Appendix A. Definitions

Exposure pathways

An exposure pathway is the way in which an individual comes into contact with a contaminant. An exposure pathway consists of the following five elements: (1) a *source* of contamination; (2) a *medium* such as air or soil through which the contaminant is transported; (3) a *point of exposure* where people can contact the contaminant; (4) a *route of exposure* by which the contaminant enters or contacts the body; and (5) a *receptor population*. A pathway is considered **complete** if all five elements are present and connected. A p**otential** exposure pathway indicates that exposure to a contaminant could have occurred in the past, could be occurring currently, or could occur in the future. A potential exposure exists when information about one or more of the five elements of an exposure pathway is missing or uncertain. An **incomplete** pathway is missing one or more of the pathway elements and it is likely that the elements were never present and are not likely to be present at a later point in time. An **eliminated** pathway was a potential or completed pathway in the past, but has had one or more of the pathway elements removed to prevent present and future exposure.

Public health hazard categories

ATSDR uses public health hazard categories to describe whether people could be harmed by conditions present at the site in the past, present, or future. One or more hazard categories might be appropriate for each site. The five public health hazard categories are defined as follows:

No public health hazard

A category used in ATSDR's assessments for sites where people have never been and will never be exposed to harmful amounts of site-related substances.

No apparent public health hazard

A category used in ATSDR's assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.

Indeterminate public health hazard

The category used in ATSDR's assessments when a professional judgment about the level of health hazard cannot be made because information critical to such a decision is lacking.

Public health hazard

A category used in ATSDR's assessments for sites that pose a public health hazard because of long-term exposures (greater than 1 year) to sufficiently high levels of hazardous substances or radionuclides that could result in harmful health effects.

Urgent public health hazard

A category used in ATSDR's assessments for sites where short-term exposure (less than 1 year) to hazardous substances or conditions could result in harmful health effects that require rapid intervention.

Appendix B. Asbestos overview

Asbestos is a general name applied to a group of silicate minerals consisting of thin, separable fibers in a parallel arrangement. Asbestos minerals fall into two classes, serpentine and amphibole. Serpentine asbestos has relatively long and flexible crystalline fibers; this class includes chrysotile, the predominant type of asbestos used commercially. Fibrous amphibole minerals are brittle and have a rod- or needle-like shape. Amphibole minerals regulated as asbestos by OSHA include five classes: crocidolite, amosite, and the fibrous forms of tremolite, actinolite, and anthophyllite. Other unregulated amphibole minerals, including winchite, richterite, and others, can also exhibit fibrous asbestiform properties [1].

Asbestos fibers do not have any detectable odor or taste. They do not dissolve in water or evaporate into the air, although individual asbestos fibers can easily be suspended in the air. Asbestos fibers do not move through soil. They are resistant to heat, fire, and chemical and biological degradation. As such, they can remain virtually unchanged in the environment over long periods of time.

Vermiculite that was mined in Libby, Montana, contains amphibole asbestos, with a characteristic composition including tremolite, actinolite, richterite, and winchite; this material will be referred to as Libby asbestos. The raw vermiculite ore was estimated to contain up to 26% Libby asbestos as it was mined [2]. For most of the mine's operation, Libby asbestos was considered a by-product of little value and was not used commercially. The mined vermiculite ore was processed to remove unwanted materials and then sorted into various grades or sizes of vermiculite that were then shipped to sites across the nation for expansion (exfoliation) or use as a raw material in manufactured products. Samples of the various grades of unexpanded vermiculite shipped from the Libby mine contained 0.3%–7% fibrous tremolite-actinolite (by mass) [2].

The following sections provide an overview of several concepts relevant to the evaluation of asbestos exposure, including analytical techniques, toxicity and health effects, and the current regulations concerning asbestos in the environment. A more detailed discussion of these topics will also be provided in ATSDR's upcoming summary report for the national review of vermiculite sites.

Methods for Measuring Asbestos Content

A number of different analytical methods are used to evaluate asbestos content in air, soil, and other bulk materials. Each method varies in its ability to measure fiber characteristics such as length, width, and mineral type. For air samples, fiber quantification is traditionally done through phase contrast microscopy (PCM) by counting fibers with lengths greater than 5 micrometers (>5 µm) and with an aspect ratio (length to width) greater than 3:1. This is the standard method by which regulatory limits were developed. Disadvantages of this method include the inability to detect fibers less than 0.25 (<0.25) µm in diameter and the inability to distinguish between asbestos and nonasbestos fibers [1].

Asbestos content in soil and bulk material samples is commonly determined using polarized light microscopy (PLM), a method which uses polarized light to compare refractive indices of minerals and can distinguish between asbestos and nonasbestos fibers and between different types of asbestos. The PLM method can detect fibers with lengths greater than approximately 1

µm (~1 µm), widths greater than ~0.25 µm, and aspect ratios (length-to-width ratios) greater than 3. Detection limits for PLM methods are typically 0.25%–1% asbestos.

Scanning electron microscopy (SEM) and, more commonly, transmission electron microscopy (TEM) are more sensitive methods that can detect smaller fibers than light microscopic techniques. TEM allows the use of electron diffraction and energy-dispersive x-ray methods, which give information on crystal structure and elemental composition, respectively. This information can be used to determine the elemental composition of the visualized fibers. SEM does not allow measurement of electron diffraction patterns. One disadvantage of electron microscopic methods is that determining asbestos concentration in soil and other bulk material is difficult [1].

For risk assessment purposes, TEM measurements are sometimes multiplied by conversion factors to give PCM equivalent fiber concentrations. The correlation between PCM fiber counts and TEM mass measurements is very poor. A conversion between TEM mass and PCM fiber count of 30 micrograms per cubic meter per fiber per cubic centimeter (µg/m3)/(f/cc) was adopted as a conversion factor, but this value is highly uncertain because it represents an average of conversions ranging from 5 to 150 (µg/m3)/(f/cc) [3]. The correlation between PCM fiber counts and TEM fiber counts is also very uncertain, and no generally applicable conversion factor exists for these two measurements [3]. Generally, a combination of PCM and TEM is used to describe the fiber population in a particular air sample.

Asbestos Health Effects and Toxicity

Breathing any type of asbestos increases the risk of the following health effects:

Malignant mesothelioma— cancer of the membrane (pleura) that encases the lungs and lines the chest cavity. This cancer can spread to tissues surrounding the lungs or other organs. The great majority of mesothelioma cases are attributable to asbestos exposure [1].

Lung cancer—cancer of the lung tissue, also known as bronchogenic carcinoma. The exact mechanism relating asbestos exposure with lung cancer is not completely understood. The combination of tobacco smoking and asbestos exposure greatly increases the risk of developing lung cancer [1].

Noncancer effects—these include asbestosis, scarring, and reduced lung function caused by asbestos fibers lodged in the lung; pleural plaques, localized or diffuse areas of thickening of the pleura (lining of the lung); pleural thickening, extensive thickening of the pleura which may restrict breathing; pleural calcification, calcium deposition on pleural areas thickened from chronic inflammation and scarring; and pleural effusions, fluid buildup in the pleural space between the lungs and the chest cavity [1].

Not enough evidence is available to determine whether inhalation of asbestos increases the risk of cancers at sites other than the lungs, pleura, and abdominal cavity [1].

Ingestion of asbestos causes little or no risk of non-cancer effects. However, some evidence indicates that acute oral exposure might induce precursor lesions of colon cancer and that chronic oral exposure might lead to an increased risk of gastrointestinal tumors [1].

ATSDR considers the inhalation route of exposure to be the most significant in the current evaluation of sites that received vermiculite from Libby. Exposure scenarios that are protective of the inhalation route of exposure should be protective of dermal and oral exposures.

The scientific community generally accepts the correlations of asbestos toxicity with fiber length as well as fiber mineralogy. Fiber length may play an important role in clearance and mineralogy may affect both biopersistence and surface chemistry.

ATSDR, responding to concerns about asbestos fiber toxicity from the World Trade Center disaster, held an expert panel meeting to review fiber size and its role in fiber toxicity in December 2002 [4]. The panel concluded that fiber length plays an important role in toxicity. Fibers with lengths <5 µm are essentially non-toxic in terms of association with mesothelioma or lung cancer promotion. However, fibers <5 µm in length may play a role in asbestosis when exposure duration is long and fiber concentrations are high. More information is needed to definitively reach this conclusion.

In accordance with these concepts, it has been suggested that amphibole asbestos is more toxic than chrysotile asbestos, mainly because physical differences allow chrysotile to break down and to be cleared from the lung, whereas amphibole is not removed and builds up to high levels in lung tissue [5]. Some researchers believe the resulting increased duration of exposure to amphibole asbestos significantly increases the risk of mesothelioma and, to a lesser extent, asbestosis and lung cancer [5]. However, OSHA continues to regulate chrysotile and amphibole asbestos as one substance, as both types increase the risk of disease [6]. Currently, EPA's Integrated Risk Information System (IRIS) assessment of asbestos also currently treats mineralogy (and fiber length) as equipotent.

Evidence suggesting that the different types of asbestos fibers vary in carcinogenic potency and site specificity is limited by the lack of information on fiber exposure by mineral type. Other data indicate that differences in fiber size distribution and other process differences can contribute at least as much as fiber type to the observed variation in risk [7].

Counting fibers using the regulatory definitions (see below) does not adequately describe risk of health effects. Fiber size, shape, and composition contribute collectively to risks in ways that are still being elucidated. For example, shorter fibers appear to deposit preferentially in the deep lung, but longer fibers may disproportionately increase the risk of mesothelioma [1,7]. Some of the unregulated amphibole minerals, such as the winchite present in Libby asbestos, can exhibit asbestiform characteristics and contribute to risk. Fiber diameters greater than 2–5 µm are considered above the upper limit of respirability (that is, too large to inhale) and thus do not contribute significantly to risk. Methods are being developed to assess the risks posed by varying types of asbestos and are currently awaiting peer review [7].

Current Standards, Regulations, and Recommendations for Asbestos

In industrial applications, asbestos-containing materials are defined as any material with >1% bulk concentration of asbestos [8]. It is important to note that 1% is not a health-based level, but instead represents the practical detection limit in the 1970s when OSHA regulations were created. Studies have shown that disturbing soil containing <1% amphibole asbestos, however, can suspend fibers at levels of health concern [9].

Friable asbestos (asbestos which is crumbly and can be broken down to suspendible fibers) is listed as a hazardous air pollutant on EPA's Toxic Release Inventory [10]. This classification requires companies that release friable asbestos at concentrations >0.1% to report the release under Section 313 of the Emergency Planning and Community Right-to-Know Act.

OSHA's permissible exposure limit (PEL) is 0.1 f/cc for asbestos fibers with lengths >5 µm and with an aspect ratio (length:width) >3:1, as determined by PCM [6]. This value represents a time-weighted average (TWA) exposure level based on 8 hours per day for a 40-hour work week. In addition, OSHA has defined an "excursion limit," which stipulates that no worker should be exposed in excess of 1 f/cc as averaged over a sampling period of 30 minutes [6]. Historically, the OSHA PEL has steadily decreased from an initial standard of 12 f/cc established in 1971. The PEL levels prior to 1983 were determined on the basis of empirical worker health observations, while the levels set from 1983 forward employed some form of quantitative risk assessment. ATSDR has used the current OSHA PEL of 0.1 f/cc as a reference point for evaluating asbestos inhalation exposure for past workers. ATSDR does not, however, support using the PEL for evaluating exposure for community members, because the PEL was developed as an occupational exposure for adult workers.

In response to the World Trade Center disaster in 2001 and an immediate concern about asbestos levels in buildings in the area, the Department of Health and Human Services, EPA, and the Department of Labor formed the Environmental Assessment Working Group. This work group was made up of ATSDR, EPA, CDC's National Center for Environmental Health, the National Institute for Occupational Safety and Health (NIOSH), the New York City Department of Health and Mental Hygiene, the New York State Department of Health, OSHA, and other state, local, and private entities. The work group set a re-occupation level of 0.01 f/cc after cleanup. Continued monitoring was also recommended to limit long-term exposure at this level [11]. In 2002, a multiagency task force headed by EPA was formed specifically to evaluate indoor environments for the presence of contaminants that might pose long-term health risks to residents in Lower Manhattan. The task force, which included staff from ATSDR, developed a health-based benchmark of 0.0009 f/cc for indoor air. This benchmark was developed to be protective under long-term exposure scenarios, and it is based on risk-based criteria that include conservative exposure assumptions and the current EPA cancer slope factor. The 0.0009 f/cc benchmark for indoor air was formulated on the basis of chrysotile fibers and is therefore most appropriately applied to airborne chrysotile fibers [12].

NIOSH set a recommended exposure limit of 0.1 f/cc for asbestos fibers longer than 5 µm. This limit is a TWA for up to a 10-hour workday in a 40-hour work week [13]. The American Conference of Government Industrial Hygienists has also adopted a TWA of 0.1 f/cc as its threshold limit value [14].

EPA has set a maximum contaminant level (MCL) for asbestos fibers in water of 7,000,000 fibers longer than 10 µm per liter, on the basis of an increased risk of developing benign intestinal polyps [15]. Many states use the same value as a human health water quality standard for surface water and groundwater.

Asbestos is a known human carcinogen. Historically, EPA's IRIS model calculated an inhalation unit risk for cancer (cancer slope factor) of 0.23 per f/cc of asbestos [3]. This value estimates

additive risk of lung cancer and mesothelioma using a relative risk model for lung cancer and an absolute risk model for mesothelioma.

This quantitative risk model has significant limitations. First, the unit risks were based on measurements with phase contrast microscopy and therefore cannot be applied directly to measurements made with other analytical techniques. Second, the unit risk should not be used if the air concentration exceeds 0.04 f/cc because the slope factor above this concentration might differ from that stated [3]. Perhaps the most significant limitation is that the model does not consider mineralogy, fiber-size distribution, or other physical aspects of asbestos toxicity. EPA is in the process of updating their asbestos quantitative risk methodology given the limitations of the IRIS model currently used and the knowledge gained since this model was implemented in 1986.

Appendix B References

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Appendix C. Exposure pathways for vermiculite processing facilities*

Pathway	Environmental media and transport mechanisms	Point of exposure	Route of exposure	Exposed population	Time
Occupational	Suspension of Libby asbestos fibers or contaminated dust into air during materials transport and handling operations or during processing operations	On the site	Inhalation	Former workers	Past
	Suspension of Libby asbestos fibers into air from residual contamination inside former processing buildings	Inside former processing buildings	Inhalation	Current workers	Present, Future
Household Contact	Suspension of Libby asbestos fibers into household air from clothing or body of workers who did not shower or change clothes after work	Workers' homes	Inhalation	Former and/or current workers' families and other household contacts	Past, present, future
Waste Piles	Suspension of Libby asbestos fibers into air by playing in or otherwise disturbing piles of vermiculite or waste rock	Waste piles on the site	Inhalation	Community members, particularly children	Past, present, future
On-site soil	Suspension of Libby asbestos fibers into air from disturbing contaminated material remaining in on-site soils (residual soil contamination, buried waste)	At areas of remaining contamination at or around the site	Inhalation	Current on-site workers, contractors, community members	Past, Present, future
Ambient Air	Stack emissions and fugitive dust from plant operations into neighborhood air	Neighborhood around site	Inhalation	Community members, nearby workers	Past
Residential Outdoor	Suspension of Libby asbestos fibers into air by disturbing contaminated vermiculite brought off the site for personal uses (gardening, paving driveways, traction, fill)	Residential yards or driveways	Inhalation	Community members	Past, present, future
Residential Indoor	Suspension of household dust containing Libby asbestos from plant emissions or waste rock brought home for personal use	Residences	Inhalation	Community members	Past, present, future
Consumer Products	Suspension of Libby asbestos fibers into air from using or disturbing insulation or other consumer products containing Libby vermiculite.	At homes where Libby asbestos-contaminated products were/are present	Inhalation	Community members, contractors, and repairmen	Past, present, future

* The contaminant source for all pathways is asbestos-contaminated vermiculite from Libby, Montana.