



# Federal Register

---

**Friday,  
July 29, 2005**

---

**Part II**

**Department of Labor**

---

**Mine Safety and Health Administration**

---

**30 CFR Parts 56, 57, and 71  
Asbestos Exposure Limit; Proposed Rule**

**DEPARTMENT OF LABOR****Mine Safety and Health Administration****30 CFR Parts 56, 57, and 71**

RIN: 1219-AB24

**Asbestos Exposure Limit**

**AGENCY:** Mine Safety and Health Administration (MSHA), Labor.

**ACTION:** Proposed rule; notice of public hearings.

**SUMMARY:** We (MSHA) are proposing to revise our existing health standards for asbestos exposure at metal and nonmetal mines, surface coal mines, and surface areas of underground coal mines. The proposed rule would reduce the full-shift permissible exposure limit and the excursion limit for airborne asbestos fibers, and make several nonsubstantive changes to add clarity to the standard. Exposure to asbestos has been associated with lung and other cancers, mesotheliomas, and asbestosis. This proposed rule would help assure that fewer miners who work in an environment where asbestos is present would suffer material impairment of health or functional capacity over their working lifetime.

**DATES:** We must receive your comments on or before September 20, 2005. We will hold public hearings on October 18 and 20. Details about the public hearings are in the **SUPPLEMENTARY INFORMATION** section of this preamble.

**ADDRESSES:** (1) To submit comments, please include "RIN: 1219-AB24" in the subject line of the message and send them to us at either of the following addresses.

• *Federal e-Rulemaking portal:* Go to <http://www.regulations.gov> and follow the online instructions for submitting comments.

• *E-mail:* [zzMSHA-comments@dol.gov](mailto:zzMSHA-comments@dol.gov). If you are unable to submit comments electronically, please identify them by "RIN: 1219-AB24" and send them to us by any of the following methods.

• *Fax:* 202-693-9441.  
• *Mail, hand delivery, or courier:* MSHA, Office of Standards, Regulations, and Variances, 1100 Wilson Blvd., Rm. 2350, Arlington, VA 22209-3939.

(2) We will post all comments on the Internet without change, including any personal information they may contain. You may access the rulemaking docket via the Internet at <http://www.msha.gov/regsinfo.htm> or in person at MSHA's public reading room at 1100 Wilson Blvd., Rm. 2349, Arlington, VA.

(3) To receive an e-mail notification when we publish rulemaking

documents in the **Federal Register**, subscribe to our *list serve* at <http://www.msha.gov/subscriptions/subscribe.aspx>.

**FOR FURTHER INFORMATION CONTACT:**

Rebecca J. Smith at 202-693-9440 (Voice), 202-693-9441 (Fax), or <mailto:smith.rebecca@dol.gov> (E-mail).

**SUPPLEMENTARY INFORMATION:****I. Introduction***A. Outline of Preamble*

We are including the following outline to help you find information in this preamble more quickly.

**I. Introduction**

- A. Outline of Preamble
- B. Dates and Locations for Public Hearings
- C. Executive Summary
- D. Abbreviations and Acronyms

**II. Background**

- A. Scope of Proposed Rule
- B. Where Asbestos Is Found at Mining Operations
- C. Asbestos Minerals

**III. History of Asbestos Regulation**

- A. MSHA's Asbestos Standards for Mining
- B. OSHA's Asbestos Standards for General Industry and Construction
- C. Other Federal Agencies Regulating Asbestos
- D. Other Asbestos-Related Activities
- E. U.S. Department of Labor, Office of the Inspector General (OIG)

**IV. Health Effects of Asbestos Exposure**

- A. Summary of Asbestos Health Hazards
- B. Factors Affecting the Occurrence and Severity of Disease
- C. Specific Human Health Effects
- D. Support from Toxicological Studies of Human Health Effects of Asbestos Exposure

**V. Characterization and Assessment of Exposures in Mining**

- A. Determining Asbestos Exposures in Mining
- B. Exposures from Naturally Occurring Asbestos
- C. Exposures from Introduced (Commercial) Asbestos
- D. Sampling Data and Exposure Calculations

**VI. The Application of OSHA's Risk Assessment to Mining**

- A. Summary of Studies Used by OSHA in Its Risk Assessment
- B. Models Selected by OSHA (1986) for Specified Endpoints and for the Determination of Its PEL and STEL
- C. OSHA's Selection of Its PEL (0.1 f/cc)
- D. Applicability of OSHA's Risk Assessment to the Mining Industry
- E. Significance of Risk

**VII. Section-by-Section Discussion of Proposed Rule**

- A. Sections 56/57.5001(b)(1) and 71.702(a): Definitions
- B. Sections 56/57.5001(b)(2) and 71.702(b): Permissible Exposure Limits (PELs)
- C. Sections 56/57.5001(b)(3) and 71.702(c): Measurement of Airborne Fiber Concentration
- D. Discussion of Asbestos Take-Home Contamination

- E. Section 71.701(c) and (d): Sampling; General Requirements
- VIII. Regulatory Analyses
  - A. Executive Order (E.O.) 12866
  - B. Feasibility
  - C. Alternatives Considered
  - D. Regulatory Flexibility Analysis (RFA) and Small Business Regulatory Enforcement Fairness Act (SBREFA)
  - E. Other Regulatory Considerations
- IX. Copy of the OSHA Reference Method (ORM)
- X. References Cited in the Preamble

*B. Dates and Locations for Public Hearings*

We will hold two public hearings. If you wish to make a statement for the record, please submit your request to us at least 5 days prior to the hearing dates by one of the methods listed in the **ADDRESSES** section above. The hearings will begin at 9 a.m. with an opening statement from MSHA, followed by statements or presentations from the public, and end after the last speaker (in any event not later than 5 p.m.) on the following dates at the locations indicated:

October 18, 2005, Denver Federal Center, Sixth and Kipling, Second Street, Building 25, Denver, Colorado 80225, Phone: 303-231-5412.

October 20, 2005, Mine Safety and Health Administration, 1100 Wilson Boulevard, Room 2539, Arlington, Virginia 22209, Phone: 202-693-9457.

We will hear scheduled speakers first, in the order that they sign in; however, you do not have to make a written request to speak. To the extent time is available, we will hear from persons making same-day requests. The presiding official may exercise discretion to ensure the orderly progress of the hearing by limiting the time allocated to each speaker for their presentation.

The hearings will be conducted in an informal manner. Although formal rules of evidence or cross examination will not apply, the hearing panel may ask questions of speakers and a verbatim transcript of the proceedings will be prepared and made a part of the rulemaking record. We also will post the transcript on MSHA's Home Page at <http://www.msha.gov>, on the *Asbestos Single Source Page*.

Speakers and other attendees may present information to the MSHA panel for inclusion in the rulemaking record. We will accept written comments and data for the record from any interested party, including those not presenting oral statements. The post-hearing comment period will close on November 21, 2005, 30 days after the last public hearing.

### C. Executive Summary

In March of 2001, the U.S. Department of Labor, Office of the Inspector General (OIG) published a report evaluating MSHA's enforcement actions at the vermiculite mine in Libby, Montana. The widespread asbestos contamination at this mine and surrounding community, together with the prevalence of asbestos-related illnesses and fatalities among persons living in this community, attracted press and public attention, which prompted the OIG investigation and report. The OIG found that MSHA had conducted regular inspections and personal exposure sampling at the mine, as required by the Federal Mine Safety and Health Act of 1977 (Mine Act). The OIG report stated, "We do not believe that more inspections or sampling would have prevented the current situation in Libby." The OIG made five recommendations to MSHA; two of which we implemented immediately. The remaining recommendations are listed below:

- Lower the existing permissible exposure limit (PEL) for asbestos to a more protective level.
- Use transmission electron microscopy (TEM) instead of phase contrast microscopy (PCM) in the initial analysis of fiber samples that may contain asbestos.
- Implement special safety requirements to address take-home contamination.

In response to the OIG's recommendations, MSHA published an advance notice of proposed rulemaking (ANPRM) on March 29, 2002 (67 FR 15134). MSHA also held seven public meetings around the country to seek input and obtain public comment on how best to protect miners from exposure to asbestos.

Following review of all public comments and testimony taken at the public meetings, and relying on OSHA's 1986 asbestos risk assessment, we determined that it is appropriate to propose reducing the PELs for asbestos and clarify criteria for asbestos sample analysis. To enhance the health and safety of miners, we are proposing to lower the existing 8-hour, time-weighted average (TWA) PEL of 2.0 f/cc to 0.1 f/cc, and to lower the short-term limit from 10.0 f/cc over a minimum sampling time of 15 minutes to an excursion limit PEL of 1.0 f/cc over a minimum sampling time of 30 minutes. To clarify the criteria for the analytical method in our existing standards, we are proposing to incorporate a reference to Appendix A of OSHA's asbestos standard (29 CFR 1910.1001). Appendix

A specifies basic elements of a PCM method for analyzing airborne asbestos samples. It includes the same analytical elements specified in our existing standards and allows MSHA's use of other methods that meet the statistical equivalency criteria in OSHA's asbestos standard.

The scope of this proposed rule, therefore, is limited to lowering the permissible exposure limits, an issue raised by the OIG; incorporating Appendix A of OSHA's asbestos standard for the analysis of our asbestos samples; and making several nonsubstantive conforming amendments to our existing rule language. After considering several regulatory approaches to prevent take-home contamination, we determined that non-regulatory measures could adequately address this potential hazard.

### D. Abbreviations and Acronyms

As a quick reference, we list below some of the abbreviations used in the preamble.

29 CFR Title 29, *Code of Federal Regulations*

30 CFR Title 30, *Code of Federal Regulations*

AFL-CIO American Federation of Labor and Congress of Industrial Organizations  
 ATSDR Agency for Toxic Substances and Disease Registry, Centers for Disease Control and Prevention, U.S. Department of Health and Human Services  
 Bureau former Bureau of Mines, U.S. Department of the Interior

cc cubic centimeter (cm<sup>3</sup>) = milliliter (mL)  
 EPA U.S. Environmental Protection Agency  
 f fiber(s)

FR **Federal Register**

Lpm liter(s) per minute

MESA former Mining Enforcement and Safety Administration, U.S. Department of the Interior (predecessor to MSHA)

MSHA Mine Safety and Health Administration, U.S. Department of Labor  
 mm millimeter = 1 thousandth of a meter (0.001 m)

mL milliliter = 1 thousandth of a liter (0.001 L) = cubic centimeter

NIOSH National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, U.S. Department of Health and Human Services

OIG Office of the Inspector General, U.S. Department of Labor

OSHA Occupational Safety and Health Administration, U.S. Department of Labor

PCM phase contrast microscopy

PEL permissible exposure limit

PLM polarized light microscopy

STEL short-term exposure limit

SWA shift-weighted average concentration

TEM transmission electron microscopy

TWA time-weighted average concentration  
 μm micron = micrometer = 1 millionth of a meter (0.000001 m)

USGS U.S. Geological Survey, U.S. Department of the Interior

## II. Background

### A. Scope of Proposed Rule

This proposed rule would apply to metal and nonmetal mines, surface coal mines, and the surface areas of underground coal mines. Because asbestos from any source poses a health hazard to miners if they inhale it, the proposed rule would cover all miners exposed to asbestos whether naturally occurring or contained in building materials, in other manufactured products at the mine, or in mine waste or tailings.

The National Institute for Occupational Safety and Health (NIOSH) and other research organizations and scientists (see Table VI-5) have observed the occurrence of cancers and asbestosis among metal and nonmetal miners involved in the mining and milling of commodities that contain asbestos. For this reason, our primary focus at metal and nonmetal mines is on asbestos in pockets or veins of mined commodities. Historically, there has been no evidence of coal miners encountering naturally occurring asbestos.<sup>1</sup> The more likely exposure to asbestos in coal mining would occur from introduced asbestos-containing products, such as asbestos-containing building materials (ACBM) in surface structures.

In 2000, the OIG investigated MSHA's activities at the vermiculite mine in Libby, Montana. The OIG's conclusions and recommendations, discussed later, are consistent with MSHA's observations and concerns that—

- Miners are exposed to asbestos at mining operations where the ore body or surrounding rock contains asbestos;
- Miners are potentially exposed to airborne asbestos at mine facilities with installed asbestos-containing material when it is disturbed during maintenance, construction, renovation, or demolition activities; and
- Family and community are potentially exposed if miners take asbestos home on their person, clothes, or equipment, or in their vehicle.

We developed this proposed rule based on our experience with asbestos, our assessment of the health risks, the OIG's recommendations, and public comments on MSHA's ANPRM addressing the OIG's recommendations. We received numerous comments in response to the ANPRM and at the

<sup>1</sup> Personal communication with Professor Kot Unrug, Department of Mining Engineering, University of Kentucky, on November 14, 2003; and with Syd S. Peng, Chairman, Department of Mining Engineering, College of Engineering and Mineral Resources, West Virginia University, the week of October 24, 2003.

public meetings, some of which suggested or supported additional requirements beyond those addressed by the OIG. We believe that the comments to the ANPRM do not justify an expansion of the scope, at this time, beyond the recommendations specifically raised in the OIG report.

On the contrary, we believe that our data support a narrowed scope in that we specifically are not proposing two of the OIG's recommendations, *i.e.*, routine use of TEM for the initial analysis of exposure samples and promulgation of standards to prevent take-home contamination. We are proposing, however, to lower our permissible exposure limits.

We have decided not to propose to change our existing definition of asbestos in this rulemaking. There are several reasons for this.

First, this rulemaking is limited in scope. We believe that a 20-fold lowering of the exposure limits, as we have proposed, together with our enhanced measures to educate the mining community about the asbestos hazard in mining, would increase protection for miners and help avoid the future development of situations such as that in Libby, Montana.

Second, interest in the definition of asbestos extends to numerous agencies in Federal, state, and local governments. Our existing definition is consistent with several Federal agencies' regulatory provisions, including OSHA's. Changing the definition would require considerable interagency consultation and coordination; additional scientific evaluation; and an unnecessary delay in providing miners access to the benefits of this proposed rule.

Third, we believe another Libby-like mining operation would not exist today because such a business arguably would not be economically viable. If a mine's ore contained significant amounts of asbestos-like minerals, there is a strong likelihood of potential liability risks, both from customers and workers, and the possibility that the mine's product would be commercially unmarketable. Such market forces are likely to compel mining companies of all sizes to sample the ore for the presence of hazardous fibrous minerals before purchasing or developing a mine site. In our view, these commercial reasons make it unlikely that a new Libby-like mining condition would arise in the future.

#### B. Where Asbestos Is Found at Mining Operations

Asbestos is no longer mined as a commodity in the United States. Even so, veins, pockets, or intrusions of

asbestos have been found in other ores in specific geographic regions, primarily in metamorphic or igneous rock.<sup>2</sup> Although less common, it is not impossible to find asbestos in sedimentary rock, soil, and air from the weathering or abrasion of other asbestos-bearing rock.<sup>3</sup> The areas where asbestos may be located can be determined from an understanding of the mineralogy of asbestos and the geology required for its formation. In some cases, visual inspection can detect the presence of asbestos. MSHA experience indicates that miners may encounter asbestos during the mining of a number of mineral commodities,<sup>4</sup> such as talc, limestone and dolomite, vermiculite, wollastonite, banded ironstone and taconite, lizardite, and antigorite. Not all mines of a specific commodity contain asbestos in the ore, however, and the mines that do have asbestos in the ore may encounter it rarely.

Asbestos also is contained in building materials and other manufactured products found at mines. Contrary to the common public perception, asbestos is not banned in the United States.<sup>5</sup> The U.S. Geological Survey (USGS) estimates that about 13,000 metric tons (29 million pounds) of asbestos were used in product manufacturing in the United States during 2001.<sup>6</sup> In addition to domestic manufacturing, the United States continues to import products that contain asbestos. Asbestos may be used for a number of purposes at a mine including insulation; reinforcement of cements; reinforcement of floor, wall, and building tile; and automotive clutch and brake linings.<sup>7</sup> If asbestos is present at the mine, miners in the vicinity are potentially at increased risk from asbestos exposure, regardless of whether or not they are actually working with asbestos.

#### C. Asbestos Minerals

To understand the scientific literature, information about asbestos, and the issues raised in the public comments, it is important to understand the terminology used to describe minerals, asbestos, and fibers. This section briefly reviews a number of key terms and concepts associated with asbestos that we use in discussing this proposed rule.

<sup>2</sup> MSHA (Bank), 1980.

<sup>3</sup> USGS, 1995.

<sup>4</sup> Roggli *et al.*, 2002; Selden *et al.*, 2001; Amandus *et al.*, Part I, 1987; Amandus *et al.*, Part III, 1987; Amandus and Wheeler, Part II, 1987.

<sup>5</sup> GETF Report, pp. 12–13, 2003.

<sup>6</sup> USGS (Virta), p. 28, 2003.

<sup>7</sup> Lemen, 2003; Paustenbach *et al.*, 2003.

#### 1. Mineralogical Classification and Mineral Names

The terminology used to refer to how minerals form and how they are named is complex. A mineral's physical properties, composition, crystalline structure, and morphology determine its classification. Asbestos minerals belong to either the serpentine (sheet silicate) or the amphibole (double-chain silicate) family of minerals. Most of the difficulties in classifying minerals as asbestos have involved the amphiboles. The formation of a particular mineral (chemical composition) or habit (morphology, crystalline structure) occurs gradually and may be incomplete, producing intermediate minerals that are difficult to classify. In the past, there have been several different systems used to classify and name minerals that, in some instances, led to inconsistent terminology and classification. Currently, there is no single, universally accepted system for naming minerals.

Asbestos is a commercial term used to describe certain naturally occurring, hydrated silicate minerals. Several Federal agencies have regulations that focus on these minerals. The properties of asbestos that give it commercial value include low electrical and thermal conductivity, chemical and crystalline stability and durability, high tensile strength, flexibility, and friability. Much of the existing health risk data for asbestos uses commercial mineral terminology. Meeker *et al.* (2003) recognized the confusion associated with asbestos nomenclature, stating—

Within much of the existing asbestos literature, mineral names are not applied in a uniform manner and are not all consistent with presently accepted mineralogical nomenclature and definitions.

##### a. Variations in Mineral Morphology.

There are many types of crystal habits, such as fibrous, acicular (slender and needle-like), massive (irregular form), and columnar (stout and column-like). The morphology of a mineral may not fit a precise definition. For example, Meeker *et al.* (2003) state that the Libby amphiboles contain “a complete range of morphologies from prismatic crystals to asbestiform fibers.” Some minerals crystallize in more than one habit. Some minerals, which can form in different habits, have a different name for each habit; others do not.<sup>8</sup> For example, crocidolite is the name for the asbestiform habit and riebeckite is the name for the same mineral in its nonasbestiform habit. Tremolite and actinolite do not have different names

<sup>8</sup> Reger and Morgan, 1990; ATSDR, p. 138, 2001.

depending on habit; therefore, to distinguish between the different habits, the descriptive term "asbestiform" or "asbestos" is added to the mineral's name. If the identifying, descriptive term is not used with the mineral name, misunderstandings or mistakes may occur.

b. *Variations in Mineral Composition.*

Atoms similar in size and valence state can replace each other within a mineral's crystal lattice, resulting in the formation of a different mineral in the same mineral series. This process is gradual and can occur to a different extent in the same mineral depending on the geological conditions during its formation. For example, tremolite contains magnesium, but no (or little) iron, and holds an end member position in its mineral series. Iron atoms can replace the magnesium atoms in tremolite and the resulting mineral may then be called actinolite. The quantity of iron needed before the mineral is called actinolite varies depending on the mineral classification scheme used. Another example is winchite, which is an intermediate member of the tremolite-glaucophane series, as well as an end member in its own sodic-calcic series.<sup>9</sup> Given the chemical similarity within the series, winchite  $[(\text{NaCa})\text{Mg}_4(\text{Al,Fe}^{3+})\text{Si}_8\text{O}_{22}(\text{OH})_2]$  often has been reported as tremolite  $[\text{Ca}_2\text{Mg}_5\text{Si}_8\text{O}_{22}(\text{OH})_2]$ .

A specific rock formation may contain a continuum of minerals from one end member of a series to the other end member, creating a solid solution of intermediate minerals. These intermediate minerals are sometimes given names, while at other times they are not. Often, when the exact chemical composition is not determined or determined to be a number of different intermediate minerals, the mineral is named by one or more of its end members, such as tremolite-actinolite or cummingtonite-grunerite. The fibrous amphiboles in the Libby ore body, for example, contain both end members and several intermediate minerals. Meeker *et al.* (2003) state that—

The variability of compositions on the micrometer scale can produce single fibrous particles that can have different amphibole names at different points of the particle.

A mineral may also undergo transition to a different mineral series. Kelse and Thompson (1989), Ross (1978), and USGS (Virta, 2002) have commented on the chemical transition of anthophyllite to talc. Stewart and Lee (1992) stated that fibrous talc might contain intermediate particles not easily

differentiated from asbestos. In the context of systems for naming and classifying fibrous amphiboles, Meeker *et al.* (2003) state that the regulatory literature often gives nominal compositions for a mineral without specifying chemical boundaries.

2. Differentiating Asbestiform and Nonasbestiform Habit

In the asbestiform habit, mineral crystals grow forming long, thread-like fibers. When pressure is applied to an asbestos fiber, it bends much like a wire, rather than breaks. Fibers can separate into "fibrils" of a smaller diameter (often less than 0.5  $\mu\text{m}$ ). This effect is referred to as "polyfilamentous," and should be viewed as one of the most important characteristics of asbestos. Appendix A of the Environmental Protection Agency's (EPA's) *Method for the Determination of Asbestos in Bulk Building Materials*<sup>10</sup> defines asbestiform as follows:

\* \* \* a mineral that is like asbestos, *i.e.*, crystallized with the habit [morphology] of asbestos. Some asbestiform minerals may lack the properties which make asbestos commercially valuable, such as long fiber length and high tensile strength. With the light microscope, the asbestiform habit is generally recognized by the following characteristics:

Mean aspect [length to width] ratios ranging from 20:1 to 100:1 or higher for fibers longer than 5 micrometers. Aspect ratios should be determined for fibers, not bundles.

Very thin fibrils, usually less than 0.5 micrometers in width, and two or more of the following:

- Parallel fibers occurring in bundles,
- Fiber bundles displaying splayed ends,
- Matted masses of individual fibers, and/or
- Fibers showing curvature.

In the nonasbestiform habit, mineral crystals do not grow in long thin fibers. They grow in a more massive habit. For example, a long thin crystal may not be polyfilamentous nor possess high tensile strength and flexibility, but may break rather than bend. When pressure is applied, the nonasbestiform crystals fracture easily into prismatic particles, which are called cleavage fragments because they result from the particle's breaking or cleavage, rather than the crystal's formation or growth. Some particles are acicular (needle shaped), and stair-step cleavage along the edges of some particles is common.

Cleavage fragments may be formed when nonfibrous amphibole minerals are crushed, as may occur in mining and milling operations. Cleavage fragments are not asbestiform and do not fall within our definition of asbestos. For some minerals, distinguishing between

asbestiform fibers and cleavage fragments in certain size ranges is difficult or impossible when only a small number of structures are available for review, as opposed to a representative population. Meeker *et al.* (2003) states that it is often difficult or impossible to determine differences between acicular cleavage fragments and asbestiform mineral fibers on an individual fiber basis. A determination as to whether a mineral is asbestiform or not must be made, where possible, by applying existing analytical methods. Although we have received comments regarding the hazards associated with cleavage fragments, we do not intend to modify our existing definition of asbestos with this rulemaking.

### III. History of Asbestos Regulation

When Federal agencies responsible for occupational safety and health began to regulate occupational exposure to asbestos, studies had already established that the inhalation of asbestos fibers was a major cause of disability and death among exposed workers. The intent of these first asbestos rules was to protect workers from developing asbestosis.<sup>11</sup>

#### A. MSHA's Asbestos Standards for Mining

*1967–1969.* In 1967, under the former Bureau of Mines, predecessor to the Mining Enforcement and Safety Administration (MESA) and then MSHA, the standard for asbestos exposure in mining was an 8-hour, time-weighted average (TWA) PEL of 5 mppcf (million particles per cubic foot of air). In 1969, the Bureau promulgated a 2 mppcf and 12 f/mL (fibers per milliliter) standard.

*1974–1976.* In 1974, MESA promulgated a 5 f/mL standard for asbestos exposure in metal and nonmetal mines (39 FR 24316). In 1976, MESA promulgated a 2 f/cc standard (41 FR 10223) for asbestos exposure in surface areas of coal mines. We retained these standards under the authority of the Federal Mine Safety and Health Act of 1977.

*1978.* In November 1978, we promulgated a 2 f/mL standard for asbestos exposure in metal and nonmetal mines (43 FR 54064). Since then, we have made only nonsubstantive changes to our asbestos standards, *e.g.*, renumbering the section of the standard in 30 CFR.

MSHA's existing standards for asbestos at metal and nonmetal mines at 30 CFR 56/57.5001 state,

<sup>9</sup> Leake *et al.*, p. 222, 1997.

<sup>10</sup> EPA, 1993.

<sup>11</sup> GETF Report, p. 33, 2003.

(b) The 8-hour time-weighted average airborne concentration of asbestos dust to which employees are exposed shall not exceed 2 fibers per milliliter greater than 5 microns in length, as determined by the membrane filter method at 400–450 magnification (4 millimeter objective) phase contrast illumination. No employees shall be exposed at any time to airborne concentrations of asbestos fibers in excess of 10 fibers longer than 5 micrometers, per milliliter of air, as determined by the membrane filter method over a minimum sampling time of 15 minutes. “Asbestos” is a generic term for a number of hydrated silicates that, when crushed or processed, separate into flexible fibers made up of fibrils. Although there are many asbestos minerals, the term “asbestos” as used herein is limited to the following minerals: chrysotile, Amosite, crocidolite, anthophyllite asbestos, tremolite asbestos, and actinolite asbestos.

The existing standard for asbestos at surface coal mines and surface work areas of underground coal mines at 30 CFR 71.702 states,

(a) The 8-hour average airborne concentration of asbestos dust to which miners are exposed shall not exceed two fibers per cubic centimeter of air. Exposure to a concentration greater than two fibers per cubic centimeter of air, but not to exceed 10 fibers per cubic centimeter of air, may be permitted for a total of 1 hour each 8-hour day. As used in this subpart, the term asbestos means chrysotile, amosite, crocidolite, anthophyllite asbestos, tremolite asbestos, and actinolite asbestos but does not include nonfibrous or nonasbestiform minerals.

(b) The determination of fiber concentration shall be made by counting all fibers longer than 5 micrometers in length and with a length-to-width ratio of at least 3 to 1 in at least 20 randomly selected fields using phase contrast microscopy at 400–450 magnification.

1989. In 1989, as part of our Air Quality rulemaking, we proposed to lower the full-shift exposure limit for asbestos from 2 f/cc to 0.2 f/cc to address the excessive risk quantified in the Occupational Safety and Health Administration’s (OSHA’s) 1986 asbestos rule (54 FR 35760). The Air Quality rulemaking, however, was withdrawn on September 26, 2002 (67 FR 60611). MSHA has not reinstated the Air Quality rulemaking at this time.

#### B. OSHA’s Asbestos Standards for General Industry and Construction

1971–1972. The initial promulgation of OSHA standards on May 29, 1971 (36 FR 10466) included a 12 f/cc PEL for asbestos. Then, on December 7, 1971, in response to a petition by the Industrial Union Department of the AFL-CIO, OSHA issued an emergency temporary standard (ETS) on asbestos that established an 8-hour, TWA PEL of 5 f/

cc and a peak exposure level (ceiling limit) of 10 f/cc. In June 1972, OSHA promulgated these limits in a final rule.

1975. In October 1975, OSHA proposed to revise its asbestos standard by reducing the 8-hour, TWA PEL to 0.5 f/cc with a ceiling limit of 5 f/cc for 15 minutes (40 FR 47652). OSHA stated that sufficient medical and scientific evidence had accumulated to warrant the designation of asbestos as a human carcinogen and that advances in monitoring and protective technology made re-examination of the standard appropriate. The final rule, however, reduced OSHA’s 8-hour, TWA asbestos PEL to 2 f/cc due to feasibility concerns. This limit remained in effect until OSHA revised it in 1986.

1983–1986. On November 4, 1983, OSHA published another emergency temporary standard (ETS) for asbestos (48 FR 51086), which would have lowered the 8-hour, TWA PEL from 2 f/cc to 0.5 f/cc. The Asbestos Information Association challenged the ETS in the U.S. Court of Appeals for the 5th Circuit. On March 7, 1984, ruling on *Asbestos Information Association/North America v. OSHA* (727 F.2d 415, 1984), the Court invalidated the ETS. Subsequent to this decision, OSHA published a proposed rule (49 FR 14116) that, together with the ETS, proposed two alternatives for lowering the 8-hour, TWA PEL: 0.2 f/cc and 0.5 f/cc.

On June 17, 1986, OSHA issued comprehensive asbestos standards (51 FR 22612) governing occupational exposure to asbestos in general industry workplaces (29 CFR 1910.1001), construction workplaces (29 CFR 1926.1101), and shipyards (29 CFR 1915.1001). The separate standards shared the same asbestos PEL and most ancillary requirements. These standards reduced OSHA’s 8-hour, TWA PEL to 0.2 f/cc from the previous 2 f/cc limit. OSHA added specific provisions in the construction standard to cover unique hazards relating to asbestos abatement and demolition jobs.

Although tremolite, actinolite, and anthophyllite exist in different forms, OSHA determined that all forms of these minerals would continue to be regulated. Following promulgation of the rule, several parties requested an administrative stay of the standard claiming that OSHA improperly included nonasbestiform minerals. A temporary stay was granted and OSHA initiated rulemaking to remove the nonasbestiform types of these minerals from the scope of the asbestos standards.

1988. Several major participants in OSHA’s rulemaking challenged various

provisions of the 1986 revised standards. In *Building Construction Trades Division (BCTD), AFL-CIO v. Brock* (838 F.2d 1258, 1988), the U.S. Court of Appeals for the District of Columbia upheld most of the challenged provisions, but remanded certain issues to OSHA for reconsideration. In partial response, on September 14, 1988, OSHA promulgated an excursion limit of 1 f/cc for asbestos as measured over a 30-minute sampling period (53 FR 35610).

1992. OSHA’s 1986 standards had applied to occupational exposure to nonasbestiform actinolite, tremolite, and anthophyllite. On June 8, 1992, OSHA deleted the nonasbestiform types of these minerals from the scope of its asbestos standards. In evaluating the record, OSHA found (57 FR 24310–24311) insufficient evidence that nonasbestiform actinolite, tremolite, and anthophyllite present “a risk similar in kind and extent” to their asbestiform counterparts. Additionally, the evidence did not show that OSHA’s removal of the nonasbestiform types of these three minerals from its asbestos standard “will pose a significant risk to exposed employees.”

1994. On August 10, 1994, OSHA published a final rule (59 FR 40964) that lowered its 8-hour, TWA PEL for asbestos to 0.1 f/cc and retained the 1 f/cc excursion limit as measured over 30 minutes.

#### C. Other Federal Agencies Regulating Asbestos

Because the health hazards of exposure to asbestos are well recognized, it is highly regulated. OSHA and MSHA have the primary authority to regulate occupational exposures to asbestos. EPA regulates asbestos exposure of state and local government workers in those states that do not have an OSHA State Plan covering them. A number of other Federal agencies, primarily EPA and the Consumer Product Safety Commission (CPSC), regulate non-occupational asbestos exposures. For example, CPSC regulates asbestos in consumer products, such as patching compounds, under the Federal Hazardous Substances Act.

EPA regulates asbestos in air and materials. EPA’s activities have focused on environmental issues and the public health by reducing emissions of hazardous gases and dusts from large industrial sources, such as taconite ore processing,<sup>12</sup> and the cleanup of contaminated waste sites. EPA also regulates asbestos in schools. The mining and processing of vermiculite in Libby, Montana, resulted in the spread

<sup>12</sup> EPA (68 FR 61868), 2003.

of asbestos to numerous homes, schools, and businesses throughout the town. In November 1999, EPA responded to a request to study the environmental contamination in the town of Libby and widespread illnesses and death among its residents. In October 2002, EPA designated the area as a Superfund site.

#### D. Other Asbestos-Related Activities

There have been increasing numbers of studies on asbestos and its hazards over the past 40 years. These efforts encompass government, industry, and academia on a local, national, and international scale. Government agencies and scientific groups in the United States, such as the National Institute for Occupational Safety and Health (NIOSH), the Agency for Toxic Substances and Disease Registry (ATSDR), the American Conference of Governmental Industrial Hygienists (ACGIH), and the National Toxicology Program (NTP), have addressed issues involving carcinogens, such as asbestos. Organizations from other countries, such as the United Kingdom (Health and Safety Executive) and Germany (Deutsche Forschungsgemeinschaft), also have addressed occupational exposure to asbestos and other carcinogens. Similarly, the International Agency for Research on Cancer (IARC) has published a monograph on asbestos that summarizes evidence of its carcinogenicity.<sup>13</sup>

#### 1. Interagency Asbestos Work Group (IAWG)

OSHA's and EPA's overlapping responsibilities and common interest in addressing asbestos hazards led to the formation of the IAWG. Participating Federal agencies include EPA, OSHA, CPSC, MSHA, NIOSH, ATSDR, USGS, and the National Institute of Standards and Technology (NIST). This work group of government agencies facilitates the sharing of information and coordination of activities, including regulatory activities, environmental assessment, technical assistance, consumer protection, and developments in environmental analysis of contaminants. The IAWG also seeks to harmonize the policies, procedures, and enforcement activities of the participating agencies, thus minimizing or eliminating potential conflicts for the regulated community. For example, the IAWG is currently discussing the Federal definition of asbestos.

#### 2. National Institute for Occupational Safety and Health (NIOSH)

The Workers' Family Protection Act of 1992 (29 U.S.C. 671A) directed NIOSH to study contamination of workers' homes by hazardous substances, including asbestos, transported from the workplace. ATSDR, EPA, OSHA, MSHA, the U.S. Department of Energy (DOE), and the Centers for Disease Control and Prevention (CDC) assisted NIOSH in conducting the study. For this proposed rule we focused on the asbestos-related results of these studies.

NIOSH (1995) published its study results in a *Report to Congress on Workers' Home Contamination Study Conducted under the Workers' Family Protection Act*. This report summarizes incidents of home contamination, including the health consequences, sources, and levels of contamination. The study documents cases of asbestos reaching workers' homes in 36 states in the United States and in 28 other countries. These cases covered a wide variety of materials, industries, and occupations. The means by which hazardous substances reached workers' homes and families included taking the substance home on the worker's body, clothing, tools, and equipment; cottage industries (*i.e.*, work performed on home property); and family visits to the workplace. In an effort to reach employers and workers, NIOSH (1997) published its recommendations in *Protect Your Family: Reduce Contamination at Home*. This pamphlet summarizes the NIOSH study and provides recommendations to prevent this contamination.

#### 3. Agency for Toxic Substances and Disease Registry (ATSDR)

The Superfund Amendments and Reauthorization Act of 1986 (SARA) directed ATSDR to prepare toxicological profiles for hazardous substances most commonly found at specific waste sites. ATSDR and EPA determined which hazardous substances pose the most significant potential threat to human health and targeted them for study. Asbestos is one of these targeted substances. ATSDR published one of the most current toxicological profiles for asbestos in September 2001, which was an update of an earlier asbestos profile.

In October 2002, ATSDR sponsored a meeting of expert panelists who presented their evaluation of state-of-the-art research concerning the relationship between fiber length and the toxicity of asbestos and synthetic vitreous fibers. We have reviewed the evidence and arguments presented in

the updated asbestos toxicological profile and the meeting proceedings and have discussed this information in this preamble, where appropriate.

#### E. U.S. Department of Labor, Office of the Inspector General (OIG)

In November 1999, a Seattle newspaper published a series of articles on the unusually high incidence of asbestos-related illnesses and fatalities among individuals who had lived in Libby, Montana. There was extensive national media attention surrounding the widespread environmental contamination and asbestos-related deaths in Libby. Dust and construction materials from the nearby vermiculite mine were the alleged cause. This mine had produced about 90 percent of the world's supply of vermiculite from 1924 until 1992.

Because MSHA had jurisdiction over the mine for two decades before it closed, the OIG investigated MSHA's enforcement actions at the mine. The OIG confirmed that the processing of vermiculite at the mine exposed miners to asbestos. The miners then, inadvertently, had carried the asbestos home on their clothes and in their personal vehicles.<sup>14</sup> In doing this, the miners continued to expose themselves and family members.

#### 1. OIG Report on MSHA's Handling of Inspections at the W.R. Grace & Company Mine in Libby, Montana

The OIG published its findings and recommendations in a report dated March 22, 2001. The OIG found that MSHA had appropriately conducted regular inspections and personal exposure sampling at the Libby mine and that there were no samples exceeding the 2.0 f/cc PEL for the 10 years prior to the mine closing in 1992. The OIG concluded, "We do not believe that more inspections or sampling would have prevented the current situation in Libby." The OIG stated its belief that there is a need for MSHA to lower its asbestos PEL.

In its report, the OIG supported the development and implementation of control measures for asbestos and vermiculite mining and milling. They also made recommendations for improving our effectiveness in controlling this hazard. This proposed rule addresses our responses to several of the OIG's recommendations.

#### 2. MSHA's Libby, Montana Experience

W.R. Grace acquired the vermiculite mine in Libby, Montana, in 1963. At that time, the amphibole in the

<sup>13</sup>IARC, 1987.

<sup>14</sup>Weis *et al.*, 2001.

vermiculite was called tremolite, soda tremolite, soda-rich tremolite, or richterite, and researchers had already linked the mine dust to respiratory disease.<sup>15</sup> The suggested exposure limit for asbestos in mining was much higher than current limits. The federal standard for asbestos in mining dropped from 5 mppcf (about 30 f/mL) in 1967 to 2 f/mL in 1978. When MESA (predecessor agency to MSHA) began inspecting the operation, the exposure limit for asbestos was 5 f/mL.

The mine operator, Federal mine inspectors, and representatives of the U.S. Public Health Service [part of the Centers for Disease Control and Prevention (CDC)] routinely sampled for asbestos at the Libby mine, starting before the mine switched to wet processing in 1974, and continued sampling periodically until the mine closed in 1992. MSHA sampling at the Libby mine found no exposures exceeding the 5.0 f/cc asbestos PEL from 1975 through 1978, and only a few over the 2.0 f/cc asbestos PEL from 1979 through 1986. Almost all the samples would have exceeded the 0.1 f/cc proposed limit. Miners' exposures continued to decrease and more recent sampling since 1986 found few exposures exceeding the OSHA PEL of 0.1 f/cc.

The results from our personal exposure sampling at the Libby mine included many of the fibrous amphiboles present. In addition, the results from TEM analysis of the air samples characterized the mineralogy of the airborne fibers as tremolite and did not distinguish between the species of amphiboles. Further characterization of the amphibole minerals using Scanning Electron Microscopy/Energy Dispersive X-ray Spectroscopy technology shows proportions of about 84 percent winchite, 11 percent richterite, and 6 percent tremolite.<sup>16</sup>

As early as 1980, MSHA had requested that NIOSH investigate health problems at all vermiculite operations, including the mine and mill in Libby, Montana. NIOSH published its study results in a series of three papers (Amandus *et al.*, Part I, 1987; Amandus and Wheeler, Part II, 1987; Amandus *et al.*, Part III, 1987). The study of Amandus *et al.* (Part I, 1987) along with that of McDonald *et al.* (1986) found that, historically, the highest exposures to fibers at the Libby operation had occurred in the mill and that exposures had decreased between the 1960's and

1970's. McDonald *et al.* (1986) reported—

In 1974, the old dry and wet mills were closed and the ore was processed in a new mill built nearby which operated on an entirely wet basis in which separation was made by vibrating screens, Humphrey separators, and flotation.

McDonald *et al.* (1986) and Amandus and Wheeler (Part II, 1987) also showed that, even at reduced exposure levels, there was still increased risk of lung cancer among the Libby miners and millers.

### 3. MSHA's Efforts To Minimize Asbestos Take-Home Contamination

"Take-home" contamination is contamination of workers' homes or vehicles by hazardous substances transported from the workplace. As discussed previously in this preamble, the widespread asbestos-related disease among the residents of Libby, Montana, was attributed, in part, to take-home contamination from the vermiculite mining and milling operation in that town. The OIG report on MSHA's activities recommended that we promulgate special safety standards similar to those in our 1989 proposed Air Quality rule (54 FR 35760) to address take-home contamination.

In our 1989 Air Quality proposed rule, we had proposed that miners wear protective clothing and other personal protective equipment before entering areas containing asbestos. Our Air Quality proposed rule also would have required miners to remove their protective clothing and store them in adequate containers to be disposed of or decontaminated by the mine operator. These proposed requirements were similar to those in OSHA's asbestos standard and to NIOSH's recommendations.

In March 2000, shortly after the series of articles on asbestos-related illnesses and deaths in Libby, Montana, we issued a Program Information Bulletin (PIB No. P00-3) about asbestos. The PIB served to remind the mining industry of the potential health hazards from exposure to airborne asbestos fibers and to raise awareness about potential asbestos exposure for miners, their families, and their communities. At that time, we also issued a Health Hazard Information Card (No. 21) about asbestos for distribution to miners to raise their awareness about the health hazards related to asbestos exposure.

The PIB included information about asbestos, its carcinogenic and other significant health effects, how miners could be exposed, where asbestos occurs naturally on mining property, and what types of commercial products

may contain asbestos. It included recommendations to help mine operators reduce miners' exposures, to prevent or minimize take-home contamination, and for the selection and use of respiratory protection. The PIB also urged mine operators to minimize exposures, to improve controls, and to train miners, listing specific training topics as essential for miners potentially exposed to asbestos.

During this same period, 2000 to 2003, we conducted an asbestos awareness campaign and increased asbestos sampling. Section VII.D of this preamble contains an additional discussion of measures to prevent asbestos "take-home" contamination.

We have decided not to pursue a regulatory approach to minimizing asbestos "take-home" contamination. Based on the existing levels of asbestos exposures in the mining industry, comments on our 2002 ANPRM, and testimony at the subsequent public meetings, we have determined that a non-regulatory approach would be effective in minimizing asbestos take-home contamination from mining operations.

### 4. Training Inspectors to Recognize and Sample for Asbestos

The OIG recommended that we increase MSHA inspectors' skills for providing asbestos compliance assistance to mine operators. In response, we developed a half-day multimedia training program that includes the following:

- A PowerPoint-based training presentation that examines MSHA's procedures for air and bulk asbestos sampling.
- An updated "Chapter 8—Asbestos Fibers" from the *Metal and Nonmetal Health Inspection and Procedures Handbook* that serves as a text for the training sessions.
- A "hands-on" segment that allows the inspectors to examine asbestos and asbestiform rock samples and the equipment used for bulk sampling, and that provides the inspectors instruction and practice in assembling and calibrating asbestos fiber air sampling apparatus.

We gave this asbestos training to journeymen inspectors from March 2002 through April 2003, and added it to the training program for entry-level inspectors.

## IV. Health Effects of Asbestos Exposure

The health hazards from exposure to asbestos were discussed extensively in the preamble to OSHA's 1983 final rule (51 FR 22615). Subsequently, researchers have confirmed and

<sup>15</sup> McDonald *et al.*, 1986; Meeker *et al.*, 2003; Peipins *et al.*, 2003.

<sup>16</sup> Meeker *et al.*, 2003

increased our knowledge of these hazards. Exposures in occupational and environmental settings are generally due to inhalation, although some asbestos may be absorbed through ingestion. While the part of the body most likely affected (target organ) is the lung, adverse health effects may extend to the linings of the chest, abdominal, and pelvic cavities, and the gastrointestinal tract. The damage following chronic exposure to asbestos is cumulative and irreversible. Workplace exposures to asbestos may be chronic, continuing for many years. The symptoms of asbestos-related adverse health effects may not become evident for 20 or more years after first exposure (latency period).

#### A. Summary of Asbestos Health Hazards

This section presents an overview of human health effects from exposure to asbestos. We are proposing to use OSHA's 1986 risk assessment to estimate the risk from asbestos exposures in mining. OSHA's risk assessment has withstood legal scrutiny and the more recent studies discussed later in this preamble support it. MSHA has placed OSHA's risk assessment in the asbestos rulemaking record. It can also be found at <http://www.osha.gov>.

Studies first identified health problems associated with occupational exposure to asbestos in the early 20th century among workers involved in the manufacturing or use of asbestos-containing products.<sup>17</sup> Early studies identified the inhalation of asbestos as the cause of asbestosis, a slowly progressive disease that produces lung scarring and loss of lung elasticity. Studies also found that asbestos caused lung and several other types of cancer. For example, mesotheliomas, rare cancers of the lining of the chest or abdominal cavities, are almost exclusively attributable to asbestos exposure. Once diagnosed, they are rapidly fatal. Asbestos-related diseases have long latency periods, commonly not producing symptoms for 20 to 30 years following initial exposure.

In the late 1960's, scientists correlated phase contrast microscopy fiber counting methods with the earlier types of dust measurements. This procedure provided a means to estimate earlier workers' asbestos exposures and enabled researchers to develop a dose-response relationship with the occurrence of disease. The British Occupational Hygiene Society reported<sup>18</sup> that a worker exposed to 100

fiber-years per cubic centimeter (e.g., 50 years at 2 f/cc, 25 years at 4 f/cc, 10 years at 10 f/cc) would have a 1 percent risk of developing early signs of asbestosis. The correlation of exposure levels with the disease experience of populations of exposed workers provided a basis for setting an occupational exposure limit for asbestos measured by the concentration of the fibers in air.

As mentioned previously, the hazardous effects from exposure to asbestos are now well known. For this reason, our discussion in this section will focus on the results of the more recent studies and literature reviews, those published since the publication of OSHA's risk assessment, and those involving miners. One such review by Tweedale (2002) stated,

Asbestos has become the leading cause of occupational related cancer death, and the second most fatal manufactured carcinogen (after tobacco). In the public's mind, asbestos has been a hazard since the 1960s and 1970s. However, the knowledge that the material was a mortal health hazard dates back at least a century, and its carcinogenic properties have been appreciated for more than 50 years.

Greenberg (2003) also published a recent review of the biological effects of asbestos and provided a historical perspective similar to that of Tweedale.

The three most commonly described adverse health effects associated with asbestos exposure are lung cancer, mesotheliomas, and pulmonary fibrosis (i.e., asbestosis). OSHA, in its 1986 asbestos rule, reviewed each of these diseases and provided details on the studies demonstrating the relationship between asbestos exposure and the clinical evidence of disease. In 2001, the ATSDR published an updated *Toxicological Profile for Asbestos* that also included an extensive discussion of these three diseases. A search of peer-reviewed scientific literature using databases, such as Gateway, PubMed, and ToxLine, accessed through the National Library of Medicine (NLM), yielded nearly 900 new references on asbestos from January 2000 to October 2003. Many of these recent articles<sup>19</sup> continue to demonstrate and support findings of asbestos-induced lung cancer, mesotheliomas, and asbestosis, consistent with the conclusions of OSHA and ATSDR. Thus, in the scientific community, there is compelling evidence of the adverse health effects of asbestos exposure. This has led some researchers and

stakeholders to recommend a worldwide ban of asbestos.<sup>20</sup>

#### B. Factors Affecting the Occurrence and Severity of Disease

The toxicity of asbestos, and the subsequent occurrence of disease, is related to its concentration (C) in the mine air and to the duration (T) of the miner's exposure. Other variables, such as the fiber's characteristics or the effectiveness of the miner's lung clearance mechanisms, also affect disease severity.

##### 1. Concentration (C)

Currently, the concentration (C) of asbestos is expressed as the number of fibers per cubic centimeter (f/cc). Some studies have also reported asbestos concentrations in the number of fibers per milliliter (f/mL), which is an equivalent concentration to f/cc. MSHA's existing PELs for asbestos are expressed in f/mL for metal and nonmetal mines and as f/cc for coal mines. To improve consistency and avoid confusion, we express the concentration of airborne fibers as f/cc in this proposed rule, for both coal and metal and nonmetal mines.

Older scientific literature (i.e., 1960's and 1970's) reported exposure concentrations as million particles per cubic foot (mppcf) and applied a conversion factor to convert mppcf to f/cc. OSHA (51 FR 22617) used a factor of 1.4 when performing these conversions. More recently, Hodgson and Darnton (2000) recommended the use of a factor of 3. In our evaluation of the scientific literature, we did not critically evaluate the impact of these and other conversion factors. We note this difference here for completeness. Because we are relying on OSHA's risk assessment, we are using OSHA's conversion factor

##### 2. Time (T)

Epidemiological and toxicological studies generally report time (T) in years (yr). The product of exposure concentration and exposure duration (i.e.,  $C \times T$ ) is referred to as "fiber-years".<sup>21</sup> When developing exposure-response relationships for asbestos-induced health effects, researchers typically use "fiber-years" to indicate the level of workplace exposure. Finkelstein<sup>22</sup> noted, however, that this product of exposure concentration times duration of exposure ( $C \times T$ ) assumes an equal weighting of each variable (C, T).

<sup>20</sup> Maltoni, 1999.

<sup>21</sup> ATSDR, 2001; Fischer *et al.*, 2002; Liddell, 2001; Pohlabein *et al.*, 2002.

<sup>22</sup> Finkelstein, 1995; ATSDR, p. 42, 2001.

<sup>17</sup> GETF Report, p. 38, 2003; OSHA (40 FR 47654), 1975.

<sup>18</sup> Lane *et al.*, 1968; OSHA (40 FR 47654), 1975.

<sup>19</sup> Baron, 2001; Bolton *et al.*, 2002; Manning *et al.*, 2002; Nicholson, 2001; Osinubi *et al.*, 2000; Roach *et al.*, 2002.

### 3. Fiber Characteristics

Baron (2001) reviewed techniques for the measurement of fibers and stated, “\* \* \* fiber dose, fiber dimension, and fiber durability are the three primary factors in determining fiber [asbestos] toxicity \* \* \*”. Manning *et al.* (2002) also noted the important roles of bio-persistence (*i.e.*, durability), physical properties, and chemical properties in defining the “toxicity, pathogenicity, and carcinogenicity” of asbestos. Roach *et al.* (2002) stated that—

Physical properties, such as length, diameter, length-to-width (aspect ratio), and texture, and chemical properties are believed to be determinants of fiber distribution [in the body] and disease severity.

Many other investigators<sup>23</sup> also have concluded that the dimensions of asbestos fibers are biologically important.

OSHA and MSHA currently specify that analysts count those fibers that are over 5.0 micrometers (µm) in length with a length to diameter aspect ratio of at least 3:1. Several recent publications<sup>24</sup> support this aspect ratio, although larger aspect ratios such as 5:1 or 20:1 have been proposed.<sup>25</sup> There is some evidence that longer, thinner asbestos fibers (*e.g.*, greater than 20 µm long and less than 1 µm in diameter) are more potent carcinogens than shorter fibers. Suzuki and Yuen (2002), however, concluded that “Short, thin asbestos fibers should be included in the list of fiber types contributing to the induction of human malignant mesotheliomas \* \* \*”. More recently, Dodson *et al.* (2003) concluded that all lengths of asbestos fibers induce pathological responses and that researchers should exercise caution when excluding a population of inhaled fibers based on their length.

We have determined that researchers have found neither a reliable method for predicting the contribution of fiber length to the development of disease, nor evidence establishing the exact relationship between them. There is suggestive evidence that the dimensions of asbestos fibers may vary with different diseases. A continuum may exist in which shorter, wider fibers produce one disease, such as asbestosis, and longer, thinner fibers produce another, such as mesotheliomas.<sup>26</sup> The scientific community continues to publish new data that will enable regulatory agencies, such as MSHA, to

<sup>23</sup> ATSDR, 2001; Osinubi *et al.*, 2000; Peacock *et al.*, 2000; Langer *et al.*, 1979.

<sup>24</sup> ATSDR, 2001; Osinubi *et al.*, 2000.

<sup>25</sup> Wylie *et al.*, 1985.

<sup>26</sup> ATSDR, pp. 39–41, 2001; Mossman, pp. 47–50, 2003.

better understand the relationship between fiber dimensions, durability, inhaled dose, and other important factors that determine the health risks of exposure not only to asbestos, but also to other fibers.

### 4. Differences in Fiber Potency

The theory that the differences among fibers have an effect on their ability to produce adverse effects on human health has received a great deal of attention. Hodgson and Darnton (2000), Browne (2001), and Liddell (2001) discuss a fiber gradient hypothesis, which is now termed the amphibole hypothesis. This hypothesis proposes that the amphiboles (*e.g.*, crocidolite, amosite) are more hazardous than the serpentine, chrysotile. ATSDR (p. 39, 2001) recently stated that—

Available evidence indicates that all asbestos fiber types are fibrogenic, although there may be some differences in relative potency among fiber types.

In its 1986 asbestos rule, OSHA (51 FR 22628) stated that—

\* \* \* epidemiological and animal evidence, taken together, fail to establish a definitive risk differential for the various types of asbestos fiber. Accordingly, OSHA has \* \* \* recognized that all types of asbestos fiber have the same fibrogenic and carcinogenic potential \* \* \*

In its comments on MSHA’s asbestos ANPRM, NIOSH stated that—

(3) experimental animal carcinogenicity studies with various minerals have provided strong evidence that the carcinogenic potential depends on the “particle” length and diameter. The consistency in tumorigenic responses observed for various mineral particles of the same size provides reasonable evidence that neither composition nor origin of the particle is a critical factor in carcinogenic potential; \* \* \*

This issue remains unresolved. Although possible differences in fiber potency are beyond the scope of this proposed rule, we will continue to monitor results of research in this area.

### 5. Lung Clearance Mechanisms

Inhaled asbestos may deposit throughout the respiratory tract, depending on the aerodynamic behavior of the fibers.<sup>27</sup> As noted by Baron (2001), “\* \* \* fiber aerodynamic behavior indicates that small diameter fibers are likely to reach into and deposit in the airways of the lungs.” Clearing the lungs of deposited asbestos occurs by several mechanisms. In the mid-airways (*i.e.*, bronchial region), small hair-like cells sweep the mucus containing asbestos toward the throat, at which time it is swallowed or

expectorated. The swallowing of mucus through this clearance mechanism can result in inhaled asbestos reaching the gastrointestinal tract.

In the air sacs deep within the lungs (the alveolar region), pulmonary macrophages engulf foreign matter, including asbestos fibers. The macrophages attempt to remove these fibers by transporting them to the circulatory or lymphatic system. Some studies have shown that groups of macrophages try to engulf longer fibers.<sup>28</sup> When asbestos fibers are not cleared, they may initiate inflammation of the cells lining the alveoli. This inflammation leads to more serious physical effects in the lungs. OSHA (1986), ATSDR (2001), and several recent papers<sup>29</sup> discuss these mechanisms for the pulmonary clearance of asbestos.

## C. Specific Human Health Effects

### 1. Lung Cancer

Lung cancer is a chronic, irreversible, and often fatal disease of the lungs. Epidemiological studies confirm, and toxicological studies support, the carcinogenicity of asbestos. (See section IV.D. below.) The form of lung cancer seen most often in asbestos-exposed individuals is bronchial carcinoma. Some of the risk factors for lung cancer include airborne asbestos concentration, duration of exposure, fiber dimensions, the age of the individual at the time of first exposure, and the number of years since the first exposure.<sup>30</sup> Another major risk factor is the smoking of tobacco products. Numerous studies have concluded that there are synergistic effects between asbestos and tobacco smoke in the development of lung cancer.<sup>31</sup> This is especially relevant to miners as NIOSH (May 2003) estimates that 33 percent of miners currently smoke.

The mechanism through which asbestos causes lung cancer is under study. Recent papers by Manning *et al.* (2002), Xu *et al.* (2002), and Osinubi *et al.* (2000) describe a scheme of cell signaling and inflammation with the release of reactive oxygen species and reactive nitrogen species.

The latency period for asbestos-related lung cancer is generally 20–30 years, although some cases have been reported within 10 years, and some up to 50 years, after initial asbestos exposure.<sup>32</sup> Lung cancer caused by

<sup>28</sup> Warheit, p. 308, 1993.

<sup>29</sup> Baron, 2001; Osinubi *et al.*, 2000.

<sup>30</sup> Yano *et al.*, 2001; ATSDR, 2001.

<sup>31</sup> Bolton *et al.*, 2002; Manning *et al.*, 2002; OSHA, 1986.

<sup>32</sup> Roach *et al.*, 2002.

<sup>27</sup> ICRP, 1966.

asbestos can progress even in the absence of continued exposure. Thus, in all of its stages, lung cancer constitutes a material impairment of human health or functional capacity.

In the preamble to its 1986 asbestos standard (51 FR 22615), OSHA stated, "Of all the diseases caused by asbestos, lung cancer constitutes the greatest health risk for American asbestos workers." OSHA (51 FR 22615–22616) also stated, "\* \* \* Asbestos exposure acts synergistically with cigarette smoking to multiply the risk of developing lung cancer." MSHA believes that the essential points of this statement remain true today.

Steenland *et al.* (2003) estimated that there were about 150,000 lung cancer deaths in 1997 in the United States, and that 6.3 to 13 percent (*i.e.*, 9,700 to 19,900) of these lung cancer deaths were occupationally-related. Steenland *et al.* (1996) also had estimated that, in the mid-1990's, there were about 5,400 asbestos-related lung cancer deaths per year. NIOSH (May 2003) identified over 10,000 lung cancer deaths in the United States during 1999 based on only 20 Census Industry Codes (CIC). This sum was computed from "selected states," not the entire United States. NIOSH (May 2003) also identified 300 lung cancer deaths among coal miners from 15 selected states.

## 2. Mesotheliomas

Mesotheliomas are malignant tumors that are rapidly fatal. They involve thin membranes that line the chest (the pleura) and that surround internal organs (the peritoneum) following asbestos exposure.<sup>33</sup> Mesotheliomas begin with a localized mass and, like other malignant tumors, they can spread (metastasize) to other parts of the body.<sup>34</sup> It does not appear that smoking is a major risk factor in the development of mesotheliomas.<sup>35</sup>

As in cases of lung cancer and asbestosis, mesotheliomas also have a latency period, varying from 15 to over 40 years.<sup>36</sup> Orenstein *et al.* (2000) reported an even wider range for the latency, from a minimum of 5 years to a maximum of 72 years. In cases involving the pleura, patients often complain of chest pain, breathing difficulties on exertion, weakness, and fatigue. Other early symptoms of this disease may also include weight loss and cough. As the disease progresses, there is increased restriction of the chest wall and highly abnormal respiration,

often characterized by a rapid and shallow breathing pattern. Mesotheliomas are rapidly progressive even in the absence of continued asbestos exposure. Mesotheliomas have a poor prognosis in most patients; death typically occurs within a year or so of diagnosis.<sup>37</sup> Thus, like lung cancer, mesotheliomas materially impair human health and functional capacity.

As noted by ATSDR (2001), OSHA (1986), and many others,<sup>38</sup> mesotheliomas are extremely rare tumors, particularly in non-asbestos exposed individuals. OSHA (1986) has stated, "\* \* \* In some asbestos-exposed occupational groups, 10 percent to 18 percent of deaths have been attributable to malignant mesotheliomas \* \* \*". NIOSH (May 2003) reported that there were about 2,500 deaths due to malignant mesotheliomas in the United States in 1999. Steenland *et al.* (2003) estimated that there were about 2,100 deaths in the United States from mesotheliomas in 1997, and that, in males, 85–90 percent of these deaths from mesotheliomas were due to occupational asbestos exposure. These tumors were generally the underlying (primary) cause of death, and not just a contributing cause of death. NIOSH found that most mesothelioma deaths were included with the categories of "all other industries" (56 percent) or "all other occupations" (57 percent). For those death certificates that included a Census Industry Code (CIC), the most frequently recorded was "construction." The 2003 NIOSH publication, *Work-Related Lung Disease Surveillance Report 2002* (WoRLD), did not provide specific data on mesotheliomas among miners.

One commenter expressed concern that the use of perchlorate in explosives might be a co-factor for increasing the incidence or shortening the latency period for mesothelioma among miners. In investigating this comment, we found that perchlorate can be a component in explosives<sup>39</sup> and that perchlorate may cause or contribute to thyroid disease.<sup>40</sup> We found no studies linking perchlorate to mesotheliomas. The California State Department of Toxic Substances Control states that perchlorate "\* \* \* has not been linked to cancer in humans \* \* \*".<sup>41</sup>

<sup>37</sup> Bolton *et al.*, 2002; Roach *et al.*, 2002; Osinubi *et al.*, 2000; West, 2003.

<sup>38</sup> Bolton *et al.*, 2002; Britton, 2002; Carbone *et al.*, 2002; Manning *et al.*, 2002; Orenstein *et al.*, 2000; Roach *et al.*, 2002; Suzuki and Yuen, 2002.

<sup>39</sup> EPA, 2002.

<sup>40</sup> ATSDR, 1998.

<sup>41</sup> <http://www.dtsc.ca.gov/ToxicQuestions/glossary.html>.

## 3. Asbestosis

Asbestosis is a chronic and irreversible disease caused by the deposition and accumulation of asbestos in the lungs. It can lead to substantial injury and may cause death from the build up of bands of scar tissue and a loss of lung elasticity (*i.e.*, pulmonary fibrosis).<sup>42</sup> It is not a tumor. Following exposure to asbestos, chronic inflammation may occur that leads to the multiplication of collagen-producing cells in the lung and the accumulation of thick collagen bundles in essential lung tissues.<sup>43</sup> These structural changes result in a hardening or stiffening of the lungs. Physicians who specialize in diseases of the lung also classify asbestosis as a restrictive lung disease due to this loss of elasticity.

In asbestosis, the lungs are unable to properly expand and contract during the breathing cycle and, thus, lung volumes, airflows, and respiratory frequencies are likely to be abnormal.<sup>44</sup> Two common symptoms of this disease are cough and breathing difficulties. Patients with asbestosis may also complain of a general feeling of discomfort, weakness, and fatigue. Breathing difficulties, weakness, and fatigue are often more severe with work or exercise. As the disease progresses, patients begin to experience symptoms even while resting and are likely to become permanently disabled.<sup>45</sup> Patients with severe asbestosis also may experience heart or circulation problems, such as heart enlargement. Like lung cancer and mesotheliomas, asbestosis may be progressive even in the absence of continued asbestos exposure. Thus, asbestosis, even in its earliest stages, constitutes a material impairment of human health and functional capacity.

NIOSH (May 2003) reported that there were about 1,200 asbestosis-related deaths in the United States in 1999. Of these, asbestosis was the underlying cause in about a third of these deaths (400) and a contributing cause in the others (800). Steenland *et al.* (2003) estimated that there were about 400 deaths from asbestosis in 1997, and that 100 percent of these asbestosis-deaths were due to occupational exposure. As shown by NIOSH (May 2003), the number of deaths related to asbestosis increased over ten-fold between 1968 and 1999. NIOSH also reported that these figures likely reflect improved diagnostic tools and the long latency period for evidence of disease that follows asbestos exposure.

<sup>42</sup> ATSDR, 2001.

<sup>43</sup> Osinubi *et al.*, 2000.

<sup>44</sup> West, 2000; West, 2003.

<sup>45</sup> OSHA, 1986.

<sup>33</sup> ATSDR, 2001.

<sup>34</sup> Roach *et al.*, 2002.

<sup>35</sup> Bolton *et al.*, 2002.

<sup>36</sup> Suzuki and Yuen, 2002.

The death certificates for most individuals who died from asbestosis lacked the Census Industry Code (CIC) and the Census Occupation Code (COC). Most asbestosis deaths were classified under "all other industries" (45 percent) and "all other occupations" (57 percent). For those death certificates that included a CIC and a COC, the most frequently recorded industry and occupation were "construction" (CIC = 060) and "plumbers, pipefitters, and steamfitters" (COC = 585), respectively. There were no specific data on asbestosis-related deaths among miners in the NIOSH WoRLD publication (May 2003).

#### 4. Other Cancers

OSHA, in its 1986 rule, reviewed epidemiologic studies of asbestos workers with cancer of the colon, rectum, kidney, larynx (voice box), throat, or stomach. Of these studies, researchers placed the greatest emphasis on those involving gastrointestinal cancers. OSHA concluded, " \* \* \* the risk of incurring cancers at these [other] sites is not as great as the increased risk of lung cancer \* \* \*". Thus, OSHA included lung and gastrointestinal cancers, and not these other cancer sites, in its 1986 risk assessment. MSHA believes that the statement remains true today, based on studies cited by ATSDR (2001) and by recent papers on kidney cancer,<sup>46</sup> laryngeal cancer,<sup>47</sup> lymphomas,<sup>48</sup> and pancreatic cancer.<sup>49</sup> We have not attempted to quantify the risks of these other cancers, which are small in comparison to lung cancer and mesotheliomas.

#### 5. Reversible Airways Obstruction (RAO)

Under normal physiological conditions, oxygen and other inhaled chemical substances pass through a branching network of airways that become narrower, shorter, and more numerous as they penetrate deeper into the lung.<sup>50</sup> The diameter of each airway has an important effect on its airflow. A reduction in airway diameter occurs temporarily on exposure to some chemical substances and permanently in some diseases. These reductions lead to temporary or permanent airflow limitations. A temporary reduction of airway diameter and the resulting difficulties in breathing have also been called broncho-constriction, acute airways constriction or obstruction, or

reversible airways obstruction (RAO). Such constriction or obstruction typically involves airways in the mid to lower respiratory tract.

Several recent studies have examined respiratory health and respiratory symptoms of asbestos-exposed workers.<sup>51</sup> Wang *et al.* (2001) reported permanent changes in airway diameters and, thus, permanent airflow limitations in diseases such as asbestosis or chronic obstructive pulmonary disease (COPD). Although patients can recover from RAO, they do not recover from asbestosis or COPD, which are typically progressive, leading to increasingly severe illness and premature death.

Delpierre *et al.* (2002) reported that RAO in asbestos workers was independent of x-ray signs of pulmonary or pleural fibrosis, as well as a worker's smoking status. The long-term implications of RAO are unknown at this time. Delpierre *et al.*, however, encouraged physicians to screen asbestos workers for RAO. Lung function tests may be useful in the early diagnosis of asbestos-disease, especially if RAO precedes the development of irreversible pulmonary disease, such as asbestosis.

#### 6. Other Nonmalignant Pleural Disease and Pleural Plaques

The pleura is the membrane lining the chest cavity. Pleural plaques are discrete, elevated areas of nearly transparent fibrous tissue (scar tissue) and are composed of thick collagen bundles. Pleural thickening and pleural plaques are biologic markers reflecting previous asbestos exposure.<sup>52</sup> They appear opaque on radiographic images and white to yellow in microscopic sections.<sup>53</sup> The American Thoracic Society (ATS, 2004) has described the criteria for diagnosis of non-malignant asbestos-related pleural disease and pleural plaques.

Pleural plaques are the most common manifestation of asbestos exposure.<sup>54</sup> Only rarely do they occur in persons who have no history or evidence of asbestos exposure. Pleural thickening and pleural plaques may occur in individuals exposed to asbestos in both occupational settings, such as miners, and non-occupational settings, such as family members. For example, the prevalence of pleural plaques ranges from 0.53 percent to 8 percent in environmentally exposed populations,

such as the residents of Libby, Montana; 3 percent to 14 percent in dockyard workers; and up to 58 percent among insulation workers.

Pleural plaques may develop within 10–20 years after an initial asbestos exposure<sup>55</sup> and slowly progress in size and amount of calcification, independent of any further exposure. There is no evidence that pleural plaques undergo malignant degeneration into mesothelioma.<sup>56</sup> Pleural thickening and pleural plaques, however, may impair lung function and may precede chronic lung disease that develops in some individuals.<sup>57</sup> Rudd (1996), for example, reported that the incidence of lung cancer in patients with pleural plaques is higher than that of other patients. These plaques are also part of the clinical picture of asbestosis.

#### 7. Asbestos Bodies

Some asbestos-exposed individuals may expel asbestos fibers from the lungs with a coating of iron and protein. These collections of coated fibers, found in sputum or broncho-alveolar lavage (BAL) fluid, are called asbestos bodies or ferruginous bodies.<sup>58</sup> Like pleural thickening and pleural plaques, these bodies indicate prior asbestos exposure.

#### D. Support From Toxicological Studies of Human Health Effects of Asbestos Exposure

Many studies are available that clearly demonstrate the toxicity of asbestos (*e.g.*, carcinogenicity, genotoxicity, pneumotoxicity) and confirm observed human responses.<sup>59</sup> Studies conducted in baboons, mice, monkeys, and rats have all demonstrated that asbestos fibers are carcinogenic.<sup>60</sup> OSHA's risk assessment, however, did not rely on data from *in vivo* or *in vitro* toxicological studies to determine the human health effects from exposure to asbestos. In the preamble to its 1986 asbestos rule (51 FR 22632), OSHA stated—

OSHA chose *not* [emphasis added] to use animal studies to predict quantitative estimates of risk from asbestos exposure because of the many high quality human studies available that were conducted in actual workplace situations \* \* \* OSHA has supplemented the human data with results from the animal studies when evaluating the

<sup>55</sup> Bolton *et al.*, 2002; OSHA, 1986.

<sup>56</sup> Peacock *et al.*, 2000; West, 2003.

<sup>57</sup> Schwartz *et al.*, 1994.

<sup>58</sup> ATSDR, 2001; Peacock *et al.*, 2000.

<sup>59</sup> OSHA, 1986; ATSDR, 2001.

<sup>60</sup> Davis *et al.*, 1986; Davis and Jones, 1988; Davis *et al.*, (in IARC) 1980; Davis *et al.*, 1980; Donaldson *et al.*, 1988; Goldstein and Coetzee, 1990; McGavran *et al.*, 1989; Reeves, *et al.*, 1974; Wagner *et al.*, 1974, 1980; Webster *et al.*, 1993.

<sup>46</sup> McLaughlin and Lipworth, 2000; Sali and Boffetta, 2000.

<sup>47</sup> Browne and Gee, 2000.

<sup>48</sup> Becker *et al.*, 2001.

<sup>49</sup> Ojajarvi *et al.*, 2000.

<sup>50</sup> West, 2000.

<sup>51</sup> Delpierre *et al.*, 2002; Eagen *et al.*, 2002; Selden *et al.*, 2001.

<sup>52</sup> ATSDR, 2001; Manning *et al.*, 2002.

<sup>53</sup> Bolton *et al.*, 2002; Manning *et al.*, 2002; Roach *et al.*, 2002; Peacock *et al.*, 2000; ATSDR, 2001.

<sup>54</sup> Cotran *et al.*, p. 732–734, 1999; Peacock *et al.*, 2000.

health information and determining the significance of risk.

Because we are relying on OSHA's 1986 asbestos risk assessment for this proposed rule, we do not use the toxicological studies for a quantitative assessment of risk, but as supportive of the causative relationship between asbestos exposure and observed human health effects.

Toxicological studies are providing important information on possible mechanism(s) through which asbestos causes disease. The ATSDR *Toxicological Profile for Asbestos* (updated 2001) contains a more detailed discussion on this topic and describes several mechanisms of action for asbestos. These include—

- Its direct interaction with cellular macromolecules,
- Its recruitment of pulmonary macrophages that produce reactive oxygen and nitrogen species, and
- Its initiation of other cellular responses (e.g., inflammation).

## V. Characterization and Assessment of Exposures in Mining

Asbestos minerals are widespread in the environment.<sup>61</sup> The use of asbestos-contaminated crushed rocks in roads, asbestos in insulation and other building materials, and the release of asbestos from brakes on vehicles contributes to its presence in the environment. Occupational asbestos exposures can be much higher than the asbestos levels the public typically encounters.

Miners may be exposed to asbestos in nature, as well as in commercial products. Mining, milling, maintenance, or other activities at the mine may result in the release or re-suspension of asbestos into the air.<sup>62</sup> In some geologic formations, asbestos may be in isolated pockets or distributed throughout the ore. Mining operations, such as blasting, cutting, crushing, grinding, or simply disturbing the ore or surrounding earth may cause the asbestos to become airborne. Milling operations may transform bulk ore containing asbestiform minerals into respirable fibers. Similarly, other activities conducted at mine sites, such as removing asbestos-containing materials during renovation or demolition of buildings and equipment repair work,<sup>63</sup> may contribute to a miner's asbestos exposure.

### A. Determining Asbestos Exposures in Mining

To evaluate asbestos exposures in mines, MSHA collects personal exposure air samples using a personal sampling pump and a filter-cassette assembly, composed of a 50-mm electrically conductive extension cowl and a 25-mm diameter mixed cellulose ester (MCE) filter. Following standard sampling procedures, we also submit blank filters for analysis. Analysts use the blanks to correct the sampling results for background fiber counts due to variations in the manufacturing and analysis of the filter.

Since 2001, we have used contract laboratories to analyze our asbestos samples by PCM. The contract laboratories report analytical results as the fiber concentration (f/cc) for each filter analyzed. Then, to evaluate a miner's full-shift exposure, MSHA calculates an 8-hour time-weighted average concentration from a consecutive series of individual filters.

Several factors complicate the evaluation of personal exposure levels in mining. Non-asbestos particles collected on the filter can hide the asbestos fibers (overloading) and, as discussed earlier (see section II.C.2), mining samples may also contain intermediate fibers that are difficult to classify. (See section II.B in this preamble.)

### B. Exposures From Naturally Occurring Asbestos

Mining and milling of asbestos-contaminated ore can release fibers into the ambient air. Beginning in January 2000, we initiated a focused effort to determine the extent of asbestos exposure among miners. We chose 124 metal and nonmetal mines for sampling based on the following:

- Geological information linking a higher probability for asbestos contamination with certain types of ores or commodities.
- Historical records identifying locations of potential problem mines.
- Complaints from miners reporting asbestos on mine property.

Asbestos tends to accumulate during the milling process, which is often in enclosed buildings. The use of equipment and machinery or other activities in these locations may re-suspend the asbestos-containing dust from workplace surfaces into the air. For this reason, we generally find higher airborne concentrations in mills than among mobile equipment operators or in ambient environments, such as pits. The following example supports this finding.

### 1. Asbestos-Contaminated Ore Case Study: Wollastonite

Wollastonite is a monocalcium silicate found in the United States, Mexico, and Finland. It occurs as prismatic crystals that can split into massive-to-acicular (needle-like) fragments when processed, and is used mainly in ceramics.<sup>64</sup>

A consumer recently sent a sample of the final bulk product from a wollastonite mine to a commercial laboratory for analysis. When the analysis indicated the presence of asbestos contamination, the consumer informed the mine operator. The mine operator contacted MSHA and informed us of this finding after their contract laboratory confirmed the presence of tremolite in product samples. MSHA then conducted industrial hygiene sampling in the mill and the pit to verify and track the source of the tremolite. We found that concentrations in the mill exceeded 2.0 f/cc as measured by PCM. Although asbestos averaged only about 1.3 percent of the total fibers, over half of the exposures in the mill exceeded 0.1 f/cc of asbestos (the OSHA 8-hour, TWA PEL). Miners' exposures in the pit were much lower and further analyses indicated that few of these samples contained asbestos.

The mine instituted an aggressive cleanup and control policy in the interest of the company and their miners' health. This wollastonite facility provides and launders uniforms for the millers, provides physical examinations to miners and their families, and uses other administrative controls to limit take-home contamination. In addition to conducting personal asbestos sampling, MSHA assisted mine management through the following compliance assistance activities:

- Assistance in developing cleanup and monitoring procedures.
- Discussion of hazards of asbestos exposure with miners and the operator.
- Identification of accredited laboratories familiar with mining samples to perform asbestos analyses.
- Assistance in implementation of a respiratory protection program.
- Instruction in recognition and avoidance of asbestos. MSHA and the mine operator worked together in recognizing the problem, evaluating the hazard, and determining ways to control exposures. This case study demonstrates successful cooperation to protect the health of miners.

<sup>61</sup> ATSDR, 2001.

<sup>62</sup> MSHA (Bank), 1980; Amandus *et al.*, Part I, 1987.

<sup>63</sup> EPA, 1986, 1993, April 2003.

<sup>64</sup> Warheit, p. 18, 1993.

## 2. Methods of Reducing or Avoiding Miners' Exposures to Naturally Occurring Asbestos

Some mine operators mining other commodities that are likely to contain asbestos, such as vermiculite, have stated that they are making an effort to avoid deposits and seams likely to contain substantial quantities of asbestos. They use knowledge of the geology of the area, visual inspections of the working face, and sample analysis to avoid encountering asbestos deposits, thus preventing asbestos contamination of their product.<sup>65</sup> In addition, some mine operators have voluntarily adopted the OSHA 8-hour, TWA PEL (0.1 f/cc), thus reducing the potential for asbestos-related illness among miners.

### C. Exposures From Introduced (Commercial) Asbestos

Asbestos is an important component in some commercial products and may be found as a contaminant in others. Due to improved technology and increased awareness, however, substitutes for asbestos in products are available for almost all uses, and manufacturers have removed the asbestos from many new products.<sup>66</sup> Nevertheless, there are mines, including coal mines, that have introduced commercial asbestos-containing products on their property. Some of these introduced products may include asbestos-containing building materials, such as Transite® board, used during construction, rehabilitation, or demolition projects. Other examples of introduced commercial products that may contain asbestos are brake linings for mining equipment, insulation, joint and packing compounds, and asbestos welding blankets.

Occasionally, miners report incidents of possible asbestos release through MSHA's Hazard Complaint Program. Inspectors also report mines with noticeably deteriorated asbestos-containing building materials (ACBM). We investigate these reported situations and take appropriate action. The following example describes an incident in which miners unsafely removed asbestos at a mining operation.

#### 1. Introduced Asbestos Case Study: Potash

In June 2003, eight miners removed siding on three transfer conveyors originally installed in 1962 at a potash mine in Utah. The siding was weathered and deteriorated to the point of being friable (crumbling). The type of siding was a commercial product named

Galbestos®, which contains 7 percent chrysotile asbestos, as indicated on the Material Safety Data Sheet (MSDS). Analysis of bulk samples of the debris left behind by the removal of the siding confirmed that it contained chrysotile asbestos. When the miners removed it without using special precautions, they released asbestos into the air. It is possible that these miners contaminated themselves with asbestos and carried it to their families and communities (*i.e.*, take-home contamination).

MSHA became aware of this asbestos-removal work when one of the miners made a hazard complaint to the MSHA District Office. We conducted an investigation and determined that the company officials had known of the potential asbestos hazard for at least 2 years. We found no asbestos in the personal air samples collected after the siding had been removed. Although we did not issue citations for overexposure to asbestos, we issued citations to the company for failure to implement special work procedures, failure to issue appropriate personal protective equipment, and failure to train the affected miners for the task. The mine operator took corrective action and we terminated these citations.

## 2. Methods of Reducing or Avoiding Miners' Exposures to Introduced (Commercial) Asbestos

Existing Federal and state standards already address the removal of asbestos-containing building materials (ACBM). If the asbestos-containing material is intact, it is preferable to leave it where it is. If the asbestos-containing material is worn or deteriorating, these standards require the use of special precautions (*e.g.*, personal protective equipment, training, decontamination) to prevent or minimize exposure of workers and the public and contamination of the environment. We train our inspectors to encourage mine operators to have worn or deteriorating asbestos-containing products removed by persons specially trained to remove the asbestos-containing material safely.

### D. Sampling Data and Exposure Calculations

After the national publicity surrounding asbestos-related diseases and death among the population of Libby, Montana, MSHA closely reviewed and updated its asbestos-related health procedures and policies for metal and nonmetal mines. We then made sure these procedures and policies were applied consistently across the country. For example, we switched from a 37-mm to a 25-mm filter cassette and recommended appropriate flow rates

and sampling times. We also allocated additional resources to asbestos sampling and analysis to verify and evaluate the extent of asbestos exposures in mining.

#### 1. Explanation of Sampling Data and Related Calculations

The time-weighted average (TWA) concentration (f/cc) for individual filters ( $n = 1, 2, \dots$ ) is calculated by dividing the number of fibers (f) collected on the filter by the volume of air (cc) drawn through the filter.  $TWA_{sum}$  is the total time-weighted average concentration for all filters in the series over the total sampling time. The exposure limits in MSHA standards are based on an 8-hour workday, regardless of the actual length of the shift. MSHA measures the miner's exposure for the entire time the miner works. We then calculate a full-shift airborne exposure concentration as if the fibers had been collected over an 8-hour shift. This allows us to compare the miner's exposure to the 8-hour TWA, full-shift exposure limit. MSHA calls this calculated 8-hour TWA a "shift-weighted average (SWA)."

We calculate the  $TWA_{sum}$  and SWA exposure levels for each miner sampled according to the following formulas, respectively.

$$TWA_{sum} = (TWA_1t_1 + TWA_2t_2 + \dots + TWA_nt_n) / (t_1 + t_2 + \dots + t_n)$$

$$SWA = (TWA_1t_1 + TWA_2t_2 + \dots + TWA_nt_n) / 480 \text{ minutes}$$

Where:

$TWA_n$  is the time-weighted average concentration for filter "n".

$t_n$  is the duration sampled in minutes for filter "n".

$TWA_nt_n$  is the time-weighted average concentration for filter "n" multiplied by the duration sampled for filter "n".

$(t_1 + t_2 + \dots + t_n)$  is the total time sampled in minutes.

MSHA defines a "sample" as the average 8-hour full-shift airborne concentration that represents an individual miner's full-shift exposure.

The following information from our database illustrates the sampling results from these calculations. For one mechanic at the potash mine in our previous example, MSHA used a series of three filter-cassettes to determine the miner's full-shift exposure. We sampled a total of 577 minutes. The highest TWA concentration for one filter-cassette in this series was 4.100 f/cc as analyzed by PCM. MSHA calculated the mechanic's full-shift exposure to report the fiber concentration as if the mechanic had received the full exposure in 8 hours

<sup>65</sup> GETF Report, pp. 17–18, 2003.

<sup>66</sup> GETF Report, pp. 12 and 15, 2003.

(480 minutes). The mechanic's shift-weighted average (SWA) was 1.982 f/cc.

TABLE V-1.—EXAMPLE OF PERSONAL SAMPLING RESULTS

Mechanic sampled 6/17/2003 at 1.7 Lpm	Sampling time (minutes)	PCM TWA fiber concentration (f/cc)
Filter-cassette 1 ...	230	4.100
Filter-cassette 2 ...	252	0.016
Filter-cassette 3 ...	95	0.045
TWA <sub>sum</sub> result .....	577	1.649
Sample (SWA) result .....	480	1.982

## 2. Summary of MSHA's Asbestos Sampling and Analysis Results

To assess exposures and present our asbestos sampling results to the public, we compiled our asbestos sampling data for the period January 1, 2000 through December 31, 2003. We formatted these data into four Excel® workbooks, one for each year, and placed them, together with additional explanatory information, on our *Asbestos Single Source Page* at <http://www.msha.gov/asbestos/asbestos.htm>.

We calculated an 8-hour full-shift exposure for each miner sampled from the TWA of individual filters, typically three filters per shift. These data include the results of 703 full-shift personal exposure samples, comprised of 2,184 filter-cassettes, and cover 163 industrial hygiene sampling visits at 125 mines (124 metal and nonmetal mines and one coal mine), including some mines and mills that are now closed. Because the last remaining asbestos mine in the

United States (Joe 5 Pit in California) closed in December 2002 and its associated mill (King City) closed in June 2003, we excluded those data in our analysis.

Of the remaining 123 mines that MSHA sampled during this 4-year period, 18 mines could be potentially impacted by the lowering of the full-shift permissible exposure limit to 0.1 f/cc as measured by PCM. These 18 mines have had at least one miner exposed to airborne fiber concentrations exceeding 0.1 f/cc during this period. Two of the 18 mines (iron ore and wollastonite) had personal asbestos exposures confirmed by TEM exceeding 0.1 f/cc. Excluding the 42 samples from the asbestos mine and mill, 8 percent of the remaining 661 personal samples had 8-hour TWA, full-shift fiber concentrations greater than the proposed 0.1 f/cc PEL, as measured by PCM. Table V-2 below summarizes these sampling results.

TABLE V-2.—PERSONAL EXPOSURE SAMPLES, ANALYZED BY PCM, AT CURRENTLY ACTIVE MINES<sup>1</sup> BY COMMODITY (1/2000–12/2003)

Commodity	Number of mines sampled	Number (%) of mines >0.1 f/cc SWA	Number of samples	Number (%) of samples >0.1 f/cc SWA <sup>2</sup>
Rock & quarry products <sup>3</sup> .....	61	4 (7%)	215	7 (3%)
Vermiculite .....	4	3 (75%)	127	5 (4%)
Wollastonite .....	1	1 (100%)	18	18 (100%)
Iron (taconite) .....	14	5 (36%)	178	17 (10%)
Talc .....	12	1 (8%)	38	2 (5%)
Boron .....	2	1 (50%)	9	4 (44%)
Other <sup>4</sup> .....	29	<sup>5</sup> 3 (10%)	76	3 (4%)
Total .....	123	<sup>6</sup> 18 (15%)	661	56 (8%)

<sup>1</sup> Excludes data from a closed asbestos mine and mill.

<sup>2</sup> MSHA uses TEM to confirm the presence of asbestos on samples showing exposures exceeding 0.1 f/cc.

<sup>3</sup> Including stone, sand and gravel mines.

<sup>4</sup> Coal, potash, gypsum, salt, cement, clay, lime, mica, metal ore NOS, olivine, shale, pumice, trona, perlite, and gold.

<sup>5</sup> Coal, potash, and gypsum (Coal and potash personal exposures are due to commercially introduced fiber release episodes, *i.e.*, not from a mineral found at the mine).

<sup>6</sup> TEM confirmed asbestos exposures exceeding 0.1 f/cc in two of the 18 mines.

MSHA is proposing to lower its 8-hour TWA, full-shift PEL from 2.0 f/cc to 0.1 f/cc to provide increased protection for miners. As noted in OSHA's risk assessment for its 1986 asbestos rule, there is significant risk of material impairment of health or functional capacity even at this lower PEL. MSHA compliance data indicate that some miners' asbestos exposures have exceeded 0.1 f/cc. Available data from death certificates in 24 states confirm that there is asbestos-related mortality among miners.<sup>67</sup>

## VI. The Application of OSHA's Risk Assessment to Mining

We are applying OSHA's risk assessment to our exposure sampling data on miners to estimate the risk from asbestos exposure in mining. In response to the ANPRM, the National Mining Association (NMA) expressed their belief that health risk is related to fiber type and that OSHA's risk assessment is no longer adequate or appropriate for us to use for the mining industry. In developing this proposed rule, we evaluated studies published over the last 20 years since OSHA completed its risk assessment, and studies that specifically focused on asbestos exposures of miners. We have

found that these additional studies confirm OSHA's conclusions.

Section VIII of this preamble contains a summary of our findings from applying OSHA's quantitative assessment of risk to the mining industry. The *Preliminary Regulatory Economic Analysis (PREA)* contains a more in-depth discussion of our methodology and conclusions. We placed our PREA in the rulemaking docket and posted it on our *Asbestos Single Source Page* at <http://www.msha.gov/asbestos/asbestos.htm>. We also placed OSHA's risk assessment in the rulemaking docket.

<sup>67</sup> NIOSH World, p. E-1, 2003.

*A. Summary of Studies Used by OSHA in Its Risk Assessment*

OSHA relied on eight non-mining and milling studies to estimate the risk of lung cancer due to asbestos exposure. They used four studies to estimate the risk of mesotheliomas, and two studies,

involving three occupational cohorts, for asbestosis. We briefly review these studies below, since they also serve as the basis of our risk assessment. For completeness, we are including Table VI-1 of some mining and milling studies that have been conducted.

EPA, in its Integrated Risk Information System (IRIS), presents a useful table summarizing data from lung cancer and mesothelioma studies. We extracted that portion of their table dealing with the studies included in OSHA's risk assessment. This is the basis for Table VI-1 below.

TABLE VI-1.—SUMMARY OF LUNG CANCER AND MESOTHELIOMA STUDIES

Human data occupational group	Fiber type	Reported average exposure (f-yr/mL)	Percent (%) increase in cancer per f-yr/mL	Reference
<b>Lung Cancer</b>				
Friction Products .....	Chrysotile .....	32	0.058	Berry and Newhouse, 1983.
Textile Products .....	Mostly Chrysotile .....	44	2.8	Dement <i>et al.</i> , 1982.
Cement Products .....	Mixed (Amosite, Chrysotile, Crocidolite).	112	6.7	Finkelstein, 1983.
Asbestos Products .....	Mixed (Amosite, Chrysotile, Crocidolite).	374	0.49	Henderson and Enterline, 1979.
Textile Products .....	Chrysotile .....	200	1.1	Peto, 1980.
Insulation Products .....	Amosite .....	67	4.3	Seidman <i>et al.</i> , 1979; Seidman, 1984.
Insulation Workers .....	Mixed (Amosite, Chrysotile, Crocidolite).	300	0.75	Selikoff <i>et al.</i> , 1979.
Cement Products .....	Mixed (Amosite, Chrysotile, Crocidolite).	89	0.53	Weill <i>et al.</i> , 1979.
<b>Mesotheliomas</b>				
Cement Products .....	Mixed (Amosite, Chrysotile, Crocidolite).	108	1.2 E-5	Finkelstein, 1983.
Textile Products .....	Chrysotile .....	67	3.2 E-6	Peto <i>et al.</i> , 1982.
Insulation Products .....	Amosite .....	400	1.0 E-6	Seidman <i>et al.</i> , 1979; Seidman, 1984.
Insulation Workers .....	Mixed (Amosite, Chrysotile, Crocidolite).	375	1.5 E-6	Selikoff <i>et al.</i> , 1979.

1. Lung Cancer

*a. Berry and Newhouse, 1983*

Berry and Newhouse (1983) conducted a retrospective mortality study (1942–1980) using data from an English factory that manufactured asbestos-containing friction materials (e.g., brake blocks, stair treads). There were 13,460 workers included in this study, of which two-thirds were men. Most had worked in this factory for 2–10 years. The asbestos exposures generally involved chrysotile, although this site also had used crocidolite for two brief periods, one from 1922–1933 and a second from 1939–1944.

Personal air sampling for the assessment of asbestos concentrations in this factory began in 1968. Fiber levels for time periods prior to 1968 were “estimated by reproducing earlier work conditions using detailed knowledge of when processes were changed and exhaust ventilation introduced.” Asbestos fiber concentrations were determined over four time periods: Pre-1931, 1932–1950, 1951–1969, and 1970–1979. Before 1931, asbestos levels

typically exceeded 20 f/mL throughout the factory. From 1932–1969, asbestos levels decreased and most exposures ranged from 2–5 f/mL. After 1970, levels decreased to below 1 f/mL.

Berry and Newhouse (1983) did not detect excessive mortality at this factory over the period 1942 to 1980. OSHA noted, however, the relatively short duration of employee exposures and the short follow-up period (e.g., less than 20 years for 33 percent of the men). In the preamble to their 1986 asbestos rule, OSHA stated,

“ \* \* \* Because of the short follow-up period used, OSHA does not believe that the non-significant increases in lung cancer mortality found by these investigators [Berry and Newhouse] contradict the findings from other studies which show that low-level exposure to asbestos has resulted in excessive mortality from lung cancer \* \* \* ”

*b. Dement et al., 1982*

Dement *et al.* (1982) conducted a retrospective cohort mortality (1930–1975) study of 768 men. These men had worked in an asbestos textile factory located in South Carolina where “only an insignificant quantity of asbestos

fiber other than chrysotile was ever processed.” The men in this study had at least 1 month of employment between January 1, 1940 and December 31, 1965. Dement *et al.* then followed the cohort for another 10 years.

Air samples were collected in this factory between 1930 and 1975 to determine asbestos levels. Impinger samples were collected prior to 1965; then membrane filter sampling was introduced. Membrane filter sampling fully replaced the impinger method in 1971. There were 193 air samples collected in 1930–1945, 183 in 1945–1960, and 5,576 in 1960–1975. The estimated mean asbestos exposure levels by job and calendar time periods, using linear regression models, were as high as 78 f/cc before 1940 and generally ranged from 5–10 f/cc after 1940.

Dement *et al.* (1982) demonstrated a linear dose-response relationship for lung cancer mortality that did not appear to have a threshold. They also found a linear dose-response relationship for non-malignant respiratory disease, other than upper respiratory infection, influenza,

pneumonia, or bronchitis. Like the lung cancer data, the dose-response relationship for non-malignant respiratory disease did not appear to have a threshold.

OSHA's 1986 rulemaking considered that Dement *et al.*'s report of excess risk at low cumulative [asbestos] exposures was well supported because of their " \* \* \* careful estimation of exposure histories for members of the cohort \* \* \*".

#### c. Finkelstein, 1983

Finkelstein (1983) studied a group of 328 men who worked in an Ontario, Canada, factory that manufactured asbestos-cement pipe and rock-wool insulation. Men selected to participate in this study began working at the factory prior to 1961 and worked for the company for at least 9 years. Finkelstein divided the men into three groups based on estimated levels of asbestos exposure: 186 in production (consistent exposure), 55 in maintenance (intermittent exposure), and 87 controls (minimal exposure). The asbestos exposures involved chrysotile and crocidolite, both of which the factory mixed with cement and silica. This study report did not indicate the proportions of asbestos and silica used in the cement.

Air samples were collected to assess asbestos levels at this cement factory. Impinger sampling was conducted between 1943 and 1968. In 1969–1970, the factory began to use the personal membrane filter sampling method and used this sampling data to classify the men who worked in cement production according to their probable cumulative asbestos exposure. They used three subgroups (A, B, C) of estimated exposure ranges and means as follows:

#### CUMULATIVE EXPOSURE

[Fiber-years/mL]

	Range	Mean
Subgroup A .....	8–69	44
Subgroup B .....	69–121	92
Subgroup C .....	122–420	180

Finkelstein also relied on detailed employment histories and medical records for each man in the study. Finkelstein (1983) found that the asbestos-exposed workers had *all-cause* mortality rates that were twice that of the general Ontario population. He also reported that the mortality rates due to malignancies and the deaths attributable to lung cancer were five and eight times those of the general population, respectively.

#### d. Henderson and Enterline, 1979

In 1979, Henderson and Enterline published an update of their 1941–1967 mortality study. The extended study provided data through 1973 and included 1,075 men who had worked for an asbestos company in the United States for an average of 25 years. Most of the workplace exposures involved chrysotile, although some involved amosite or crocidolite.

Henderson and Enterline conducted impinger sampling to determine asbestos levels for this study and reported asbestos concentrations in millions of particles per cubic foot (mppcf). They also identified five cumulative exposure categories (87, 255, 493, 848, and 1,366 fiber-years/cc) by converting their original data, reported in mppcf, to f/cc using a factor of 1:1.4 as discussed in the 1986 OSHA asbestos rule (51 FR 22617).

For the period 1941–1973, Henderson and Enterline (1979) found that this cohort had an overall mortality rate that was about 20 percent higher than that of males in the general population. This increase in mortality rate was mainly due to lung cancer and other respiratory diseases.

OSHA (1986) noted that the excess mortality risk found by Henderson and Enterline (1979) was less than that found by Dement *et al.* (1982). Henderson and Enterline, however, studied retired asbestos workers, which "constitute a select group of survivors" (51 FR 22617), and which might explain the difference in results of these two mortality studies.

#### e. Peto, 1980

Peto (1980) continued the study of workers in an asbestos textile factory in England. His paper, published in 1980, was an extension of two earlier reports, one by Doll (1955) and a second by Peto *et al.* (1977). In this updated study (1980), Peto included 679 men who were hired in 1933 or later, and who had been employed by the company for at least 10 years by 1972. Peto divided the workers into two cohorts: those first exposed before 1951 (Cohort 1, n = 424 men) and those first exposed during or after 1951 (Cohort 2, n = 255 men). The National Health Central Register and factory personnel followed the workers until 1978. The exposures in this textile factory involved chrysotile.

Although routine measurements of asbestos levels were not made prior to 1951, Peto *et al.* (1977) had estimated the workers' exposures in an earlier study. Between 1951 and 1961, a thermal precipitator was used to sample for asbestos, then was gradually

replaced by membrane filters. In this study, Peto revised earlier estimates of asbestos exposure concentrations and reported mean levels in fibers/mL for six selected years as follows: 32.4 (1951), 23.9 (1956), 12.2 (1961), 12.7 (1966), 6.7 (1971), and 1.1 (1974). Peto *et al.* then used these values to calculate cumulative exposures. The average cumulative exposure for men first exposed to asbestos during or after 1951 (*i.e.*, Cohort 2) was 200–300 fiber-years/mL.

Peto (1980) confirmed earlier conclusions by Doll (1955) and Peto *et al.* (1977) that there was excess lung cancer mortality in this asbestos textile factory. Although Peto *et al.* (1977) suggested a dose-response relationship for lung cancer using measurements from a static dust sampler, Peto did not demonstrate such a dose-response relationship in this later study (1980).

#### f. Seidman *et al.*, 1979 (With Update to OSHA in 1984)

Seidman *et al.* (1979) conducted a mortality study (1946–1977) of 820 men who worked in an amosite factory in New Jersey. This factory supplied the U.S. Navy with insulation for pipes, boilers, and turbines. The men in this study were first employed between 1941 and 1945 and were followed for 35 years. Due to wartime conditions, however, there was a changing composition of the workforce. Seidman *et al.* (1979) stated that—

This resulted in a unique experience; men with a very limited duration of intense exposure to Amosite asbestos, followed by long observation \* \* \*

The men were classified according to the time in which they came into direct contact with the amosite: Less than 1 month, 1 month, 2 months, 3–5 months, 6–11 months, 1 year, or 2 or more years. Thus, this cohort is unlike those of other studies where workers were exposed to asbestos for long periods, often 20 or more years.

In this amosite factory, there were no direct measurements of asbestos levels. The determination of asbestos concentrations was made solely by analogy with another factory in which air sampling was done in the late 1960's and in the 1970's. Seidman *et al.* reported that, in samples taken in this latter factory in October of 1971, asbestos counts averaged as high as 23 f/mL.

Seidman *et al.* (1979) demonstrated that the amosite workers were at risk of developing lung cancer and dying from this disease. Seidman *et al.* (1979) concluded that—

- Prolonged follow-up is necessary to evaluate the effects of asbestos on

health, especially with lower concentration or shorter duration exposures.

- Asbestos retained in tissues may continue to produce adverse effects long after the exposure may have stopped.

- The length of the latency period for asbestos-related diseases depends directly on the dosage and the age at which exposure takes place. For example, older workers will show a more pronounced and quicker effect than younger workers with the same level of exposure.

- The longer the time after first exposure to asbestos, the more pronounced the excesses in mortality.

- Reducing the asbestos exposure (lowering the dosage) can both delay the occurrence of adverse effects (e.g., time to death) and lower the frequency of their occurrence (e.g., fewer deaths).

In 1984, Seidman updated his earlier work by adding 593 cases involving deaths that occurred 5–40 years beyond each man’s first amosite exposure. Seidman again developed a classification scheme, but now he based it on cumulative exposure to amosite and not on time alone. The exposure categories were less than 6, 6–11.9, 12–24.9, 25–49.9, 50–99.9, 100–149.9, 150–249.9, and 250 or more fiber-years/cc. Using this new information, he was able to demonstrate an exposure-response relationship for lung cancer mortality.

*g. Selikoff et al., 1979.*

Selikoff *et al.* (1979) conducted a mortality study (1943–1976) of 17,800 men who belonged to the insulation workers’ union. Members of this insulation union worked mainly in construction in the United States and Canada, but some worked in refineries, industrial plants, shipyards, and power plants. Selikoff *et al.* (1979) described the content of the asbestos insulation as follows.

Until approximately the early 1940s, chrysotile alone was utilized in the manufacture of the asbestos insulation products used by these men. Amosite began to be used in the mid-1930s in small quantities but became more widely utilized during World War II and subsequently.

The ages of men in this study ranged from 15 to over 85 years and Selikoff *et al.* (1979) established a series of “age

categories,” each including a 5-year age span (e.g., 15–19 years, 20–24 years, etc.) Those men age 85 or older were grouped together. The investigators identified the time at which each man was first exposed to asbestos and then separated the data into a series of categories based on how long it had been since their first exposure (e.g., less than 20, 20–34, and 35 or more years ago).

Selikoff *et al.* (1979) reported that few measurements were made to assess asbestos levels in insulation work until the mid-1960’s. For this reason, they estimated exposure levels using reconstructions of past work conditions and extrapolations of more current measurements to past conditions. They concluded that insulation workers would have been exposed to TWA concentrations of 4–12 f/mL.

Selikoff *et al.* (1979) concluded that the asbestos insulation workers were at “extraordinary increased risk of death of cancer and asbestosis.” The study had found an excessive number of lung cancers (486) in this cohort, particularly at 15–35 years after the first exposure to asbestos. This figure was even more striking when compared to the expected number of lung cancer cases (106) for this same group of men.

*h. Weill et al., 1979.*

Weill *et al.* (1979) conducted a mortality study of 5,645 men who had at least 1 month of continuous employment before January 1, 1970 in one of two asbestos cement building materials plants in New Orleans, Louisiana. The men in this study had worked at some time during the 1940’s to the mid-1970’s. The investigators followed this cohort for at least 20 years and found that—

For both plants, 7 percent [of the men] were initially employed before 1940, 76 percent during the 1940s, and 17 percent during 1950 to 1954. Sixty percent were employed for less than one year, 24 percent for one to 10 years, and 16 percent for more than 10 years.

The asbestos exposures mainly involved chrysotile, although the two plants also processed crocidolite and amosite. The cement products were comprised of about 15–28 percent asbestos and some silica. Weill *et al.*

(1979), however, did not provide the proportion of silica in the asbestos cement mixture.

Impinger sampling was conducted in this factory to determine asbestos levels. The sampling results were reported in millions of particles per cubic foot (mppcf). Based on sampling data, Weill *et al.* (1979) defined five categories of exposure in mppcf/year as follows: Less than 10, 11–50, 51–100, 101–200, and more than 200. OSHA (51 FR 22618) converted the original data of Weill *et al.* (1979) from mppcf/year to fiber-years/cc using a factor of 1:1.4, as given in the 1986 OSHA rule (51 FR 22617). This yielded the following exposure categories in fiber-years/cc: Less than 14, 15–70, 71–140, 141–280, more than 280.

Weill *et al.* (1979) found excess mortality due to cancers, mainly lung cancer, in men whose cumulative exposures were moderate (141–280 fiber-years/cc) to high (greater than 280 fiber-years/cc). About 25 percent of their cohort, however, was lost in the follow-up period. For the purpose of the study, Weill *et al.* assumed they were alive. This assumption may have led to an underestimation of lung cancer risk. For this reason, OSHA (51 FR 22618) stated its opinion as follows:

\* \* \* the presence of an excess risk of mortality from lung cancer could not be ruled out for the cohorts in these exposure categories. [The other three, lower exposure categories defined by Weill *et al.*, 1979.]

2. Mesotheliomas

a. *Finkelstein, 1983.*

We reviewed the most important aspects of this study above. (See section VI.A.1.) Based on death records, Finkelstein (1983) found 11 mesotheliomas among the total of 58 deaths in his study. The mean age at which these men were first exposed to asbestos was 25 years, and their mean latency period for mesotheliomas was 25 years. The mean age at death was 51 years, and none was over 60 years. This demonstrates that death follows quickly after this disease becomes evident.

Finkelstein noted that the rates of death from mesotheliomas were proportional to the magnitude of cumulative asbestos exposure, as shown in Table VI–2 below.

TABLE VI–2.—MESOTHELIOMAS MORTALITY RATES COMPARED TO EXPOSURE

Mesotheliomas mortality rates (per 1,000 man-years)	Estimated exposure range (fiber-years/mL)	Estimated mean exposure (fiber-years/mL)
1.9	8–69	44

TABLE VI-2.—MESOTHELIOMAS MORTALITY RATES COMPARED TO EXPOSURE—Continued

	Mesotheliomas mortality rates (per 1,000 man-years)	Estimated exposure range (fiber-years/mL)	Estimated mean exposure (fiber-years/mL)
4.9 .....		70–121	92
11.9 .....		122–420	180

Based on the exposure-response data, Finkelstein concluded, “\* \* \* the relation is compatible with a linear function through the origin \* \* \*.” Accordingly, Finkelstein’s data suggest the lack of a threshold for mesotheliomas.

b. *Peto et al., 1982.*

*Peto et al.* (1982) evaluated mesothelioma mortality (1967–1979) in the same group of 17,800 insulation workers previously described by Selikoff *et al.* (1979). We reviewed the salient features of Selikoff *et al.* (1979) above. (See section VI.A.1.) Members of this insulation workers’ union worked in the United States and Canada and were exposed to chrysotile and amosite.

*Peto et al.* (1982) reported “a high incidence” of mesotheliomas in this cohort. There were 236 deaths from mesotheliomas, of which 87 were pleural and 149 were peritoneal. They closely examined each man’s age at the first asbestos exposure and the number of years since his first exposure. *Peto et al.* (1982) concluded that mesothelioma mortality was strongly dependent on the number of years since the first asbestos exposure, but was independent of the age at the first exposure. They stated—

Mesothelioma death rates in asbestos workers appear to be proportional to the third or fourth power of time \* \* \* Age at first exposure has little or no influence, however, which supports the multi-stage model of carcinogenesis \* \* \* mesotheliomas may constitute a high proportion of cancer deaths resulting from early exposure to asbestos.

*Peto et al.* (1982) also reviewed mesothelioma mortality data from several other studies in addition to those from Selikoff *et al.* (1979). They were interested in determining if they could establish a relationship between deaths from mesotheliomas and fiber type. Although there were some data to suggest that deaths from mesotheliomas were more common in men who worked with amphiboles (*e.g.*, crocidolite), *Peto et al.* (1982) were cautious when drawing conclusions. They stated that—

Chemical [and physical] differences between different fibre types may also be

important, but until carcinogenic effects of such differences have been demonstrated, it would seem sensible to concentrate on fibre dimension rather than mineral type in developing dose-response relationships. \* \* \* It may therefore be dangerously optimistic to attribute the substantial incidence of pleural mesothelioma among chrysotile factory workers to occasional crocidolite exposure \* \* \*

c. *Seidman et al. 1979 (With Update to OSHA in 1984).*

We reviewed the salient features of this study and its update above. (See section VI.A.1.) Based on death records, *Seidman et al.* (1979) found 14 mesotheliomas among the total 528 deaths in their study. They reported an additional three mesotheliomas in their update. OSHA commented that this was “a finding of great significance given the rarity of the disease” (51 FR 22617).

d. *Selikoff et al. (1979).*

The salient features of this study were reviewed above. (See section IV.A.1.) Based on death records, Selikoff *et al.* (1979) found 38 mesotheliomas (pleural and peritoneal) in their initial cohort of 632 asbestos insulation workers. There were 223 deaths in this part of their study (1943–1976). Some of these deaths from mesotheliomas occurred 20–34 years after the first exposure to asbestos, described by the authors as “duration from onset.” For most men who died from mesotheliomas, however, it was 35 or more years after their first exposure.

In the second and much larger cohort (n = 17,800) of Selikoff *et al.* (1979), there were 175 deaths due to mesotheliomas of the total 2,271 deaths in this group. Some (14) of these deaths caused by mesotheliomas occurred 15–24 years after the first asbestos exposure, while most (161) were recorded 25 or more years after the first exposure. Selikoff *et al.* (1979) had been unable to provide expected death rates for mesotheliomas due to their rarity in the general population. This study demonstrated an unequivocal association between mesotheliomas and prior asbestos exposure. In the 25 years since this paper was published, there has been no evidence to the contrary.

3. Asbestosis

a. *Berry and Lewinsohn, 1979.*

*Berry and Lewinsohn* (1979) studied the same group of textile workers that was originally described by *Berry et al.* (1979) and, thus, a short summary of the original paper is presented here.

*Berry et al.* (1979) studied a group of 379 men who worked in an asbestos textile factory located in northern England. Most of the worker exposures involved chrysotile, although this site also used crocidolite. Asbestos fiber levels were measured in this factory since 1951 and had been estimated since 1936. *Berry et al.* defined two cohorts. One included men who were first employed between 1933 and 1950, and were still working in this textile factory in 1966. The other included men who were employed after 1966, and had worked for at least 10 years in this textile factory. *Berry et al.* (1979) found relationships between cumulative asbestos exposure and crepitations (abnormal lung sounds), possible asbestosis, and certified asbestosis.

As noted above, *Berry and Lewinsohn* (1979) used data from the same textile factory as that described by *Berry et al.* (1979); but *Berry and Lewinsohn* (1979) defined two different cohorts. One included men who were first employed before 1951. The other included men first employed after 1950. *Berry and Lewinsohn* (1979) plotted the incidence of cases of possible asbestosis against the cumulative asbestos exposure up to 1966. They stated—

The data are compatible with a linear relationship through the origin [indicating no threshold], with no statistically significant difference between the two groups [cohorts].

b. *Finkelstein, 1982.*

*Finkelstein* (1982) studied a group of 201 men who worked in a factory in Ontario, Canada, that manufactured asbestos-cement pipe and rock-wool insulation. *Finkelstein* defined two subsets in his study population: A group of 157 production workers and a group of 44 maintenance workers. The men selected to participate in this study worked in the pipe or board shop for at least one year prior to 1961 and had been employed at least 15 years. Most of the asbestos exposures involved

chrysotile and crocidolite, both of which were mixed with cement and silica.

Between the 1940's and 1968, impinger sampling was conducted to assess total dust levels. In 1969/1970, the company began to conduct quarterly personal sampling for asbestos using the membrane filter method. Finkelstein used the results of such sampling as baseline values for various jobs.

Of the workers in this study, 39 percent of those in production and 20 percent of those in maintenance had certified asbestosis. Finkelstein demonstrated that there was a relationship between cumulative asbestos exposure and certified asbestosis. He describes the exposure-response curve as sigmoidal, a shape commonly observed in toxicology. The curve also appears to intersect the origin, which suggests a lack of threshold.

*B. Models Selected by OSHA (1986) for Specified Endpoints and for the Determination of Its PEL and STEL*

Based on their critical review of the studies described above (see section VI.A), OSHA (51 FR 22631) concluded—

\* \* \* asbestos exposure causes lung disease, respiratory cancer, mesothelioma, and gastrointestinal cancer. \* \* \* excess disease risk has been observed at cumulative exposures at or below those permitted by the existing OSHA 8-hour permissible exposure limit [PEL] of 2 f/cc. In addition, OSHA has made risk estimates of the excess mortality from lung cancer, mesothelioma, gastrointestinal cancer, and the incidence of asbestosis using mathematical models \* \* \*

The following is a summary of the mathematical models that OSHA used in its asbestos risk assessment.

1. Lung Cancer

For lung cancer, OSHA (1986) relied on a relative risk model that was linear in dose, as described by the following equation:

$$R_L = R_E[1 + (K_L)(f)(d_{t-10})]$$

Where:

- R<sub>L</sub> = Predicted lung cancer mortality.
- R<sub>E</sub> = Expected lung cancer mortality in the absence of asbestos exposure.
- K<sub>L</sub> = Slope of the dose-response relationship for lung cancer.
- f = Asbestos fiber concentration (f/cc).
- d = Duration of the exposure (minus 10 years to account for latency).

The following list gives the K<sub>L</sub> values for the eight studies used by OSHA. OSHA (51 FR 22637) used K<sub>L</sub> = 0.01, the geometric mean of these eight studies, in their risk assessment.

Study	K <sub>L</sub>
Berry and Newhouse, 1983 ....	0.0006
Dement <i>et al.</i> , 1982 .....	0.042
Finkelstein, 1983 .....	0.048
Henderson and Enterline, 1979 .....	0.0047
Peto, 1980 .....	0.0076
Seidman <i>et al.</i> , 1979; Seidman, 1984 .....	0.045
Selikoff <i>et al.</i> , 1979 .....	0.020
Weill <i>et al.</i> , 1979 .....	0.0033

2. Mesotheliomas

For mesotheliomas, OSHA (1986) relied on an absolute risk model that is linear in dose, but exponentially related to the time after the first exposure to asbestos. The following three equations describe the risk.

$$AR_M = (f)(K_M)[(t-10)^3 - (t-10-d)^3], \text{ for } t > 10 + d$$

$$AR_M = (f)(K_M)[(t-10)^3], \text{ for } 10 + d > t > 10$$

$$AR_M = 0, \text{ for } 10 > t$$

Where:

- R<sub>M</sub> = Excess risk of mesotheliomas.
- f = Asbestos fiber concentration.
- K<sub>M</sub> = Slope of the dose-response relationship for mesotheliomas.
- d = Duration of the exposure.
- t = Time after the first exposure to asbestos.

The following list gives the K<sub>M</sub> values for the four studies used by OSHA. OSHA (51 FR 22640 and 22642) used K<sub>M</sub> = 1 × 10<sup>-8</sup>, the ratio of K<sub>M</sub>/K<sub>L</sub>, rather than K<sub>M</sub> = 2.91 × 10<sup>-8</sup>, the geometric mean of these four studies, to account

for the bias in its analysis and avoid overestimation of mesotheliomas in their risk assessment.

Study	K <sub>M</sub> (10 <sup>-8</sup> )
Finkelstein, 1983 .....	12
Peto <i>et al.</i> , 1982 .....	0.7
Seidman <i>et al.</i> , 1979; Seidman, 1984 .....	5.7
Selikoff <i>et al.</i> , 1979 .....	1.0

3. Asbestosis

For asbestosis, OSHA (1986) relied on an absolute risk model that was linear in cumulative dose. The following equation describes the lifetime incidence of asbestosis:

$$R_A = m(f)(d)$$

Where:

R<sub>A</sub> = Predicted lifetime incidence of asbestosis.

f = Asbestos fiber concentration.

d = Duration of the exposure.

m = Slope of the linear regression.

OSHA stated (48 FR 51132), “the best estimates of asbestosis incidence are derived from the Finkelstein data “and OSHA did not rely on the values for the slope as determined by Berry and Lewinsohn (1979). Thus, based on Finkelstein’s data (1982) alone, the slope (m) is 0.055 and the equation becomes R<sub>A</sub> = 0.055(f)(d).”

Using this linear model, OSHA also calculated estimates of lifetime asbestosis incidence at five exposure levels of asbestos (*i.e.*, 0.5, 1, 2, 5, 10 f/cc) and published Table VI-3 (48 FR 51132), which we have reproduced below. OSHA concluded that for lifetime exposures to asbestos at concentrations of 2 or 0.5 f/cc, there would be a 5 percent or a 1.24 percent incidence of asbestosis, respectively (48 FR 51132). Based on Finkelstein’s linear relationship for lifetime asbestosis incidence, OSHA later stated (51 FR 22646) that, “Reducing the exposure to 0.2 f/cc [a concentration not included in Table VI-3] would result in a lifetime incidence of asbestosis of 0.5%.”

TABLE VI-3.—ESTIMATES OF LIFETIME ASBESTOSIS INCIDENCE

Exposure level, fiber/cc	Percent (%) Incidence		
	Finkelstein	Berry (employed before 1951)	Berry (first employed after 1950)
0.5 .....	1.24	0.45	0.35
1 .....	2.49	0.89	0.69
2 .....	4.97	1.79	1.38
5 .....	12.43	4.46	*3.45
10 .....	24.86	8.93	6.93
Slope .....	0.055	0.020	0.015

TABLE VI-3.—ESTIMATES OF LIFETIME ASBESTOSIS INCIDENCE—Continued

Exposure level, fiber/cc	Percent (%) Incidence		
	Finkelstein	Berry (em- ployed before 1951)	Berry (first em- ployed after 1950)
R <sup>2</sup> .....	0.975	0.901	0.994

\* Note: 1.38 in original table was a typographical error. The text (48 FR 51132) and the regression formula indicate that 3.45 is the correct percent.

C. OSHA's Selection of Its PEL (0.1 f/cc)

Using the models described above in section VI.B., OSHA estimated cancer mortality for workers exposed to asbestos at various cumulative exposures (i.e., combining exposure

concentration and duration of exposure). These data were published in its 1986 risk assessment (51 FR 22644), which we have reproduced in the following Table VI-4.

It is clear from Table VI-4 that the estimated mortality from asbestos-

related cancer decreases significantly by lowering exposure. This is true regardless of the type of cancer: lung, pleural, peritoneal, or gastrointestinal. Although excess relative risk is linear in dose, the excess mortality rates in Table VI-4 are not strictly linear in dose.<sup>68</sup>

TABLE VI-4.—ESTIMATED ASBESTOS-RELATED CANCER MORTALITY PER 100,000 BY NUMBER OF YEARS EXPOSED AND EXPOSURE LEVEL

Asbestos fiber concentration (fiber/cc)	Cancer Mortality per 100,000 Exposed			
	Lung	Mesothelioma	Gastro- intestinal	Total
1-year exposure				
0.1 .....	7.2	6.9	0.7	14.8
0.2 .....	14.4	13.8	1.4	29.6
0.5 .....	36.1	34.6	3.6	74.3
2.0 .....	144	138	14.4	296.4
4.0 .....	288	275	28.8	591.8
5.0 .....	360	344	36.0	740.0
10.0 .....	715	684	71.5	1,470.5
20-year exposure				
0.1 .....	139	73	13.9	225.9
0.2 .....	278	146	27.8	451.8
0.5 .....	692	362	69.2	1,123.2
2.0 .....	2,713	1,408	271.3	4,392.3
4.0 .....	5,278	2,706	527.8	8,511.8
5.0 .....	6,509	3,317	650.9	10,476.9
10.0 .....	12,177	6,024	1,217.7	13,996.7
45-year exposure				
0.1 .....	231	82	23.1	336.1
0.2 .....	460	164	46.0	670.0
0.5 .....	1,143	407	114.3	1,664.3
2.0 .....	4,416	1,554	441.6	6,411.6
4.0 .....	8,441	2,924	844.1	12,209.1
5.0 .....	10,318	3,547	1,031.8	14,896.8
10.0 .....	18,515	6,141	1,851.5	26,507.5

OSHA's PEL for asbestos was 2 f/cc in 1983. Table VI-4 shows that after 45 years of exposure to asbestos at this concentration, there would be an estimated 6,411.6 deaths (per 100,000 workers). This is the sum of deaths from 4,416 lung cancers, 1,554 mesotheliomas, and 441.6 gastrointestinal cancers. By lowering its PEL to 0.1 f/cc, OSHA decreased the risk of cancer mortality to an estimated

336.1 deaths (per 100,000 workers), which is the sum of deaths from 231 lung cancers, 82 mesotheliomas, and 23.1 gastrointestinal cancers.

As shown above in Table VI-3, there is also a significant reduction in the incidence of asbestosis by lowering exposures. For example, the lifetime incidence of asbestosis would be reduced from 4.97 percent (4,970 cases per 100,000 workers) at 2 f/cc to 1.24

percent (1,240 cases per 100,000 workers) at 0.5 f/cc. Using the linear model described above [ $R_A = 0.055(f)(d)$ ], the incidence of asbestosis can also be calculated at a concentration of 0.1 f/cc (not included by OSHA in Table VI-4) following 45 years of exposure to asbestos. This yields 0.25 percent, or 250 cases per 100,000 workers. Thus, by lowering the 8-hour TWA PEL from 2 f/cc to 0.1 f/cc, we

<sup>68</sup>Nicholson, p. 53, 1983.

would reduce the lifetime asbestosis risk from 4,970 cases to 250 cases per 100,000 exposed miners.

Based on these reductions in cancer deaths and asbestosis cases, OSHA demonstrated that a lowering of the PEL below 2 f/cc would “substantially reduce that risk” (51 FR 22612). OSHA also noted—

Evidence in the record “has shown that employees exposed at the revised standards” PEL of 0.2 fiber/cc [OSHA’s 1986 standard] remain at significant risk of incurring a chronic exposure-related disease, but considerations of feasibility have constrained OSHA to set the revised PEL at the 0.2 fiber/cc level.

When OSHA further reduced its PEL from 0.2 to 0.1 f/cc in 1994, this statement was still true and the PEL continued to reflect technical feasibility issues. OSHA stated (59 FR 40967)—

The 0.1 f/cc level leaves a remaining significant risk. However as discussed below [in OSHA’s 1994 Final Rule] and in earlier documents, OSHA believes that this is the

practical lower limit of feasibility for measuring asbestos levels reliably.

*D. Applicability of OSHA’s Risk Assessment to the Mining Industry*

In its asbestos emergency temporary standard, and in its proposed, amended, and final asbestos rules (1983, 1984, 1986, 1992, 1994), OSHA discussed few mining and milling studies and excluded these data in their risk assessment. OSHA (51 FR 22637) stated,

The distinct nature of mining-milling data (and hence the estimate of KL from these data) has been considered earlier. There is some evidence that risks in the asbestos mining-milling operations are lower than other industrial operations due to differences in fiber size. “Thus, in determining the KL for the final rule, the data from mining and milling processes were not considered.

OSHA suggested that the proportionality constants (*i.e.*,  $K_L$ ,  $K_M$ ), also known as the slopes of the respective dose response curves, from mining and milling studies are lower

than the slopes for the studies included in its risk assessment (51 FR 22632 and 22637). This difference in slopes may suggest that the risk of asbestos-related cancers is lower in miners and millers. Because there is remaining significant risk of asbestos-related cancer at the OSHA PEL of 0.1 f/cc, we may be accepting a higher estimate of risk by relying on OSHA’s quantitative risk assessment that excluded mining and milling studies.

Although we are relying on OSHA’s risk assessment, we also reviewed the scientific literature to identify studies that involved the exposure of miners and millers to asbestos. Most of these studies were conducted in Canada, although some have been conducted in Australia, India, Italy, South Africa, and the United States. Table VI–5 lists some of these mining and milling studies, in chronological order, and gives the salient features of each study. These studies are in the rulemaking docket.

TABLE VI–5.—SELECTED STUDIES INVOLVING MINERS EXPOSED TO ASBESTOS

Author(s), year of publication	Study group, type of asbestos	Major finding(s) or conclusion(s)
Rossiter <i>et al.</i> , 1972	Canadian miners and millers, Chrysotile	Radiographic changes (opacities) related to age and exposure.
Becklake, 1979	Canadian miners and millers, Chrysotile	Weak relationship between exposure and disease.
Gibbs and du Toit, 1979	Canadian and South African miners, Chrysotile.	Need for workplace epidemiologic surveillance and environmental programs.
Irwig <i>et al.</i> , 1979	South African miners, Amosite and crocidolite	Parenchymal radiographic abnormalities preventable by reduced exposure.
McDonald and Liddell, 1979	Canadian miners and millers, Chrysotile	Lower risk of mesotheliomas and lung cancer from chrysotile than crocidolite.
Nicholson <i>et al.</i> , 1979	Canadian miners and millers, Chrysotile	Miners and millers: At lower risk of mesotheliomas, at risk of asbestosis (as factory workers and insulators), at risk of lung cancer (as factory workers).
Rubino <i>et al.</i> , <i>Ann NY Ac Sci</i> 1979	Italian miners, Chrysotile	Role of individual susceptibility in appearance and progression of asbestosis.
Rubino <i>et al.</i> , <i>Br J Ind Med</i> 1979	Italian miners, Chrysotile	Elevated risk of lung cancer.
Solomon <i>et al.</i> , 1979	South African miners, Amosite and Crocidolite	Sign of exposure to asbestos: Thickened interlobar fissures.
McDonald <i>et al.</i> , 1980	Canadian miners and millers, Chrysotile	No statistically significant increases in SMRs.
McDonald <i>et al.</i> , 1986	U.S. miners, Tremolite	A. Increased risk of mortality from respiratory cancer. B. Increased prevalence of small opacities by retirement age.
McDonald <i>et al.</i> , 1980	U.S. miners, Tremolite	B. Increased prevalence of small opacities by retirement age.
Cookson <i>et al.</i> , 1986	Australian miners and millers, Crocidolite	No threshold dose for development of radiographic abnormality.
Amandus <i>et al.</i> , 1987	U.S. miners, and millers, Tremolite-Actinolite	Part I: Increased prevalence of radiographic abnormalities associated with past exposure. Part II: Increased mortality from nonmalignant respiratory disease and lung cancer. Part III: Exposures below 1 f/cc after 1977, up to 100–200X higher in 1960’s and 1970’s.
Amandus and Wheeler, 1987	U.S. miners, and millers, Tremolite-Actinolite	Part II: Increased mortality from nonmalignant respiratory disease and lung cancer.
Amandus <i>et al.</i> , 1987	U.S. miners, and millers, Tremolite-Actinolite	Part III: Exposures below 1 f/cc after 1977, up to 100–200X higher in 1960’s and 1970’s.
Armstrong <i>et al.</i> , 1988	Australian miners and millers, Crocidolite	Increased mortality from mesotheliomas and lung cancer.
Enarson <i>et al.</i> , 1988	Canadian miners, Chrysotile	Increased cough, breathlessness, abnormal lung volume and capacity.
McDonald <i>et al.</i> , 1988	U.S. miners, and millers, Tremolite	Low exposure and no statistically significant SMRs.
McDonald <i>et al.</i> , 1993	Canadian miners and millers, Chrysotile	Increased SMRs for lung cancer and mesotheliomas as cohort aged.

TABLE VI-5.—SELECTED STUDIES INVOLVING MINERS EXPOSED TO ASBESTOS—Continued

Author(s), year of publication	Study group, type of asbestos	Major finding(s) or conclusion(s)
Dave <i>et al.</i> , 1996 .....	Indian miners and millers, Chrysotile .....	Higher exposures in surface than underground mines; higher exposures in mills than mines; restrictive lung impairment and radiologic parenchymal changes more common in millers.
McDonald <i>et al.</i> , 1997 .....	Canadian miners and millers, Chrysotile .....	Risk of mesotheliomas related to geography and mineralogy of region; mesotheliomas caused by amphiboles.
Nayebzadeh <i>et al.</i> , 2001 .....	Canadian miners and millers, Chrysotile .....	Respiratory disease related to regional differences in fiber concentration and not dimension.
Ramanathan and Subramanian, 2001 .....	Indian miners and millers, Chrysotile and tremolite.	Increased risk of cancer, restrictive lung disease, radiologic changes, and breathing difficulties; more common in milling.

These studies of miners and millers provide further evidence of potential adverse health effects from asbestos exposure. MSHA found that many of the observations presented in these studies (e.g., age of first exposure, latency, radiologic changes) are consistent with those from studies of factory and insulation workers. The exposure to asbestos, a known human carcinogen, results in similar disease endpoints regardless of the occupation that has been studied.

#### E. Significance of Risk

##### 1. Defining “Significant” Risk: The Benzene Case

We (MSHA) believe that this proposed rule for asbestos meets the requirements set forth by the OSHA Benzene Case described below. We have relied on OSHA’s risk assessment, the studies used by OSHA in its development, and our review of more recent studies and mining studies, which further support OSHA’s findings.

In the Benzene Case, *Industrial Union Department, AFL-CIO v. American Petroleum Institute et al.* (448 U.S. 607, 1980), the U.S. Supreme Court ruled that, prior to the issuance of a new or revised standard regulating occupational exposures to toxic materials, such as asbestos, OSHA is required to make two findings:

- They must determine that a “significant” health risk exists, and
- They must demonstrate that the new standard will reduce or eliminate that risk.

In the preamble to its 1994 final asbestos rule (59 FR 40966, 1994), OSHA provided an interpretation of a “significant health risk”. They stated,

OSHA has always considered that a working lifetime risk of death of over 1 per 1000 from occupational causes is significant. This has been consistently upheld by the courts.

When OSHA lowered its PEL for asbestos from 2 to 0.2 f/cc (1986), and then to 0.1 f/cc (1994), they used this definition of a “significant health risk” and made the two findings as outlined in the Benzene Case. With respect to the first finding, OSHA estimated the excess lifetime cancer risk to be 3.4 deaths per 1,000 workers exposed to asbestos at 0.1 f/cc for a working lifetime. OSHA stated (51 FR 22646),

The finding that a significant risk exists is supported by OSHA’s quantitative risk assessment, which is based upon studies of asbestos-exposed worker populations.

With respect to the second finding, OSHA went on to say (51 FR 22647),

In accordance with the second element [finding, *sic*] of the Supreme Court’s Benzene decision on the determination of significant risk, OSHA has determined that reducing the permissible exposure limit for asbestos [from 2 f/cc, *sic*] to 0.2 f/cc is reasonably necessary to reduce the cancer mortality risk from exposure to asbestos. \* \* \* significant risks of asbestos-related cancer mortality and asbestosis are not eliminated at the exposure level that is permitted under the new standard [0.2 f/cc, *sic*]; however, the reduction in the risk of asbestos-related death and disease brought about by promulgation of the new standard is both significant and dramatic.

OSHA concluded that the lowering of their PEL from 0.2 to 0.1 f/cc would “further reduce a significant health risk” (59 FR 40966–40967).

##### 2. Demonstrating Significant Health Risk for the Miner

The Federal Mine Safety and Health Act of 1977 (Mine Act), Title I, section 101(a), requires MSHA

\* \* \* to develop, promulgate, and revise as may be appropriate, improved mandatory health or safety standards for the protection of life and prevention of injuries in coal or other mines.

Furthermore, section 101(a)(6)(A) of the Mine Act requires MSHA to set health or safety standards—

\* \* \* on the basis of the best available evidence that no miner shall suffer material impairment of health or functional capacity even if such miner has regular exposure to the hazards \* \* \* for the period of his working lifetime.

A significant health risk exists for miners exposed to asbestos at our existing 8-hour full-shift exposure limit of 2 f/cc. Miners, like the insulation workers in the studies cited by OSHA, are at risk of developing lung cancer, mesotheliomas, and asbestosis. These effects are significant and clearly constitute a material impairment of health and functional capacity. They also emphasize the need for us to lower our PEL. By lowering the 8-hour full-shift exposure limit to 0.1 f/cc, we would significantly reduce the risk of asbestos-related lung cancers, mesotheliomas, and asbestosis.

##### 3. Using the Experience of OSHA and Current Studies to Demonstrate Significant Risk

Under the Mine Act, section 101(a)(6)(A), MSHA must base its health and safety standards on—

\* \* \* the latest available scientific data in the field, the feasibility of the standards, and experience gained under this and other health and safety laws.

In our proposed rule for asbestos, we have relied heavily on the experience of OSHA, which demonstrates the feasibility of a 0.1 f/cc exposure limit for asbestos. We believe that this limit is technically and economically feasible for the mining industry. (See section VIII.B. Feasibility.) We also have obtained and reviewed the latest available scientific data on the health effects of asbestos exposure. MSHA concludes that these studies provide further support of the significant risk of

adverse health effects following exposure to asbestos.

Using OSHA's risk assessment, we have demonstrated that a lowering of our 8-hour full-shift exposure limit from 2 to 0.1 f/cc would significantly reduce the risk of asbestos-related disease in miners. MSHA believes that other existing standards help reduce the remaining significant risk at this new 0.1 f/cc PEL. For example, MSHA requires the use of engineering and work practice controls to reduce a miner's exposure to the PEL and, until this concentration is reached, the use of an approved respirator. MSHA also requires the use of personal protective clothing and equipment, as necessary, for equipment repair and for construction or demolition activities<sup>69</sup> and hazard communication and task training.<sup>70</sup> As long as miners are likely to encounter asbestos, miners and mine operators will need to follow adequate safety procedures to ensure a reduction of exposures. We anticipate risk reduction to occur by the use of engineering controls and accepted industrial hygiene administrative controls that effectively avoid disturbing asbestos on mine property.

## VII. Section-by-Section Discussion of Proposed Rule

In the ANPRM, we asked commenters for supporting information to help us evaluate whether or not to—

- Lower our asbestos PEL,
- Revise our analytical methods and criteria to make them more appropriate for the mining industry, and
- Implement safeguards to limit take-home exposures.

We received almost 100 comments, considered the commenters' concerns, and discussed them in the following sections.

To make the standard easier to read, we have divided the requirements in the proposed standards into three paragraphs: *Definitions*, *Permissible Exposure Limits (PELs)*, and *Measurement of Airborne Fiber Concentration*. For §§ 56/57.5001(b), the metal and nonmetal asbestos standards, we numbered the paragraphs (b)(1), (b)(2), and (b)(3). For § 71.702, the coal asbestos standard, we assigned the paragraphs letters (a), (b), and (c).

### A. Sections 56/57.5001(b)(1) and 71.702(a): Definitions

Our existing definition of asbestos is consistent with several Federal agencies' regulatory provisions, including OSHA's. As discussed in

section II.B of this preamble and in the existing regulatory language, asbestos is not a definitive mineral name, but rather a commercial name for a group of minerals with specific characteristics. Our existing standards clearly state that, "when crushed or processed, [asbestos] separate[s] into flexible fibers made up of fibrils" [§§ 56/57.5001(b)]; and "does not include nonfibrous or nonasbestiform minerals" (§ 71.702). Although there are many asbestiform minerals, the term "asbestos" in our existing standards is limited to the following six (*Federal Six*):<sup>71</sup>

- Chrysotile (serpentine asbestos, white asbestos);
- Amosite (cummingtonite-grunerite asbestos, brown asbestos);
- Crocidolite (riebeckite asbestos, blue asbestos);
- Anthophyllite asbestos (asbestiform anthophyllite);
- Tremolite asbestos (asbestiform tremolite); and
- Actinolite asbestos (asbestiform actinolite).

Substantive changes to the definition of asbestos are beyond the scope of this proposed rule. We recognize that there are limitations in the general analytical methods, such as PCM and TEM, used to identify and quantify the *Federal Six*. Without the use of more complicated and costly analyses, it may not always be possible to differentiate other chemically similar amphiboles from the *Federal Six*. Also, the International Minerals Association has proposed more specific nomenclature in the literature to classify some of the amphiboles.<sup>72</sup> We decline to adopt such classifications here, because they are beyond the scope of this proposed rule, and propose to continue to use the existing regulatory designations. However, we are proposing a few nonsubstantive changes to the existing regulatory language to clarify the standard. These wording changes would have no impact on the minerals that we regulate as asbestos from that contained in the existing standards. This proposed rule would—

- Clarify the term "amosite," a name tied to asbestos from a specific geographical region, by adding the mineralogical term "cummingtonite-grunerite asbestos" parenthetically.
- Add a definition for fiber to be more consistent with OSHA. This change would clarify that the dimensional criteria in our existing standards refer to the asbestiform habit of the listed minerals.

- Conform the asbestos standards for metal and nonmetal mines, surface coal mines, and the surface work areas of underground coal mines by using the same structure and wording in the rule text. For example, we retain the descriptive language "Asbestos is a generic term for a number of hydrated silicates that, when crushed or processed, separate into flexible fibers made up of fibrils" from the metal and nonmetal standards rather than the comparable language from the coal standards. We believe that this descriptive language assists mine operators in understanding the scope of the standard.

MSHA's ANPRM did not specifically solicit information about which asbestiform minerals we should regulate. Even so, some commenters suggested that MSHA should expand its definition of asbestos to include other asbestiform minerals, so long as our analytical method excluded the counting of cleavage fragments. One commenter recommended that the PEL be reduced not only for the six currently regulated asbestos minerals, but also for other amphibole minerals in their asbestiform habit. NIOSH commented that cleavage fragments of the serpentine minerals antigorite and lizardite and amphibole minerals contained in the series cummingtonite-grunerite, tremolite-ferro-actinolite, and glaucophane-riebeckite should be counted as asbestos if they meet the counting requirements for a fiber (3:1 aspect ratio and greater than 5 µm in length). Another commenter asked that MSHA not include nonasbestiform fibrous minerals and mineral cleavage fragments when we perform microscopic analysis of samples.

Most commenters did not want MSHA to make changes to the fibers regulated as asbestos in the existing standards. Specifically, they do not want us to address other asbestiform amphiboles found in mineral deposits because they may not pose the same health problems that asbestos does. Some said that it would be unreasonable and expensive to try to meet exposure limits for all these minerals. Other commenters at MSHA's public hearing in New York (2002) stated that, whatever they are called, these minerals cause illness.

At this time, we decline to propose substantive changes to the definition of asbestos as suggested by some commenters. These changes are beyond the scope of this rulemaking. We will continue to monitor the toxicological, epidemiological, and mineralogical research studies and other new

<sup>71</sup> ATSDR, p.136, 2001; NIOSH Pocket Guide, 2003.

<sup>72</sup> Leake *et al.*, 1997.

<sup>69</sup> 30 CFR 56/57.5005, 56/57.15006, and 71.701

<sup>70</sup> 30 CFR parts 46, 47, and 48.

information relevant to protecting the health of miners.

*B. Sections 56/57.5001(b)(2) and 71.702(b): Permissible Exposure Limits (PELs)*

MSHA currently limits a miner's 8-hour TWA, full-shift exposure to 2.0 f/cc over a full shift; and limits a miner's short-term exposure to 10 f/cc over a 15-minute sampling period for metal and nonmetal miners and 10 f/cc for a total of one hour in an 8-hour day for miners at surface work areas of coal mines. We are proposing to adopt OSHA's 8-hour TWA, full-shift exposure limit of 0.1 f/cc and their 30-minute excursion limit of 1.0 f/cc for the mining industry. These actions would reduce by almost 20-fold the risk of asbestos-related deaths from a lifetime exposure at MSHA's existing permissible exposure limits. The proposed exposure limits, however, were based on feasibility and would not completely eliminate the risk. We believe that the proposed excursion limit would help reduce the residual risk from long-term exposure at the 0.1 f/cc 8-hour TWA, full-shift exposure limit.

As noted by the OIG, the continued occurrence of asbestos-related diseases and deaths among miners emphasizes the need to reduce asbestos exposures. MSHA's recent field sampling data (2000 through 2003) show that 2 percent of the total number of MSHA's samples exceed OSHA's PEL of 0.1 f/cc based on TEM analysis. This same data indicate that 10 percent of the samples exceed OSHA's PEL of 0.1 f/cc based on PCM.

MSHA's asbestos ANPRM requested information to help us determine appropriate exposure limits for the mining industry, considering the health risk and technological and economic feasibility. We specifically asked what would be an appropriate agency action considering these levels, and if OSHA's asbestos exposure limits would afford sufficient protection to miners. Most commenters supported our adoption of OSHA's exposure limits.

As discussed below in section VII.C of this preamble, we are proposing to incorporate the generic elements of PCM analytical methods for asbestos exposure monitoring by referencing Appendix A of OSHA's asbestos standard (29 CFR 1910.1001). Appendix A lists both NIOSH 7400 and OSHA ID 160 as examples of analytical methods that meet the equivalency criteria in OSHA's asbestos standard. The evaluation or inclusion of other protocols that deviate from the criteria for counting fibers in our existing standards is beyond the scope of this rulemaking.

1. Sections 56/57.5001(b)(2)(i) and 71.702(b)(1): 8-Hour Time-Weighted Average (TWA), Full-Shift Exposure Limit

Our sampling results indicate that there is not widespread overexposure to asbestos in the mining industry. Recognizing this low exposure, many industry commenters generally supported reducing the PEL for asbestos to the OSHA level of 0.1 f/cc, if MSHA also ensured that the analytical method only counted asbestos fibers. Labor representatives supported reducing the PEL for asbestos to the OSHA level of 0.1 f/cc and recommended that MSHA propose additional requirements from the OSHA asbestos standard.

Even though there was general agreement among the commenters to the ANPRM that MSHA should adopt OSHA's asbestos exposure limits, some commenters from a community association expressed concern about asbestos originating at a local mine. They seemed concerned not only with the health of miners, but also with exposures of people in relative proximity to the mining operations. They believe that any level of airborne asbestos is unacceptable.

While we are concerned about the spread of asbestos from mine sites into the atmosphere, asbestos occurs naturally in many types of soils and ore bodies. Although comments concerning the asbestos exposure of those living close to a mining operation fall outside the scope of this rule, the proposed reduction in the permissible exposure limits may reduce environmental levels as well.

We are proposing an 8-hour TWA, full-shift exposure limit of 0.1 f/cc. This limit would significantly reduce the risk of material impairment of health or functional capacity for miners exposed to asbestos.

2. Sections 56/57.5001(b)(2)(ii) and 71.702(b)(2): Excursion Limit

As previously discussed, asbestos poses a long-term health risk to exposed workers. There are no toxicological data identifying a "dose-rate"<sup>73</sup> health effect from exposure to airborne concentrations of asbestos. "Dose-rate" effect means that a specific dose can cause different health problems depending on the length of exposure. For example, asbestos does not seem to have a "dose-rate" effect because exposure to a high concentration over a short time period poses no greater risk of an adverse health effect than if the worker received the same dose at a

lower concentration over a longer time period. An excursion limit sets boundaries for peak episodes of exposure that are not based on toxicological data. We are proposing an excursion limit for asbestos to help maintain the average airborne concentration below the full-shift exposure limit. For example, the 8-hour, TWA airborne asbestos concentration would be 0.06 f/cc for miners exposed to one 30-minute excursion per day at 1.0 f/cc and 0.13 f/cc for miners exposed to two 30-minute excursions per day at 1.0 f/cc.

In the ANPRM, we requested comments on an appropriate level for a short-term exposure limit (67 FR 15134). We specifically asked whether adopting the OSHA limit of 1 f/cc over 30 minutes would afford sufficient protection to miners in light of the health risk and the technical and economic feasibility of such a limit. Commenters offered no objections to adopting OSHA's excursion limit for airborne asbestos, and some agreed that this level is appropriate.

a. *OSHA's Short-Term Exposure Limit.*

When OSHA issued its 1986 asbestos standard, it decided not to issue an explicit short-term exposure limit (STEL). OSHA stated the basis for its decision (51 FR 22709) as follows.

To summarize, OSHA is not promulgating a short-term exposure limit for asbestos because toxicological and dose-response evidence fail to show that short-term exposure to asbestos is associated with an independent or greater adverse health effect than is exposure to the corresponding 8-hour TWA level; that is, there is no evidence that exposure to asbestos results in a "dose-rate" effect. This is reflected in OSHA's risk models for lung cancer and mesothelioma, which associate health risk with cumulative dose. The decision not to promulgate a short-term exposure limit for asbestos is consistent with OSHA's recent policy decision described in the Supplemental Statement of Reasons for the Final Rule for Ethylene Oxide (50 FR 64) in which OSHA established that short-term exposure limits for toxic substances are not warranted in the absence of health evidence demonstrating a dose-rate effect.

OSHA's decision not to issue a STEL was challenged in *Public Citizen Health Research Group v. OSHA* (796 F.2d 1505), 1986. The U.S. Court of Appeals for the District of Columbia held that the Occupational Safety and Health Act compels OSHA to adopt a short-term limit, if the rulemaking record shows that it would further reduce a significant health risk and is feasible to implement, regardless of whether the record supports a "dose-rate" effect. Subsequently, OSHA found that

<sup>73</sup> OSHA (51 FR 22709), 1986.

compliance with a short-term limit would further reduce a significant health risk remaining after complying with the 8-hour TWA, full-shift exposure limit. OSHA also found that the lowest excursion level which is feasible both to measure and to achieve primarily through engineering and work practice controls is 1 f/cc measured over 30 minutes. For these reasons, in 1988, OSHA promulgated an asbestos excursion limit of 1 f/cc over a sampling period of 30 minutes (53 FR 35610).

*b. Minimum Detectable Level and Feasibility of Measuring Short-Term Excursions.*

As discussed in OSHA's 1986 asbestos final rule (51 FR 22686), the key factor in sampling precision is fiber loading. To determine whether the analytical method described in Appendix A of its asbestos standard could be used to analyze short-term samples, OSHA calculated the lowest reliable limit of quantification using the following formula:

$$C = [(f/[(n)(A_f)])(Ac)]/[(V)(1,000)]$$

where:

C is fiber concentration (in f/cc of air);

f is the total fiber count;

n is the number of microscope fields examined;

A<sub>f</sub> is the field area (0.00785 mm<sup>2</sup>) for a properly calibrated Walton-Beckett graticule;

Ac is the effective area of the filter (in mm<sup>2</sup>); and

V is the sample volume (liters).

Table VII-1 was generated from the above equation. The table shows that 1.0 f/cc measured over 30 minutes can be reliably measured when pumps are used at the higher flow rates of 1.6 Lpm or more, using the 25-mm filters.

TABLE VII-1.—RELATIONSHIP OF SAMPLING METHOD TO MEASUREMENT OF ASBESTOS

Flow rate (Lpm)	Sampling time	Lowest level reliably measured (f/cc) using 25-mm filters
2.5 .....	15 minutes ..	1.05
2.0 .....	.....	1.31
1.6 .....	.....	1.63
1.0 .....	.....	2.61
0.5 .....	.....	5.23
2.5 .....	30 minutes ..	0.51
2.0 .....	.....	0.65
1.6 .....	.....	0.82
1.0 .....	.....	1.31
0.5 .....	.....	2.61

We recognize that in some situations, such as low background dust levels, lower exposures could be measured; however, the risk of overloading the filter with debris increases when using the higher flow rates. We can be confident that we are measuring the actual airborne concentrations of asbestos, within a standard sampling and analytical error (±25 percent), when we use the minimum loading suggested by the OSHA Reference Method (29 CFR 1910.1001, Appendix A). The excursion limit of 1.0 f/cc for 30 minutes is the lowest concentration that we can measure reliably for determining compliance with the excursion limit.

Some commenters supported MSHA's adoption of OSHA's asbestos excursion limit of 1.0 f/cc for 30-minutes. Many other commenters offered no objections, choosing to remain silent on this issue. We have considered the comments and are proposing an asbestos excursion limit of 1.0 f/cc over a minimum sampling time of 30 minutes.

*C. §§ 56/57.5001(b)(3) and 71.702(c): Measurement of Airborne Fiber Concentrations*

We currently require asbestos samples to be analyzed by PCM for the initial determination of exposure and compliance with the PELs. We are

proposing to retain this requirement for PCM analysis. The proposed rule would require fiber concentration to be determined by PCM using a method statistically equivalent to the OSHA Reference Method in OSHA's asbestos standard (29 CFR 1910.1001, Appendix A).

The OIG recommended that we use TEM for the initial analysis of samples collected to evaluate a miner's personal exposure to asbestos. In our 2002 asbestos ANPRM, we requested information to help us determine the benefits and feasibility of changing our asbestos analytical method from PCM to TEM for evaluating a miner's exposure to asbestos. For the reasons discussed in this preamble, we cannot justify using a TEM analytical method for the initial determination of compliance with our asbestos PELs.

1. Brief Description and Comparison of Three Analytical Techniques

To ease understanding of the discussion that follows, this section briefly describes the three analytical techniques that MSHA has used for analyzing asbestos samples. All three techniques involve counting fibers. MSHA has used—

- *Phase contrast microscopy* (PCM) on air samples to determine a miner's exposure for comparison with our permissible exposure limits (PELs) for asbestos.
- *Transmission electron microscopy* (TEM) on the same air samples analyzed by PCM when we need to confirm the presence of asbestos and distinguish asbestos from other fibers in the sample.
- *Polarized light microscopy* (PLM) to analyze bulk samples collected from an area suspected of having asbestos in the ore or dust, not for air samples collected to determine a miner's exposure.

Table VII-2 below presents a brief summary of various features of these three analytical techniques. The values listed are approximate.

TABLE VII-2.—MSHA'S COMPARISON OF THREE ANALYTICAL TECHNIQUES<sup>74</sup> USED TO ANALYZE ASBESTOS SAMPLES

Criteria	PCM	TEM	PLM
Magnification .....	Up to 1,000X; typically 400–450X	Up to 1,000,000X; typically 10,000X.	Up to 1,000X; typically 10–45X.
Resolution .....	0.2 µm .....	0.001 µm <sup>75</sup> .....	0.2 µm.
Sample Area Examined .....	Minimum: 100 fibers & 20 fields; or 100 fields (0.157–0.785 mm <sup>2</sup> ).	100 fibers or 4.4 mm <sup>2</sup> minimum (0.06–0.4 mm <sup>2</sup> )*.	Scan entire prepared sample (1 cm <sup>2</sup> ).
Additional information .....	None .....	Crystal structure & elemental composition.	Refractive index.
Microscope cost .....	\$1,500–\$2,000 .....	\$200,000–\$300,000 .....	\$1,500–\$2,000.
Analysis cost/filter .....	\$10–\$15 .....	\$100–\$400 .....	\$10–\$15.
Analysis time/filter .....	0.25–0.5 hour .....	3–4 hours or more .....	0.25–0.5 hour.

TABLE VII-2.—MSHA'S COMPARISON OF THREE ANALYTICAL TECHNIQUES<sup>74</sup> USED TO ANALYZE ASBESTOS SAMPLES—Continued

Criteria	PCM	TEM	PLM
Degree of expertise of analysts .....	Requires a moderate level of expertise; 40 hours training minimum.	Requires a high level of expertise and experience.	Requires a moderate level of expertise; 40 hours training minimum.

\* NIOSH 7402 depends on loading: light—40 fields; medium—40 fields or 100 fibers; heavy—6 fields and 100 fibers.

## 2. Fiber Identification Using Transmission Electron Microscopy (TEM)

### a. *Advantages and Disadvantages of TEM Analysis*

The transmission electron microscope (TEM), equipped with an energy dispersive x-ray spectrometer (EDS) and using selected area electron diffraction (SAED) is generally capable of identifying the mineralogy of individual asbestos fibers. Even so, TEM does not always have sufficient precision to make definitive distinctions between closely related minerals, such as between winchite

$[(\text{NaCa})\text{Mg}_4(\text{Al}, \text{Fe}^{3+})\text{Si}_8\text{O}_{22}(\text{OH})_2]$  and tremolite  $[\text{Ca}_2\text{Mg}_5\text{Si}_8\text{O}_{22}(\text{OH})_2]$ .<sup>76</sup>

Because electron microscopes provide greater magnification and greater image clarity, including sharper three-dimensional images than light microscopes, TEM can detect fibers that are undetectable using PCM. Routine use of TEM analysis, however, would have some significant disadvantages.

- Epidemiological data correlating TEM asbestos exposure levels with asbestos-related diseases is not available for conducting a new risk assessment.

- TEM analysis is time consuming and expensive, requiring highly skilled personnel for instrument operation and data interpretation, especially when applied as the primary analytical method.

- Few facilities offer TEM analysis for asbestos air samples collected in a mining environment.

Another disadvantage of TEM is that it uses an even smaller amount of sample than is used in PLM or PCM analysis. Asbestos fibers may not be present in the small portion of sample examined under the electron microscope, even when it is present in the larger sample examined by PLM or PCM. Despite its disadvantages, TEM allows us to better identify asbestos minerals in air samples collected in a mine.

### b. *Use of TEM to Determine Compliance with MSHA's PELs.*

<sup>74</sup> MSHA's summary of its literature reviews and experience.

<sup>75</sup> Clark, p. 5, 1977.

<sup>76</sup> Leake *et al.*, 1997.

The OIG recommended that MSHA use TEM for its initial analysis to determine if an asbestos sample is over the PEL. MSHA believes that analyzing an airborne dust sample from a mine, which might contain asbestos, requires additional expertise not readily developed through experience analyzing samples known to contain asbestos. We recognize that EPA routinely uses TEM for the analysis of air samples collected for asbestos abatement under the Asbestos Hazard Emergency Response Act (AHERA) and requires the use of TEM to characterize workers' asbestos exposures (40 CFR part 763). MSHA currently uses TEM on a limited basis, when necessary, to verify the presence of asbestos in samples. These samples often contain few fibers among much dust and a variety of other interferences.

In the ANPRM, we requested comments on the use of TEM including cost, availability, comparisons of PCM to TEM, and a possible relationship of TEM to a PEL. In response to the ANPRM, some commenters suggested that MSHA use TEM to augment PCM measurements. Overall, industry commenters did not recommend the use of TEM for the initial analysis of fiber samples for comparison to the PELs. Commenters did not dispute additional, confirmatory analysis of samples that show possible exposure to asbestos in excess of the PELs. NIOSH also did not believe that TEM should be used for routine monitoring even though they consider TEM a valuable tool in mineral identification. NIOSH comments stated the reasons for not using TEM as the primary method for determining compliance with the PELs as (i) the lack of health risk data associated with TEM, (ii) the level of expertise required, and (iii) the high cost.

#### (i) *Lack of Health Risk Data Based on TEM.*

OSHA did not use analytical results based on TEM in its original risk assessment for asbestos. Although attempts have been made,<sup>77</sup> researchers have not reported a strong, consistent correlation between PCM and TEM analyses. The relationships that are reported are specific to the fiber type

<sup>77</sup> Snyder *et al.*, 1987.

and environment sampled.<sup>78</sup> To set a meaningful permissible exposure limit based on TEM analysis, we must have either—

- Peer-reviewed epidemiology or toxicology studies relating TEM analysis and adverse health effects, or
- A predictive relationship correlating TEM and PCM for samples collected in a mining environment.

#### (ii) *Level of Expertise.*

One commenter representing an industry association at MSHA's public hearing in Charlottesville, Virginia (2002) testified that TEM was not a method for routine monitoring. This commenter also pointed out—

\* \* \*that very few commercial TEM labs are competent to perform valid analyses of the complicated mineralogical mixtures that you find in mining and quarrying operations.

Another commenter at the Charlottesville public hearing testified that TEM is fallible. This commenter said that electron diffraction patterns for structurally similar minerals can be difficult to distinguish from one another. Each particle in the sample may be of a different composition and the analyst cannot assume that every particle with the same shape is the same mineral.

#### (iii) *High Cost of TEM Analysis.*

Several commenters representing an industry association each commented on the high cost of TEM analysis. One commenter stated that, because the variability of the measurement increases at the lower concentrations, when the PEL is lowered it is important to increase the frequency of monitoring and, therefore, the cost of sample analysis becomes an issue.

## 3. Phase Contrast Microscopy (PCM) for the Analysis of Personal Exposure Samples

The use of PCM for quantitative analysis of samples does not differentiate between mineral species. There is industry concern that misidentification of fibers as asbestos can lead to incorrect conclusions, resulting in unnecessary expenses for mining companies. PCM counting schemes address the key problem of

<sup>78</sup> Verma and Clark, 1995.

needing to make a relatively fast, cost-effective evaluation of a situation in a mine so as to protect miners from danger to their health. PCM maintains the integrity, meaning, and usefulness of the analytical method for evaluating samples relative to the historic health data.<sup>79</sup>

*a. Discussion of Microscope Properties.*

One issue commenters mentioned repeatedly concerning PCM is the limited resolution and magnification of light microscopes compared to electron microscopes.

*(i) Resolution.*

The resolution of the microscope is the smallest separation between two objects that will allow them to be distinctly visible. The higher the resolving power of a microscope, the smaller the distance can be between two particles and have them still appear as two distinct particles. Resolution is about 0.22  $\mu\text{m}$  using PCM and 0.00025  $\mu\text{m}$  using TEM. This means that where the analyst sees a single fiber using PCM, that same analyst might see a number of thinner fibers using TEM.

*(ii) Magnification.*

The level of magnification is another PCM microscopy issue. Magnification is the ratio of the size that the object appears under the microscope to its actual size. PCM analytical methods specify a magnification of 400 to 450 times ( $\times$ ) the object's actual size. The magnification using TEM can be 10,000X to 1,000,000X. This means that the analyst sees a smaller amount of the sample using TEM than when using PCM.

*b. Health Risk Data Based on PCM.*

Historically, asbestos samples have been analyzed by mass (weighing), counting (microscopy), or a qualitative property (spectroscopy). When recommending an exposure standard for chrysotile asbestos, the British Occupational Hygiene Society contended<sup>80</sup> that the microscopic counting of particles greater than 5  $\mu\text{m}$  in length would show a relationship with the prevalence of asbestosis similar to those based on the mass of respirable asbestos. Many scientific papers have suggested that counting only fibers longer than 5  $\mu\text{m}$  would minimize variations between microscopic techniques<sup>81</sup> and improve the precision of the results.<sup>82</sup> Nonetheless, this criterion was accepted as an index of exposure, even though some believed that, due to their possible health effects,

the smaller fibers should not be excluded.<sup>83</sup>

In recommending an asbestos standard in 1972, NIOSH suggested using the same size criteria that the British adopted. They also recommended reevaluating these criteria when more definitive information on the biologic response and precise epidemiologic data were developed. When exposure data were not obtained using PCM, NIOSH applied a conversion factor to the non-PCM data to estimate PCM concentrations for use as the basis of a recommended permissible occupational exposure level.

A number of commenters testified (Charlottesville, 2002) that PCM methodology includes more than asbestos when determining fiber concentration in air. The commenters suggested that the lower risk seen in epidemiological studies relating PCM to adverse health outcomes in miners was possibly due to the background material inherent in air samples taken in a mining environment. They speculated that the background material had been counted and included in the estimated asbestos concentrations. This may have overestimated exposures and resulted in a dilution of the dose-response relationship presented in scientific publications.

*c. Subjectivity and Consistency of Counting Asbestos Fibers*

The fiber count obtained using the PCM method is dependent on several factors. These factors include the analyst's interpretation of the counting rules, the analyst's visual acuity, the optical performance of the microscope, and the optical properties of the prepared sample.<sup>84</sup> Much of the variability is attributed to the ability of the analyst to observe and size fibers.

The American Industrial Hygiene Association (AIHA) Proficiency Analytical Testing Program (PAT), operated in cooperation with NIOSH, maintains a database for historical data relating to asbestos fiber counting using PCM. This program, begun in 1972, provides statistical evaluation of laboratory performance on test samples. At its inception in 1968, the method used by laboratories participating in this program was the U.S. Public Health Service method (USPHS 68).<sup>85</sup> The counting rules for this method were vague and required little microscope standardization.

Work has been done to modify the PCM method to address these

consistency issues.<sup>86</sup> Commenters to our asbestos ANPRM suggested that we consider thoracic sampling to minimize interference from large particles. Testimony at MSHA's public hearing in Charlottesville (2002) presented a counting technique based on the typical characteristics of asbestos in air. Another commenter stated that several approaches have been tried to remove non-asbestos minerals from samples, such as low temperature ashing or dissolution, but they would not be useful for mining samples. Another commenter suggested using a higher aspect ratio to increase the probability that the structures counted are fibers. Several commenters suggested the development of a new analytical method.

Overall, commenters recognized that it takes far less time to develop expertise in counting fibers using PCM than in developing expertise using TEM. NIOSH has developed a 40-hour training course for teaching analysts to count asbestos fibers.

The availability of analyst training courses, and the formation of accreditation bodies requiring laboratory quality assurance programs, helps minimize the variations in measurements between and within laboratories. Accreditation bodies require laboratories to use standardized analytical methods. AIHA also has the Asbestos Analyst Registry that specifies criteria for competence, education, and performance for analysts. In addition to these programs, our incorporation of OSHA's Appendix A would help minimize the subjectivity and increase consistency of measuring airborne asbestos concentrations by specifying core elements of acceptable analytical PCM methods.

**4. MSHA's Incorporation of OSHA's Appendix A**

Commenters generally supported the use of PCM for the initial analysis of fiber samples for determining compliance with the PELs. Commenters' major concerns focused on fiber counting procedures. Commenters suggested that differential counting techniques be developed to analyze air samples for asbestos using PCM and taking into consideration the fiber morphology and the distributions or populations of distinct fiber groups with characteristic dimensions. Other commenters stated that particle characteristics could not reliably be used to differentiate fibers from cleavage fragments when examining relatively small numbers of fibers.

<sup>79</sup> Wylie *et al.*, 1985.

<sup>80</sup> Lane *et al.*, 1968.

<sup>81</sup> ACGIH-AIHA, 1975.

<sup>82</sup> Wylie, 2000.

<sup>83</sup> ACGIH-AIHA, 1975; NIOSH, 1972.

<sup>84</sup> Rooker *et al.*, 1982.

<sup>85</sup> Schlecht and Shulman, 1995.

<sup>86</sup> Pang, 2000; Harper and Bartolucci, 2003.

In this rulemaking, we propose to continue to use PCM to determine asbestos concentrations. PCM was used in the development of past exposure assessments and risk estimates and is relatively quick and cost-effective. Thus, with respect to analytical methods, this proposed rule is not substantively different than our existing standards. We also have added language to allow for our acceptance of other asbestos analytical methods that are at least as effective in identifying potential overexposures.

The OSHA Reference Method, mandatory Appendix A to the OSHA asbestos standard (29 CFR 1910.1001), specifies the elements of an acceptable analytical method for asbestos and the quality control procedures that laboratories performing the analysis must implement. Paragraph (d)(6)(iii) of OSHA's asbestos standard (29 CFR 1910.1001) requires employers, who must monitor for asbestos exposure, to use a method for collecting and analyzing samples that is equivalent to the OSHA Reference Method (ORM), and also describes the criteria for equivalency. For the purpose of this proposed rule, MSHA would consider a method equivalent if it meets the following criteria:

[from 29 CFR 1910.1001(d)(6)(iii)]

(A) Replicate exposure data used to establish equivalency are collected in side-by-side field and laboratory comparisons; and

(B) The comparison indicates that 90% of the samples collected in the range 0.5 to 2.0 times the permissible limit have an accuracy range of plus or minus 25 percent of the ORM results at a 95% confidence level as demonstrated by a statistically valid protocol; and

(C) The equivalent method is documented and the results of the comparison testing are maintained.

Appendix A of OSHA's asbestos standard lists NIOSH 7400 and OSHA ID-160 as examples of analytical methods that meet these criteria. In addition, there are other PCM analytical methods for asbestos:

- The Asbestos International Association (AIA), AIA RTM1, "Airborne Asbestos Fiber Concentrations at Workplaces by Light Microscopy (Membrane Filter Method)."
- The International Organization for Standardization (ISO), ISO 8672:1993(E), "Air quality—Determination of the number concentration of airborne inorganic fibres by phase contrast microscopy—Membrane filter method."

MSHA recognizes that there are advantages and disadvantages of various PCM analytical methods, especially as

they relate to the processing of samples collected in a mining environment. For example, the ASTM dilution method (D 5755-95) for overloaded samples has allowed laboratories to recover useable results from airborne exposure samples that, in the past, had been invalidated. We note that both ASTM and the National Stone Sand and Gravel Association are pursuing the development of an analytical method for asbestos in mining samples. We would consider analytical methods that afford a better measurement alternative as they become available. We believe that allowing statistically equivalent analytical methods would remove barriers to innovation and technological advancement.

We specifically request information on additional criteria for equivalency for use in evaluating alternative analytical methods for the determination of asbestos in air samples collected in a mining environment. We also request information about analytical methods for which equivalency has already been demonstrated.

#### 5. MSHA Asbestos Control Program

In the ANPRM, we asked whether or not our current sampling methods met the needs of the mining community and how mineral dust interferences could be removed from mining samples. The ANPRM also asked for comments on other ways to reduce miners' exposures, such as increased awareness of potential asbestos hazards at the mine site and the provision of adequate protection. We also asked for suggestions on what educational and technical assistance MSHA could provide and what other factors, circumstances, or measures we should consider when engineering controls are unable to reduce asbestos exposure below the PEL.

We received some criticism concerning our sampling and analysis procedures from a few commenters who believed that we should develop specific test procedures for the sampling and analysis of bulk samples for the mining environment, as well as specific air sampling procedures (including pump flow rates, cassette types, and filter matrix). They also believed that we should improve our reports by including inspection field notes, location, purpose, and procedure followed, as well as descriptions of the accuracy, meaning, and limitations of the results. In its comments to the ANPRM, one trade association recommended that we maintain our current, established asbestos monitoring protocols with emphasis on full-shift monitoring for comparison to the PEL. Another trade association stated that our

current field sampling methods are adequate for most mines and quarries, particularly when no significant amount of asbestos is found. They also suggested that respirable dust sampling using a cyclone might be a means to remove interfering dust from the sample. NIOSH suggested that we could use thoracic samplers, but that studies performed on their use did not include mines and further positive test results would be needed before they could promote their use in mining.

We believe that our current sampling procedures are adequate and we are proposing to continue using them. Our current procedures, which we updated in 2000, specify using several, typically three, 25-mm filter-cassettes in series to collect a full-shift sample. Depending on the amount of visible dust in the air, these procedures allow the setting of pump flow rates to optimize fiber loading and minimize or eliminate mixed dust overload. We are not considering the use of a cyclone to capture respirable dust because research indicates that larger durable fibers also could cause adverse health effects.

#### 6. Bulk Sample Analysis Using Polarized Light Microscopy (PLM)

In the ANPRM, we asked what method was most appropriate for MSHA to use to analyze bulk samples for asbestos in the mining industry. The presence of asbestos in a bulk sample does not mean that it poses a hazard. The asbestos must become airborne and be respirable, or contaminate food or water, to pose a health hazard to miners. The detection of asbestos in a bulk sample serves to alert mine operators, miners, and MSHA to the possible presence of asbestos. One mining association stated that air monitoring is not the preferred scheme to screen for possible asbestos exposure. They believe, and we agree, that knowledge of the geology of asbestos and identification of asbestos in bulk samples may be a useful step in determining whether asbestos is present in the ore or host rock.

We are not proposing to use bulk samples to determine asbestos exposures in mining. We are requesting comments on whether MSHA's use of routine, periodic bulk sampling would be useful in determining whether or not we should take personal exposure air samples to evaluate miners' exposures to asbestos at mines suspected of having naturally occurring asbestos.

MSHA also uses the detection of asbestos in bulk samples as a trigger for its compliance assistance activities. We have trained MSHA inspectors on ways to identify asbestos in the ore and

surrounding rock formations at mines and to pass this information on to mine operators. Analysis of samples of accumulated settled dust from a mill or construction debris can identify areas or activities that would require special precautions. After considering the results of the bulk sample analysis, together with its strengths and weaknesses, the mine operator, miners, and MSHA can take appropriate action to reduce the risk of exposure, which would help reduce the risk of asbestos-related diseases among miners.

Analysis of bulk samples is usually performed using PLM. Commenters to the ANPRM expressed concern that the PLM analysis may not detect asbestos when it is present. A particle must be at least 0.5  $\mu\text{m}$  in diameter to refract light and many asbestos fibers are too thin to refract light. Asbestos may be a small percentage of the parent material or not uniformly dispersed in the sample and, therefore, may not be seen in the small portion of sample that is examined under the microscope. In addition, the method could detect asbestos erroneously because a nonasbestiform mineral could have a refractive index similar to one of the asbestos minerals. Another problem with identifying asbestos using PLM is that all varieties of a mineral show the same refractive index. For example, even an experienced analyst might not differentiate between the asbestiform and nonasbestiform varieties of a mineral based on their refractive indices.

Although a trained individual may be able to identify bulk asbestos by its appearance and physical properties, the identification can be more difficult when the asbestos is dispersed in a dust sample or is present in low concentration in a rock. A commenter at MSHA's hearing in Charlottesville (2002) testified that none of the existing methods for bulk sample analysis (EPA, NIOSH, ASTM) were designed for complex mine environments.

#### *D. Discussion of Asbestos Take-Home Contamination*

This proposed rule does not include standards to address asbestos take-home contamination. We recognize the important role of take-home exposures in contributing to asbestos disease of workers and their family members. We believe that a combination of enforcement and compliance assistance activities, together with increased education and training of mine inspectors, mine operators, and miners, coupled with the lowering of the PELs, would be effective in preventing asbestos take-home contamination.

Mine operators are encouraged to measure the potential for take-home contamination and provide protective measures where necessary to minimize secondary take-home exposures.

#### 1. MSHA's Request for Information

MSHA's ANPRM for measuring and controlling asbestos exposures at mines included requests for information and data to help us evaluate what we could do to eliminate or minimize take-home contamination. We asked how and/or should MSHA be addressing take-home contamination. We also asked about provisions for the special needs of small mine operators and what assistance (e.g., step-by-step instructions, model programs, certification of private programs) we could provide. We also requested information on the types of protective clothing miners currently use when working in areas where asbestos may be present, and the types of preventive measures currently in use when miners leave the area, to prevent the spread of asbestos exposure.

#### 2. Commenters' Responses to the Take-Home Contamination Issue in MSHA's Asbestos ANPRM

Commenters expressed concern that we would apply the requirements in OSHA's and EPA's standards to trace levels of fibrous mineral exposures at mines, pits, and quarries. Many industry commenters urged MSHA to limit protective measures for take-home contamination to those activities involving known asbestos and asbestos-containing products, such as those regulated by OSHA and EPA. For example, commenters suggested that MSHA adopt appropriate provisions from the OSHA asbestos standard for construction workers, for asbestos abatement workers, and for those miners whose exposures exceed MSHA's PEL.

Commenters cautioned MSHA to be mindful of the definitions of asbestos when analyzing samples to determine compliance. They also urged MSHA to acknowledge the presence of interferences in mining samples, as well as the differences between nonasbestiform amphiboles and their asbestos analogues. Some commenters cautioned that, unless MSHA constructed the provisions for reducing take-home contamination carefully, the consequences for the mining industry might be costly with little or no benefit to miners.

NIOSH encouraged MSHA to adopt measures included in its 1995 Report to Congress on their *Workers' Home Contamination Study Conducted under the Workers' Family Protection Act*. Labor participants also supported

protective measures, such as personal protective equipment and showers before leaving work, to prevent take-home contamination.

#### 3. MSHA's Considerations in Making Its Decision To Use Non-Regulatory Methods To Address the Hazard From Take-Home Contamination

In determining an appropriate proposed action for preventing take-home contamination, we considered the comments to the ANPRM, OSHA's and EPA's requirements, and the recommendations of NIOSH and the OIG. We based our determination to propose to address asbestos take-home contamination through non-regulatory measures on the following factors:

- Existing standards requiring engineering controls for airborne contaminants, respiratory protection, personal protective clothing, hazard communication, and housekeeping, together with a lower PEL, would provide sufficient enforcement authority to assure that mine operators take adequate measures when necessary to prevent asbestos take-home contamination.

- There are no asbestos mines or mills currently operating in this country and different ore bodies of the same commodity, such as vermiculite mining, are not consistent in the presence, amount, or dispersion of asbestiform minerals. Currently, asbestos exposures in mining are low. As discussed in section V.D.2 of this preamble, only two of the 123 mines sampled for asbestos in the ore show personal asbestos exposures exceeding 0.1 f/cc. This is less than 2 percent of the sampled mines.

- Some mines with asbestos minerals in the ore or host rock have implemented protective measures voluntarily. MSHA experience in the recent past indicates that mine operators and mining companies are increasingly aware of asbestos hazards and have been willing to cooperate with MSHA to eliminate this hazard.

- The measures taken to prevent take-home contamination are varied, and mine operators would have the freedom to eliminate this hazard in a manner based on site-specific exposure measurements and the nature of the asbestos exposures at the mine. For example, mine operators could minimize or prevent asbestos take-home contamination by providing disposable coveralls or on-site shower facilities coupled with clothing changes.

#### 4. MSHA's Activities for Eliminating the Risk of Asbestos Take-Home Contamination

We believe that mine operators and miners would take action to eliminate any possible recurrence of a disaster, such as that in Libby, Montana, if they understand the hazards and ways to minimize the risk. To that end, we are placing special emphasis on the potential hazard from asbestos take-home contamination in our enforcement, compliance assistance, and educational activities as follows.

##### a. Enforcement Activities.

- Enforce the new, lower PELs when they become effective.

- Continue enforcement of standards applicable to providing special protective equipment and clothing whenever environmental hazards are encountered in a manner capable of causing injury or impairment, *e.g.*, § 56.15006.

- Ensure that mine operators provide miners, who are at risk of being exposed, with information about the signs, symptoms, and risk for developing asbestos-related illness as required by the hazard communication standard.

##### b. Compliance Assistance.

- Continue to monitor targeted mines for the presence of asbestos.

- Encourage mine operators to comply with OSHA's asbestos standard, or hire professionals skilled and certified in working with asbestos, when they engage in construction, demolition, or renovation activities at the mine.

- Issue an updated *Program Information Bulletin (PIB)* on asbestos to include a greater emphasis on protective measures to reduce take-home contamination. We expect distribution this year.

##### c. Educational Activities.

- Continue outreach to mine operators through training courses, informational materials, and topical local meetings.

- Issue an updated *Health Hazard Information Card* for miners this year to increase miners' awareness of the hazards of take-home contamination from asbestos or other asbestiform minerals and to suggest measures that the miners can take to prevent it.

- Continue specialized asbestos hazard and sampling training for mine inspectors.

#### E. Section 71.701(c) and (d): Sampling; General Requirements [Controlling Asbestos Exposures in Coal Mines]

For surface coal mines and surface worksites at underground coal mines, we are proposing to add a reference to

§ 71.702 (the asbestos standard for coal mines) in paragraphs (c) and (d) of § 71.701, which contain the requirements for controls and sampling. The existing language in § 71.701(c) and (d) references the Threshold Limit Values (TLVs®) and excursion limits in § 71.700, but not the asbestos exposure limits in § 71.702. MSHA regulations currently require mine operators to control miners' exposures to airborne contaminants and to sample for airborne contaminants, as necessary, to determine when and where such controls may be needed. In developing this proposed rule, we determined that § 71.701 was unclear as to its applicability to asbestos exposures. This proposed rule would clarify our intent that coal mine operators control miners' exposures to asbestos.

### VIII. Regulatory Analyses

#### A. Executive Order (E.O.) 12866

In our ANPRM on asbestos exposure, we specifically requested information, data, and comments on the costs and benefits of an asbestos rule, including what engineering controls and personal protective equipment are being used to protect miners from exposure to asbestos and to prevent take-home contamination. Considering the public comments, and MSHA data and experience, we assessed both the costs and benefits of this proposed rule in accordance with Executive Order 12866. The following sections summarize the analysis of benefits and costs presented in the Preliminary Regulatory Economic Analysis (PREA) for this proposed rule. The PREA contains a full disclosure of our methodology and the basis for our estimates.

##### 1. Discussion of Benefits

The benefits of a rulemaking addressing measurement and control of asbestos would be the reduction or elimination of diseases arising from exposure to asbestos. Exposure to airborne asbestos can cause the development of lung cancer, mesothelioma, gastrointestinal cancer, and asbestosis. Other associated adverse health effects include cancers of the larynx, pharynx, and kidneys. A person with an asbestos-related disease suffers material impairment of health or functional capacity.

##### a. Summary of Benefits.

We estimate that between 1 and 19 deaths could be avoided during the next 65 years by lowering the 8-hour TWA, full-shift exposure limit from 2.0 f/cc to 0.1 f/cc. This equates to a reduction of between 9 and 84 percent of occupationally related deaths caused by

asbestos exposures. Additional deaths would be avoided by decreasing miners' exposures to short-term bursts of airborne asbestos undetectable by the proposed 8-hour TWA, full-shift exposure limit. We estimate that lowering the excursion limit from 10 f/cc over 15 minutes to 1 f/cc over 30 minutes would reduce the risk of death from lung cancer, mesothelioma, or gastrointestinal cancer by 1 additional avoidable death for every 1,000 miners exposed to asbestos at the proposed PELs.

We are aware that lowering our PELs would not completely eliminate the risk of asbestos-related material impairment of health or functional capacity. We expect some additional risk reduction from mine operators' management directives to avoid disturbing asbestos on mine property.

##### b. Calculation of Deaths Avoided.

The benefits resulting from the lowered PELs depend on several factors including—

- Existing and projected exposure levels,
- Age of the miner at first exposure,
- Number of workers exposed, and
- Risk associated with each exposure level.

We estimate the number of miners currently exposed and their level of exposure from personal exposure information gathered during our inspections between January 2000 and December 2003. These data are available on our Web site at <http://www.msha.gov>. Section V of this preamble contains the characterization and assessment of exposures in mining.

Laboratory results indicate that exposure concentrations are unevenly distributed across mines and miners. We use four fiber concentration levels to estimate the risk to miners. The break points for these exposure levels are the proposed and existing exposure limits. Observations show that 90 percent of the sampling results are below 0.1 f/cc.

To estimate the expected number of asbestos-related deaths, we applied OSHA's linear, no-threshold, dose-response risk assessment model to our existing and proposed PELs. The upper exposure limit is 10 f/cc because the range of information derived from the epidemiological studies used to determine the dose-response relationship in OSHA's quantitative risk assessment does not include higher levels. The expected reduction of deaths resulting from lowering the PELs would

be the differences between the expected deaths at each exposure level.<sup>87</sup>

OSHA estimated cancer mortality for workers exposed to asbestos and published these data in their 1986 final rule (51 FR 22644). We discuss OSHA's asbestos risk assessment in section VI of this preamble and have reproduced OSHA's mortality data in Table VI-4.

c. Benefit of the Proposed 0.1 f/cc 8-hour TWA, Full-Shift Exposure Limit.

The current deaths from lung cancer, mesotheliomas, gastrointestinal cancer, and asbestosis are the result of past exposures to much higher air concentrations of asbestos than those found in mines today. The risks of these diseases still exist, however, and these risks are significant for miners exposed to lower air concentrations of asbestos. Most diseases resulting from a current asbestos exposure may not become evident for another 20 to 30 years. Most likely, the full benefits will occur over a 65-year period following implementation of the lower PELs. The rate at which the incidence of the cancers decreases depends on several factors including—

- Latency of onset of cancer,
- Attrition of the mining workforce,
- Changing rates of competing causes of death,
- Dynamics of other risk factors,
- Changes in life expectancy, and
- Advances in cancer treatments.

It is not possible to quantify accurately the complete dynamics of this process.

Supplemental examination of MSHA's personal exposure samples using TEM analysis indicates that not all fibers counted by PCM are the currently regulated asbestos minerals. This is especially true for operations mining and processing wollastonite. We distinguish between different mineralogical fibers using TEM and

combine this supplemental information with PCM information to calculate our lower estimate of benefits.

We estimate that there would be from 0.5 to 13.1 lung cancer deaths avoided, 0.2 to 4.4 mesothelioma deaths avoided, and 0.1 to 1.3 gastrointestinal cancer deaths avoided. The total number of cancer deaths avoided by this rule would be the sum of cancer deaths avoided at all the mines included in the exposure data, that is, the mines we have sampled. Based on the best available information, we expect a reduction of between 1 and 19 deaths avoided due to lowering the 8-hour TWA PEL to 0.1 f/cc.

d. *Benefits of the Proposed 1.0 f/cc Excursion Limit.*

We are proposing an asbestos excursion limit of 1.0 f/cc as measured over a 30-minute period for metal and nonmetal miners and coal miners working at surface work areas. We intend that the excursion limit protect miners from the adverse health risks associated with brief fiber-releasing episodes. We anticipate that some mining operations will be subject to brief fiber-releasing episodes even after lowering airborne asbestos concentrations to the 8-hour TWA, full-shift exposure limit. We have insufficient data, however, to obtain a meaningful estimate of the frequency of these episodes, the actual exposure concentrations, or the numbers of miners exposed. Miners may encounter brief fiber-releasing episodes from exposure to commercial asbestos in asbestos-containing building materials (ACBM) or as settled dust containing asbestos; while working on equipment that may have asbestos-containing parts; and while drilling, dozing, blasting, or roof bolting in areas of naturally occurring asbestos.

Because we have little information from short-term exposure measurements, we estimate the benefit

of an excursion limit from the difference in concentration between the 8-hour TWA, full-shift exposure limit (0.1 f/cc) and the excursion limit averaged over the full shift  $[(1 \text{ f/cc}) / (16 \text{ 30-minute periods}) = 0.063 \text{ f/cc}]$ . The lifetime risk associated with an exposure to 0.1 f/cc from either of the three types of cancer is 0.00336, if first exposed at age 25 and exposure continues every work day at that level for a duration of 45 years. The risk associated with exposure to 0.063 f/cc using the same age and duration of exposure is 0.00212. The difference in lifetime risk is 0.00124. This risk equates to 1.24 additional deaths avoided for every 1,000 miners exposed to asbestos at a concentration afforded by the proposed excursion limit.

e. *Further Consideration of Benefits.*

We believe that the pressure of public scrutiny and government intervention has prompted mine operators to take precautionary measures to limit miners' exposures to asbestos. If public pressures were to subside, and we did not have a regulation limiting exposures to 0.1 f/cc over an 8-hour shift, we would not have a means to enforce the same level of protection provided in other industries.

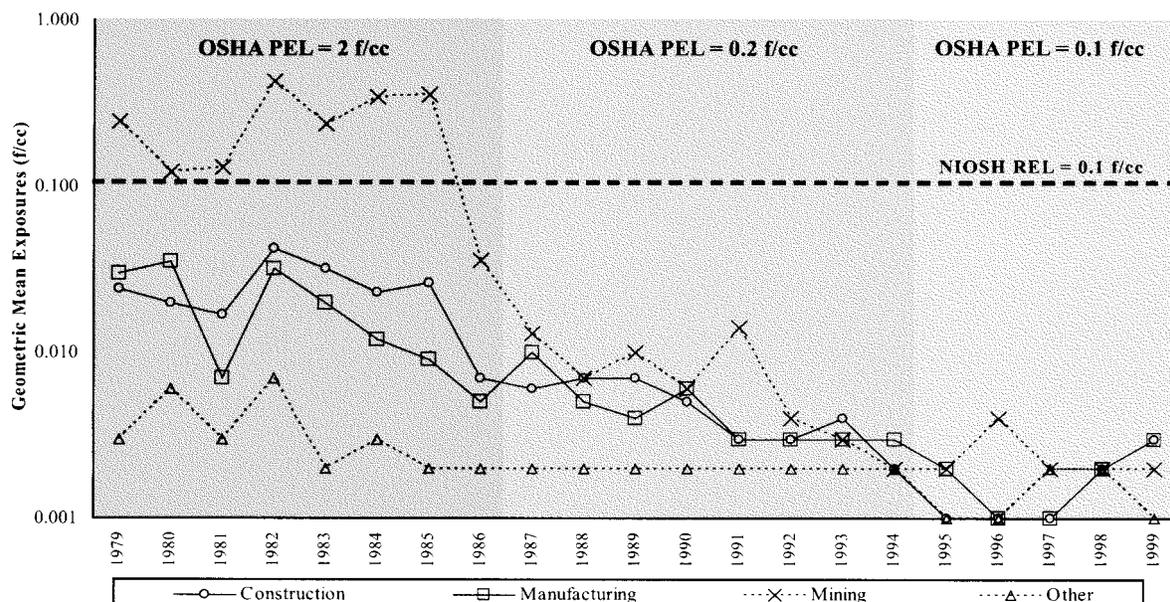
Enforcement of the lower PELs together with the direct support from the federal government in education, identification, and elimination of the asbestos hazard would increase awareness and attention to the presence of asbestos on mine property. These activities also would help focus efforts on preventing exposures, thus providing miners with added health benefits. As seen in Chart VIII-1, mining operations with ore containing naturally occurring asbestos seem to have reduced miners' exposures, perhaps due to their awareness of the lower exposure limits OSHA promulgated in 1986.<sup>88</sup>

<sup>87</sup> Nicholson, 1983; JRB Associates, 1983; OSHA (51 FR 22612), 1986; OSHA (53 FR 35609), 1988; OSHA (59 FR 40964), 1994.

<sup>88</sup> NIOSH WoRLD pp. 16-17 and 19-23, 2003.

<sup>89</sup> NIOSH WoRLD, 2003.

Chart VIII-1: Industry Trends of Airborne Asbestos Concentrations.<sup>89</sup>



The estimates of the cancer deaths avoided by reducing the PELs understate the total amount of benefit gained from this rule. These benefits do not include the reduced incidence of asbestosis-related disabilities. Asbestosis cases often lead to tremendous societal costs in terms of health care utilization, loss of worker productivity, and a decrease in the quality of life of the affected individual. Similarly, MSHA's analysis does not quantify benefits among groups incidentally exposed, such as miners' family members. We note that several

published articles document and discuss the health effects resulting from exposure to asbestos incident to living with a miner.<sup>90</sup>

This analysis overstates health benefits to the extent that we do not account for differential risks posed by different types of fibers as identified by PCM, and differences in the cancer mortality risk for asbestos-exposed workers who smoke and those who do not.

2. Discussion of Costs

The proposed rule would result in total yearly costs of about \$136,100. The

cost would be about \$91,500 per year for metal and nonmetal mines and about \$44,600 per year for coal mines. These costs represent less than 0.001 percent of the yearly revenues of \$38.0 billion for the metal and nonmetal mining industry and \$10.1 billion for the surface coal mining industry.

Table VIII-1 presents our estimate of the total yearly compliance costs by compliance strategy and mine size. The total costs reported are projected costs, in 2002 dollars, based on our knowledge, experience, and available information.

TABLE VIII-1.—SUMMARY OF YEARLY COMPLIANCE COSTS

Metal and nonmetal mine size	Compliance strategy				Total for metal and nonmetal mines
	Selective mining	Wet methods	Mill ventilation	Removal of introduced asbestos	
Small (<20) .....	\$1,058	\$1,235	\$747	\$1,750	\$4,790
Large (20-500) .....	4,922	8,614	12,916	21,000	47,452
Large (>500) .....	1,641	2,871	19,001	15,750	39,264
<b>Total .....</b>	<b>7,622</b>	<b>12,721</b>	<b>32,664</b>	<b>38,500</b>	<b>91,506</b>
Coal mine size	Compliance strategy				Total for coal mines
	Selective mining	Wet methods	Mill ventilation	Removal of introduced asbestos	
Small (<20) .....	.....	.....	.....	\$875	\$875
Large (20-500) .....	.....	.....	.....	12,250	12,250
Large (>500) .....	.....	.....	.....	31,500	31,500

<sup>90</sup> NIOSH Publication No. 2002-113, May 2002.

Coal mine size	Compliance strategy				Total for coal mines
	Selective mining	Wet methods	Mill ventilation	Removal of introduced asbestos	
Total .....	.....	.....	.....	44,625	44,625

**B. Feasibility**

MSHA has concluded that the requirements of this proposed rule would be both technologically and economically feasible. This proposed rule is not a technology-forcing standard and does not involve activities on the frontiers of scientific knowledge. All devices that would be required by the proposed rule are already available in the marketplace and have been used in either the United States or the international mining community. We have concluded, therefore, that this proposed rule is technologically feasible.

As previously estimated, the mining industry would incur costs of about \$136,100 yearly to comply with this proposed rule. These compliance costs represent well less than 0.001 percent of the yearly revenues of the mines covered by this rule, thus providing convincing evidence that the proposed rule is economically feasible.

**C. Alternatives Considered**

In our discussion of PELs in section VII.B of this preamble, we recognize that there is a remaining residual risk of adverse health effects for miners exposed at the proposed asbestos 8-hour TWA PEL. We considered proposing a lower PEL as a regulatory alternative to further reduce the risk of adverse health effects from a working lifetime of exposure. Assuming 0.05 f/cc, for example, and interpolating the data from OSHA's risk assessment summarized in Table VI-4 of this preamble, there would be about 1.68 cancer deaths per 1,000 miners exposed to asbestos at 0.05 f/cc for 45 years. The 1.68 cancer mortality rate is 50 percent less than the rate of 3.36 cancer deaths per 1,000 exposed miners calculated for the proposed 0.1 f/cc PEL; and about 97 percent less than we estimate for our existing standard (64.12 cancer deaths per 1,000 exposed miners). We also project that reducing miner's exposure to an 8-hour TWA of 0.05 f/cc would reduce the expected cases of asbestosis to about 50 percent less than at the proposed 8-hour TWA PEL.

About 85 percent of the 123 sampled mines are already well in compliance with the 0.1 f/cc proposed PEL. We believe that, theoretically, almost all of the mining industry could be in

compliance with a lower alternative PEL (0.05 f/cc 8-hour TWA). However, we cannot enforce an 8-hour TWA limit below 0.1 f/cc. The diversity of airborne particles prevalent in mining environments can interfere with sample analysis. Our existing standardized sampling techniques minimize interferences, but also impose limitations of accuracy below concentrations of 0.1 f/cc. We address these limitations in more detail in Chapter III of the PREA that accompanies this proposed rule. These accuracy issues make it infeasible for us to enforce a concentration lower than 0.1 f/cc airborne asbestos.

Although TEM provides greater characterization of asbestos fibers than PCM methodology, there is no predictable relationship between PCM and TEM measures of exposure using either method alone. We do not know of a risk assessment correlating TEM measures of exposure with adverse health effects. TEM measurements, therefore, cannot be used as the basis for an occupational exposure limit at this time. Additionally, TEM is much more expensive and time consuming than PCM. If we were to analyze each of the 2,184 personal exposure filters (collected by us to determine full-shift asbestos exposures from 2000 through 2003) using TEM, rather than PCM, it would cost us about \$186,000 to \$852,000 more. The mine operator's costs would increase in so far as the operator would do comparable sampling. We expect the operator to sample to determine whether control measures are needed, what controls might be needed, and the effectiveness of controls when implemented. A number of commenters supported our continued use of PCM for the initial analysis of asbestos samples.

We conclude that it is not feasible to regulate the mining industry below the proposed limit at this time. We welcome comments on the exposure limit proposed and the rationale used for choosing it over the alternative discussed above.

**D. Regulatory Flexibility Analysis (RFA) and Small Business Regulatory Enforcement Fairness Act (SBREFA)**

Based on our data, our experience, and information submitted to the

record, we determined, and here certify, that this proposed rule would not have a significant economic impact on a substantial number of small entities. The PREA for this proposed rule (RIN: 1219-AB24), *Measuring and Controlling Asbestos Exposure*, contains the factual basis for this certification as well as complete details about data, equations, and methods used to calculate the costs and quantified benefits. We have placed the PREA in the rulemaking docket and posted it on MSHA's Web site at <http://www.msha.gov>.

**E. Other Regulatory Considerations**

**1. The National Environmental Policy Act of 1969 (NEPA)**

We have reviewed this proposed rule in accordance with the requirements of NEPA (42 U.S.C. 4321 *et seq.*), the regulations of the Council on Environmental Quality (40 CFR 1500), and the Department of Labor's NEPA procedures (29 CFR 11) and have assessed its environmental impacts. We found that this proposed rule would have no significant impact on air, water, or soil quality; plant or animal life; the use of land; or other aspects of the human environment.

**2. Paperwork Reduction Act of 1995**

This proposed rule contains no information collection or recordkeeping requirements. Thus, there are no additional paperwork burden hours and related costs associated with the proposed rule. Accordingly, the Paperwork Reduction Act requires no further agency action or analysis.

**3. The Unfunded Mandates Reform Act of 1995**

This proposed rule does not include any Federal mandate that may result in increased expenditures by State, local, or tribal governments; nor would it significantly or uniquely affect small governments. It would not increase private sector expenditures by more than \$100 million annually. Accordingly, the Unfunded Mandates Reform Act requires no further agency action or analysis.

4. Treasury and General Government Appropriations Act of 1999, (Section 654: Assessment of Impact of Federal Regulations and Policies on Families)

This proposed rule would have no effect on family well-being or stability, marital commitment, parental rights or authority, or income or poverty of families and children. Accordingly, the Treasury and General Government Appropriations Act requires no further agency action, analysis, or assessment.

5. Executive Order 12630: Government Actions and Interference With Constitutionally Protected Property Rights

This proposed rule would not implement a policy with takings implications. Accordingly, Executive Order 12630 requires no further agency action or analysis.

6. Executive Order 12988: Civil Justice Reform

We have drafted and reviewed this proposed rule in accordance with Executive Order 12988. We wrote this proposed rule to provide a clear legal standard for affected conduct and carefully reviewed it to eliminate drafting errors and ambiguities, thus minimizing litigation and undue burden on the Federal court system. MSHA has determined that this proposed rule would meet the applicable standards in section 3 of Executive Order 12988.

7. Executive Order 13045: Protection of Children From Environmental Health Risks and Safety Risks

This proposed rule would have no adverse impact on children. This proposed asbestos standard might benefit children by reducing occupational exposure limits, thus reducing their risk of disease from take-home contamination. Accordingly, Executive Order 13045 requires no further agency action or analysis.

8. Executive Order 13132: Federalism

This proposed rule would not have "federalism implications," because it would not "have substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government." Accordingly, Executive Order 13132 requires no further agency action or analysis.

9. Executive Order 13175: Consultation and Coordination With Indian Tribal Governments

This proposed rule would not have "tribal implications," because it would

not "have substantial direct effects on one or more Indian tribes, on the relationship between the Federal government and Indian tribes, or on the distribution of power and responsibilities between the Federal government and Indian tribes." Accordingly, Executive Order 13175 requires no further agency action or analysis.

10. Executive Order 13211: Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use

In accordance with Executive Order 13211, we have reviewed this proposed rule for its impact on the supply, distribution, and use of energy. This proposed rule would regulate both the coal and metal and nonmetal mining sectors. Because this proposed rule would result in negligible yearly costs of less than 0.001 percent of revenues to the coal mining industry, the proposed rule would neither significantly reduce the supply of coal nor significantly increase its price. Regulation of the metal and nonmetal sector of the mining industry has no significant impact on the supply, distribution, or use of energy.

This proposed rule is not a "significant energy action," because it would not be "likely to have a significant adverse effect on the supply, distribution, or use of energy" "(including a shortfall in supply, price increases, and increased use of foreign supplies)." Accordingly, Executive Order 13211 requires no further agency action or analysis.

11. Executive Order 13272: Proper Consideration of Small Entities in Agency Rulemaking

In accordance with Executive Order 13272, we have thoroughly reviewed this proposed rule to assess and take appropriate account of its potential impact on small businesses, small governmental jurisdictions, and small organizations. As discussed in section VIII.C. above and in chapter V of the PREA, MSHA has determined and certified that this proposed rule would not have a significant economic impact on a substantial number of small entities.

#### **IX. Copy of the OSHA Reference Method (ORM)**

MSHA's existing asbestos standards require that the analyst determine fiber concentrations using a phase contrast microscopy analytical method with 400–450X magnification and count fibers 5 µm or longer having a length to diameter aspect ratio of at least 3:1. The

OSHA Reference Method contains these requirements.

#### **29 CFR 1910.1001 Appendix A: OSHA Reference Method—Mandatory**

This mandatory appendix specifies the procedure for analyzing air samples for asbestos and specifies quality control procedures that must be implemented by laboratories performing the analysis. The sampling and analytical methods described below represent the elements of the available monitoring methods (such as Appendix B of their regulation, the most current version of the OSHA method ID–160, or the most current version of the NIOSH Method 7400). All employers who are required to conduct air monitoring under paragraph (d) of the [OSHA] standard are required to utilize analytical laboratories that use this procedure, or an equivalent method, for collecting and analyzing samples.

##### *Sampling and Analytical Procedure*

1. The sampling medium for air samples shall be mixed cellulose ester filter membranes. These shall be designated by the manufacturer as suitable for asbestos counting. See below for rejection of blanks.

2. The preferred collection device shall be the 25-mm diameter cassette with an open-faced 50-mm electrically conductive extension cowl. The 37-mm cassette may be used if necessary but only if written justification for the need to use the 37-mm filter cassette accompanies the sample results in the employee's exposure monitoring record. Do not reuse or reload cassettes for asbestos sample collection.

3. An air flow rate between 0.5 liter/min and 2.5 liters/min shall be selected for the 25-mm cassette. If the 37-mm cassette is used, an air flow rate between 1 liter/min and 2.5 liters/min shall be selected.

4. Where possible, a sufficient air volume for each air sample shall be collected to yield between 100 and 1,300 fibers per square millimeter on the membrane filter. If a filter darkens in appearance or if loose dust is seen on the filter, a second sample shall be started.

5. Ship the samples in a rigid container with sufficient packing material to prevent dislodging the collected fibers. Packing material that has a high electrostatic charge on its surface (e.g., expanded polystyrene) cannot be used because such material can cause loss of fibers to the sides of the cassette.

6. Calibrate each personal sampling pump before and after use with a representative filter cassette installed between the pump and the calibration devices.

7. Personal samples shall be taken in the "breathing zone" of the employee (i.e., attached to or near the collar or lapel near the worker's face).

8. Fiber counts shall be made by positive phase contrast using a microscope with an 8 to 10 X eyepiece and a 40 to 45 X objective for a total magnification of approximately 400 X and a numerical aperture of 0.65 to 0.75. The microscope shall also be fitted with a green or blue filter.

9. The microscope shall be fitted with a Walton-Beckett eyepiece graticule calibrated

for a field diameter of 100 micrometers (+/- 2 micrometers).

10. The phase-shift detection limit of the microscope shall be about 3 degrees measured using the HSE phase shift test slide as outlined below.

a. Place the test slide on the microscope stage and center it under the phase objective.

b. Bring the blocks of grooved lines into focus.

**Note:** The slide consists of seven sets of grooved lines (ca. 20 grooves to each block) in descending order of visibility from sets 1 to 7, seven being the least visible. The requirements for asbestos counting are that the microscope optics must resolve the grooved lines in set 3 completely, although they may appear somewhat faint, and that the grooved lines in sets 6 and 7 must be invisible. Sets 4 and 5 must be at least partially visible but may vary slightly in visibility between microscopes. A microscope that fails to meet these requirements has either too low or too high a resolution to be used for asbestos counting.

c. If the image deteriorates, clean and adjust the microscope optics. If the problem persists, consult the microscope manufacturer.

11. Each set of samples taken will include 10 percent blanks or a minimum of 2 field blanks. These blanks must come from the same lot as the filters used for sample collection. The field blank results shall be averaged and subtracted from the analytical results before reporting. A set consists of any sample or group of samples for which an evaluation for this standard must be made. Any samples represented by a field blank having a fiber count in excess of the detection limit of the method being used shall be rejected.

12. The samples shall be mounted by the acetone/triacetin method or a method with an equivalent index of refraction and similar clarity.

13. Observe the following counting rules.

a. Count only fibers equal to or longer than 5 micrometers. Measure the length of curved fibers along the curve.

b. In the absence of other information, count all particles as asbestos that have a length-to-width ratio (aspect ratio) of 3:1 or greater.

c. Fibers lying entirely within the boundary of the Walton-Beckett graticule field shall receive a count of 1. Fibers crossing the boundary once, having one end within the circle, shall receive the count of one half (1/2). Do not count any fiber that crosses the graticule boundary more than once. Reject and do not count any other fibers even though they may be visible outside the graticule area.

d. Count bundles of fibers as one fiber unless individual fibers can be identified by observing both ends of an individual fiber.

e. Count enough graticule fields to yield 100 fibers. Count a minimum of 20 fields; stop counting at 100 fields regardless of fiber count.

14. Blind recounts shall be conducted at the rate of 10 percent.

#### Quality Control Procedures

1. *Intralaboratory program.* Each laboratory and/or each company with more than one

microscopist counting slides shall establish a statistically designed quality assurance program involving blind recounts and comparisons between microscopists to monitor the variability of counting by each microscopist and between microscopists. In a company with more than one laboratory, the program shall include all laboratories and shall also evaluate the laboratory-to-laboratory variability.

2.a. *Interlaboratory program.* Each laboratory analyzing asbestos samples for compliance determination shall implement an interlaboratory quality assurance program that as a minimum includes participation of at least two other independent laboratories. Each laboratory shall participate in round robin testing at least once every 6 months with at least all the other laboratories in its interlaboratory quality assurance group. Each laboratory shall submit slides typical of its own work load for use in this program. The round robin shall be designed and results analyzed using appropriate statistical methodology.

2.b. All laboratories should also participate in a national sample testing scheme such as the Proficiency Analytical Testing Program (PAT), or the Asbestos Registry sponsored by the American Industrial Hygiene Association (AIHA).

3. All individuals performing asbestos analysis must have taken the NIOSH course for sampling and evaluating airborne asbestos dust or an equivalent course.

4. When the use of different microscopes contributes to differences between counters and laboratories, the effect of the different microscope shall be evaluated and the microscope shall be replaced, as necessary.

5. Current results of these quality assurance programs shall be posted in each laboratory to keep the microscopists informed.

[57 FR 24330, June 8, 1992; 59 FR 40964, Aug. 10, 1994]

#### X. References Cited in the Preamble

Agency for Toxic Substances and Disease Registry (ATSDR). *Perchlorate Contamination in the Citizens Utilities' Suburban and Security Park Water Service Areas*, Prepared by California Department of Health Services under CERCLIS No. CAD980358832, March 18, 1998.

Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological Profile for Asbestos (Update)*, Prepared by Syracuse Research Corp. under Contract No. 205-1999-00024, U.S. Department of Health and Human Services, Public Health Service, September 2001.

Agency for Toxic Substances and Disease Registry (ATSDR). *Report on the Expert Panel on Health Effects of Asbestos and Synthetic Vitreous Fibers: The Influence of Fiber Length* (Proceedings of panel discussion, October 29-30, 2002, New York City), Prepared by Eastern Research Group, Inc., March 17, 2003.

Amandus, H.E., R. Wheeler, J. Jankovic, and J. Tucker. "The Morbidity and Mortality of Vermiculite Miners and Millers Exposed to Tremolite-Actinolite: Part I. Exposure Estimates," *American Journal of Industrial Medicine*, 11(1):1-14, 1987.

Amandus, H.E., and R. Wheeler. "The Morbidity and Mortality of Vermiculite Miners and Millers Exposed to Tremolite-Actinolite: Part II. Mortality," *American Journal of Industrial Medicine*, 11(1):15-26, 1987.

Amandus, H.E., R. Althouse, W.K.C. Morgan, E.N. Sargent, and R. Jones. "The Morbidity and Mortality of Vermiculite Miners and Millers Exposed to Tremolite-Actinolite: Part III. Radiographic Findings," *American Journal of Industrial Medicine*, 11(1):27-37, 1987.

American Conference of Governmental Industrial Hygienists-American Industrial Hygiene Association, Joint ACGIH-AIHA Aerosol Hazards Evaluation Committee. "Background Documentation on Evaluation of Occupational Exposure to Airborne Asbestos," *American Industrial Hygiene Association Journal*, February 1975, pp. 91-103.

American Thoracic Society. "Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos," *American Journal of Respiratory and Critical Care Medicine*, 170:691-715, 2004.

Armstrong, B.K., N.H. de Klerk, A.W. Musk, and M.S.T. Hobbs. "Mortality in Miners and Millers of Crocidolite in Western Australia," *British Journal of Industrial Medicine*, 45:5-13, 1988.

Asbestos International Association (AIA). "Airborne Asbestos Fiber Concentrations at Workplaces by Light Microscopy (Membrane Filter Method)," *AIA Health and Safety Recommended Technical Method No. 1 (RTM1)*, London, September 7, 1979.

Baron, Paul A. "Measurement of Airborne Fibers: A Review," *Industrial Health*, 39:39-50, 2001.

Becker, Nikolaus, Jurgen Berger, and Ulrich Bolm-Audorff. "Asbestos Exposure and Malignant Lymphomas— a Review of the Epidemiological Literature," *International Archives of Occupational and Environmental Health*, 74:459-469, 2001.

Becklake, Margaret R. "Clinical Measurements in Quebec Chrysotile Miners: Use for Future Protection of Workers," *Annals New York Academy of Sciences*, pp. 23-29, 1979.

Berry, G., and H.C. Lewinsohn. "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part I. Morbidity," *Annals New York Academy of Sciences*, pp. 185-194, 1979.

Berry, G., and M.L. Newhouse. "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine*, 40:1-7, 1983.

Bolton, C., A. Richards, and P. Ebdon. "Asbestos-Related Disease," *Hospital Medicine*, 63(3):148-151, March 2002.

Britton, Mark. "The Epidemiology of Mesothelioma," *Seminars in Oncology*, 29(1):18-25, February 2002.

Browne, Kevin. "The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure," *Annals of Occupational Hygiene* (Letters to the Editor), 45(4):327-329, 2001.

Browne, Kevin, and J. Bernard L. Gee. "Asbestos and Laryngeal Cancer," *Annals*

- of *Occupational Hygiene*, 44:239–250, 2000.
- Carbone, Michele, Robert A. Kratzke, and Joseph R. Testa. “The Pathogenesis of Mesothelioma,” *Seminars in Oncology*, 29(1):2–17, February 2002.
- Clark, R.L. “Metallic Substrates as an Internal Standard for Transmission Electron Microscope,” *Annals of the Institute for the Certification of Engineering Technicians*, 1977.
- Cookson, W.O.C.M., N.H. de Klerk, A.W. Musk, B.K. Armstrong, J.J. Glancy, and M.S.T. Hobbs. “Prevalence of Radiographic Asbestosis in Crocidolite Miners and Millers at Wittenoom, Western Australia,” *British Journal of Industrial Medicine*, 43:450–457, 1986.
- Cotran, Ramzi S., Vinay Kumar, and Tucker Collins. *Robbins Pathological Basis of Disease, Sixth Edition* (W.B. Saunders Company, Philadelphia), pp. 732–734, 1999.
- Crane, Dan. Letter to Kelly Bailey dated May 16, 1989. (1219–AB24–COMM–28–10).
- Dave, S.K., L.J. Bhagia, P.K. Mazumdar, G.C. Patel, P.K. Kulkarni, and S.K. Kashyap. “The Correlation of Chest Radiograph and Pulmonary Function Tests in Asbestos Miners and Millers,” *Indian Journal of Chest Disease and Allied Sciences*, 38:81–89, 1996.
- Davis, J.M.G., S.T. Beckett, R.E. Bolton, and K. Donaldson. “The Effects of Intermittent High Asbestos Exposure (Peak Dose Levels) on the Lungs of Rats,” *British Journal of Experimental Pathology*, 61:272–280, 1980.
- Davis, J.M.G., S.T. Beckett, R.E. Bolton, and K. Donaldson. “A Comparison of the Pathological Effects in Rat of the UICC Reference Samples of Amosite and Chrysotile with Those of Amosite and Chrysotile Collected from the Factory Environment,” In: *Biological Effects of Mineral Fibres*, J.C. Wagner (Editor-in-Chief), IARC Scientific Publications No. 30 (2 volumes), pp. 285–292, 1980.
- Davis, J.M.G., J. Addison, R.E. Bolton, K. Donaldson, A.D. Jones and T. Smith. “The Pathogenicity of Long versus Short Fibre Samples of Amosite Administered to Rats by Inhalation and Intraperitoneal Injection,” *British Journal of Experimental Pathology*, 67:415–430, 1986.
- Davis, J.M.G., and A.D. Jones. “Comparison of the Pathogenicity of Long and Short Fibres of Chrysotile Asbestos in Rats,” *British Journal of Experimental Pathology*, 69:717–737, 1988.
- Delpierre, Stephane, Yves Jammes, Marie Jose Delvogo-Gori, and Marion Faucher. “High Prevalence of Reversible Airway Obstruction in Asbestos-Exposed Workers,” *Archives of Environmental Health*, 57(5):441–445, September/October, 2002.
- Dement, J.M., R.L. Harris, M.J. Symons, and C. Shy. “Estimates of Dose-Response for Respiratory Cancer among Chrysotile Asbestos Textile Workers,” *Annals of Occupational Hygiene*, 26(14):869–887, 1982.
- Dodson, Ronald F., Mark A.L. Atkinson, and Jeffrey L. Levin. “Asbestos Fiber Length as Related to Potential Pathogenicity: A Critical Review,” *American Journal of Industrial Medicine*, 44:291–297, 2003.
- Doll, Richard. “Mortality from Lung Cancer in Asbestos Workers,” *British Journal of Industrial Medicine*, 12:81–86, 1955.
- Donaldson, K., R.E. Bolton, A. Jones, G.M. Brown, M.D. Robertson, J. Slight, H. Cowie, and J.M.G. Davis. “Kinetics of the Bronchoalveolar Leucocyte Response in Rats during Exposure to Equal Airborne Mass Concentrations of Quartz, Chrysotile Asbestos, or Titanium Dioxide,” *Thorax*, 43:525–533, 1988.
- Eagen, Tomas M.L., Amund Gulsvik, Geir E. Eide, and Per S. Bakke. “Occupational Airborne Exposure and the Incidence of Respiratory Symptoms and Asthma,” *American Journal of Respiratory Critical Care Medicine*, 166:933–938, 2002.
- Enarson, D.A., Valerie Embree, Lonia Maclean, and S. Grzybowski. “Respiratory Health in Chrysotile Asbestos Miners in British Columbia: A Longitudinal Study,” *British Journal of Industrial Medicine*, 45:459–463, 1988.
- Finkelstein, Murray M. “Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory,” *American Review of Respiratory Disease*, 125:496–501, 1982.
- Finkelstein, M.M. “Mortality among Long-Term Employees of an Ontario Asbestos-Cement Factory,” *British Journal of Industrial Medicine*, 40:138–144, 1983.
- Finkelstein, Murray M. “Potential Pitfall in Using Cumulative Exposure in Exposure-Response Relationships: Demonstration and Discussion,” *American Journal of Industrial Medicine*, 28:41–47, 1995.
- Fischer, M., S. Gunther, and K.-M. Muller. “Fibre-Years, Pulmonary Asbestos Burden and Asbestosis,” *International Journal of Hygiene and Environmental Health*, 205:245–248, 2002.
- Gibbs, Graham W., and R.S.J. du Toit. “Environmental Considerations in Surveillance of Asbestos Miners and Millers,” *Annals New York Academy of Sciences*, pp. 163–178, 1979.
- Global Environmental & Technology Foundation (GETF). “Report of Findings and Recommendations on the Use and Management of Asbestos,” *Asbestos Strategies*, 2003.
- Goldstein, B., and F.S.J. Coetzee. “Experimental Malignant Mesotheliomas in Baboons,” *South African Journal of Science*, 86:89–93, February 1990.
- Greenberg, Morris. “Biological Effects of Asbestos: New York Academy of Sciences 1964,” *American Journal of Industrial Medicine* (Historical Perspective), 43:543–552, 2003.
- Harper, Martin, and Al Bartolucci. “Preparation and Examination of Proposed Consensus Reference Standards for Fiber-Counting,” *American Industrial Hygiene Association Journal*, 64:283–287, 2003.
- Henderson, Vivian L., and Philip E. Enterline. “Asbestos Exposure Factors Associated with Excess Cancer and Respiratory Disease Mortality,” *Annals New York Academy of Sciences* (prepublication copy), 1979.
- Hodgson, John T., and Andrew Darnton. “The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure,” *Annals of Occupational Hygiene*, 44(8):565–601, 2000.
- International Agency for Research on Cancer (IARC). “Asbestos,” *Monographs* (Volume 14), Supplement 7, p. 106, 1987.
- International Commission on Radiation Protection (ICRP), Prepared by the Task Group on Lung Dynamics for Committee II of the ICRP. “Deposition and Retention Models for Internal Dosimetry of the Human Respiratory Tract,” *Health Physics*, 12:173–207, 1966. [“Errata and Revisions to Health Physics 12, 173 (1966),” *Health Physics*, 13:1251, 1967.]
- International Organization for Standardization (ISO). “Air quality—Determination of the number concentration of airborne inorganic fibres by phase contrast microscopy—Membrane filter method,” ISO 8672:1993(E).
- Irwig, L.M., R.S.J. du Toit, G.K. Sluis-Cremer, A. Solomon, R. Glyn Thomas, P.P.H. Hamel, I. Webster, and T. Hastie. “Risk of Asbestosis in Crocidolite and Amosite Mines in South Africa,” *Annals New York Academy of Sciences*, pp. 35–52, 1979.
- JRB Associates. “Benefits Assessment of Emergency Temporary and Proposed Asbestos Standards, Final Report,” Prepared by Marthe B. Kent, William G. Perry, and Christine B. New for OSHA Office of Regulatory Analysis, November 3, 1983.
- Kelse, John W., and C. Sheldon Thompson. “The Regulatory and Mineralogical Definitions of Asbestos and their Impact on Amphibole Dust Analysis,” *American Industrial Hygiene Association Journal*, 50:613–622, 1989.
- Lane, R.E. (Chairman) et al., Subcommittee on Asbestos, Committee on Hygiene, British Occupational Hygiene Society. “Hygiene Standards for Chrysotile Asbestos Dust,” *Annals of Occupational Hygiene*, 11:47–69, 1968. (1219–AB24–COMM–29–2)
- Langer, Arthur M., Arthur N. Rohl, Mary Snow Wolf, and Irving J. Selikoff. “Asbestos, Fibrous Minerals and Acicular Cleavage Fragments: Nomenclature and Biological Properties,” *Dusts and Disease*, 1979. (1219–AB24–COMM–29–11)
- Leake, Bernard E. (Chairman), et al. “Nomenclature of Amphiboles: Report of the Subcommittee on Amphiboles of the International Mineralogical Association, Commission on New Minerals and Mineral Names,” *Canadian Mineralogist*, 35:219–246, 1997.
- Lemen, Richard A. “Asbestos in Brakes,” October 16, 2003. [Paper received from Ralph D. Zumwalde (NIOSH) via Tom Simons (EPA), December 5, 2003.]
- Liddell, Douglas. Letter to the Editor, “Asbestos and Cancer,” *Annals of Occupational Hygiene*, 45(4):329–335, 2001.
- Maltoni, Cesare. “Call for an International Ban on Asbestos,” *Toxicology and Industrial Health*, 15:529–531, 1999.
- Manning, Christopher B., Val Vallyathan, and Brooke Mossman. “Diseases Caused by Asbestos: Mechanisms of Injury and Disease Development,” *International Immunopharmacology*, 2:191–200, 2002.
- McDonald, J. Corbett, and F. Douglas K. Liddell. “Mortality in Canadian Miners and Millers Exposed to Chrysotile,” *Annals*

- New York Academy of Sciences*, pp. 1–9, 1979.
- McDonald, J.C., F.D.K. Liddell, G.W. Gibbs, G.E. Eyssen, and A.D. McDonald. "Dust Exposure and Mortality in Chrysotile Mining, 1910–75," *British Journal of Industrial Medicine*, 37:11–24, 1980.
- (A) McDonald, J.C., A.D. McDonald, B. Armstrong, and P. Sebastien. "Cohort study of mortality of vermiculite miners exposed to tremolite," *British Journal of Industrial Medicine*, 43:436–444, 1986.
- (B) McDonald, J.C., P. Sebastien, and B. Armstrong. "Radiological Survey of Past and Present Vermiculite Miners Exposed to Tremolite," *British Journal of Industrial Medicine*, 43:445–449, 1986.
- McDonald, J.C., A.D. McDonald, P. Sebastien, and K. Moy. "Health of Vermiculite Miners Exposed to Trace Amounts of Fibrous Tremolite," *British Journal of Industrial Medicine*, 45:630–634, 1988.
- McDonald, J.C., F.D.K. Liddell, A. Dufresne, and A.D. McDonald. "The 1891–1920 Birth Cohort of Quebec Chrysotile Miners and Millers: Mortality 1976–1988," *British Journal of Industrial Medicine*, 50:1073–1081, 1993.
- McDonald, A.D., B.W. Case, A. Churg, A. Dufresne, G.W. Gibbs, P. Sebastien, and J.C. McDonald. "Mesothelioma in Quebec Chrysotile Miners and Millers: Epidemiology and Aetiology," *Annals of Occupational Hygiene*, 41(6):707–719, 1997.
- McGavran, Patricia D., Charles J. Butterick, and Arnold R. Brody. "Tritiated Thymidine Incorporation and the Development of an Interstitial Lesion in the Bronchiolar-Alveolar Regions of the Lungs of Normal and Complement Deficient Mice after Inhalation of Chrysotile Asbestos," *Journal of Environmental Pathology, Toxicology and Oncology (JEPTO)*, 9(5)/9(6):377–392, 1989.
- McLaughlin, Joseph K., and Loren Lipworth. "Epidemiologic Aspects of Renal Cell Cancer," *Seminars in Oncology*, 27(2):115–123, April 2000.
- Meeker, G.P., A.M. Bern, I.K. Brownfield, H.A. Lowers, S.J. Sutley, T.M. Hoefen, and J.S. Vance. "The Composition and Morphology of Amphiboles from the Rainy Creek Complex, Near Libby, Montana," *American Mineralogist*, 88:1955–1969, 2003.
- Mine Safety and Health Administration (MSHA). Walter Bank, "Asbestiform and/or Fibrous Minerals in Mines, Mills, and Quarries," *Informational Report IR 1111*, 1980.
- Mine Safety and Health Administration (MSHA). "Asbestos Hazards in the Mining Industry," *Health Hazard Information Card No. 21*, March 2000.
- Mine Safety and Health Administration (MSHA). "Chapter 8 Asbestos Fibers," *Metal/Nonmetal Health Inspection Procedures Handbook (PH04–IV–5)*, November 2003; and "Chapter 8 Asbestos," *MSHA Handbook Series: Coal Mine Health Inspection Procedures (Handbook No. 89–V–1)*, February 1989.
- Mine Safety and Health Administration (MSHA). Program Information Bulletin No. P00–3 from J. Davitt McAteer, re: Potential Exposure to Airborne Asbestos on Mining Properties, dated March 2, 2000.
- Mossman, Brooke. In *Report of the Expert Panel on Health Effects of Asbestos and Synthetic Vitreous Fibers: The Influence of Fiber Length*, (Proceedings of Panel, October 29–30, 2002, New York City), Prepared by Eastern Research Group for the Agency for Toxic Substances and Disease Registry (ATSDR), March 17, 2003.
- National Institute for Occupational Safety and Health (NIOSH). *Criteria for a Recommended Standard Occupational Exposure to Asbestos*, U.S. Department of Health, Education, and Welfare, 1972.
- National Institute for Occupational Safety and Health (NIOSH). *Report to Congress on Workers' Home Contamination Study Conducted Under the Workers' Family Protection Act*, DHHS (NIOSH) Publication No. 95–123 (September 1995).
- National Institute for Occupational Safety and Health (NIOSH). *Protect Your Family: Reduce Contamination at Home*, DHHS (NIOSH) Publication No. 97–125 (1997).
- National Institute for Occupational Safety and Health (NIOSH). *Protecting Workers' Families: A Research Agenda: Report of the Workers' Family Protection Task Force*, DHHS (NIOSH) Publication No. 2002–113 (2002).
- National Institute for Occupational Safety and Health (NIOSH). Division of Respiratory Disease Studies, *Work Related Lung Disease Surveillance Report 2002* [World 2003], DHHS (NIOSH) Publication No. 2003–111, May 2003.
- National Institute for Occupational Safety and Health (NIOSH). *Pocket Guide to Chemical Hazards*, DHHS (NIOSH) Publication No. 2004–103, October 2003.
- Nayebzadeh, Ataollah, Andre Dufresne, Bruce Case, Hojatolah Vali, A.E. Williams-Jones, Robert Martin, Charles Normand, and James Clark. "Lung Mineral Fibers of Former Miners and Millers from Thetford-Mines and Asbestos Regions: A Comparative Study of Fiber Concentration and Dimension," *Archives of Environmental Health*, 56(1):65–76, January/February 2001.
- Nicholson, William J., Irving J. Selikoff, Herbert Seidman, Ruth Lillis, and Paul Formby. "Long-Term Mortality Experience of Chrysotile Miners and Millers in Thetford Mines, Quebec," *Annals New York Academy of Sciences*, pp. 11–21, 1979.
- Nicholson, William J. "Quantitative Risk Assessment for Asbestos Related Cancers," Prepared in conjunction with U.S. Department of Labor, Occupational Safety and Health Administration (OSHA), Office of Carcinogen Standards, under OSHA Contract No. J–9–F–2–0074, October 1983.
- Nicholson, William J. "The Carcinogenicity of Chrysotile Asbestos—A Review," *Industrial Health*, 39:57–64, 2001.
- Nolan, R.P., A.M. Langer, and Richard Wilson. "A Risk Assessment for Exposure to Grunerite Asbestos (Amosite) in an Iron Ore Mine," Paper presented at the National Academy of Sciences Colloquium "Geology, Mineralogy, and Human Welfare," Irvine, CA, November 8–9, 1998.
- In: *Proceedings of the National Academy of Science*, 96(7):3412–3419, March 1999.
- Ojajarvi, I. Anneli, Timo J. Partanen, Anders Ahlbom, Paolo Boffetta, Timo Hakulinen, Nadia Jourenkova, Timo P. Kauppinen, Manolis Kogevinas, Miquel Porta, Harri U. Vainio, Elisabete Weiderpass, and Catharina H. Wesseling. "Occupational Exposures and Pancreatic Cancer: A Meta-Analysis," *Occupational and Environmental Medicine*, 57:316–324, 2000.
- Orenstein, Marla R., and Marc B. Schenker. "Environmental Asbestos Exposure and Mesothelioma," *Pulmonary Medicine (Current Opinion)*, 6:371–377, 2000.
- Osinubi, Omowunmi Y.O., Michael Gochfeld, and Howard M. Kipen. "Health Effects of Asbestos and Nonasbestos Fibers," *Environmental Health Perspectives*, 108 (Supplement 4): 665–674, 2000.
- Pang, Thomas W.S. "Precision and Accuracy of Asbestos Fiber Counting by Phase Contrast Microscopy," *American Industrial Hygiene Association Journal*, 61:529–538, 2000.
- Paustenbach, Dennis J., Richard O. Richter, Brent L. Finley, and Patrick J. Sheehan. "An Evaluation of the Historical Exposures of Mechanics to Asbestos in Brake Dust," *Applied Occupational and Environmental Hygiene*, 18:786–804, 2003.
- Peacock, C., S.J. Copley, and D.M. Hansell. "Asbestos-Related Benign Pleural Disease," *Clinical Radiology (Review)*, 55:422–432, 2000.
- Peipins, Lucy A., Michael Lewin, Sharon Campolucci, Jeffrey A. Lybarger, Aubrey Miller, Dan Middleton, Christopher Weis, Michael Spence, Brad Black, and Vikas Kapil. "Radiographic Abnormalities and Exposure to Asbestos-Contaminated Vermiculite in the Community of Libby, Montana, USA," *Environmental Health Perspectives*, 111(14):1753–1759, November 2003.
- Peto, J., R. Doll, S.V. Howard, L.J. Kinlen, and H.C. Lewinsohn. "A Mortality Study among Workers in an English Asbestos Factory," *British Journal of Industrial Medicine*, 34:169–173, 1977.
- Peto, Julian. "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," In: *Biological Effects of Mineral Fibres*, J.C. Wagner (Editor-in-Chief), *IARC Scientific Publications No. 30* (2 volumes), pp. 829–836, 1980.
- Peto, J., H. Seidman, and I.J. Selikoff. "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," *British Journal of Cancer*, 45:124–135 (prepublication copy), 1982.
- Pohlmann, H., P. Wild, W. Schill, W. Ahrens, I. Jahn, U. Bolm-Audorff, and K-H Jockel. "Asbestos Fibre Years and Lung Cancer: A Two Phase Case-Control Study with Expert Exposure Assessment," *Occupational and Environmental Medicine*, 59:410–414, 2002.
- Ramanathan, A.L., and V. Subramanian. "Present Status of Asbestos Mining and Related Health Problems in India—A Survey," *Industrial Health*, 39:309–315, 2001.
- Reeves, Andrew L., Henry E. Puro, and Ralph G. Smith. "Inhalation Carcinogenesis from

- Various Forms of Asbestos," *Environmental Research*, 8:178–202, 1974.
- Reger, Robert, and W.K. Morgan. "On talc, tremolite, and tergilversation," *British Journal of Industrial Medicine*, 47:505–507, 1990.
- Roach, Huw D., Gareth J. Davies, Richard Attanoos, Michael Crane, Haydn Adams, and Sian Phillips. "Asbestos: When the Dust Settles—An Imaging Review of Asbestos-Related Disease," *RadioGraphics*, 22:S167–S184, 2002.
- Roggli, Victor L., Robin T. Vollmer, Kelly J. Butnor, and Thomas A. Sporn. "Tremolite and Mesothelioma," *Annals of Occupational Hygiene*, 46(5):447–453, 2002.
- Rooker, Stephen J., Nicholas P. Vaughan, and Jean M. Le Guen. "On the Visibility of Fibers by Phase Contrast Microscopy," *American Industrial Hygiene Association Journal*, 43:505–515, July 1982. (1219–AB24–COMM–29–19)
- Ross, Malcom. "The 'Asbestos' Minerals: Definitions, Description, Modes of Formation, Physical and Chemical Properties, and Health Risk to the Mining Community," *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, November 1978.
- Rossiter, Charles E., Leonard J. Bristol, Paul H. Cartier, John G. Gilson, T. Roger Grainger, Gerald K. Sluis-Cremer, and J. Corbett McDonald. "Radiographic Changes in Chrysotile Asbestos Mine and Mill Workers of Quebec," *Archives of Environmental Health*, 24:388–400, June 1972.
- Rubino, G.F., M. Newhouse, R. Murray, G. Scansetti, G. Piolatto, and G. Aresini. "Radiologic Changes after Cessation of Exposure among Chrysotile Asbestos Miners in Italy," *Annals New York Academy of Sciences*, pp. 157–161, 1979.
- Rubino, G.F., G. Piolatto, M.L. Newhouse, G. Scansetti, G.A. Aresini, and R. Murray. "Mortality of Chrysotile Asbestos Workers at the Balangero Mine, Northern Italy," *British Journal of Industrial Medicine*, 36:187–194, 1979.
- Rudd, Robin M. "New Developments in Asbestos-Related Pleural Disease," *Thorax*, 51:210–216, 1996.
- Sali, Davide, and Paolo Boffetta. "Kidney Cancer and Occupational Exposure to Asbestos: A Meta-Analysis of Occupational Cohort Studies," *Cancer Causes and Control*, 11:37–47, 2000.
- Schlecht, Paul C., and Stanley A. Shulman. "Phase Contrast Microscopy Asbestos Fiber Counting Performance in the Proficiency Analytical Testing Program," *American Industrial Hygiene Association Journal*, 56:480–489, 1995.
- Schneider, Andrew. "A P–I Special Report: Uncivil Action," *Seattle Post-Intelligencer*, November 18–19, 1999.
- Schwartz, David A., Charles S. Davis, James A. Merchant, W. Bruce Bunn, Jeffrey R. Galvin, D. Scott Van Fossen, Charles S. Dayton, and Gary W. Hunninghake. "Longitudinal Changes in Lung Function among Asbestos-Exposed Workers," *American Journal of Respiratory Critical Care Medicine*, 150:1243–1249, 1994.
- Seidman, Herbert, Irving J. Selikoff, and E. Cuyler Hammond. "Short-Term Asbestos Work Exposure and Long-Term Observation," *Annals New York Academy of Sciences*, pp. 61–89, 1979.
- Seidman, Herbert. "Short-Term Asbestos Work Exposure and Long-Term Observation," from OSHA Asbestos Docket (Exh-261-A), July 1984 Updating.
- Selden, A.I., N.P. Berg, E.A.L. Lundgren, G. Hillerdal, N.-G. Wik, C.-G. Ohlson, and L.S. Bodin. "Exposure to Tremolite Asbestos and Respiratory Health in Swedish Dolomite Workers," *Occupational and Environmental Medicine*, 58:670–677, 2001.
- Selikoff, Irving J., E. Cuyler Hammond, and Herbert Seidman. "Mortality Experience of Insulation Workers in the United States and Canada, 1943–1976," *Annals New York Academy of Sciences*, pp. 91–116, 1979.
- Snyder, J.G., R.L. Virta, and J.M. Segreti. "Evaluation of the Phase Contrast Microscopy Method for the Detection of Fibrous and Other Elongated Mineral Particulates by Comparison with a STEM Technique," *American Industrial Hygiene Association Journal*, 48(5):471–477, 1987.
- Solomon, A., L.M. Irwig, G.K. Sluis-Cremer, R. Glyn Thomas, and R.S.J. du Toit. "Thickening of Pulmonary Interlobar Fissures: Exposure-Response Relationship in Crocidolite and Amosite Miners," *British Journal of Industrial Medicine*, 36:195–198, 1979.
- Steenland, Kyle, Dana Loomis, Carl Shy, and Neal Simonsen. "Review of Occupational Carcinogens," *American Journal of Industrial Medicine*, 29:474–490, 1996.
- Steenland, Kyle, Carol Burnett, Nina Lalich, Elizabeth Ward, and Joseph Hurrell. "Dying for Work: The Magnitude of U.S. Mortality from Selected Causes of Death Associated with Occupation," *American Journal of Industrial Medicine*, 43:461–482, 2003.
- Stewart, I.M., and R.J. Lee. *Considerations in the Regulation of Actinolite, Tremolite and Anthophyllite*, Society for Mining, Metallurgy and Exploration, Inc., 1992.
- Suzuki, Yasunosuke, and Steven R. Yuen. "Asbestos Fibers Contributing to the Induction of Human Malignant Mesothelioma," *Annals New York Academy of Sciences*, 982:160–176, 2002.
- Tweedale, Geoffrey. "Asbestos and Its Lethal Legacy," *Nature Reviews/Cancer* (Perspectives), 2:1–5, April 2002.
- U.S. Department of Labor, Office of the Inspector General. *Evaluation of MSHA's Handling of Inspections at the W.R. Grace & Company Mine in Libby, Montana*, Report No. 2E-06-620-0002, March 22, 2001.
- U.S. Environmental Protection Agency (EPA). *Guidance for Preventing Asbestos Disease Among Auto Mechanics*, EPA-560-OPTS-86-002, June 1986.
- U.S. Environmental Protection Agency (EPA). *Method for the Determination of Asbestos in Bulk Building Materials*, EPA Report No. EPA/600/R-93/116 (NTIS/PB93-218576), July 1993. [Updates and replaces Interim version in 40 CFR 763, Subpart F, App A].
- U.S. Environmental Protection Agency (EPA, Region 8). "Environmental News " Asbestos in Libby, EPA Proposes Libby as a National Priority," *EPA Action Update* # 12, February 26, 2002.
- U.S. Environmental Protection Agency (EPA, Region 2). *World Trade Center Background Study Report*, Prepared for U.S. Federal Emergency Management Agency, IAG No.: EMW-2002-IA-0127, April 2003.
- U.S. Environmental Protection Agency (EPA). "40 CFR Part 63, National Emission Standards for Hazardous Air Pollutants: Taconite Iron Ore Processing; Final Rule," **Federal Register** (68 FR 61868), October 30, 2003.
- U.S. Geological Survey (USGS). "Preliminary Compilation of Descriptive Geoenvironmental Mineral Deposit Models," *Open-file Report 95-831*, 1995.
- U.S. Geological Survey (USGS). Robert L. Virta, "Talc and Pyrophyllite," *U.S. Geological Survey Minerals Yearbook*, 2002.
- U.S. Geological Survey (USGS). Robert L. Virta, "Asbestos," *U.S. Geological Survey 2003 Mineral Commodity Summary*, online at <http://minerals.er.usgs.gov/minerals/pubs/commodity/asbestos/070303.pdf>.
- Verma, Dave K., and Nancy E. Clark. "Relationships between Phase Contrast Microscopy and Transmission Electron Microscopy Results of Samples from Occupational Exposure to Airborne Chrysotile Asbestos," *American Industrial Hygiene Association Journal*, 56:866–873, 1995.
- Wang, Xiao-Rong, Eiji Yano, Mianzheng Wang, Zhiming Wang, and David C. Christiani. "Pulmonary Function in Long-Term Asbestos Workers in China," *Journal of Occupational and Environmental Health*, 43(7):623–629, July 2001.
- Wagner, J.C., G. Berry, J.W. Skidmore, and V. Timbrell. "The Effects of the Inhalation of Asbestos in Rats," *British Journal of Cancer*, 29:252–269, 1974.
- Wagner, J.C., G. Berry, J.W. Skidmore, and F.D. Pooley. "The Comparative Effects of Three Chrysotiles by Injection and Inhalation in Rats," In: *Biological Effects of Mineral Fibres*, J.C. Wagner (Editor-in-Chief), *IARC Scientific Publications No. 30* (2 volumes), pp. 363–372, 1980.
- Warheit, David B. (Editor). *Fiber Toxicology*, Academic Press, Inc., 1993.
- Webster, Ian, Bertie Goldstein, Frans Coetzee, and Gerhardus C.H. van Sittert. "Malignant Mesothelioma Induced in Baboons by Inhalation of Amosite Asbestos," *American Journal of Industrial Medicine*, 24:659–666, 1993.
- Weill, Hans, Janet Hughes, and Carmel Waggenspack. "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease*, 120:345–354, 1979.
- Weis, Christopher P., Aubrey K. Miller, and Paul Peronard. "Task-based exposure monitoring for residential exposure to amphibole asbestos in Libby, Montana," U.S. Environmental Protection Agency, Paper presented at 129th Annual APHA Meeting, October 21–25, 2001.
- West, John B. *Respiratory Physiology, The Essentials* (Sixth Edition), Lippincott Williams & Wilkins: Baltimore, MD, pp. 4–6 and 131–133, 2000.

West, John B. *Pulmonary Pathophysiology, The Essentials* (Sixth Edition), Lippincott Williams & Wilkins: Baltimore, MD, pp. 82–91 and 126–137, 2003.

Wylie, Ann G., Robert L. Virta, and Estelle Russek. "Characterizing and Discriminating Airborne Amphibole Cleavage Fragments and Amosite Fibers: Implications for the NIOSH Method", *American Industrial Hygiene Association Journal*, 46(4):197–201, 1985.

Wylie, Ann G. "The Habit of Asbestiform Amphiboles: Implications for the Analysis of Bulk Samples," *Advances in Environmental Measurement Methods for Asbestos*, ASTM STP 1342, M.E. Beard and H.L. Rooks (editors), American Society for Testing and Materials (ASTM), West Conshohocken, PA, 2000.

Xu, An, Hongning Zhou, Dennis Zengliang Yu, and Tom K. Hei. "Mechanisms of the Genotoxicity of Crocidolite Asbestos in Mammalian Cells: Implication from Mutation Patterns Induced by Reactive Oxygen Species," *Environmental Health Perspectives*, 110:1003–1008, 2002.

Yano, Eiji, Zhi-Ming Wang, Xiao-Rong Wang, Mian-Zheng Wang, and Ya-Jia Lan. "Cancer Mortality among Workers Exposed to Amphibole-Free Chrysotile Asbestos," *American Journal of Epidemiology*, 154(6):538–542, 2001.

**List of Subjects**

*30 CFR Parts 56 and 57*

Air quality, Asbestos, Chemicals, Hazardous substances, Metals, Mine safety and health.

*30 CFR Part 71*

Air quality, Asbestos, Chemicals, Coal mining, Hazardous substances, Mine safety and health.

Dated: July 14, 2005.

**David G. Dye,**

*Deputy Assistant Secretary of Labor for Mine Safety and Health.*

For the reasons set out in the preamble, and under the authority of the Federal Mine Safety and Health Act of 1977, we are proposing to amend chapter I of title 30 of the Code of Federal Regulations as follows.

**PART 56—[AMENDED]**

1. The authority citation for part 56 would continue to read as follows:

**Authority:** 30 U.S.C. 811.

2. Section 56.5001 would be amended by revising paragraph (b) to read as follows:

**§ 56.5001 Exposure limits for airborne contaminants.**

\* \* \* \* \*

(b) *Asbestos standard.* (1) *Definitions.* Asbestos is a generic term for a number of hydrated silicates that, when crushed or processed, separate into flexible fibers made up of fibrils. As used in this part—

*Asbestos* means chrysotile, amosite (cummingtonite-grunerite asbestos), crocidolite, anthophyllite asbestos, tremolite asbestos, and actinolite asbestos.

*Fiber* means a particulate form of asbestos 5 micrometers (µm) or longer with a length-to-diameter ratio of at least 3-to-1.

(2) *Permissible Exposure Limits (PELs).*

(i) *Full-shift exposure limit.* A miner's personal exposure to asbestos shall not exceed an 8-hour time-weighted average, full-shift airborne concentration of 0.1 fibers per cubic centimeter of air (f/cc).

(ii) *Excursion limit.* No miner shall be exposed at any time to airborne concentrations of asbestos in excess of 1.0 fiber per cubic centimeter of air (f/cc) as averaged over a sampling period of 30 minutes.

(3) *Measurement of airborne fiber concentration.* Fiber concentration shall be determined by phase contrast microscopy using a method statistically equivalent to the OSHA Reference Method in OSHA's asbestos standard found in 29 CFR 1910.1001, appendix A.

\* \* \* \* \*

**PART 57—[AMENDED]**

3. The authority citation for part 57 would continue to read as follows:

**Authority:** 30 U.S.C. 811.

4. Section 57.5001 would be amended by revising paragraph (b) to read as follows:

**§ 57.5001 Exposure limits for airborne contaminants.**

\* \* \* \* \*

(b) *Asbestos standard.* (1) *Definitions.* Asbestos is a generic term for a number of hydrated silicates that, when crushed or processed, separate into flexible fibers made up of fibrils. As used in this part—

*Asbestos* means chrysotile, amosite (cummingtonite-grunerite asbestos), crocidolite, anthophyllite asbestos, tremolite asbestos, and actinolite asbestos.

*Fiber* means a particulate form of asbestos 5 micrometers (µm) or longer with a length-to-diameter ratio of at least 3-to-1.

(2) *Permissible Exposure Limits (PELs).*

(i) *Full-shift exposure limit.* A miner's personal exposure to asbestos shall not exceed an 8-hour time-weighted average, full-shift airborne concentration of 0.1 fibers per cubic centimeter of air (f/cc).

(ii) *Excursion limit.* No miner shall be exposed at any time to airborne concentrations of asbestos in excess of 1.0 fiber per cubic centimeter of air (f/cc) as averaged over a sampling period of 30 minutes.

(3) *Measurement of airborne fiber concentration.* Fiber concentration shall be determined by phase contrast microscopy using a method statistically equivalent to the OSHA Reference Method in OSHA's asbestos standard found in 29 CFR 1910.1001, appendix A.

\* \* \* \* \*

**PART 71—[AMENDED]**

5. The authority citation for part 71 would be revised to read as follows:

**Authority:** 30 U.S.C. 811, 951, 957.

6. Section 71.701 would be amended by revising paragraphs (c) and (d) to read as follows:

**§ 71.701 Sampling; general requirements.**

\* \* \* \* \*

(c) Where concentrations of airborne contaminants in excess of the applicable threshold limit values, permissible exposure limits, or permissible excursions are known by the operator to exist in a surface installation or at a surface worksite, the operator shall immediately provide necessary control measures to assure compliance with § 71.700 or § 71.702, as applicable.

(d) Where the operator has reasonable grounds to believe that concentrations of airborne contaminants in excess of the applicable threshold limit values, permissible exposure limits, or permissible excursions exist, or are likely to exist, the operator shall promptly conduct appropriate air sampling tests to determine the concentration of any airborne contaminant which may be present and immediately provide the necessary control measures to assure compliance with § 71.700 or § 71.702, as applicable.

7. Section 71.702 would be revised to read as follows:

**§ 71.702 Asbestos standard.**

(a) *Definitions.* Asbestos is a generic term for a number of hydrated silicates that, when crushed or processed, separate into flexible fibers made up of fibrils. As used in this part—

*Asbestos* means chrysotile, amosite (cummingtonite-grunerite asbestos), crocidolite, anthophyllite asbestos, tremolite asbestos, and actinolite asbestos.

*Fiber* means a particulate form of asbestos 5 micrometers (µm) or longer with a length-to-diameter ratio of at least 3-to-1.

(b) *Permissible Exposure Limits (PELs)*. (1) *Full-shift exposure limit*. A miner's personal exposure to asbestos shall not exceed an 8-hour time-weighted average, full-shift airborne concentration of 0.1 fibers per cubic centimeter of air (f/cc).

(2) *Excursion limit*. No miner shall be exposed at any time to airborne concentrations of asbestos in excess of 1.0 fiber per cubic centimeter of air (f/cc) as averaged over a sampling period of 30 minutes.

(c) *Measurement of airborne fiber concentration*. Fiber concentration shall

be determined by phase contrast microscopy using a method statistically equivalent to the OSHA Reference Method in OSHA's asbestos standard found in 29 CFR 1910.1001, appendix A.

[FR Doc. 05-14510 Filed 7-28-05; 8:45 am]

**BILLING CODE 4510-43-P**