentation for drinking water guidelines (edited 1989). Health and Welfare Canada, Ottawa.

USEPA, 1980. Ambient Water Quality Criteria for Asbestos. US Environmental Protection Agency, Washington D.C.

USEPA, 1988. Asbestos. IRIS Database, last updated 7/1/1993.

USEPA, 1998. Drinking Water and Health: Consumer Factsheet on Asbestos. US Environmental Protection Agency Office of Water, Washington D.C.

WHO, 1987. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Supplement 7 (p. 106). International Agency for Research on Cancer, Lyon.

WHO, 1996. Consensus Report, IARC Group of Expert Meeting, 9-11 January 1996. International Agency for Research on Cancer, Lyon.

WHO, 1996. Guidelines for Drinking Water Quality, Volume 2. World Health Organisation, Geneva (p.167)