Asbestos CAS No. 1332-21-4

Known to be a human carcinogen First Listed in the *First Annual Report on Carcinogens* (1980)

Carcinogenicity

Asbestos and all commercial forms of asbestos are known to be human carcinogens based on sufficient evidence of carcinogenicity in humans. Studies in humans have demonstrated that exposure to asbestos causes respiratory-tract cancer, pleural and peritoneal mesothelioma (tumors of the membranes lining the chest and abdominal cavities and surrounding internal organs), and other cancers. Case reports and epidemiological studies have found that occupational exposure to chrysotile, amosite, anthophyllite, mixtures containing crocidolite, and various complex mixtures of asbestos increases the risk of lung cancer (the various forms of asbestos are identified and described below, under "Properties"). The risk of lung cancer was increased up to 6-fold among vermiculite miners exposed to tremolite and actinolite. Mesothelioma and digestive-tract cancer were observed in workers occupationally exposed to crocidolite, amosite, and chrysotile; however, the results for digestive-tract cancer were inconsistent among studies. An excess of laryngeal cancer was reported in studies of shipyard workers, chrysotile miners, insulation workers, and other workers exposed to asbestos. People living near asbestos factories or mines or living with asbestos workers also developed mesothelioma; however, no clear association was found between cancer risk and exposure to asbestos in drinking water. Asbestos exposure and smoking increased the risk of lung cancer in a synergistic manner (i.e., the effects of co-exposure on risk were multiplicative, rather than additive). The International Agency for Research on Cancer (IARC) concluded that there was sufficient evidence for the carcinogenicity of asbestos in humans (IARC 1987).

Since asbestos was reviewed for listing in the First Annual Report on Carcinogens and by IARC, additional information has been published regarding asbestos exposure and cancer other than mesothelioma and lung cancer (mainly lymphoma and cancer of the larynx, digestive tract, and kidney); however, the evidence that asbestos causes cancer at these other tissue sites remains inconclusive. A meta-analysis (statistical overview) of epidemiological studies on malignant lymphoma found a small increase in the risks of non-Hodgkin's lymphoma, chronic lymphatic leukemia, and multiple myeloma or plasmocytoma (cancer of white blood cells in the bone marrow); however, the findings were not consistent across all studies (Becker et al. 2001). Some, but not all, epidemiological studies have reported increased risks of laryngeal cancer among asbestos-exposed workers; however, reviewers have noted that many of these studies did not adequately control for confounding factors known to increase the risk of laryngeal cancer, such as alcohol use and smoking (Griffiths and Malony 2003). The results of studies on digestive-tract and kidney cancer also were conflicting (Homa et al. 1994, Weiss 1995, Goodman et al. 1999, Sali and Boffetta 2000). A meta-analysis of studies on pleural mesothelioma reported increased risks (7- to 8-fold) following environmental (household or neighborhood) exposure to asbestos at relatively high levels (Bourdes et al. 2000).

The findings in humans are supported by carcinogenicity studies in experimental animals. All commercial forms of asbestos have been shown to cause cancer in multiple species by various exposure routes (IARC 1977, 1987). Inhalation exposure to chrysotile, crocidolite, amosite, anthophyllite, or tremolite caused mesothelioma and lung carcinoma in rats. Intrapleural injection of various types of asbestos caused mesothelioma in rats and hamsters, and intraperitoneal injection of chrysotile, crocidolite, or amosite caused peritoneal

tumors, including mesothelioma, in mice and rats. The incidence of abdominal tumors was increased by intraperitoneal injection of crocidolite in hamsters and actinolite or tremolite in rats. When filter material containing chrysotile was added to the diet of rats, the incidence of total malignant tumors (which included kidney, lung, and liver tumors) was increased. Oral administration of amosite, tremolite, or crocidolite did not cause tumors in rats, nor did oral administration of amosite or chrysotile in hamsters (NTP 1985, IARC 1987). Dietary administration of chrysotile asbestos fibers of short or intermediate lengths did not cause tumors in female rats, but dietary exposure to the intermediate-length fibers resulted in a low incidence of benign adenomatous polyps of the large intestine in male rats (NTP 1985). Asbestos and the polycycylic aromatic hydrocarbon (PAH) benzo[a]pyrene administered alone by intratracheal injection did not cause tumors in rats, but when co-administered caused lung tumors and mesothelioma (IARC 1977). Synergistic effects on tumor induction also were observed following co-administration of asbestos and benzo[a]pyrene or asbestos and N-nitrosodiethylamine to hamsters (IARC 1987). IARC (1977, 1987) concluded that there was sufficient evidence in experimental animals for the carcinogenicity of asbestos, including the following forms: actinolite, amosite, anthophyllite, chrysotile, crocidolite, and tremolite. Since asbestos was reviewed for listing in the First Annual Report on Carcinogens and by IARC, intrabronchial instillation of chrysotile was shown to cause pulmonary and pleural mesothelioma in rats (Fasske 1988).

Properties

Asbestos is the generic name for a group of six naturally occurring fibrous silicate minerals, including the fibrous serpentine mineral chrysotile and the five fibrous amphibole minerals actinolite, amosite, anthophyllite, crocidolite, and tremolite. Asbestos minerals possess a number of properties useful in commercial applications, including heat stability, thermal and electrical insulation, wear and friction characteristics, tensile strength, the ability to be woven, and resistance to chemical and biological degradation. The forms are ranked from greatest to least tensile strength as follows: crocidolite, chrysotile, amosite, anthophyllite, tremolite, and actinolite. Their ranking from greatest to least acid resistance is tremolite, anthophyllite, crocidolite, actinolite, amosite, and chrysotile. The forms that have been used commercially are chrysotile, anthophyllite, amosite, and crocidolite (IARC 1977, ATSDR 2001, HSDB 2003).

Chrysotile, the most abundant form of asbestos, occurs naturally in fiber bundle lengths of several mm to more than 10 cm (Virta 2002a). It has an idealized chemical composition of Mg₃Si₂O₅(OH)₄ and occurs as a curled sheet silicate, which wraps around itself in a spiral, forming a hollow tubular fiber. The hydroxyl group may, rarely, be replaced by oxygen, fluorine, or chlorine. In addition, small amounts of iron, aluminum, nickel, calcium, chromium, manganese, sodium, and potassium may be present as impurities. Natural chrysotiles occur with a range of physical properties. Chrysotiles may be white, gray, green, or yellowish, with a silky luster. Although chrysotile fibers are more flexible than the amphiboles, fibers from different geological locations may differ in flexibility. Chrysotile fibers have a net positive surface charge and form a stable suspension in water. The fibers degrade in dilute acids (IARC 1973, 1977, IPCS 1986).

The amphibole forms of asbestos consist of chain structures, with nine structural sites that accommodate cations. Amphibole crystals consist of two chains based on $\mathrm{Si_4O_{11}}$ units, linked by a band of cations. The principal cations are magnesium, iron, calcium, and sodium, and their ratios determine the mineral species. The chemical composition and physical properties vary over a wide range and the chemical composition of a field sample seldom matches the idealized formula. Amphibole fibers do not divide into fibrils as small in diameter or as symmetrical as chrysotile fibers, and they do not have a

hollow central core. They have a negative surface charge in water (IPCS 1986, HSDB 2003).

Amosite is ash gray, greenish, or brown and is somewhat resistant to acids. It tends to occur with more iron than magnesium, at a ratio of approximately 5.5 to 1.5. The fibers are long, straight, coarse, and somewhat flexible (less so than chrysotile or crocidolite) (IARC 1973, 1977, IPCS 1986).

Anthophyllite is grayish white, brown-gray, or green and is very resistant to acids. It is relatively rare and occasionally occurs as a contaminant in talc deposits. The fibers are short and very brittle (IARC 1973, 1977, IPCS 1986).

Crocidolite is lavender or blue and has good resistance to acids, but less heat resistance than other asbestos fibers. Its fibers typically are shorter and thinner than those of other amphiboles, but not as thin as chrysotile fibers. The fibers have fair to good flexibility and fair spinnability. Crocidolite usually contains organic impurities, including low levels of PAHs (IARC 1973, 1977, IPCS 1986).

Tremolite is a calcium-magnesium amphibole and actinolite is an iron-substituted derivative of tremolite. Both occur in asbestos and non-asbestos forms. Tremolite is a common contaminant in chrysotile and talc deposits, and actinolite is a common contaminant in amosite deposits. Tremolite is white to gray, and actinolite is pale to dark green. Both are brittle; tremolite is resistant to acids, but actinolite is not (IARC 1977, IPCS 1986).

Use

Although asbestos use dates back at least 2,000 years, modern industrial use began around 1880. Use of asbestos peaked in the late 1960s and early 1970s, when more than 3,000 industrial applications or products were listed. Asbestos has been used in roofing, thermal and electrical insulation, cement pipe and sheets, flooring, gaskets, friction materials, coatings, plastics, textiles, paper, and other products (ATSDR 2001, HSDB 2003). The U.S. Consumer Product Safety Commission banned use of asbestos in general-use garments, but asbestos may be used in firefighting garments if they are constructed to prevent release of asbestos fibers (HSDB 2003). Domestically used asbestos fibers are classified into seven quality categories or grades. Grades 1, 2, and 3 include the longer, maximum-strength fibers and generally are used in the production of textiles, electrical insulation, and pharmaceutical and beverage filters. Grades 4, 5, and 6 are medium-length fibers used in the production of asbestos-cement pipes and sheets, clutch facings, brake linings, asbestos paper, packaging, gaskets, and pipe coverings. Grade 7 includes short fibers generally used as reinforcers in plastics, floor tiles, coatings and compounds, some papers, and roofing felts (OSHA 1986).

The four commercially important forms of asbestos have been chrysotile, amosite, anthophyllite, and crocidolite (IARC 1973); however, commercial use of anthophyllite was discontinued by the 1980s (IPCS 1986, HSDB 2003). Chrysotile, amosite, and particularly crocidolite all have extremely high tensile strengths and are used extensively as reinforcers in cements, resins, and plastics. Although chrysotile is most adaptable to industrial use, crocidolite and amosite are particularly useful in combination with chrysotile for adding specific properties, such as rigidity (OSHA 1986). By the 1990s, chrysotile accounted for more than 99% of U.S. asbestos consumption (ATSDR 2001) and was the only type of asbestos used in the United States in 2002 (Virta 2002b).

In 1973, when U.S. consumption of asbestos was at its peak, the major markets included asbestos cement pipe (24%), flooring (22%), roofing (9%), friction products such as automobile brakes and clutches (8%), and packing and gaskets (3%) (Virta 2000). In 2002, U.S. consumption of asbestos was distributed as follows: roofing products (50%), coatings and compounds (32%), gaskets (8%), friction products (4%), and electrical and thermal insulation (4%). Ninety-one percent of the chrysotile used in 2002 was categorized as grade 7

asbestos (with fiber lengths less than 3 µm), followed by grades 4, 5, 6, and 3 (Virta 2002b). See erratum, January 26, 2009.

Erratum-replacement text: Ninety-one percent of the chrysotile used in 2002 was categorized as grade 7 asbestos (with fiber lengths less than 3 mm), followed by grades 4, 5, 6, and 3 (Virta 2002a,b).

Production

U.S. demand for asbestos increased dramatically from 1900 to the early 1970s. By 1950, the United States was the world's largest user of asbestos. However, as health and liability issues became apparent, asbestos demand declined rapidly after 1973 (Virta 2000). Before the 1980s, asbestos was produced in California, Arizona, North Carolina, and Vermont; however, most of these facilities suspended mining operations in the 1970s, and the last U.S. asbestos mine closed in 2002 (ATSDR 2001, Virta 2002b). In 2003, there were two U.S. suppliers of asbestos (ChemSources 2003).

U.S. production of asbestos decreased considerably from a high of 136,000 metric tons (300 million pounds) in 1973 (Buckingham and Virta 2002) to 3,000 metric tons (6.6 million pounds) in 2002 (Virta 2003). Domestic consumption (production plus imports minus exports and adjustments for government and industry stocks) declined from 803,000 metric tons (1.8 billion pounds) in 1973 to 13,100 metric tons (28.9 million pounds) in 2000. Most of the asbestos used in the United States is imported from Canada. The amounts imported declined from 718,000 metric tons (1.6 billion pounds) in 1973 to 13,100 metric tons (28.9 million pounds) in 2001. Asbestos exports also peaked in 1973 at 59,900 metric tons (132 million pounds) and declined to 21,700 metric tons (47.8 million pounds) in 2001 (Buckingham and Virta 2002).

Exposure

The primary routes of potential human exposure to asbestos are inhalation and ingestion. Dermal absorption of asbestos is minimal, but dermal contact may lead to secondary ingestion or inhalation of dust. Asbestos fibers vary with respect to size (length and diameter) and chemical composition. These differences are known to affect the deposition, movement, and clearance from the body and the carcinogenic potency. Fiber diameter is the most important factor controlling penetration and deposition in the lungs. Thin fibers have the greatest inhalation potential and deposit deep within the lungs. Fiber length, surface chemistry, and other properties affect biological activity. Fibers longer than 8 µm with a diameter less than 1.5 µm have shown the greatest carcinogenic potency (IPCS 1986).

Asbestos is released to the environment from both natural and anthropogenic sources and has been detected in indoor and outdoor air, soil, drinking water, food, and medicines (ATSDR 2001). Because asbestos products were used so widely, the entire U.S. population potentially is exposed to some degree; however, the potential for exposure continues to decline, because asbestos mining has stopped and asbestos products are being eliminated from the market.

Releases from asbestos materials in buildings and vehicle brake linings account for substantial emissions of asbestos into the air. Demolition of buildings with asbestos insulation or fireproofing may cause high atmospheric concentrations for relatively short periods. Environmental asbestos concentrations vary widely; therefore, it is not possible to accurately calculate human exposure levels except on a site-by-site basis (ATSDR 2001).

In the past, occupational exposure occurred primarily during the mining and milling of asbestos, during the manufacture of all asbestos products, and in the construction and shipbuilding industries. Occupational exposure still occurs among workers who use asbestos

end products, such as asbestos insulation workers, brake repair and maintenance workers, building demolition workers, and asbestos abatement workers (IARC 1977, ATSDR 2001, HSDB 2003). The National Occupational Exposure Survey (1981–1983) estimated that 215,265 workers, including 9,727 women, potentially were exposed to asbestos and that 92,033 workers, including 13,262 women, potentially were exposed to chrysotile (NIOSH 1984). In 1990, the U.S. Occupational Safety and Health Administration estimated that approximately 568,000 workers in production and services industries and 114,000 workers in construction industries potentially were exposed to asbestos (ATSDR 2001). No more recent occupational exposure estimates were found.

People may be exposed to higher-than-average levels of asbestos in air if they live near asbestos-containing waste sites or asbestos-related industries, if they use asbestos-containing products, or if they live or work in buildings with deteriorating asbestos insulation or that have undergone poorly performed asbestos removal (ATSDR 2001). In the past, families of asbestos workers potentially were exposed to high fiber levels from contaminated clothing brought home for laundering. People living in households with asbestos workers were found to have significantly elevated lung burdens of asbestos, often in the same range as found in individuals occupationally exposed to asbestos, such as shipyard workers. The asbestos-fiber burdens of occupants of a building containing asbestos insulation, on the other hand, were comparable to those of individuals with no known occupational exposure to asbestos (IARC 1977, Roggli and Longo 1991).

According to the U.S. Environmental Protection Agency's Toxics Release Inventory, 57 industrial facilities (mostly waste-management companies) reported releasing or disposing of about 20.5 million pounds (9,300 metric tons) of friable (readily crumbled) asbestos in 2001. Almost all asbestos disposal is to landfills. Reported asbestos releases declined about 80% from 1988 to 2001 (TRI01 2003).

Regulations

Consumer patching compounds containing intentionally-added respirable, free-form asbestos are banned

Artificial emberizing materials (ash and embers) containing respirable free-form asbestos are banned

General use garments containing asbestos (other than those needed for personal protection and constructed so that asbestos fibers will not become airborne) are banned.

Certain household products containing intentionally added asbestos that release asbestos fibers are subject to cautionary labeling requirements

EPA

Clean Air Act

NESHAP: Listed as a Hazardous Air Pollutant (HAP)

Clean Water Act

Effluent Guidelines: Listed as a Toxic Pollutant

Water Quality Criteria: Based on fish/shellfish and water consumption = 7 million fibers/L

Comprehensive Environmental Response, Compensation, and Liability Act

Reportable Quantity (RQ) = 1 lb

Emergency Planning and Community Right-To-Know Act

Toxics Release Inventory: Listed substance subject to reporting requirements

Safe Drinking Water Act

Maximum Contaminant Level (MCL) = 7 million fibers per liter

Toxic Substances Control Act

Rules have been set for identifying, analyzing, and disposing of asbestos found in schools and prohibitions on the manufacturing and import of asbestos products have been established

OSHA

Ceiling Concentration = 1 fiber/cm³ (excursion limit) as averaged over a sampling period of 30 minutes

Permissible Exposure Limit (PEL) = 0.1 fiber/cm³ (fibers longer than 5 micrometers having a length-to-diameter ratio of at least 3 to 1)

"Comprehensive Standards" for occupational exposure to this substance have been developed

Guidelines

ACGIH

Threshold Limit Value - Time-Weighted Average Limit (TLV-TWA) = 0.1 respirable fibers/cc

Listed as a potential occupational carcinogen

Recommended Exposure Limit (REL) = 0.1 fiber/cm³ (fibers longer than 5 micrometers)

REFERENCES

ATSDR. 2001. Toxicological Profile for Asbestos. NTIS Accession No. PB/2001/109101. Atlanta, GA: Agency for Toxic Substances and Disease Registry. 146 pp.

Becker, N., J. Berger and U. Bolm-Audorff. 2001. Asbestos exposure and malignant lymphomas--a review of the epidemiological literature. Int Arch Occup Environ Health 74(7): 459-69.

Bourdes, V., P. Boffetta and P. Pisani. 2000. Environmental exposure to asbestos and risk of pleural mesothelioma: review and meta-analysis. Eur J Epidemiol 16(5): 411-7.

Buckingham, D. A. and R. L. Virta. 2002. Asbestos Statistics. Historical Statistics for Mineral Commodities in the United States. U.S. Geological Survey. http://minerals.usgs.gov/minerals/pubs/commodity/ asbestos/index.html.

ChemSources. 2003. Asbestos. Chemical Sources International, Inc. http://www.chemsources.com and search CAS number 1332-21-4.

Fasske, E. 1988. Experimental lung tumors following specific intrabronchial application of chrysotile asbestos. Longitudinal light and electron microscopic investigations in rats. Respiration 53(2): 111-27.

Goodman, M., R. W. Morgan, R. Ray, C. D. Malloy and K. Zhao. 1999. Cancer in asbestos-exposed occupational cohorts: a meta-analysis. Cancer Causes Control 10(5): 453-65.

Griffiths, H. and N. C. Malony. 2003. Does asbestos cause laryngeal cancer? Clin Otolaryngol 28(3): 177-82. Homa, D. M., D. H. Garabrant and B. W. Gillespie. 1994. A meta-analysis of colorectal cancer and asbestos exposure. Am J Epidemiol 139(12): 1210-22.

HSDB. 2003. Hazardous Substances Database. National Library of Medicine. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlqen?HSDB.

IARC. 1973. Some Inorganic and Organometallic Compounds. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, vol. 2. Lyon, France: International Agency for Research on Cancer. 181 pp.

IARC. 1977. Asbestos. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, vol. 14. Lyon, France: International Agency for Research on Cancer. 106 pp.

IARC. 1987. Overall Evaluations of Carcinogenicity. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, Supplement 7. Lyon, France: International Agency for Research on Cancer. 440 pp.

IPCS. 1986. Environmental Health Criteria No. 53. Asbestos and Other Natural Mineral Fibers. World Health Organization. http://www.inchem.org/documents/ehc/ehc/shc53.htm.

NIOSH. 1984. National Occupational Exposure Survey (1981-83). Cincinnati, OH: U. S. Department of Health and Human Services. http://www.cdc.gov/noes/noes3/empl0003.html.

NTP. 1985. Carcinogenesis Studies of Chrysotile Asbestos (CAS no. 12001-29-5) in F344/N Rats and B6C31F Mice (Feed Studies). Technical Report Series No 295. NIH Publication No. 86-2551. Research Triangle Park, NC and Bethesda, MD: National Toxicology Program. 390 pp.

OSHA. 1986. Final Regulatory Impact and Regulatory Flexibility Analysis of the Revised Asbestos Standard. Occupational Safety and Health Administration.

Roggli, V. L. and W. E. Longo. 1991. Mineral fiber content of lung tissue in patients with environmental exposures: household contacts vs. building occupants. Ann N Y Acad Sci 643: 511-8.

Sali, D. and P. Boffetta. 2000. Kidney cancer and occupational exposure to asbestos: a meta-analysis of occupational cohort studies. Cancer Causes Control 11(1): 37-47.

TRI01. 2003. Toxics Chemical Release Inventory 2001. Data contained in the Toxics Chemical Release Inventory (TRI). U. S. Environmental Protection Agency Office of Environmental Information. http://www.epa.gov/triexplorer/.

Virta, R. L. 2000. Asbestos. Mineral Yearbook. U.S. Geological Survey. http://minerals.usgs.gov/minerals/pubs/commodity/asbestos/index.html.

Virta, R. L. 2002a. Asbestos: Geology, Mineralogy, Mining and Uses. Open File Report 02-149. U.S. Geological Survey. Last updated: 10/21/02. http://pubs.usgs.gov/of/2002/of02-149/index.html. Last accessed: 2/20/04.

Virta, R. L. 2002b. Asbestos. Mineral Yearbook. U.S. Geological Survey. http://minerals.usgs.gov/minerals/pubs/commodity/asbestos/index.html.

Virta, R. L. 2003. Asbestos. Mineral Commodities Summaries. U.S. Geological Survey. http://minerals.usgs.gov/minerals/pubs/commodity/asbestos/index.html.

Weiss, W. 1995. The lack of causality between asbestos and colorectal cancer. J Occup Environ Med 37(12): 1364-73.