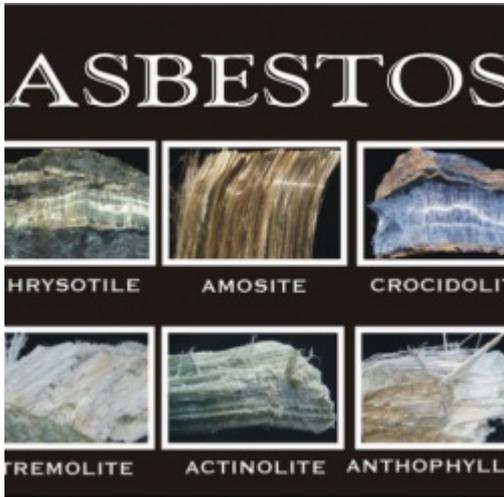


ACSH Explains: What's The Story On Asbestos?



By Michael Dourson — July 18, 2018



Credit: Asbestorama [1]

The Frank R. Lautenberg Chemical Safety for the 21st Century Act amends the [Toxic Substances Control Act \(TSCA\)](#) [2] and was signed into law June 22, 2016. It created a mandatory requirement for EPA to evaluate existing chemicals with clear and enforceable deadlines, to do so in a transparent fashion, and to do so using risk-based chemical assessments rather than rely on simple epidemiological correlations.

EPA selected the first 10 chemicals to undergo risk evaluation under the amended TSCA and to make those understandable for the public, the American Council on Science and Health is producing risk-based evaluations of each, which will then be compiled into a free downloadable book for consumers.

What is Asbestos?

The US Agency for Toxic Substances and Disease Registry (ATSDR, 2001), US Environmental Protection Agency (EPA, 2018), and the US National Center for Biotechnology Information (NCBI, 2018), uses the name asbestos to a group of six different mineral fibers. These fibers are amosite (brown), chrysotile (white), crocidolite (blue), and the fibrous varieties of tremolite (silicic acid), actinolite, and anthophyllite (azbolen). All of these fibers occur naturally in rock and soil and are composed of some of the same starting material as sand. Chrysotile (white), the predominant commercial form of asbestos, belongs to the serpentine family of minerals, while all of the other forms belong to the amphibole family. Asbestos is used in numerous building construction materials and vehicle products for its strength and ability to resist heat, fire, and corrosion, as well as chemical and biological degradation.

Asbestos fibers do not have any detectable odor or taste, nor do they dissolve in water or

evaporate into the air. They are resistant to heat, fire, chemical and biological degradation. The fibrous minerals are composed of magnesium and calcium silicates that may contain other elements (US National Library of Medicine's Toxnet database, 2018). Pieces of fibers can enter the air and water from the weathering of natural deposits and the wearing down of manufactured products. When broken into smaller pieces and inhaled, they may get trapped in the lungs.

Levels of the fibers can build up over time in the lung tissues, with amphibole fibers being retained in the lungs longer than chrysotile fibers.

Nearly all ingested fibers pass through the intestines within a few days and are excreted in the feces. A small number of the fibers may penetrate into cells lining the stomach or intestines, while a few may penetrate all the way through and get into the bloodstream. However, very few of these fibers will pass through the skin into the body.

Exposure to Asbestos

According to both ATSDR (2001) and EPA (1988, 2014), asbestos can be released into the air, water, and soil from the erosion of asbestos-bearing natural deposits, piles of waste asbestos, asbestos mine or factory, or from the wearing down or disturbance of manufactured products that contain asbestos. The primary route of exposure of asbestos fibers to humans is through the air. Asbestos can also move into groundwater by being eroded from natural deposits or piles of waste asbestos, from asbestos-containing cement pipes used to carry drinking water, or from asbestos containing filters. Asbestos does not break down in water, nor does it move through the soil.

ATSDR (2001) has reported asbestos levels in air in rural areas of 10 fibers per cubic meter (fibers/m³). In cities, typical levels are about 10-fold higher. Levels may reach up to 10,000 fibers/m³ or higher close to a mine or factory. Levels could also be above average near a building that contains asbestos products and that is being torn down or renovated, or near a waste site where asbestos is not properly covered up or stored to protect it from wind erosion.

Concentrations measured in homes, schools, and other buildings that contain asbestos range from about 30 to 6,000 fibers/m³.

In the United States, most water supplies have some small concentrations of asbestos, but a few supplies have more in areas with deposits or with cement water supply pipes. In a few locations, water samples may contain 10–300 million fibers per liter or even higher. The average person drinks about 2 liters of water per day.

Asbestos Health Effects

Both ATSDR (2001) and EPA (2014) consider **inhalation** to be the main route of human exposure to asbestos mineral fibers, and also the route with the most severe consequences to adverse health. Ingestion occurs mainly through the swallowing of material removed from the respiratory tract in the normal process of clearing one's throat. To a lesser degree, ingestion occurs through drinking water contaminated with asbestos, or eating, drinking, or smoking in asbestos-contaminated work environments. Exposure to the skin does not result in the absorption of asbestos.

Asbestos is not metabolized (or broken down) into other chemicals inside most organisms.

Animal and human data indicate that long fibers (more than 5 or 10 μm) are cleared from the lower airways more slowly than shorter fibers. Nearly all fibers that are ingested and those that are swallowed after clearing one's throat following inhalation are ultimately excreted in the feces, but a small number may end up being excreted in the urine. Fibers that are not cleared from the lungs lead to a gradual accumulation and persistence over time; persistence determines cumulative exposure and toxicity of fibers. Amphibole fibers appear to be retained in the lungs for longer periods of time than chrysotile fibers.

Like exposure to any chemical, toxicity of asbestos depends on the level to which one is exposed and the length of time of exposure. According to ATSDR (2001), limited data are available on the effects of higher short-term inhalation or oral exposure in humans. Furthermore, oral exposure to high levels of asbestos is unlikely. The available data suggest that health effects from asbestos ultimately are functions of fiber dose, fiber dimension (length and diameter), and fiber durability or persistence in the lung (ATSDR, 2003). Fibers with lengths greater than 5.0 μm are more likely to cause damage than fibers with lengths less than 2.5 μm (1 μm is about 1/25,000 of an inch.)

Workers who are exposed to fibers have an increase in mortality from noncancer respiratory diseases, including fibrosis (thickening and scarring of the lungs) or asbestosis and deficit in pulmonary function (ATSDR, 2001; EPA, 1988, 2014). These effects have been reported in workers exposed repeatedly over a longer term through inhalation of fibers with lengths greater than or equal to 5 μm and at concentrations ranging from about 5 to 20 f/mL (e.g., 5,000 to 20,000 fibers per m^3) (ATSDR, 2001; EPA, 2014). Asbestos workers have increased chances of developing two principal types of cancer: cancer of the lung tissue itself, and the almost always fatal mesothelioma, a cancer of the thin membrane that surrounds the lungs and other internal organs. Workers can also develop cancer in other locations, for example, the stomach, intestines, esophagus, pancreas, ovaries, and kidneys, although this is less common. Exposure to asbestos via drinking water may result in cancer of the esophagus, stomach, and intestines, but it is difficult to determine whether this is caused by asbestos or something else. Epidemiological studies also demonstrate associations between exposure to airborne asbestos and autoimmunity (EPA, 2014).

Limited data in animals suggest that single high inhalation exposures may cause fibrosis of the lungs or lung tumors. In animals, short-term oral exposure to high levels of asbestos have been shown to cause abnormal colon and rectum structure, which is one of the earliest changes seen in the colon that may lead to cancer. Lung lesions (inflammation and fibrosis) have also been reported in animals following longer-term exposures to asbestos, and tumors that were observed in animal tissues following these exposures, such as mesothelioma and lung cancer, are consistent with the cancer effects observed in humans. However, animals exposed to very high doses of asbestos in food did not generally develop more fatal cancers than usual.

Other experimental animal studies also support human studies demonstrating potential autoimmune effects of exposure. Available data in both humans and animals indicate that the amphibole types of asbestos may be more harmful than the chrysotile type, particularly for mesothelioma (ATSDR, 2001; EPA, 2014).

Asbestos Safe Or Virtually Levels

Federal and state governments develop regulations and recommendations to protect public health. Regulations and recommendations for chemicals are often expressed as a safe or virtually safe level, that is, a level of a substance in air, water, soil, or food that is not expected to cause any adverse health effect, even in sensitive people. These safe levels are usually based on information from experiments with animals (usually rodents) at much higher levels of the chemicals than humans would typically encounter. However, in the case of asbestos, we have sufficient information which to determine the virtually safe level. Sometimes these safe levels differ among federal and state organizations because they assume different exposures, use different experimental animal studies, or employ slightly different methods. The recommendations for safe levels may also differ because new science develops that suggests different levels are toxic or safe. Recommendations and regulations are also updated periodically as more information becomes available.

Recently, the EPA (2014) derived a non-cancer safe level of 9×10^{-5} fiber/cc (or 9 fibers/m³). For cancer, the EPA derived a unit risk level of 1.7×10^{-1} per fiber/cc (0.17 per mg/m³) for inhalation exposure based on cancer mortality from lung cancer and mesothelioma. No virtually safe level has been derived for asbestos in water or air. The weight of evidence indicates that asbestos ingestion does not cause any significant noncarcinogenic effects in the gastrointestinal tract or other tissues. This supports the generally held perception that oral exposure to asbestos does not present a high priority public health concern for noncancer effects (ATSDR, 2001).

Why Is EPA Looking At Asbestos Under The Lautenberg Chemical Safety Act?

EPA scientists are currently looking at the likely routes of exposure to asbestos in the environment and will be developing exposure scenarios, or pathways, for the public.

These exposure pathways will be studied by EPA scientists by comparing the amount of asbestos exposure in the pathway to its safe or virtually safe level. If exposure in the pathway is at or below this safe or virtually safe dose, then asbestos exposure from the pathway is not considered to be a health concern. If exposure is above this safe or virtually safe level, then the pathway might be considered as a possible health concern, and regulations might be developed to lessen the exposure of asbestos from this pathway. Small excesses of the safe or virtually safe dose are seldom cause for concern since these safety levels are developed from conservative assumptions, including the use of safety factors that tend to exaggerate risk or exposure pathways that tend to exaggerate exposure.

See also EPA (2018) for specific questions related to the assessment of asbestos under the new Lautenberg Chemical Safety Act (LCSA).

Controversy Over Asbestos

Three general controversies exist with asbestos. The first has to do with the extrapolation of cancer findings to commonly encountered asbestos exposures. The second controversy is whether asbestos fibers smaller than 5 μm are considered toxic. The third has to do with whether all uses of asbestos could be banned.

Regarding the first controversy, several government and other agencies have evaluated the carcinogenicity of asbestos in humans. Epidemiological evidence shows a significant association between exposure to all forms of asbestos and increased lung cancer and mesothelioma mortality following exposure in humans. This association is supported by experimental animal studies. Based on this evidence, asbestos is considered *carcinogenic to humans* (EPA, 2014; International Agency for Research on Cancer, 2012). Although this statement is not controversial, EPA (2014) describes multiple ways in which tumors can be formed from asbestos exposure, including mutation, DNA toxicity, chronic inflammation, cell toxicity, and regeneration of tissue from severe injury. Although EPA determined that the available evidence is generally supportive of chronic inflammation or cell toxicity and regeneration in the formation of tumors, and that the available data are not sufficient to conclude that asbestos is mutagenic, it still defaulted to a conservative linear extrapolation for estimating the virtually safe dose for asbestos. In such cases, EPA could have conducted a dose response assessment with both mutagenic and threshold ways of tumor formation as per its guidelines (EPA, 2005, page 3-22). However, EPA chose not to do so.

Regarding the second controversy, available studies indicate that inhalation of fibers greater than 5 μm in length are associated with both pulmonary fibrosis (i.e., asbestosis) and malignancies (carcinoma of the lung and mesothelioma), while there is a strong weight of evidence that asbestos fibers shorter than 5 μm are unlikely to cause cancer in humans (ATSDR, 2003). ATSDR (2003) indicated that there are several notable exceptions, for example, in laboratory animals short asbestos at sufficiently high doses have been shown to cause inflammation and lung effects, the doses needed to cause these effects in humans may not be relevant to environmental exposures.

Several authors have also expressed concerns regarding the prevailing scientific opinion that short fibers (less or equal to 5 μm in length) have not been demonstrated to be pathogenic (see Roggli, 2015). In reviewing these studies and the available scientific literature, Roggli (2015) noted that there is no convincing scientific evidence from studies in both experimental animal models and human tissue samples to support a pathogenic role for fibers 5 μm or less in length. According to Roggli (2015), unlike the short fibers, clearance from the respiratory system is very slow for the long, thin persistent fibers, which become concentrated in the lungs and in the membrane surrounding the lungs.

Regarding the third controversy, EPA (2018) interprets the mandates within the LCSA to conduct risk evaluations on current and prospective uses of asbestos for which “manufacture, processing, or distribution in commerce is intended, known or reasonably foreseen, rather than reaching back to evaluate the risks associated with legacy uses, associated disposal, and legacy disposal...” That is to say that EPA’s evaluation process focuses on the “continuing flow of chemical substances from manufacture, processing and distribution in commerce into the use and disposal stages of their life cycle.” In contrast, some groups are calling for the complete ban of

asbestos products, including legacy uses. Such a request, while not inconsistent with the uses of asbestos that were banned under TSCA in 1989 (*Asbestos: Manufacture, Importation, Processing, and Distribution in Commerce Prohibitions; Final Rule* (40 CFR Part 763), also known as Asbestos Ban and Phase-out Rule Remanded), would be extremely difficult to manage under LCSA, but which otherwise is being managed in other areas. Unfortunately, the controversy continues to be ongoing.

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More analyses in our series on the Lautenberg Chemical Safety Act compounds:

[ACSH Explains: What's The Story On Dioxane?](#) [3]

[ACSH Explains: What's The Story On Trichloroethylene \(TCE\)?](#) [4]

[ACSH Explains: What's The Story On Methylene Chloride \(DCM\)?](#) [5]

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[2] <https://www.epa.gov/laws-regulations/summary-toxic-substances-control-act>

[3] <https://www.acsh.org/news/2018/06/18/acsh-explains-whats-story-dioxane-13088>

[4] <https://www.acsh.org/news/2018/06/21/acsh-explains-whats-story-trichloroethylene-tce-13098>

[5] <https://www.acsh.org/news/2018/06/26/acsh-explains-whats-story-methylene-chloride-dcm-13122>

[6] <http://monographs.iarc.fr/ENG/Monographs/vol100C/mono100C.pdf>

[7] <https://www.atsdr.cdc.gov/toxprofiles/tp61.pdf>

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