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**NIOSH**

# Asbestos Publications



U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Public Health Service  
Centers for Disease Control  
National Institute for Occupational Safety and Health

# **ASBESTOS PUBLICATIONS**

**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Public Health Service  
National Institute for Occupational Safety and Health  
Division of Standards Development and Technology Transfer  
4676 Columbia Parkway  
Cincinnati, Ohio 45226**

**June 1992**

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# CONTENTS

Preface .....	v
Part I. Full-Text or Abstracted Copies of Selected NIOSH References on Asbestos .....	1
A. Occupational Safety and Health Guideline for Asbestos .....	3
B. Revised Recommended Asbestos Standard (Abstract) .....	9
C. Workplace Exposure to Asbestos - Review and Recommendations (Abstract) .....	21
D. Occupational Respiratory Diseases .....	29
Asbestosis .....	31
Lung Cancer .....	73
Mesothelioma .....	85
E. Work-Related Lung Disease Surveillance Report (Abstract) .....	103
F. NIOSH Manual of Analytical Methods - Third Edition .....	117
Method #7400 - Fibers .....	119
Method #7402 - Asbestos Fibers .....	133
Method #9000 - Chrysotile Asbestos .....	141
Method #9002 - Asbestos (Bulk) .....	149
G. Testimony of the National Institute for Occupational Safety and Health on the Occupational Safety and Health Administration's Proposed Rule on Occupational Exposure to Asbestos, Tremolite, Anthophyllite and Actinolite, January 24, 1991 .....	159
H. Statement of the National Institute for Occupational Safety and Health before the Subcommittee on Toxic Substances, Environmental Oversight, Research and Development, Committee on Environment and Public Works, April 26, 1990 .....	175
Part II. NIOSH Bibliography on Asbestos .....	179
NTIS Order Form .....	191
GPO Order Form .....	193





## PREFACE

The National Institute for Occupational Safety and Health (NIOSH) has assembled this publication to aid in the dissemination of NIOSH information about asbestos.

This publication is divided into two parts. Part I consists of full-text or abstracted copies of selected NIOSH documents on asbestos. These documents include NIOSH publications and testimony that summarize both NIOSH research on health hazards of asbestos and NIOSH recommendations on workplace exposure to asbestos.

Part II contains a complete list of NIOSH documents on asbestos. Each document citation includes the title and year of publication, the number of pages, a NIOSH identifying number, and ordering information (see below). The citations are arranged alphabetically by document title or author within one of the following six categories: numbered publications; testimony; journal articles and conference proceedings; contract reports; grant reports; and miscellaneous reports. A brief description of each of these categories precedes the listing in that section. Those documents that appear in Part I are so identified in the appropriate citation in Part II.

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**PART I**

**FULL-TEXT OR ABSTRACTED COPIES OF  
SELECTED NIOSH REFERENCES  
ON ASBESTOS**



# OCCUPATIONAL SAFETY AND HEALTH GUIDELINE FOR ASBESTOS POTENTIAL HUMAN CARCINOGEN

## INTRODUCTION

This guideline summarizes pertinent information about asbestos for workers, employers, and occupational safety and health professionals who may need such information to conduct effective occupational safety and health programs. Recommendations may be superseded by new developments in these fields; therefore, readers are advised to regard these recommendations as general guidelines.

## SUBSTANCE IDENTIFICATION

Data in the following section are presented for various forms of asbestos: (1) Asbestos (mixed forms);

- (2) Chrysotile;
- (3) Amosite;
- (4) Crocidolite;
- (5) Tremolite;
- (6) Anthophyllite;
- (7) Actinolite.

If unspecified, data apply to all forms.

• **Composition:** (1) Not Available;

- (2)  $3\text{MgO} \cdot 2\text{SiO}_2 \cdot 2\text{H}_2\text{O}$ ;
- (3)  $(\text{FeMg})\text{SiO}_3$ ;
- (4)  $\text{NaFe}(\text{SiO}_3)_2 \cdot \text{FeSiO}_3 \cdot \text{H}_2\text{O}$ ;
- (5)  $\text{Ca}_2\text{Mg}_3\text{Si}_8\text{O}_{22}(\text{OH})_2$ ;
- (6)  $(\text{MgFe})_7\text{Si}_8\text{O}_{22}(\text{OH})_2$ ;
- (7)  $\text{CaO} \cdot 3(\text{MgFe})\text{O} \cdot 4\text{SiO}_2$

• **Synonyms:** (1) Asbestos fiber, serpentine, amphibole;

- (2) Canadian chrysotile, white asbestos, serpentine;
- (3) Brown asbestos, fibrous grunerite;
- (4) Blue asbestos;
- (5) Fibrous tremolite;
- (6) Azbolen asbestos;
- (7) Not available

• **Identifiers:** (1) CAS 1332-21-4; RTECS CI6475000; DOT 2212 (blue) 2590 (white);

- (2) CAS 12001-29-5; RTECS CI6478500; DOT 2590;
- (3) CAS 12172-73-5; RTECS CI6477000; DOT Not assigned;
- (4) CAS 12001-28-4; RTECS CI6479000; DOT 2212;

- (5) CAS 14567-73-8; RTECS CI6560000; DOT Not assigned;
- (6) CAS 17068-78-9; RTECS CI6478000; DOT Not assigned;
- (7) CAS 13768-00-8; RTECS CI6476000; DOT Not assigned

• **Appearance and odor:** A fiber or filament, asbestos may have a "fluffy" appearance. Colors may vary from white, gray, blue, brown, green or yellow. Positive identification requires microscopic examination.

## CHEMICAL AND PHYSICAL PROPERTIES

• **Physical data**

- 1. Molecular weight: (2) 277.13; (5) 185.03
- 2. Specific gravity (water = 1): 2.5-3.0
- 3. Noncombustible solid

• **Warning properties**

Evaluation of warning properties for respirator selection: Warning properties are not considered in recommending respirators for use with carcinogens.

## EXPOSURE LIMITS

Only asbestos fibers greater than 5 micrometers ( $\mu\text{m}$ ) in length are considered for the following exposure limits. The current Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) for asbestos is 0.2 fiber per cubic centimeter (cc) of air as a time-weighted average (TWA) concentration over an 8-hour workshift with an action level of 0.1 fiber/cc as an hour TWA. The National Institute for Occupational Safety and Health (NIOSH) recommends that asbestos be controlled and handled as a potential human carcinogen in the workplace and that exposure be minimized to the lowest feasible limit. The NIOSH recommended exposure limit (REL) is 0.1 fiber/cc (in 40-liter air sample) as a TWA concentration for up to an 8-hour workshift, 40-hour workweek. The American Conference of Governmental Industrial Hygienists (ACGIH) has designated asbestos as an A1 substance (suspected human carcinogen, with an assigned threshold limit value/ TLV<sup>®</sup>) of 2 fibers/cc for chrysotile, 0.5 fiber/cc for amosite, 0.2 fiber/cc for crocidolite, and 2 fibers/cc for other forms, as a TWA for a normal 8-hour workday and a 40-hour workweek (Table 1).

### U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

Public Health Service    Centers for Disease Control  
National Institute for Occupational Safety and Health  
Division of Standards Development and Technology Transfer



**Table 1.—Occupational exposure limits for asbestos**

	Exposure limits mg/m <sup>3</sup> *
OSHA PEL TWA	0.2
Action level	0.1
NIOSH REL TWA (Ca)†	0.1
ACGIH TLV® TWA (Ala)§	
Chrysotile	2.0
Amosite	0.5
Crocidolite	0.2
Other forms	2.0

\* Fibers greater than 5  $\mu$ m in length.

† (Ca): NIOSH recommends treating as a potential human carcinogen.

§ (Ala): Human carcinogen with an assigned TLV®.

## HEALTH HAZARD INFORMATION

### • Routes of exposure

Asbestos may cause adverse health effects following exposure via inhalation or ingestion.

### • Summary of toxicology

1. *Effects on animals:* Single intrapleural injections of asbestos in rats, rabbits, and hamsters produced mesothelioma (cancer of the chest or abdominal linings). In rats, chronic inhalation or oral administration of asbestos produced cancers of the lungs, stomach, kidneys, liver, or mammary glands. All forms of asbestos were found to be carcinogenic in treated animals.

2. *Effects on humans:* Exposure to asbestos has been found to significantly increase the risks of contracting asbestosis, lung cancer, and mesothelioma.

### • Signs and symptoms of exposure

1. *Short-term (acute):* Exposure to asbestos can cause shortness of breath, chest or abdominal pain, and irritation of the skin and mucous membranes.

2. *Long-term (chronic):* Exposure to asbestos can cause reduced pulmonary function, breathing difficulty, dry cough, broadening and thickening of the ends of the fingers, and bluish discoloration of the skin and mucous membranes.

## RECOMMENDED MEDICAL PRACTICES

### • Medical surveillance program

Workers with potential exposures to chemical hazards should be monitored in a systematic program of medical surveillance intended to prevent or control occupational injury and disease. The program should include education of employers and workers about work-related hazards, placement of workers in jobs that do not jeopardize their safety and health, earliest possible detection of adverse health effects, and referral of workers for diagnostic confirmation and treatment. The occurrence of disease (a "sentinel health event," SHE) or other work-related adverse health effects should prompt immediate evaluation of primary preventive measures (e.g., industrial hygiene monitoring, engineering controls, and personal protective equipment). A medical surveillance program is intended to supplement, not replace, such measures.

A medical surveillance program should include systematic collection and epidemiologic analysis of relevant environmental

and biologic monitoring, medical screening, and morbidity and mortality data. This analysis may provide information about the relatedness of adverse health effects and occupational exposure that cannot be discerned from results in individual workers. Sensitivity, specificity, and predictive values of biologic monitoring and medical screening tests should be evaluated on an industry-wide basis prior to application in any given worker group. Intrinsic to a surveillance program is the dissemination of summary data to those who need to know, including employers, occupational health professionals, potentially exposed workers, and regulatory and public health agencies.

### • Preplacement medical evaluation

Prior to placing a worker in a job with a potential for exposure to asbestos, the physician should evaluate and document the worker's baseline health status with thorough medical, environmental, and occupational histories, a physical examination, and physiologic and laboratory tests appropriate for the anticipated occupational risks. These should concentrate on the function and integrity of the respiratory system using the principles and methods recommended by NIOSH and the American Thoracic Society (ATS).

A preplacement medical evaluation is recommended in order to detect and assess preexisting or concurrent conditions which may be aggravated or result in increased risk when a worker is exposed to asbestos at or below the NIOSH REL. The examining physician should consider the probable frequency, intensity, and duration of exposure, as well as the nature and degree of the condition, in placing such a worker. Such conditions, which should not be regarded as absolute contraindications to job placement, include cigarette smoking, preexisting asbestos-related disease, and significant breathing impairment due to preexisting chronic lung diseases. In addition to the medical interview and physical examination, the means to identify these conditions may include the methods recommended by NIOSH and ATS.

### • Periodic medical screening and/or biologic monitoring

Occupational health interviews and physical examinations should be performed at regular intervals. Additional examinations may be necessary should a worker develop symptoms that may be attributed to exposure to asbestos. The interviews, examinations, and appropriate medical screening and/or biologic monitoring tests should be directed at identifying an excessive decrease or adverse trend in the physiologic function of the respiratory system as compared to the baseline status of the individual worker or to the expected values for a suitable reference population. The following tests should be used and interpreted according to standardized procedures and evaluation criteria recommended by NIOSH and ATS: standardized questionnaires, tests of lung function, and chest X-rays.

### • Medical practices recommended at the time of job transfer or termination

The medical, environmental, and occupational history interviews, the physical examination, and selected physiologic and laboratory tests which were conducted at the time of placement should be repeated at the time of job transfer or termination. Any changes in the worker's health status should be compared to those expected for a suitable reference population. Because

occupational exposure to asbestos may cause diseases of prolonged induction-latency, the need for medical surveillance may extend well beyond termination of employment.

- **Sentinel health events**

Delayed-onset SHE's include: Scarring of the lungs (asbestosis) and its lining (pleural fibrosis) and cancer of the lungs (bronchogenic lung cancer) and its lining (mesothelioma).

## MONITORING AND MEASUREMENT PROCEDURES

- **TWA exposure evaluation**

Measurements to determine worker exposure to asbestos should be taken so that the TWA exposure is based on a single entire workshift sample or an appropriate number of consecutive samples collected during the entire workshift. Under certain conditions, it may be appropriate to collect several short-term interval samples (up to 30 minutes each) to determine the average exposure level. Air samples should be taken in the worker's breathing zone (air that most nearly represents that inhaled by the worker).

- **Method**

Sampling and analysis for airborne asbestos may be performed by collecting asbestos fibers with membrane filters and analyzing by phase contrast microscopy. A detailed sampling and analytical method for asbestos may be found in the *NIOSH Manual of Analytical Methods* (method number 7400).

## PERSONAL PROTECTIVE EQUIPMENT

Chemical protective clothing (CPC) should be selected after utilizing available performance data, consulting with the manufacturer, and then evaluating the clothing under actual use conditions.

Workers should be provided with and required to use CPC, gloves, and other appropriate protective clothing necessary to prevent skin contact with asbestos.

## SANITATION

Clothing which is contaminated with asbestos should be removed at the end of the work period and placed in nonreusable, impermeable containers for storage, transport, and disposal until it can be discarded or until provision is made for the removal of asbestos from the clothing. These containers should be marked "Asbestos-Contaminated Clothing" in easy-to-read letters. If the clothing is to be laundered or cleaned, the person performing the operation should be informed of asbestos's hazardous properties. Reusable clothing and equipment should be checked for residual contamination before reuse or storage.

A change room with showers, washing facilities, and lockers that permit separation of street and work clothes should be provided.

Workers should be required to shower following a workshift and prior to putting on street clothes. Clean work clothes should be provided daily.

Skin that becomes contaminated with asbestos should be promptly washed with soap and water.

The storage, preparation, dispensing, or consumption of food or beverages, the storage or application of cosmetics, the storage or smoking of tobacco or other smoking materials, or the storage or use of products for chewing should be prohibited in work areas.

Workers who handle asbestos should wash their faces, hands, and forearms thoroughly with soap and water before eating, smoking, or using toilet facilities.

## COMMON OPERATIONS AND CONTROLS

Common operations in which exposure to asbestos may occur and control methods which may be effective in each case are listed in Table 2.

**Table 2.—Operations and methods of control for asbestos**

Operations	Controls
During asbestos removal	Process enclosure, wet process (when possible), personal protective equipment
During the production of asbestos or the manufacture of products containing asbestos	Process enclosure, local exhaust ventilation, wet process (when possible), personal protective equipment
During the demolition of buildings	Water spray, personal protective equipment

## EMERGENCY FIRST AID PROCEDURES

In the event of an emergency, remove the victim from further exposure, send for medical assistance, and initiate emergency procedures.

- **Eye exposure**

Where there is any possibility of a worker's eyes being exposed to asbestos, an eye wash fountain should be provided within the immediate work area for emergency use.

If asbestos gets into the eyes, flush them immediately with large amounts of water for 15 minutes, lifting the lower and upper lids occasionally. Get medical attention as soon as possible. Contact lenses should not be worn when working with this substance.

- **Skin exposure**

If asbestos gets on the skin, wash it immediately with soap and water.

- **Rescue**

If a worker has been incapacitated, move the affected worker from the hazardous exposure. Put into effect the established emergency rescue procedures. Do not become a casualty. Understand the facility's emergency rescue procedures and know the locations of rescue equipment before the need arises.

## SPILLS AND LEAKS

Workers not wearing protective equipment and clothing should be restricted from areas of spills or leaks until cleanup has been completed.

If asbestos is spilled or leaked, the following steps should be taken:

Asbestos dust may be collected by vacuuming with an appropriate high-efficiency filtration system or by using wet methods and placed in an appropriate container.

## WASTE REMOVAL AND DISPOSAL

U.S. Environmental Protection Agency, Department of Transportation, and/or state and local regulations shall be followed to assure that removal, transport, and disposal are in accordance with existing regulations.

## RESPIRATORY PROTECTION

It must be stressed that the use of respirators is the least preferred method of controlling worker exposure and should not normally be used as the only means of preventing or minimizing exposure during routine operations. However, there are some exceptions for which respirators may be used to control exposure: when engineering and work practice controls are not technically feasible, when engineering controls are in the process of being installed, or during emergencies and certain maintenance operations including those requiring confined-space entry (Table 3).

In addition to respirator selection, a complete respiratory protection program should be instituted which as a minimum complies with the requirements found in the OSHA Safety and Health Standards 29 CFR 1910.134. A respiratory protection program should include as a minimum an evaluation of the worker's ability to perform the work while wearing a respirator, the regular training of personnel, fit testing, periodic environmental monitoring, maintenance, inspection, and cleaning. The implementation of an adequate respiratory protection program, including selection of the correct respirators, requires that a knowledgeable person be in charge of the program and that the program be evaluated regularly.

Only respirators that have been approved by the Mine Safety and Health Administration (MSHA, formerly Mining Enforcement and Safety Administration) and by NIOSH should be used. **Remember! Air-purifying respirators will not protect from oxygen-deficient atmospheres.**

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**Table 3.—Respiratory protection for asbestos**

Condition	Minimum respiratory protection*
Any detectable concentration	Any self-contained breathing apparatus with a full facepiece and operated in a pressure-demand or other positive pressure mode  Any supplied-air respirator with a full facepiece and operated in a pressure-demand or other positive pressure mode in combination with an auxiliary self-contained breathing apparatus operated in a pressure-demand or other positive pressure mode
Planned or emergency entry into environments containing unknown or any detectable concentration	Any self-contained breathing apparatus with a full facepiece and operated in a pressure-demand or other positive pressure mode  Any supplied-air respirator with a full facepiece and operated in a pressure-demand or other positive pressure mode in combination with an auxiliary self-contained breathing apparatus operated in a pressure-demand or other positive pressure mode
Firefighting	Any self-contained breathing apparatus with a full facepiece and operated in a pressure-demand or other positive pressure mode
Escape only	Any air-purifying full facepiece respirator with a high-efficiency particulate filter  Any appropriate escape-type self-contained breathing apparatus

\* Only NIOSH/MSHA-approved equipment should be used.



# **REVISED RECOMMENDED ASBESTOS STANDARD**



**U. S. DEPARTMENT OF HEALTH, EDUCATION, AND  
WELFARE  
Public Health Service  
Center for Disease Control  
National Institute for Occupational Safety and Health**

**DECEMBER 1976**

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The Division of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health (NIOSH), having primary responsibility for development of a NIOSH position paper on health effects of occupational asbestos exposure, has critiqued all available data and prepared the following document for publication and transmittal to the Occupational Safety and Health Administration (OSHA), as requested by the Assistant Secretary of Labor. Primary responsibility for development of this document was shared by Richard A. Lemen and John M. Dement, with technical consultation provided by Dr. Joseph K. Wagoner. Individuals who served as the NIOSH review committee were:

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**DHEW (NIOSH) Publication No. 77-169**

REVISED RECOMMENDED  
ASBESTOS STANDARD

Table of Contents

	<u>Page</u>
I. INTRODUCTION	1
II. BIOLOGIC EFFECTS OF EXPOSURE ON ANIMALS	3
Carcinogenicity	3
Mutagenicity	12
References	13
Summary Table of Asbestos-induced Carcinogenicity in Animals	17
Tables and Figure	21
III. EFFECTS ON HUMANS	26
Nonmalignant Respiratory Disease	26
Carcinogenicity	30
Synergism	38
Fiber Analysis in Tissue	39
References	43
Tables	53
IV. SAMPLING METHODS AND ENVIRONMENTAL DATA	58
Review of Sampling and Analysis Techniques for Asbestos	58
Comparisons of Asbestos Mass Concentrations (mg/m <sup>3</sup> ) and Fiber Number Concentrations (fibers/cc)	71
Nonoccupational Exposures - Ambient Levels	73
References	78
Tables	82
V. BASIS FOR THE RECOMMENDED STANDARD	88
VI. THE RECOMMENDED STANDARD	92
References	95
Table	96

## I. INTRODUCTION

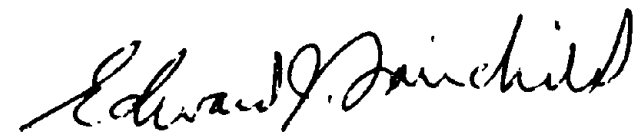
When the asbestos criteria document was first published in 1972, the National Institute for Occupational Safety and Health (NIOSH) recommended a standard of 2.0 asbestos fibers/cubic centimeter (cc) of air based on a count of fibers greater than 5 micrometers ( $\mu\text{m}$ ) in length. This standard was recommended with the stated belief that it would "prevent" asbestosis and with the open recognition that it would not "prevent" asbestos-induced neoplasms. Furthermore, data were presented which supported the fact that technology was available to achieve that standard and that the criteria would be subject to review and revision as necessary. Since the time that the asbestos criteria were published in 1972, sufficient additional data regarding asbestos-related disease have been developed to warrant reevaluation.

On June 7, 1972, the Occupational Safety and Health Administration (OSHA) promulgated a standard for occupational exposure to asbestos containing an 8-hour time-weighted average (TWA) concentration exposure limit of 5 fibers longer than 5  $\mu\text{m}$ /cc of air, with a ceiling limitation against any exposure in excess of 10 such fibers/cc. The standard further provided that the 8-hour TWA was to be reduced to 2 fibers/cc on July 1, 1976.

As the result of a court case, OSHA decided that to achieve the most feasible occupational health protection, a reexamination of the standard's general premises and general structure was necessary. To this end, on October 9, 1975, OSHA announced a proposed rule-making to lower the exposure limit to an 8-hour TWA concentration of 0.5 asbestos fibers longer

than 5  $\mu\text{m}/\text{cc}$  of air with a ceiling concentration of 5 fibers/cc of air determined by a sampling period of up to 15 minutes. On December 2, 1975, OSHA requested NIOSH to reevaluate the information available on the health effects of occupational exposure to asbestos fibers and to advise OSHA on the results of this study.

This document contains an updated review of the available information on the health effects of exposure to asbestos. In addition, NIOSH's proposal for a new numerical exposure limit is included.

  
for John F. Finklea, M.D.  
Director, National Institute for  
Occupational Safety and Health

## V. BASIS FOR THE RECOMMENDED STANDARD

The first modern approach to the setting of an asbestos standard was proposed by the British Occupational Hygiene Society (BOHS 1968) in terms of fiber concentration. In 1968, a subcommittee of the Society evaluated data on 290 men at work in an asbestos factory. These data were provided by company sources. All the men had been employed after January 1933, following implementation of dust control measures mandated by the Factory Inspectorate in 1931. Estimates of the fiber exposure of these workmen were also provided by the company. Of the 290 individuals, 8 were stated to have x-ray evidence of asbestos disease and 16 had rales. Noteworthy in the 1968 data was the preponderance of individuals who had been employed less than 20 years. Only 118 of the 290 persons had worked for longer than 20 years and a scant 13 has been employed for 30 or more years.

After a review of these data, the BOHS proposed a standard which was adopted with minor modifications by the British government in 1969, and implemented in May 1970. All fibers between 5 and 100 microns in length were counted by light microscopy. The standard required no action to be taken below 2 fibers/cc. Between 2 fibers/cc and 12 fibers/cc, control measures commensurate with the exposure circumstances (time and frequency of worker exposure) were prescribed; above 12 fibers/cc, full application of control measures, including respiratory protection, was mandatory. The BOHS predicted that the risk of being affected, to the extent of having the earliest clinical signs of asbestos exposure (rales), would be less than 1% for an accumulated exposure of 100

fiber-years/cc (2 fibers/cc for 50 years, 4 fibers/cc for 25, etc.). Data (Lewinsohn, 1972) from the same factory which formed the basis for the BOHS standard demonstrate that a greater prevalence of abnormalities now exist (Table V-1). These data, in addition to demonstrating a dose-response relationship for radiographically detected abnormalities consistent with asbestosis, further showed a 17% prevalence of abnormal radiographic findings (6% consistent with asbestosis) in individuals employed since 1950.

Weill et al (1975), when considering lung function and irregular small opacities, reported that there was little evidence of a dose-response relationship below 100 mppcf-years. They further concluded that a concentration of 5 fibers/cc could be cautiously considered as "safe". Ayer and Berg (1976), however, reported data which suggest that the BOHS standard, of an average cumulative exposure of 100 fiber-years/cc, for chrysotile asbestos may prevent significant decreases in pulmonary function only when combined with periodic spirometry and further reduction of exposure for affected workers. Holmes (1973) has since stated that the data upon which the BOHS standard was based were inadequate to set a standard to prevent asbestosis. The BOHS-recommended standard of 2 fibers/cc was based on data related only to asbestosis and the Society clearly cautioned that, since a quantitative relationship between asbestos exposure and cancer risk was not known, it was not possible at that time to specify an air concentration which was known to be free of increased cancer risk. (BOHS 1968)

Howard et al (1976), in a follow-up examination of the textile workers previously studied by Doll (1955) and Knox et al (1965, 1968) for cancer, and by Lewinsohn (1972) for asbestosis, reported a statistically significant increase in the risk of developing lung cancer (1.8 times the expected) among those first entering scheduled areas from 1933 to



1950. In the same study, they also reported an excess of deaths due to lung cancer (1.9 times the expected) after 15 or more years from initial exposure among those who started work subsequent to 1950, a period of improved industrial engineering control technology and regulation.

In a study of miners exposed to amphibole fibers (amosite) in the cummingtonite-grunerite ore series, with airborne concentrations of less than 2.0 fibers/cc (average concentration, 0.25 fibers/cc) and 94% of the fibers shorter than 5  $\mu\text{m}$  in length, Gillam et al (1976) have demonstrated threefold increases in the risks of mortality from both malignant and nonmalignant respiratory diseases.

Newhouse (1969, 1973) and Newhouse et al (1972) have shown that the cancer risk to factory workers following mixed exposure to chrysotile, amosite, and crocidolite is dose-related. The women reported to have heavier exposures (as judged by their occupations) showed a sixfold excess of cancer following only 15 years' latency, whereas those with moderate or low exposures required 25 years' latency to demonstrate an excess. The rate of mesothelioma increased with both the severity and the length of exposure. However, even with as little as two years of asbestos exposure, six mesotheliomas occurred among female employees.

McDonald (1973) stated that the risk of developing lung cancer was essentially confined to persons with a dust index above 200 mppcf-years, and Enterline et al (1973) showed no direct dose-response for respiratory cancer below 125 mppcf-years. In a review of these two papers, Schneiderman (1974) concluded that, instead of being consistent with a threshold level at which no cancer risk exists, these data did not provide evidence for a threshold or for a "safe" level of exposure. He pointed out that in

the paper by Enterline et al (1973) there is no dose group for which the Standardized Mortality Ratio (SMR) is below 100 (100 = normal), but that the 95% confidence limits on the SMR's included 100 for two of the three dose groups below 125 mppcf-years. One of the dose groups (25-62.4) had a statistically significant excess mortality from lung cancer, whereas for the other two this mortality rate was insignificantly elevated above the expected values. Regarding McDonald's paper, Schneiderman stated that it is hard to determine what is excess since no expected numbers for each group were given upon which to base this comparison.

Among amosite workers with employment of 3 months or less, Selikoff (1976) reported excess cancer risks of 3.87, 1.68, and 1.65 times those expected for cancer of the lung, colon and rectum, and all sites, respectively.

Anderson et al (1976) have reported a significant excess of radiographic abnormalities of the chest characteristic of asbestos exposure (pleural and/or parenchymal) 25-30 years after the onset of household contamination. These abnormalities were observed in 35% of 326 otherwise healthy workers who had household contacts with amosite asbestos. In addition, four pleural mesotheliomas were found in this group.

## VI. THE RECOMMENDED STANDARD

Available studies provide conclusive evidence that exposure to asbestos fibers causes cancer and asbestosis in man. Lung cancers and asbestosis have occurred following exposure to chrysotile, crocidolite, amosite, and anthophyllite. Mesotheliomas, lung and gastrointestinal cancers have been shown to be excessive in occupationally exposed persons, while mesotheliomas have developed also in individuals living in the neighborhood of asbestos factories and near crocidolite deposits, and in persons living with asbestos workers. Asbestosis has been identified among persons living near anthophyllite deposits.

Likewise, all commercial forms of asbestos are carcinogenic in rats, producing lung carcinomas and mesotheliomas following their inhalation, and mesotheliomas after intrapleural or ip injection. Mesotheliomas and lung cancers were induced following even 1 day's exposure by inhalation.

The size and shape of the fibers are important factors; fibers less than  $0.5\ \mu\text{m}$  in diameter are most active in producing tumors. Other fibers of a similar size, including glass fibers, can also produce mesotheliomas following intrapleural or ip injection.

There are data that show that the lower the exposure, the lower the risk of developing cancer. Excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a "safe" level of asbestos exposure.

In view of the above, the standard should be set at the lowest level detectable by available analytical techniques, an approach consistent with NIOSH's most recent recommendations for other carcinogens (ie, arsenic and vinyl chloride). Such a standard should also prevent the development of asbestosis.

Since phase contrast microscopy is the only generally available and practical analytical technique at the present time, this level is defined as 100,000 fibers  $>5\ \mu\text{m}$  in length/ $\text{m}^3$  (0.1 fibers/cc), on an 8-hour-TWA basis with peak concentrations not exceeding 500,000 fibers  $>5\ \mu\text{m}$  in length/ $\text{m}^3$  (0.5 fibers/cc) based on a 15-minute sample period. Sampling and analytical techniques should be performed as specified by NIOSH publication USPHS/NIOSH Membrane Filter Method for Evaluating Airborne Asbestos Fibers - T.R. 84 (1976).

This recommended standard of 100,000 fibers  $>5\ \mu\text{m}$  in length/ $\text{m}^3$  is intended to (1) protect against the noncarcinogenic effects of asbestos, (2) materially reduce the risk of asbestos-induced cancer (only a ban can assure protection against carcinogenic effects of asbestos) and (3) be measured by techniques that are valid, reproducible, and available to industry and official agencies.

However, some difficulties arise in that specific work practices and innovative engineering control or process changes are needed. But because of the well-documented human carcinogenicity from all forms of asbestos, these difficulties should not be cited as cause for permitting continued exposure to asbestos at concentrations above 100,000 fibers  $>5\ \mu\text{m}$  in length/ $\text{m}^3$ .

This standard was not designed for the population-at-large, and any extrapolation beyond general occupational exposures is not warranted. The standard was designed only for the processing, manufacturing, and use of asbestos and asbestos-containing products as applicable under the Occupational Safety and Health Act of 1970.

**WORKPLACE EXPOSURE TO ASBESTOS**  
**Review and Recommendations**

DHHS (NIOSH) Publication No. 81-103

NIOSH-OSHA  
Asbestos Work Group  
April 1980

**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES**  
**Public Health Service**  
**Centers for Disease Control**  
**National Institute for Occupational Safety and Health**

**U.S. DEPARTMENT OF LABOR**  
**Occupational Safety and Health Administration**

**November 1980**

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## CONTENTS

	<i>PAGE</i>
MEMORANDUM ON ASBESTOS UPDATE AND RECOMMENDED OCCUPATIONAL STANDARD	1
I. ASBESTOS NOMENCLATURE/DEFINITIONS	9
Review	9
General Definition	10
II. ASBESTOS SAMPLING AND ANALYSIS	12
III. BIOLOGIC EFFECTS OF EXPOSURE TO ASBESTOS IN ANIMALS	14
In Vivo	14
In Vitro	16
IV. BIOLOGIC EFFECTS OF EXPOSURE TO ASBESTOS IN HUMANS	16
Amosite	16
Chrysotile	18
Crocidolite	20
Mixed Fiber Types	21
Malignant Neoplasms other than Mesothelioma and Cancer of the Lung	24
V. SMOKING AND ASBESTOS	25
VI. EXPOSURE TO ASBESTIFORM MINERALS OTHER THAN COMMERCIALLY MINED ASBESTOS	28
VII. NON-OCCUPATIONAL EXPOSURE TO COMMERCIAL SOURCES OF ASBESTOS	30
VIII. DOSE-RESPONSE RELATIONSHIPS	30
REFERENCES	33

**MEMORANDUM FOR:** *Dr. Eula Bingham*  
*Assistant Secretary for*  
*Occupational Safety and Health*  
*Dr. Anthony Robbins*  
*Director, National Institute for*  
*Occupational Safety and Health*

**FROM:** *Asbestos Work Group*

**SUBJECT:** *The Updated Scientific*  
*Information on Asbestos and*  
*Recommended Occupational*  
*Standard for Asbestos Exposure*

In the fall of 1979, a NIOSH/OSHA committee was formed at the direction of Dr. Eula Bingham, Assistant Secretary of Labor for Occupational Safety and Health, and Dr. Anthony Robbins, Director of the National Institute for Occupational Safety and Health (NIOSH), to review the scientific information concerning asbestos-related disease and assess the adequacy of the current OSHA occupational health standard of <sup>\*</sup>2,000,000 fibers per cubic meter greater than 5  $\mu\text{m}$  in length ( $2\text{Mf}/\text{m}^3$ ). Since the 1972 promulgation of this  $2,000,000 \text{ f}/\text{m}^3$  standard, OSHA, in 1975, proposed lowering the standard to  $500,000 \text{ f}/\text{m}^3$ ; NIOSH, in 1976, recommended lowering the standard to  $100,000 \text{ f}/\text{m}^3$ ; and the British Advisory Committee on Asbestos, in 1979, recommended lowering its occupational exposure standards. The NIOSH/OSHA committee has reviewed the most recent scientific information, including documents concerning the above developments and the 1977 International Agency for Research on Cancer (IARC) review of the carcinogenicity hazards of asbestos, and presents the following major conclusions and recommendations. A detailed updating of significant scientific literature since the 1976 NIOSH Criteria Document and the 1977 IARC Monograph is attached.

<sup>\*</sup>Effective January 19, 1989, the OSHA Permissible Exposure Limit (PEL) was changed to  $0.2 \text{ f}/\text{cc}$ .  
 Federal Register, Vol. 54, No. 12, pp. 2332-2983.

1. *Definition of Asbestos.* Having considered the many factors involved in specifying which substances should be regulated as asbestos, the committee recommends the following definition:

Asbestos is defined to be chrysotile, crocidolite, and fibrous cummingtonite-grunerite including amosite, fibrous tremolite, fibrous actinolite, and fibrous anthophyllite. The fibrosity of the above minerals is ascertained on a microscopic level with fibers defined to be particles with an aspect ratio of 3 to 1 or larger.

2. *Sampling and Analysis of Airborne Asbestos.* The committee concludes that the membrane filter-phase contrast microscopy method represents the only technique available that can reasonably be used for routine monitoring of occupational exposures and sampling for compliance purposes. However, the committee recognizes the lack of specificity of this method for fiber identification, and recommends the use of supplementary methods such as electron microscopy for fiber identification in cases of mixed fiber exposures. In recommending the primary use of light microscopy, the committee also wants to stress the inability of this method to detect short asbestos fibers to which workers are exposed. The toxicity of asbestos fibers shorter than the 5-micrometer detection limit of light microscopy cannot be dismissed on the basis of current scientific information.

3. *Biologic Effects of Exposure to Asbestos.* Animal studies demonstrate that all commercial forms and several non-commercial forms of asbestos produce pulmonary fibrosis, mesothelioma, and lung neoplasms. Chrysotile is as likely as crocidolite and other amphiboles to induce mesotheliomas after intrapleural injection, and also as likely to induce lung neoplasms after inhalation exposures.

Human occupational exposures to all commercial asbestos fiber types, both individually and in various combinations, have been associated with high rates of asbestosis, lung cancer, and mesothelioma. While significant excesses of cancer of several other sites have been observed in exposed workers, presently available information is insufficient to determine the role of specific fiber types.

On the basis of available information, the committee concludes that there is no scientific basis for differentiating between asbestos fiber types for regulatory purposes. Accordingly, the committee recommends that a single occupational health standard be established and applied to all asbestos fiber types.

Available data show that the lower the exposure, the lower the risk of developing asbestosis and cancer. Excessive cancer risks, however, have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a "safe" level of asbestos exposure. Accordingly, the committee recommends that, to the extent uses of asbestos cannot be eliminated or less toxic materials substituted for asbestos, worker exposures to asbestos must be controlled to the maximum extent possible.

*4. Inadequacy of Current 2,000,000-Fiber Occupational Standard.* The committee concluded that a variety of factors demonstrates that the current 2,000,000-fiber standard is grossly inadequate to protect American workers from asbestos-related disease. First, the 2,000,000-fiber standard was designed in 1969 by the British Occupational Hygiene Society (BOHS) for the limited purpose of minimizing asbestosis. Disease prevalence data from the BOHS study population collected subsequent to 1969 strongly suggest that this standard is insufficient to prevent a large incidence of asbestosis. Second, all levels of asbestos exposure studied to date have demonstrated asbestos-related disease, and a linear relationship appears to best describe the shape of the dose-response curve. These considerations led the committee to conclude that there is no level of exposure below which clinical effects do not occur. Third, the absence of a threshold is further indicated by the dramatic evidence of asbestos-related disease in members of asbestos-worker households and in persons living near asbestos-contaminated areas. These household and community contacts involved low level and/or intermittent casual exposure to asbestos. Studies of duration of exposure suggest that even at very short exposure periods (1 day to 3 months) significant disease can occur.

Although various models can be and have been fashioned to postulate possible dose-response relationships involving asbestos, the committee believes that the limited current data preclude the creation of any one empirical curve to describe *the exact* dose-response relationship. Over the last three decades, measurement techniques for asbestos have changed in several crucial respects, and there have been no suitable methods available to date to compare the results of prior techniques to current methods.

In addition, no adequate epidemiological information is available on the disease experience of workers exposed below the current standard and followed for a sufficient period to identify long latent effects. Consequently, the committee cannot present a precise dose-response relationship for the variety of asbestos-related diseases. However, the committee firmly believes that compelling evidence demonstrates that prevention of asbestos-related diseases requires that an occupational standard minimize all asbestos exposures, and definitely be set far below the current 2,000,000-fiber standard.

*5. Recommended Occupational Standard for Asbestos Exposure.* Given the inadequacy of the current 2,000,000-fiber standard, the committee urges that a new occupational standard be promulgated which is designed to eliminate non-essential asbestos exposures, and which requires the substitution of less hazardous and suitable alternatives where they exist. Where asbestos exposures cannot be eliminated, they must be controlled to the lowest level possible. A significant consideration in establishing a permissible exposure limit should be the lowest level of exposure detectable using currently available analytical techniques. At present this level would be 100,000 fibers greater than 5  $\mu\text{m}$  in length per cubic meter averaged over an 8-hour workday. Regardless of the choice of a permissible exposure limit, the best engineering controls and work practices should be instituted, and protective clothing and hygiene facilities should be provided and their use required of all workers exposed to asbestos. Respirators are not a suitable substitute for these control measures. The committee also reiterates its judgment that even where exposure is controlled to levels below 100,000 fibers, there is no scientific basis for concluding that all asbestos-related cancers would be prevented.

*6. Medical Surveillance Program.* Appropriate medical surveillance is crucial to detect and minimize the progression of some asbestos-related diseases. Considerable emphasis should be placed on baseline medical examinations for all workers potentially exposed or who have been exposed to asbestos at any level. These examinations should include the following: (1) a 14" x 17" postero-anterior chest X-ray; (2) spirometry including forced vital capacity (FVC) and forced expiratory volume in one second ( $\text{FEV}_1$ ); (3) a physical examination of the chest including auscultation for the presence or absence of rales, rhonchi,

and wheezing; (4) an assessment of the presence or absence of finger clubbing; and (5) a history of respiratory symptoms and conditions including tobacco smoking.

An occupational history should include a history of exposure to asbestos and exposure to other substances of real or potential medical significance. Performance criteria for these procedures, including the periodicity of subsequent medical surveillance, should be developed by NIOSH in consultation with OSHA and professional societies and organizations concerned with the diagnosis and prevention of respiratory diseases. The committee does not recommend comprehensive annual medical examinations as presently required. Sputum cytology should be evaluated in the development of an improved medical surveillance program. The committee believes that sputum cytology may prove to be a valuable supplement to X-ray evaluation.

It is also crucial that all required medical surveillance be promptly evaluated and the results reported to the employee. Furthermore, the standard should provide for periodic reporting of aggregate medical information concerning an employer's entire workforce. Results at a minimum should be displayed in a non-identifiable, aggregate format so that the employer, employees, and OSHA can see the prevalence of abnormalities possibly associated with asbestos-related disease, and also see how this prevalence has changed over time.

The committee recognizes that OSHA's recent lead standard contains a multiple physician review mechanism whereby workers can get independent medical evaluations by physicians of their choice. The lead standard also contains a medical removal protection program whereby workers can obtain special health protection where necessary, accompanied by appropriate economic protection. The committee feels that these programs are relevant to asbestos workers and should be considered as part of a new occupational asbestos standard.

Medical records generated due to the standard's medical surveillance program should be maintained for at least 40 years or for 20 years after termination of employment, whichever is longer.

*7. Other Recommendations.* The committee further recommends the following: (1) Due to the widespread current and past uses of asbestos products in the maritime and construction in-

dustries, it is vital that any new asbestos standard address these industry sectors as well as other workplaces with employees exposed to asbestos. Regulation of these industries should be structured around the principle that where work must be done using asbestos, only those employees needed to do this work should be present, and only for the minimum period of time needed to complete this work.

(2) Due to the sampling and analytical difficulties concerning asbestos, manufacturers of asbestos-containing products such as construction materials should perform detailed monitoring of exposures which could result from all foreseeable uses of their products, including misuse. This monitoring should include electron microscopy to identify fiber type mix and exposures to fibers less than 5  $\mu\text{m}$  in length. This monitoring data should accompany these products downstream so the users not only know that asbestos exposures may occur, but also know the nature of potential exposures. This monitoring data could, if appropriate, avoid the need for small employers who use asbestos-containing products to have to conduct monitoring on their own.

(3) Due to the fact that other agencies regulate occupational exposures to asbestos (such as the Mine Safety and Health Administration), these agencies should be urged to participate in the development of a new standard and adopt this new standard.

(4) Because cigarette smoking enhances the carcinogenic effect of asbestos exposure on the lung, particular emphasis should be placed on this in any educational program developed under a new standard.

# **OCCUPATIONAL RESPIRATORY DISEASES**

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## ASBESTOSIS

*John M. Dement  
James A. Merchant  
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### INTRODUCTION

Occupational exposure to asbestos minerals constitutes a major health hazard in the United States and in most industrialized nations of the world. Because of their unique properties such as resistance to heat and chemical attack, asbestos minerals have long been used by man. Finnish potters are known to have used soils containing anthophyllite asbestos dating from 2500 B.C. (103). Use of asbestos in lamp wick was described by Theophrastus, Strabo, and Plutarch. Herodotus (456 B.C.) described cremation clothes made of woven asbestos. Marco Polo described tablecloths of asbestos seen during his journeys (66).

Despite early uses, large scale use of asbestos came with industrialization and particularly the steam engine which required heat resistant materials for packings and seals. The first asbestos textile mill in the United States began production in about 1896. Today, commercial uses of asbestos are countless and nearly every manufacturing sector may be involved with production or use of asbestos-containing products.

The term "asbestos" is applied to a group of naturally occurring fibrous silicate minerals. Although many minerals are fibrous in nature, only six are regulated by Occupational Safety and Health Administration (OSHA) standards. These minerals fall into two major mineralogical subdivisions: chrysotile, which belongs to the serpentines; and the amphiboles, including crocidolite, asbestiform actinolite, asbestiform tremolite, amosite, and anthophyllite. Only amosite, chrysotile, and crocidolite are of economic importance. Chrysotile is basically a sheet silicate mineral rolled into itself to form a hollow tube. This tube constitutes the basic fibril of chrysotile.

All amphibole asbestos types are similar in crystal structure: they consist of double chains of linked silicon oxygen tetrahedra between which metallic ions are sandwiched (128). Chemical composition and trace metal contamination (Cr, Co, Mn, Ni associated with chrysotile) of asbestos fibers may vary considerably between deposits from different mining regions (43).

More than 90% of all asbestos used in the United States is of the chrysotile variety. Total U.S. consumption of asbestos in 1977 was 610,000 metric tons, down from peak consumption of 795,000 metric tons in 1973 (12). By contrast, only 93,000 metric tons were produced in U.S. mines and mills; Canada furnished 95% of all imported raw asbestos fiber. U.S. asbestos consumption by end use for 1978 is shown in Table II-9. Asbestos cement products constitute the major use of asbestos followed closely by floor products or materials used in the construction industry. Materials containing asbestos have been extensively used in construction and shipbuilding for purposes of fireproofing and for decoration. These have often been applied by spray application.

### DEFINITION

Asbestosis is the name of the pneumoconiosis produced by the inhalation of asbestos fibers. It is characterized by diffuse interstitial fibrosis of the lung parenchyma, often accompanied by thickening of the visceral pleura and sometimes calcification of the pleura. Clinical findings include dyspnea on exertion, non-productive cough, rales at the lung bases, bronchi, and in advanced cases, finger clubbing. Lung function measurements usually demonstrate a restrictive impairment with reduced diffusing capacity.

**Table II-9**  
**U.S. ESTIMATED ASBESTOS CONSUMPTION IN 1978 BY END USE CATEGORY**

Product	Consumption (Metric Tons)			
	Chrysotile	Crocidolite	Amosite	Anthophyllite
Asbestos cement pipe	119,800	23,300	2,700	
Asbestos cement sheet	28,400		800	
Flooring products	122,400			
Roofing products	58,200	100		
Packing and Gaskets	23,200	100		
Thermal insulation	14,300			
Electrical insulation	3,200			
Friction products	81,000			600
Coating and compounds	29,100			
Plastics	5,300	500		
Textiles	5,700			
Paper	28,400	700		
Other	33,100			2,100
<b>Total</b>	<b>552,100</b>	<b>24,700</b>	<b>3,500</b>	<b>2,700</b>

Source: (12)

### CAUSATIVE AGENTS

Asbestosis is perhaps the most widely studied of the known occupational hazards; however, its mechanisms are still not fully understood. Both clinical and epidemiological data have conclusively shown that asbestos is associated with asbestosis and respiratory cancer in man. Animal bioassay data fully support these findings and suggest that pathological responses to asbestos may be more related to physical characteristics of the fibers than to chemical composition. Animal data have shown a wide variety of fibrous minerals and small diameter glass fibers to be capable of producing tumors upon pleural injection or implantation (110)(111)(139). Interstitial fibrosis has also been produced in animals intratracheally injected with small diameter glass fibers (63).

### POPULATION AT RISK

Asbestos has over 3,000 commercial uses and is ubiquitous in the general environment. Because of the mineral's resistance to thermal and chemical degradation, exposures may take place starting from initial mining of the fibers through manufacture, use, and eventual burial of asbestos containing waste.

Mining and milling of asbestos in the United States is not extensive: fewer than a thousand workers are employed (148). However, amphibole

minerals and, to a lesser extent, serpentines, are sometimes found as contaminants of other types of ore bodies, such as talc, vermiculite, crushed stone aggregates, and in ores from various metal mining operations (19)(64)(115) (140). There have been no systematic studies of mining operations in the United States to identify specific ores containing asbestos as contaminants and the degree to which workers are exposed.

Estimates of the number of workers exposed to asbestos in primary manufacturing of asbestos products are given in Table II-10. In the primary manufacturing sector approximately 18,000 workers are estimated to be potentially exposed; however, this number could be as high as 37,000 (17). A large variety of asbestos products and materials produced in primary manufacturing are fabricated and processed with other materials in secondary industries to produce the more than 3,000 end products containing asbestos. The secondary fabrication and processing industry is very large and has been estimated to employ more than 300,000 workers (17).

By far the largest number of workers with potential asbestos exposures may be found in industries which utilize asbestos products such as the construction industry, the automobile servicing industry (including remanufacturing of

**Table II-10**  
ESTIMATES OF WORKERS EXPOSED  
TO ASBESTOS IN PRIMARY  
MANUFACTURING

Manufacturing Sector	Estimated Number of Potential Exposed Workers
Asbestos cement pipe	1,755
Asbestos cement sheet	980
Friction materials	5,605
Floor coverings	3,500
Asbestos paper products	2,120
Packing and gaskets	1,125
Paint, coating and sealant	815
Asbestos textiles	1,800
Total	17,700

Source: (17)

asbestos containing parts), and the shipbuilding and repair industry. In the construction industry, including those doing demolition and repair, an estimated 180,000 to 408,000 workers are potentially exposed to asbestos. The automobile servicing industry includes brake and clutch servicing garages, rebuilding and refacing friction components, and repackaging of friction products. Within this sector, 2 million workers are potentially exposed to asbestos (17). Approximately 3,800 workers are potentially exposed to asbestos in shipbuilding and repair.

A total of 2.3 to 2.5 million workers are estimated to be currently (potentially) exposed to asbestos. However, because of the long latency (20 to 30 years) required before asbestos related diseases become clinically manifest, past asbestos workers must also be considered at risk. These estimates are especially difficult to develop and are subject to controversy (29). Nonetheless, large numbers of previous asbestos workers are now completing their latency period and are at risk of asbestos related diseases.

## EPIDEMIOLOGY

### Early Observations

#### Asbestosis

The first well documented case of asbestosis was reported by H. Montague Murray in 1906, although there were several anecdotal reports prior to this time (66)(95). Murray documented

a case of pulmonary fibrosis at autopsy in a worker engaged in the production of asbestos textiles. This worker reported that he was the sole survivor of 10 men who started with him in the carding room; the others had died.

Following the report by Murray, Pancoast et al. (1917) reported 17 cases of pulmonary fibrosis in a Pennsylvania plant (105). In 1924, Cooke published another detailed autopsy report of a 33-year-old woman suffering from asbestosis (14). Necropsy findings included pulmonary fibrosis, pleural thickening, pleural calcification, and heart enlargement. Further cases were reported by Mills in 1930, Donnelly (1933), Lynch and Smith (1931), Seiler and Gilmour (1931), Wood and Gloyne (1930), Oliver (1927), Simson (1928), Stewart (1928), and Pancoast and Pendergrass (1926) (21)(70)(88)(104)(106)(120)(134)(141)(164). By 1930, more than 75 asbestosis cases had been reported in the literature.

Early case reports stimulated concern and in 1928 the first detailed epidemiologic study of asbestos workers was undertaken by the Ministry of Labour in Great Britain. Results were published by Merewether and Price in 1930 (84). This was a cross-sectional chest x-ray study of 363 workers engaged in production of asbestos textiles. Of this group, 95 (26.2%) were found to have pulmonary fibrosis and the prevalence of fibrosis with 20 or more years employment was over 80%.

In the United States, Donnelly (1936) reported a cross-sectional chest x-ray study of 151 asbestos workers which found a pulmonary fibrosis prevalence of 59% among workers employed 4 years or more (22). Schull (1936) reported chest x-ray studies of 100 workers dismissed from North Carolina asbestos plants due to disability and found a 55% prevalence of moderate or advanced asbestosis (131).

In 1937 the U.S. Public Health Service undertook the first detailed epidemiologic study of asbestos workers in the United States with results published by Dreessen et al. in 1938 (23). A total of 511 employees were studied in this cross-sectional study and worker exposures were estimated by the impinger method. A relationship was found between extent of asbestos exposure and clinical symptoms of asbestosis although many workers had only short periods of exposure at the time of the study. This study resulted in a recommended occupational exposure

limit of 5 million particles per cubic foot of air (mppcf) in the United States.

### *Lung Cancer and Mesothelioma*

The first indication that asbestos might be a human carcinogen came in 1935. Lynch and Smith (in the United States) and Gloyne (in England) independently reported three cases of lung cancer detected during autopsy studies of asbestos workers (34)(71). All three workers had died of asbestosis. Other case reports followed by Egbert and Geiger in 1936, Gloyne in 1936, and Nordmann in 1938 (26)(33)(102). In the 1947 annual report of the Chief Inspector of Factories in England, Merewether stated that of 365 asbestosis deaths, 65 (17.8%) also had cancer of the lung at autopsy (83). This compared to a prevalence of lung cancer of only 1.3% for cases certified at death as having silicosis.

Despite early suggestions, the first detailed epidemiologic study to conclusively demonstrate an association between asbestos exposure and lung cancer was not published until 1955 by Doll (20). Doll studied the mortality experience of a cohort of 113 asbestos textile workers employed more than 20 years. Among this group, 11 lung cancer deaths were observed compared to only 0.8 expected—based on the mortality experience of England and Wales.

Asbestos exposure is associated with mesothelial tumors of pleural and peritoneal tissues. Lee and Selikoff have reviewed early reports associating asbestos exposures and mesothelioma (66). The first cases were reported in 1946 by Wyers (165). However, conclusive evidence of an association between asbestos exposure and mesothelioma was not available until 1960 when Wagner et al. reported 33 pleural mesotheliomas in the crocidolite mining area of South Africa (152).

### *Mortality*

Epidemiologic studies have repeatedly demonstrated an association between asbestos exposure and increased mortality due to asbestosis, lung cancer, pleural and peritoneal mesothelioma, and gastrointestinal cancer. In some studies, asbestos exposure has also been associated with increased risks for laryngeal cancer and cancer of the buccal cavity and pharynx. Table II-11 contains a brief summary of important mortality studies and significant findings. In this section, mortality studies are reviewed with emphasis on

asbestosis and lung cancer risk differences by fiber type, industry, and smoking patterns.

### *Mixed Fiber Exposures*

In most plants processing asbestos, several different types of asbestos may be used or have been used in the past. Typically, chrysotile and one or more amphiboles are used.

Asbestos insulation workers have been extensively studied in the United States and other countries. Selikoff et al. studied the mortality experience of 632 insulation workers followed between 1943 and 1962 and observed 45 lung cancer deaths whereas only 6.6 were expected (123). Of the 255 deaths in this cohort, 28 (11%) were due to asbestosis and 3 (1.2%) to mesothelioma. An SMR of 309 was observed for cancer of the stomach, colon, and rectum (although it was based on a small number of observed cases).

A much larger cohort of 17,800 insulation workers was followed by Selikoff et al. between 1967 and 1976 (126)(127). Among this cohort, 2,271 deaths were observed including 429 lung cancers (SMR-406), 78 asbestosis deaths, and 49 deaths due to mesotheliomas. Significant increased mortality was also observed for cancers of the esophagus, stomach, colon-rectum, larynx, buccal cavity and pharynx, and kidney. Only 2 of the 78 asbestosis deaths occurred prior to 20 years from onset of employment, based on death certificate information. Review of all available autopsy, surgical, and clinical material indicated an additional 90 deaths were due to asbestosis, 57 to lung cancer, and 126 to mesothelioma.

Elmes and Simpson studied the mortality of 162 insulation workers in Belfast between 1940 and 1975 (27)(28). Among this cohort, 122 deaths were observed including 16 (13.1%) due to asbestosis and 13 (10.7%) to mesothelioma. A large excess due to respiratory cancer was observed.

There are several important studies of mortality among textile workers exposed to mixed asbestos types. In an early study in the United States published in 1963, Mancuso and Coulter observed more than a threefold excess risk of lung cancer among workers producing textile and friction products (73). Fourteen percent of 195 deaths were due to asbestosis and 2 (1%) were due to mesotheliomas.

Mortality among employees in the plant initially studied by Doll in 1955 has been in-

**Table II-11**  
**SUMMARY OF MORTALITY STUDIES OF ASBESTOS EXPOSED POPULATIONS**

<b>Author(s)</b>	<b>Date</b>	<b>Study Population</b>	<b>Fiber Type</b>	<b>Study Design</b>	<b>Summary of Important Findings</b>
Doll	1955	113 textile workers employed 20 or more years	Mixed	Retrospective cohort 1922-1953	11 lung cancers observed versus 0.8 expected, 14 death certificates mentioned asbestosis.
Mancuso and Coulter	1963	1,495 workers producing textile, friction products	Mostly chrysotile	Retrospective cohort, 1940-1960	28 asbestosis deaths, 19 lung cancers observed versus 5.6 expected, 5 peritoneal neoplasms (2 were mesotheliomas).
Selikoff, Churg and Hammond	1964	632 insulation workers with 20 or more years employment	Mixed	Retrospective cohort, 1943-1962	12 asbestosis deaths, 45 lung cancers observed versus 6.6 expected. Increased gastrointestinal cancer, 3 pleural mesotheliomas.
Knox et al.	1965, 1968	1,014 textile workers	Mixed	Retrospective cohort, 1922-1966	27 lung cancers observed versus 10.75 expected, 42 with asbestosis on death certificate. Authors suggested reduced risks after controls added in 1933.
Newhouse	1969, 1973	4,500 textile workers	Mixed	Retrospective cohort, 1933-1968	Significant excesses for lung cancer among workers in highest exposure category; 24 mesotheliomas among males.
Newhouse et al.	1972	922 female textile and friction product workers	Mixed	Retrospective cohort, 1942-1968	14 lung cancers observed versus 0.5 expected in those working 2 years in highest exposure jobs. Approximately threefold excess of respiratory disease mortality in this group. Overall 1 mesothelioma.

**Table II-11**  
**SUMMARY OF MORTALITY STUDIES OF ASBESTOS EXPOSED POPULATIONS (Continued)**

<b>Author(s)</b>	<b>Date</b>	<b>Study Population</b>	<b>Fiber Type</b>	<b>Study Design</b>	<b>Summary of Important Findings</b>
Selikoff, Hammond and Churg	1968	370 insulation workers with >20 years employment	Mixed	Retrospective cohort, 1963-1967	Observed strong interactive effect between asbestos exposure and smoking for lung cancer; 10 mesothelioma deaths observed and 15 asbestosis deaths.
Elmes and Simpson	1971, 1977	162 insulation workers	Mixed	Retrospective cohort, 1940-1975	16 asbestosis deaths, 13 mesotheliomas. Large excess risk for respiratory cancer throughout follow-up period.
McDonald, et al.	1971, 1974, 1979, 1980,	11,379 asbestos miners and millers	Chrysotile	Retrospective cohort, 1926-1975	Among those achieving >20 years latency, overall lung cancer SMR = 125, with 42 pneumoconiosis deaths and 11 mesothelioma deaths. Linear dose-response observed for lung cancer and pneumoconiosis.
Enterline and Henderson	1972, 1978	1,075 retired asbestos product worker's	Chrysotile and amphiboles	Retrospective cohort, 1941-1973	Lung cancer SMR = 270; 19 asbestos deaths. Linear dose-response observed for lung cancer with SMR = 198 at 62 mppcf-yrs. and SMR = 778 at 976 mppcf-yrs.; 2 mesothelioma deaths.
Selikoff et al.	1973, 1979	17,800 insulation workers	Mixed	Retrospective cohort, 1967-1976	429 lung cancers observed versus 105.6 expected; 78 asbestosis deaths and 49 mesotheliomas.
Meurman et al.	1974	1,092 asbestos mine and mill workers	Anthophyllite	Retrospective cohort, 1936-1974	21 lung cancers observed versus 13 expected; 13 asbestosis deaths but no mesotheliomas. A strong interactive effect on lung cancer with smoking and asbestos exposure was observed.

**Table II-11**  
**SUMMARY OF MORTALITY STUDIES OF ASBESTOS EXPOSED POPULATIONS (Continued)**

Author(s)	Date	Study Population	Fiber Type	Study Design	Summary of Important Findings
Peto et al. and Peto	1977, 1979	1,106 textile workers employed >10 years	Mixed	Retrospective cohort	36 respiratory cancers observed versus 19.3 expected among those only employed in controlled areas. Significant excess of non-malignant respiratory diseases.
Weiss	1977	264 paper and millboard workers	Chrysotile	Retrospective cohort, 1945-1974	2 asbestosis deaths among a total of 66 deaths. No excess of lung cancer but numbers were small; no mesotheliomas reported.
Jones et al.	1976, 1979	1,088 gas mask workers during WW II	Crocidolite	Retrospective cohort, 1939-1976	12 lung cancers observed versus 6.3 expected in women; 17 mesothelioma deaths. Linear dose-response for mesothelioma with employment duration; 3 mesotheliomas observed among those exposed 5-10 months.
Edge	1976, 1979	429 shipyard workers with pleural plaques	Mixed	Prospective follow-up 1968-1974	19 bronchogenic cancers observed versus 4.0 expected; 23 mesotheliomas observed. Shipyard workers with plaques had 2.5 times lung cancer risk when compared to matched controls without plaques.
Hughes and Weill	1979	5,645 asbestos cement workers >20 years latency	Chrysotile and crocidolite	Retrospective cohort, 1940-1973	23 lung cancers observed versus 9.3 expected among those with cumulative fiber exposures >100 mppcf/yr.; 2 pleural mesotheliomas observed versus 4.4 expected among those not exposed to crocidolite.



**Table II-11**  
**SUMMARY OF MORTALITY STUDIES OF ASBESTOS EXPOSED POPULATIONS (Continued)**

<b>Author(s)</b>	<b>Date</b>	<b>Study Population</b>	<b>Fiber Type</b>	<b>Study Design</b>	<b>Summary of Important Findings</b>
Sheers	1979	410 dockyard workers with pleural plaques or pleural fibrosis	Mixed	Prospective follow-up 1967-1976	6 mesothelioma deaths among those with plaques and 2 with only pleural fibrosis. Author suggested pleural plaques are of greater biological significance than simply a marker of exposure.
Seidman, Selikoff and Hammond	1979	820 men producing insulation between 1941-1945	Amosite	Retrospective cohort, 1961-1975	83 lung cancers observed versus 23.9 expected. Among 61 men employed <1 month, 3 lung cancers observed versus 1.3 expected. 4 mesotheliomas by death certificate diagnosis but an additional 10 identified using necropsy data. 15 deaths observed due to asbestosis.
Hammond, Selikoff	1979	12,051 insulation workers with >20 years latency	Mixed	Retrospective cohort, 1967-1976	Asbestos workers who did not smoke had a fivefold risk of lung cancer compared to nonsmoking controls. Smoking asbestos workers had 53 times the lung cancer risk of nonasbestos exposed persons who also did not smoke.
Robinson, Lemen and Wagner	1979	3,276 workers producing textile, friction products	Mostly chrysotile	Retrospective cohort, 1940-1975	Overall lung cancer SMR = 136 for males and 824 among females. Some increasing trends in lung cancer with employment duration. Large excesses due to asbestosis. 17 mesothelioma deaths observed.

Table II-11

## SUMMARY OF MORTALITY STUDIES OF ASBESTOS EXPOSED POPULATIONS (Continued)

Author(s)	Date	Study Population	Fiber Type	Study Design	Summary of Important Findings
Nicholson et al.	1979	544 chrysotile miners and millers, >20 years employment	Chrysotile	Retrospective cohort, 1961-1977	28 lung cancers observed versus 11.1 expected; 26 cases of asbestosis observed; 1 pleural mesothelioma observed.
Dement et al.	1980	768 textile workers	Chrysotile	Retrospective cohort, 1940-1975	26 lung cancers observed versus 7.47 expected; 15 asbestosis deaths and 1 mesothelioma death. Linear dose-response for lung cancer with SMR = 223 at cumulative exposures <30 fiber/cc x yrs.
Brown, Dement, and Wagoner	1979	398 talc miners and millers	Anthophyllite and tremolite	Retrospective cohort, 1947-1975	9 lung cancers observed versus 3.3 expected. Significant excess due to nonmalignant respiratory diseases; 1 mesothelioma death.

vestigated by Knox et al. (59)(60), and more recently by Peto et al. (108)(109). Peto studied 1,106 men and women who had worked 20 or more years in asbestos exposed areas. Among those who were first employed after 1933 (when control regulations were enacted), 31 lung cancer deaths were observed whereas 19.3 were expected. Additionally, 35 deaths were observed due to nonmalignant respiratory disease versus 25 expected, and there were 5 deaths due to pleural mesothelioma. Dust exposures in this plant were reported to be generally above 5 fiber/cc until about 1970.

Newhouse (96)(97) and Newhouse et al. (98) have studied patterns of mortality among 4,600 male and 922 female workers in a plant which chiefly produced asbestos textiles but later asbestos insulation products. Exposures were classified as low to moderate (5-10 fibers/cc) and severe (>10 fibers/cc). Among males, there were 46 mesothelial tumors and an SMR for lung cancer of 538 was observed for those employed more than ten years in the severe exposure group. In those with lowest exposure, a lung cancer SMR of 154 was observed. Deaths from chronic respiratory diseases were 1.8 times expected in the highest exposure group. A remarkable cancer SMR was observed among females in the highest exposure group (21 observed versus 0.8 expected). Both males and females were found to have smoked more than the comparison population; however, this could only account for 10% to 20% of the observed excess lung cancer mortality.

The asbestos cement product industry is one of the largest consumers of asbestos in the United States. In addition to their asbestos exposure, workers in this industry may also be exposed to low levels of crystalline silica and other materials associated with cement dust. Weill et al. reported mortality patterns among 5,645 asbestos cement product workers with a minimum of 20 years since initial employment (156). Exposures for the cohort were estimated and expressed as mppcf  $\times$  yrs. Among those exposed to greater than 100 mppcf  $\times$  yrs., 23 lung cancers were observed versus 9.3 expected. No excess lung cancer risk was reported among those with cumulative exposures less than 100 mppcf  $\times$  yrs. Two pleural mesothelioma deaths were observed. Weill et al. reported that exposure to crocidolite in addition to the (predominant)

chrysotile used in cement products increased the lung cancer risk in comparison to chrysotile exposure alone. The unusually low SMRs for all causes regardless of exposure category suggest that cohort follow-up and death certificate ascertainment was less complete than desired.

### *Crocidolite*

Wagner et al., in 1960, reported 33 pleural mesotheliomas among men working in crocidolite mines and mills and the population living in the vicinity of these mills in the Northwest Cape Province of South Africa (152). The high incidence of mesotheliomas in this area has been confirmed by other investigations (13)(39)(155).

Crocidolite was commonly used in the production of gas mask canisters during World War II and mortality among these workers has been investigated. Jones et al. studied the mortality of 1,088 workers exposed between 1940 and 1945 and followed through 1976 (46)(47). Twenty-two pleural and 7 peritoneal mesotheliomas were observed and a linear relationship was observed between employment duration and the risk of mesothelioma. There was also a modest excess of bronchial carcinoma. Similar results have been reported by McDonald and McDonald who studied a smaller cohort of gas mask workers in Canada and found that 7% of all deaths were due to mesotheliomas (75).

### *Amosite*

Mortality patterns among a cohort of workers producing amosite asbestos insulation between 1941 and 1945 have been reported by Selikoff et al. (125) and more recently by Seidman et al. (118)(119). This group of 820 men were observed over a 35 year period during which 528 deaths occurred: by death certificate information 15 (2.8%) were due to asbestosis and 1 was due to mesothelioma. Review of available surgical, pathological, and clinical data for this group identified 13 additional mesotheliomas and 15 additional cases of asbestosis not listed on death certificates. Overall there were 83 lung cancers observed whereas 23.1 were expected and among those employed less than one month, 3 lung cancers were observed versus 1.3 expected. Anderson et al. have observed four confirmed cases of mesothelioma among household contacts of workers at this plant (1).

### *Anthophyllite and Tremolite*

The only location in the world where anthophyllite has been commercially mined and processed is Finland. These ores are also known to contain smaller quantities of tremolite. Mortality among workers in two Finnish mines and mills has been studied by Meurman et al. (86) (87). In their first report, 1,092 workers were followed from 1936 until 1974. A relative risk for lung cancer of 1.6 was observed and there were 13 (5.2%) asbestosis deaths but no deaths due to mesothelioma. Their subsequent study concerned 793 workers with known smoking histories with 10 additional years of follow-up. A relative risk for lung cancer of 19 was observed for smoking asbestos workers and 1.6 for asbestos workers who did not smoke. Asbestosis mortality was found to be equally frequent among smokers and nonsmokers. All lung cancer cases with more than 10 years of exposure were also found to have asbestosis.

### *Chrysotile*

Chrysotile is the major asbestos fiber type used in the United States, but most of this fiber is imported from Canada. The mortality of Quebec chrysotile miners and millers has been extensively studied by McDonald et al. (76) (79-81). The most recent report for this cohort included 10,939 men who had been employed one or more months and followed between 1926 and 1975. An overall SMR for lung cancer of 125 was observed; 42 deaths were due to asbestosis and 11 to mesothelioma. A nearly linear dose-response relationship was reported for lung cancer. Increased mortality was also observed for cancer of the stomach and esophagus but no other gastrointestinal sites. Similar patterns of lung cancer and asbestosis mortality have been reported by Rubino et al. in Italian chrysotile miners and millers where an SMR for lung cancer of 206 was observed among those with sufficient latency (117).

The McDonald et al. studies demonstrated a low lung cancer risk even in the highest exposure group. Nicholson et al. have reported larger excesses from lung cancer and asbestosis in their study of chrysotile miners and millers in Quebec (99). This latter study cohort consisted of 544 miners and millers with at least 20 years seniority and followed between 1961 and 1977. A total of 28 lung cancers were observed versus 11.1 expected (SMR = 252). There were 30

deaths due to noninfectious respiratory diseases whereas only 6.7 were expected. Of these 30 deaths, 26 were due to asbestosis. Only one mesothelioma (pleural) was observed.

Mortality among chrysotile asbestos miners and millers in the Urals has been investigated by Kogan et al. (61). The overall cancer mortality risk was found to be 1.6 times that for the general male population and was higher in mining than in milling. Among males, the relative risk for lung cancer was 2.0 and ranged from 1.4 to 2.1 for females. The lung cancer risk was considerably greater in older age groups having the longest latency. No mesotheliomas were reported; however, Kogan et al. attributed this to insufficient experience of pathologists in that geographic area (61). Nonetheless, the low mesothelioma risk is consistent with other studies of chrysotile-exposed populations.

There have been several studies of factory populations exposed only to chrysotile. Weiss studied a small cohort of 264 workers in a plant producing asbestos millboard and reported no excess cancer mortality (160). However, there were only 66 deaths (2 of which were due to asbestosis) and cancer latency was not taken into account in the analysis.

A facility manufacturing asbestos textile, friction, and packing products has been studied by Robinson et al. (113). Chrysotile constituted over 99% of the total quantity of asbestos processed per year in this plant except during World War II; the remaining 1% was crocidolite and amosite. The cohort consisted of 2,722 males and 544 females followed between 1940 and 1975. Among males, an overall lung cancer SMR of 135 was observed but among females the excess lung cancer risk was much higher with an overall SMR of 824. There were 76 deaths in males due to noninfectious respiratory disease but only 16.4 expected. Again, the chronic respiratory disease risk was higher among females with an SMR of 1,555. There were 4 mesotheliomas among females and 13 in males.

Dement et al. have reported mortality among a cohort of asbestos textile workers exposed only to chrysotile (18). This cohort consisted of 768 white males employed at least 6 months and followed between 1940 and 1975. There were 26 lung cancers observed versus 7.47 expected. Of the 191 deaths in this cohort, 15 (7.9%) were due to asbestosis or pulmonary fibrosis and 1 (0.5%) was due to a peritoneal

mesothelioma. Linear relationships were demonstrated between cumulative fiber dose and the risk of mortality for lung cancer and noninfectious respiratory diseases. An SMR for lung cancer of 223 was observed for the lowest cumulative exposure category of less than 30 fibers/cc  $\times$  years.

### *Fibers and Asbestos-like Contamination of Other Minerals*

Both serpentines and amphiboles may be found as contaminants in other mined and processed ores and may result in significant fiber exposures to workers in these operations.

Fibers and cleavage fragments of fibrous grunerite occur where ore from some iron formations are crushed and comminuted and have been found in high concentrations in Lake Superior as a result of mining and milling operations (64). Gillam et al. studied mortality among gold miners exposed to cummingtonite-grunerite and found a threefold excess risk of lung cancer and a twofold excess of nonmalignant respiratory disease, excluding influenza and pneumonia (32). However, workers in this mine were also exposed to silica. McDonald et al., in a subsequent study of the same mine, examined the mortality experience of persons with at least 21 years of employment with the company (78). This study demonstrated excess mortality due to pneumoconiosis (mainly silicosis), tuberculosis, and heart disease but no overall excess of malignant diseases was found. However, when the population was stratified by exposure, respiratory cancer was elevated (but was not statistically significant) in the highest exposure group.

Commercial talc deposits are sometimes found to contain serpentines (chrysotile, antigorite, and lizardite) and fibrous and nonfibrous amphiboles. Kleinfeld et al. demonstrated significantly increased proportionate mortality due to lung cancer and nonmalignant respiratory disease among talc miners and millers in New York State exposed to fibrous anthophyllite and fibrous tremolite (53)(58). Brown et al. have reported a further mortality of talc miners and millers in one company mining this same ore body (9). This cohort consisted of 398 workers followed between 1947 and 1975. Among this cohort, 10 respiratory cancers were observed whereas only 3.5 were expected. Approximately a threefold excess risk of nonmalignant respiratory disease was reported; however, only one

death due to mesothelioma was observed.

### *Effects of Smoking*

Smoking and asbestos exposure are more than additive in their combined ability to increase the risk of lung cancer. Hammond et al. reported results of their 10-year follow-up of 8,220 asbestos insulation workers with known smoking status (38). The mortality experience of these workers was compared with that expected among smokers and nonsmokers of the American Cancer Society's prospective cancer prevention study. Asbestos workers who did not smoke showed approximately a fivefold risk of lung cancer compared to the nonsmoking control population. On the other hand, a more than sixtyfold risk of lung cancer was observed for smoking asbestos workers compared to nonsmoking controls. A similar multiplicative effect was observed by Selikoff et al. among a factory cohort producing amosite insulation (129).

Although less striking, cigarette smoking may also contribute to the risk of death due to asbestosis. Hammond et al. reported that asbestosis death rates of smoking asbestos workers were 2.8 times as high as that of nonsmoking asbestos workers. Meurman found less association between asbestosis mortality and smoking; he reported 7 of 42 asbestosis deaths among nonsmokers (86).

### *Mortality and Pleural Radiographic Changes*

The relationship between pleural thickening and calcification and subsequent mortality is important insofar as surveillance of asbestos workers is concerned. Edge studied the mortality of 429 shipyard workers with plaques and compared this to matched controls without plaques (25). Among those with plaques, 23 mesotheliomas were observed and workers with plaques had 2.5 times the lung cancer risk of those without plaques. Sheers observed 6 mesothelioma deaths among 410 dockyard workers with plaques, but he found just 2 mesotheliomas in those with only pleural fibrosis (130). Neither of these studies established causality between pleural changes and subsequent development of mesothelioma or lung cancer because neither asbestos exposure or latency were controlled for in the analysis. Meurman has shown that anthophyllite asbestos workers have a high prevalence

of pleural changes but a minimal mesothelioma risk (86)(87). However, plaques and pleural thickening do indicate an asbestos exposure and this fact alone places the workers at an increased risk for lung cancer and asbestosis.

### *Respiratory Morbidity*

All types of asbestos have been shown in epidemiologic studies to be associated with asbestosis, pleural thickening, and pleural calcification. Available evidence from cross-sectional and prospective respiratory disease studies provide little evidence that any one type of asbestos is more biologically active than another insofar as x-ray or clinical changes are concerned (149) (164). These findings are fully supported by animal bioassay data.

Important epidemiologic studies of respiratory morbidity among asbestos workers are summarized in Table II-12. In these studies, various objective measures of effect or disease outcome have been used including chest roentgenographs, spirometry, measures of diffusion capacity, and chest auscultation. Subjective data such as respiratory symptoms obtained by questionnaire have also been used. In the diagnosis of "definite asbestosis," most studies have relied upon combinations of objective and subjective data.

### *Mixed Fiber Exposures*

Early cross-sectional studies of chest roentgenographs of asbestos workers by Merewether and Price, Donnelly, Schull, and Dreessen et al. demonstrated a striking prevalence of pulmonary fibrosis of as much as 80% for workers employed more than 20 years (22)(23)(84)(131).

Several studies have been conducted among insulation workers. Selikoff et al. studied chest films of 1,117 insulation workers exposed to chrysotile and amosite (122)(124). A 50% overall prevalence of pulmonary fibrosis was observed increasing to 90% among those employed more than 30 years. Pleural calcification showed an increasing prevalence with latency reaching 57.9% at 40 years since initial employment. Pleural fibrosis (thickening) occurred earlier than calcification. Murphy et al. also studied shipyard insulation workers and found a prevalence of asbestosis 11 times that of age matched, non-exposed controls (92)(93). Exposures among this group were thought to be low.

Cross-sectional data from an asbestos textile plant processing a mixture of asbestos types were

used by the British Occupational Hygiene Society (BOHS) in establishing occupational exposure standards (8). Among 290 workers employed after dust controls were installed in 1933, only 8 workers (2.7%) demonstrated x-ray changes considered consistent with asbestosis. Basal rales was taken as an early disease marker with a 1% risk estimated for a working lifetime of 50 years at an average exposure of 2 fibers cc. Workers at this same plant were subsequently studied cross-sectionally by Lewinsohn (67). This latter and much larger study demonstrated a significantly greater prevalence of pulmonary fibrosis; reaching 40.5% among workers employed from 30-39 years. Pleural fibrosis (thickening) was observed in 1.6% of those employed 1-9 years and in 50% of workers employed more than 40 years.

Berry et al. reported the results of a prospective study of workers employed in the same plant studied by Lewinsohn (67). This study consisted of 379 persons completing 10 or more years employment by 1971. Possible asbestosis was diagnosed based on one or more combinations of basal rales or crepitations, radiological changes, a falling transfer factor and restrictive lung function changes. Among these 379 men, 60 cases of possible asbestosis were diagnosed by the factory medical officer, whereas 85 cases were diagnosed by an independent clinician. Using plant exposure data, it was estimated that the cumulative dose necessary for a 1% incidence for crepitations, possible asbestosis, and certified asbestosis was 43 fiber/cc-yr, 55 fiber/cc-yr, and 72 fiber/cc-yr, respectively. Two cases of certified asbestosis were observed among nonsmokers and nine among ex-smokers, suggesting a contributory smoking role. Weiss reported similar findings in his study of 100 asbestos textile workers where a 24% prevalence of pulmonary fibrosis was observed in nonsmokers versus 40% for smokers (159)(161). Gregor et al. demonstrated a progression of radiological changes in asbestos workers referred to the British Pneumoconiosis Medical Panel without further asbestos exposures (36).

Lung function and chest film effects of exposure to asbestos cement dust have been studied by Weill et al. (157)(158). This study included 859 workers in two asbestos cement plants who were administered respiratory symptom questionnaires, spirometry, and chest films. Cumulative dust exposures were estimated and expressed as mppcf-yr. Both small rounded and linear opac-

**Table II-12**  
**SUMMARY OF RESPIRATORY MORBIDITY STUDIES OF ASBESTOS EXPOSED POPULATIONS**

<b>Author(s)</b>	<b>Date</b>	<b>Study Population</b>	<b>Fiber Type</b>	<b>Study Design</b>	<b>Summary of Important Findings</b>
Selikoff, Churg, and Hammond	1965	1,117 insulation workers	Chrysotile and amosite	Cross-sectional, no external controls	50% prevalence of pulmonary fibrosis. Increasing prevalence of all chest film changes with employment duration increasing to 90% prevalence at >30 years
Kiviluoto et al.	1960, 1965, 1979	Persons in Central Finland	Anthrophyllite tremolite	Case series	Pleural calcification observed in persons only secondarily exposed to asbestos. Pleural changes unrelated to lung cancer mortality.
Selikoff	1965	1,117 insulation workers	Chrysotile and amosite	Cross-sectional, no external controls	Pleural calcification showed increasing prevalence reaching 57.9% among those with 40 years since first exposure. Pleural fibrosis occurred earlier than calcification, 50% of cases were bilateral.
McDonald et al.	1972	1,015 chrysotile miners and millers	Chrysotile	Cross-sectional, no externals	Shortness of breath increased with estimated cumulative dust exposure but bronchitis showed little correlation.
Becklake et al.	1972	1,105 chrysotile miners and millers	Chrysotile	Cross-sectional, no externals	FVC found to decrease with estimated cumulative dust exposure in smokers and non-smokers. Same trends seen in FEV <sub>1</sub> . Obstructive impairment seen in high exposure group. Few trends in diffusing capacity.

**Table II-12**  
**SUMMARY OF RESPIRATORY MORBIDITY STUDIES OF ASBESTOS EXPOSED POPULATIONS (Continued)**

<b>Author(s)</b>	<b>Date</b>	<b>Study Population</b>	<b>Fiber Type</b>	<b>Study Design</b>	<b>Summary of Important Findings</b>
McDonald et al.	1974	5,082 miners and millers with chest films	Chrysotile	Mortality follow-up	Increased mortality observed for those with parenchymal changes but not in those with only pleural changes, 32 deaths observed due to all respiratory diseases versus 8 expected.
Liddell et al.	1977	267 miners and millers with chest films	Chrysotile	Prospective follow-up	During 20-year period, the following cumulative incidence was reported: small opacities 16%, pleural thickening 5.3%, pleural calcification 5.3%, obliteration of c/p angle 7.3%
Weiss	1971	100 asbestos textile workers	Unknown	Cross-sectional, no external controls	Overall prevalence of fibrosis 36% with 24% prevalence in non-smokers and 40% in smokers. None of 11 nonsmokers with exposures less than 20 years showed fibrosis.
BOHS	1968	290 asbestos textile workers	Mixed	Cross-sectional, no external controls	Basal rates used as early disease marker, 1% risk estimated for a working lifetime of 50 years at 2 fibers/cc.
Lewinsohn	1972	1,287 asbestos textile workers	Mixed	Cross-sectional, no external controls	Prevalence of pulmonary fibrosis 0% with 0-9 years exposure up to 40.5% with 30-39 years exposure. Pleural fibrosis prevalence 1.6% in 0-9 years and 50% in 40-49 years exposure group.



**Table II-12**  
**SUMMARY OF RESPIRATORY MORBIDITY STUDIES OF ASBESTOS EXPOSED POPULATIONS (Continued)**

<b>Author(s)</b>	<b>Date</b>	<b>Study Population</b>	<b>Fiber Type</b>	<b>Study Design</b>	<b>Summary of Important Findings</b>
Berry et al.	1979	379 asbestos textile workers	Mixed	Prospective follow-up	6.6% of workers had "possible" asbestosis after 16 years follow-up and an average exposure of 5 fibers/cc. Cumulative exposure for 1% incidence of "possible asbestosis" for 40 years employment estimated to be 55 fibers/cc X years.
Weill et al.	1973	908 asbestos cement workers	Mixed	Cross-sectional, no external controls	Overall prevalence of small rounded opacities 1/0 or greater was 3.1%, for small irregular opacities prevalence was 2.5%. Reduced FEV <sub>1</sub> , FEF <sub>25-75</sub> and FEV <sub>1</sub> /FVC ratio found in those with x-ray abnormalities.
Weill et al.	1975	859 asbestos cement workers	Mixed	Cross-sectional, no external controls	Prevalence of small rounded and irregular opacities, 4% in lowest exposure group and 30% in highest. Pleural changes 11% in lowest exposure group and 30% in highest. FVC and FEV <sub>1</sub> reduced in those with x-ray changes.
Weiss and Theodas	1978	98 workers age 40 or over in two plants	Chrysotile and amosite	Cross-sectional, no external controls	Prevalence of profusion (1/1) 17.5% in chrysotile workers and 16.5% in mixed fiber workers. Pleural thickening prevalence, 17.5% in chrysotile workers and 35.4% in mixed fiber workers. Smoking found to be significant factor in those exposed to amosite.

**Table II-12**  
**SUMMARY OF RESPIRATORY MORBIDITY STUDIES OF ASBESTOS EXPOSED POPULATIONS (Continued)**

<b>Author(s)</b>	<b>Date</b>	<b>Study Population</b>	<b>Fiber Type</b>	<b>Study Design</b>	<b>Summary of Important Findings</b>
Selikoff et al.	1977	485 miners and millers	Chrysotile	Cross-sectional, no external controls	10% prevalence of all radiographic abnormalities. Pleural changes seen in 3% of all workers. Prevalence of abnormalities among those employed less than 5 years was 5% with 3% being parenchyma changes (profusion $\geq 1/0$ ).
Jones et al.	1979	204 asbestos cement workers	Mixed	Prospective follow-up 1970-1976	Progression of small opacities dependent upon both average and cumulative exposure. Lung function declines were associated with smoking and cumulative exposure. Pleural abnormalities progressed more as a function of time with little association with additional exposure.
Anderson	1979	Household contacts of factory workers	Amosite	Cross-sectional, age, sex matched controls	35.9% prevalence of x-ray abnormalities compared to a 4.6% prevalence in the control group. Pleural abnormalities more prevalent than parenchymal changes.
Gamble, Fellner, and DiMeo	1979	121 talc miners and millers	Anthophyllite and tremolite	Cross-sectional, external comparison populations	Talc workers with greater than 15 years employment had increased prevalence of pleural abnormalities compared to comparison populations, FEV <sub>1</sub> and FVC reduced in association with dust and fiber exposures.

**Table II-12**  
**SUMMARY OF RESPIRATORY MORBIDITY STUDIES OF ASBESTOS EXPOSED POPULATIONS (Continued)**

Author(s)	Date	Study Population	Fiber Type	Study Design	Summary of Important Findings
Irwig et al.	1979	1,801 miners and millers with chest films	Crocidolite and Amosite	Cross-sectional, no external controls	Prevalence of pleural changes increased from 2.5% for workers with less than 1 year employment to 33.6% for workers with 15 or more years. Parenchymal changes (>1/0 ILO) found in 2.3% of workers employed less than 1 year and 26.7% in workers employed more than 15 years.
Gregor et al.	1979	119 asbestos workers referred to Pneumoconiosis Medical Panel	Mixed	Prospective follow-up	One-third of workers showed progression after 6 years follow-up and no further asbestos exposure. Progression frequency higher among those with profusion > 1/1 or 1/2 (ILO).
Rubino et al.	1979	56 retired chrysotile miners and millers surviving > 3 years	Chrysotile	Prospective follow-up	39% of persons with abnormal films (profusion > 1/0 ILO) showed progression after an average follow-up of 8 years. 7.9% of workers with normal initial films developed radiographic changes.
Murphy et al.	1971, 1978	101 shipyard pipe coverers and 95 controls	Mixed	Cross-sectional with further follow-up matched controls	Prevalence ratio of asbestosis 11 times greater than controls. Asbestosis evident after cumulative exposures of 60 mppcf-years.

ities were observed, indicating the possible role of small quantities of silica present in cement dust. Among those with a cumulative exposure less than 50 mppcf-yr, and approximately 4% prevalence of small opacities (rounded or irregular, profusion  $\geq 1/0$  was observed; the prevalence of these changes increased to 30% with an exposure of more than 400 mppcf-yr. Pleural changes were seen in 11% of those in the lowest exposure category. Both FVC and FEV<sub>1</sub> were reduced in those with x-ray changes. There was no apparent interaction effect of cigarette smoking on the development of diffuse fibrosis.

Jones et al. studied the progression of radiographic abnormalities and lung function changes among 204 asbestos cement workers between 1970 and 1976 (48). Films were read side by side in known order and ranked according to progression. These authors concluded that: (1) progression of small opacities depended upon both average and cumulative exposure; (2) declines in lung function were related to both smoking and cumulative exposure; and (3) pleural abnormalities progressed as a function of time. Disease incidence was not estimated in relation to exposure.

### *Anthophyllite and Tremolite*

Respiratory morbidity among Finnish anthophyllite miners and millers has been studied by Meurman et al. (87). Among 787 active employees, a threefold excess of dyspnea and a twofold excess of cough was observed among asbestos workers compared to controls. The prevalence of dyspnea was not found to be associated with smoking habits.

A high prevalence of pleural plaques has been reported among persons residing near anthophyllite mines and mills in Finland (51)(85). In two mining communities where mass roentgenological surveys were conducted, prevalences of pleural plaques of 9% and 6.5% were observed compared to less than 0.1% for the Finnish population.

Talc deposits found in upper New York State contain both anthophyllite and tremolite. Workers in talc mines and mills in this area have been shown to experience pulmonary fibrosis, pleural changes, and restrictive lung function changes (52)(54-57)(107)(132)(133). A recent cross-sectional study of lung function and chest

x-rays among talc workers in this area was reported by Gamble et al. (31). Compared with coal and potash miners, talc miners and millers were found to have an increased prevalence of cough and dyspnea along with reduced FEV<sub>1</sub>, FVC, and flow rates. Talc workers with more than 15 years employment were found to have a 33% prevalence of pleural calcification and pleural thickening. Recent exposures in these operations were reported by Dement and Zumwalde (19). Time-weighted-average fiber exposures were found to range from 0.8 to 16.0 fibers/cc with 12-19% identified as tremolite and 38-45% anthophyllite.

**Chrysotile**—Radiological changes, lung function, and respiratory symptoms among Canadian chrysotile miners and millers have been extensively studied by McDonald et al. (76) (77) and Becklake et al. (4). A total of 1,015 current employees were given chest x-rays, underwent pulmonary function studies, and were administered a standard British Medical Research Council Questionnaire on respiratory symptoms. Both persistent cough and phlegm (bronchitis) and breathlessness on exercise were found to increase with exposure. The prevalence of bronchitis rose to 50% among smokers in the highest dust exposure categories. The prevalence of breathlessness was not affected by smoking but rose to greater than 40% in those with cumulative dust exposures over 800 mppcf-years. The prevalence of irregular small opacities ( $>1/0$  ILO/UC) in the lowest exposure category was found to be 1.8% for the Thetford mine and 6.4% for the Asbestos mine. Prevalences increased to 26.4% for Thetford and 10.9% for Asbestos in the group with exposures more than 800 mppcf-yr. The prevalence of pleural thickening was found to be less strongly related to exposure. Among various lung function parameters measured, both FVC and FEV<sub>1</sub> declined more with exposure. Those with small opacities of category 2/1 or greater were found to have significantly reduced functional residual capacity, residual volume, and single breath diffusing capacity at rest. Only FVC and FEV<sub>1</sub> were reduced in those with earliest roentgenographic changes.

Cross-sectional respiratory disease studies have been conducted among chrysotile miners and millers in Newfoundland and Corsica (7) (121). Selikoff studied 485 current employees

of a chrysotile mine in Newfoundland and found a 5% prevalence of parenchymal abnormalities (ILO U/C  $\geq 1/0$ ) (121). This prevalence increased to 11.5% among those employed more than 10 years. The prevalence of pleural changes was less than that observed for parenchymal changes.

Boutin et al. studied chest films of 16 ex-workers of chrysotile mines and mills in Corsica which had been closed in 1965 (7). Compared with controls, chrysotile miners and millers had 2.4 times the risk of parenchymal abnormalities and 2 times the risk of pleural abnormalities. Exposure levels among those workers were extremely high, ranging from 85 to 267 mppcf.

The above studies of chrysotile asbestos workers have been cross-sectional by design and have likely underestimated risks since: (1) those who develop severe disease are likely to have already left employment, and (2) chest film changes may develop after termination of employment, or changes may be progressive without additional exposure. Liddell et al. studied chest film changes in a 20-year longitudinal study of chrysotile miners and millers (62). These authors observed a 20-year cumulative incidence for small irregular opacities of 16%, a pleural calcification incidence of 5.3%, and a pleural thickening incidence of 5.3%. Only the incidence of small opacities was strongly associated with smoking. Rubino et al. studied the progression of chest film changes among retired chrysotile asbestos miners and millers and found that 39% of those who had initial films with a profusion of 1/0 or greater, demonstrated progression without further exposure (116). Becklake et al. also studied radiological changes after withdrawal from asbestos exposure (5). Parenchymal progression was observed in 7% of the films, pleural progression in 19.8%, and both parenchymal and pleural progression in 2.3%. These changes were found to be independent of age and smoking, but parenchymal "attacks" occurred more among those with higher asbestos exposure prior to employment termination.

Relationships between radiological findings and subsequent mortality among chrysotile miners and millers have been studied by Liddell and McDonald (69). This study consisted of 4,559 whose latest film had been read according to the UICC/Cincinnati classification system with mortality follow-up from time of film assessment through 1975. Overall, this cohort ex-

perience significantly increased mortality for all causes (SMR = 144), lung cancer (SMR = 177), pneumoconiosis (31 cases), other respiratory diseases (SMR = 127), diseases of the heart (SMR = 136), cancer of the esophagus or stomach (SMR = 170), and cerebrovascular diseases. There were 5 pneumoconiosis deaths among those classified as having normal radiographs; however, the risk of death due to pneumoconiosis was 11.75 times greater among those with "less-than-normal" films. The lung cancer relative risk for those with chest film changes was 3.24 and most who died of lung cancer were found to be smokers. Small parenchymal opacities were present in most but not all persons whose deaths were attributed to lung cancer. The authors concluded that the chest radiograph was useful for surveillance of asbestos workers but was limited due to radiological progression after withdrawal from exposure and by the carcinogenic risk associated with dust retained in the lung.

## PATHOLOGY

### Pleural Plaques

Hyaline plaques of the parietal pleura occur in association with exposure to all commercial types of asbestos. They are more common than the pulmonary parenchymal lesions of asbestosis, thus their presence does not necessarily imply coexistent asbestosis. The majority occur in men, 20 years or more after first exposure. The plaques almost invariably involve the parietal pleura; less commonly they are found on the visceral pleura or parietal pericardium. They are usually bilaterally symmetrical and appear as well circumscribed, pearly white or creamy, fibrotic elevations of the pleura (Figure II-11). Their surface is smooth and glistening with either a flat, plateau-like or nodular contour. They range in size from a few millimeters to several centimeters in diameter. Most commonly they are found following the lines of the lower ribs posteriorly or on the diaphragm. On cut section, they have the consistency of cartilage. Histologically, the plaques are composed of avascular and acellular bundles of hyalinized collagen arranged in a reticulated mesh or "basket weave" pattern (Figure II-12). Some of the more nodular plaques show a whorled pattern of collagen fibers. Focal

calcification is fairly common and elastic fibers are sometimes demonstrable within the plaque (112). Although the plaques are almost acellular, lymphocytes and plasma cells may be present around blood vessels beneath the plaque. The origin of the plaque is not known; histological studies suggest an extrapleural rather than a pleural origin (145). Asbestos bodies are rarely seen in pleural plaques, though they can usually be detected in the underlying pulmonary parenchyma (40)(112). Short, uncoated fibers may be present in a proportion of plaques (40) (65). Pleural plaques rarely, if ever, undergo malignant change.

### Asbestosis

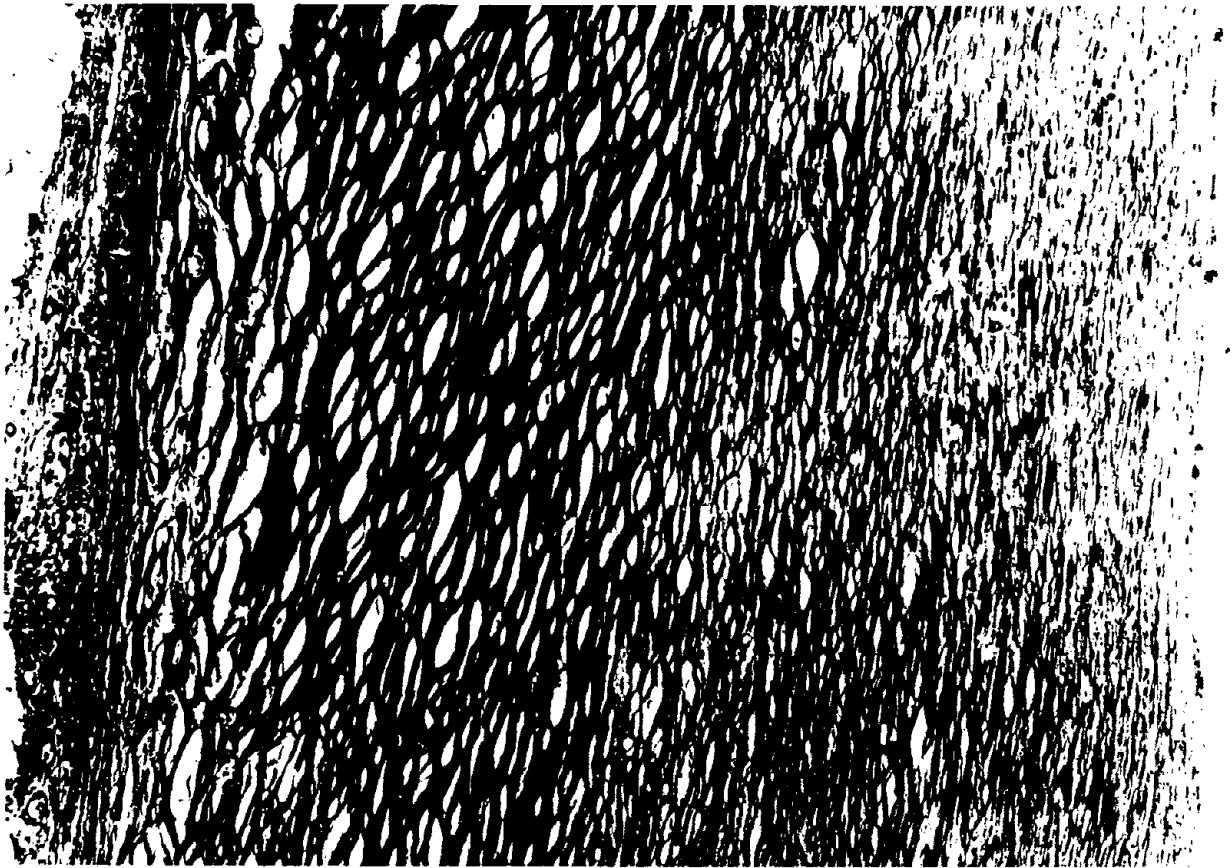
In early or mild cases of asbestosis, the lungs may be of normal size and shape; in advanced

cases, they show a marked reduction in volume. The visceral pleura is usually pale, opaque, and thickened, particularly over the lower lobes. Adhesions between the visceral and parietal pleura may be present. In the absence of other exposures, pleural pigmentation is usually slight.

The lungs may appear grossly normal in cases showing histological evidence of mild disease. However, on careful palpation, it is usually possible to detect an increased firmness of the parenchyma. With advancing disease, the lungs are dark tan in color and show a pale reticular fibrosis. Characteristically, the fibrosis is most prominent in the lower lobes and dependent parts of the upper and middle lobes. In the late stages of the disease, the lungs have firm, spongy texture and show dense fibrosis with areas of cyst formation (honeycombing). The honeycomb



**Figure 11-11.** Diaphragmatic pleura of 68-year-old ex-construction worker. Numerous dome shaped and flattened, ivory colored plaques are seen over both hemidiaphragms.



**Figure II-12. Histological section of pleural plaque. The plaque is composed of acellular bundles of collagen fibers arranged in a "basket weave" pattern. Hematoxylin and eosin  $\times 64$ .**

cysts vary in size from a few millimeters to a centimeter or more in diameter and are most prominent in the lower lobes and subpleural areas of the lungs (Figure II-13, A & B). Emphysema is unusual and, when present, is not related to asbestos exposure. Massive fibrosis is a less common feature of asbestosis and probably results from mixed dust exposure. Necrotic nodules similar to Caplan's lesions in coal workers have been described in patients with asbestosis and circulating rheumatoid factor (91).

Microscopically, the earliest lesion attributable to asbestos inhalation involves the respiratory bronchiole. Fibers deposited on the walls of respiratory bronchioles and adjacent alveoli stimulate a macrophage response. Depending on fiber size, giant cells may form. The macrophagic response is followed by the deposition of

reticulin and collagen in the walls of the respiratory bronchioles (Figure II-14). Asbestos bodies and fibers are found in association with the lesions of the respiratory bronchioles and within alveoli. A similar lesion has been described in cigarette smokers (100). The early lesion of asbestosis differs from the respiratory bronchiolitis of cigarette smokers only with respect to the presence of asbestos bodies. The diagnosis, therefore, of asbestosis depends upon the recognition of asbestos bodies within the lesion.

As the disease evolves, the fibrosis extends out to involve the walls of adjacent alveoli. Eventually, adjacent acini are affected resulting in a diffuse interstitial fibrosis (Figure II-15). With further progression of the disease, the pulmonary architecture becomes distorted. Intra-alveolar fibrosis leads to obliteration of alveolar spaces



**Figure II-13 (A).** Freeze dried whole lung section from 51-year-old male plumber exposed to asbestos lagging for 16 years. There is marked honeycombing of the mid and lower zones.

and eventually to areas of conglomerate fibrosis (Figure II-16). Despite the obliteration of alveolar spaces, the outline of the walls of the alveoli usually remain intact and can be demonstrated with elastic stains (138). Eventually, fibrous-walled (honeycomb) cysts form (Figure II-17). The cysts are lined by flattened or metaplastic epithelial cells of ciliated cuboidal, goblet, or squamous type. These changes are nonspecific and may occur in the late stages of pulmonary fibrosis, whatever the etiology. This pathogenetic

sequence of events forms the basis for a grading system developed by a committee of U.S. pulmonary pathologists assembled under the auspices of the National Institute for Occupational Safety and Health and the College of American Pathologists (16).

The above features appear to be common to all the commercially available types of asbestos. Several other types of tissue response have been described in association with asbestosis. These include chronic inflammatory cell infiltrates, desquamative interstitial pneumonia (15), and the formation of intra-epithelial eosinophilic hyaline bodies (62). These features are not specific for asbestos.

### **Asbestos Bodies and Fibers**

Two types of fibers are encountered in the lungs; uncoated fibers that resemble the inhaled particle and coated fibers or asbestos bodies. The ratio of uncoated fibers to coated bodies is high, ranging from 5:1 to 10,000:1 (10).

Asbestos bodies are an index of asbestos exposure and are considered an essential feature for the histological diagnosis of asbestosis (16). They may be formed in the lungs as early as two months after first exposure (135). Asbestos bodies tend to form on the larger fibers, i.e., those greater than  $5\mu\text{m}$  in length and result from the deposition of iron-protein complexes on the core fiber by alveolar macrophages (143). In hematoxylin and eosin stained sections they appear as golden brown segmented structures with a clear central core fiber. In Perl's iron stained sections they appear blue. The morphology of the coating is variable, with club-shaped or beaded bodies predominating (Figure II-18). Similar structures may form around other minerals such as carbon, ceramic aluminum silicate fibers, and fiberglass, and they have been termed ferruginous bodies (37)(42). They usually lack the clear central core of a typical asbestos body. These types of bodies are relatively uncommon, however, and for practical purposes, it can be assumed that a typical asbestos body contains an asbestos fiber. Although all major commercial types of asbestos can produce asbestos bodies, the majority of the core fibers, when analyzed by selected area electron diffraction, are found to be amphibole asbestos (11). Several procedures exist for the quantification and identification of fibers in tissues (11)(16)(137)(150). The majority of these fibers are too small ( $<5\mu\text{m}$  in length) to be





**Figure II-13 (B).** Roentgenogram showing marked interstitial disease with honeycombing which is most severe in the mid zones.

resolved by the light microscope. Electron microscopical studies on selected cases have shown that occupationally exposed workers have pulmonary asbestos fiber counts orders of magnitude greater than the general population (16)(163). The value of these techniques is to establish exposure and to identify the mineral type and should not be considered a substitute for more conventional diagnostic methods. Currently, the role of the short fibers in the pathogenesis of asbestosis and asbestos-associated lung cancer has not been resolved.

#### **Lung Cancer**

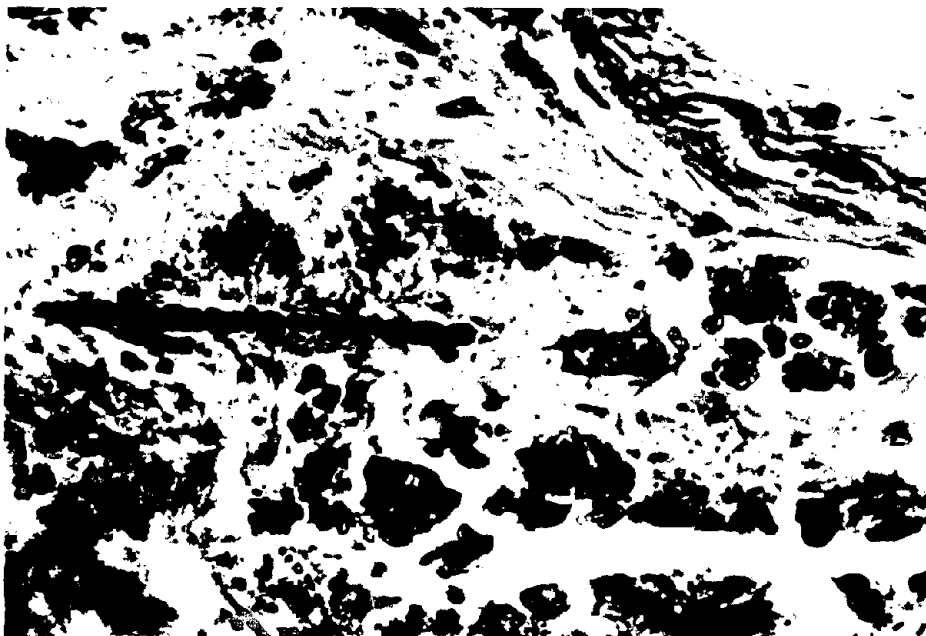
The association between asbestos exposure,

smoking, and lung cancer is now firmly established. The majority of asbestos-associated bronchial carcinomas arise in lungs that also show asbestosis. Autopsy and mortality studies indicate that the prevalence of lung cancer in persons with asbestosis ranges from 12-55% (42) (136).

The lung cancers associated with asbestos exposure occur at a slightly earlier age than in nonexposed individuals (74). They arise in relation to the fibrotic lesions and are thus more common in the periphery of the lower lobes (49)(162). All histological types of cancer occur with most (41)(42)(162), but not all (49), studies



**Figure II-14.** Section of lung from 68-year-old asbestos insulation worker showing the histological features of mild asbestosis. The lesion is characterized by peribronchiolar fibrosis in which there are numerous asbestos bodies. Inset shows an asbestos body. Hematoxylin and eosin  $\times 100$ .



**Figure II-14 (Inset).**

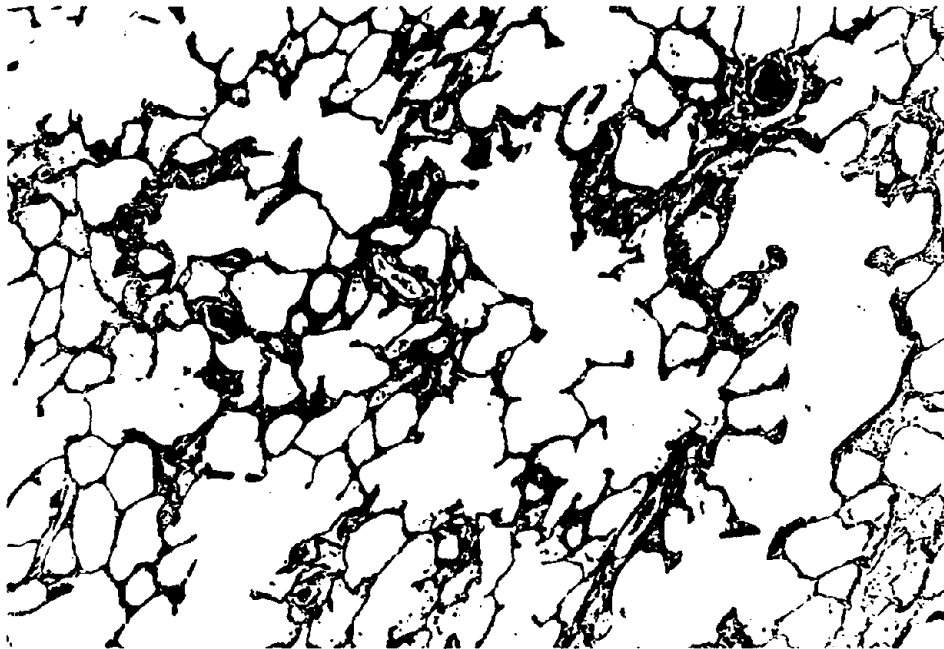


Figure II-15. Section of lung from 48-year-old worker in an asbestos textile mill showing diffuse interstitial and peribronchiolar fibrosis. Hematoxylin and eosin  $\times 40$ .

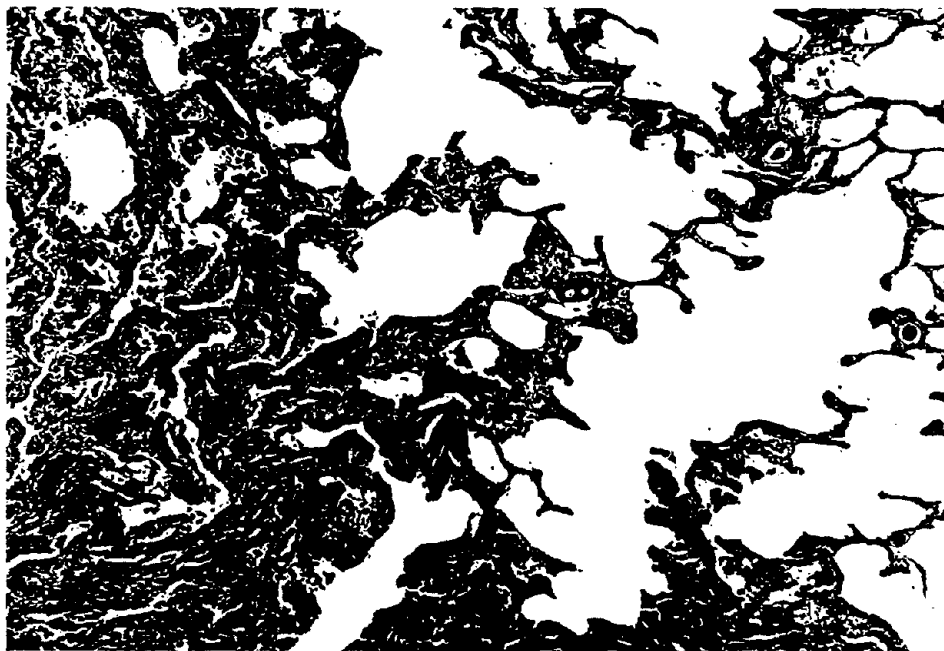
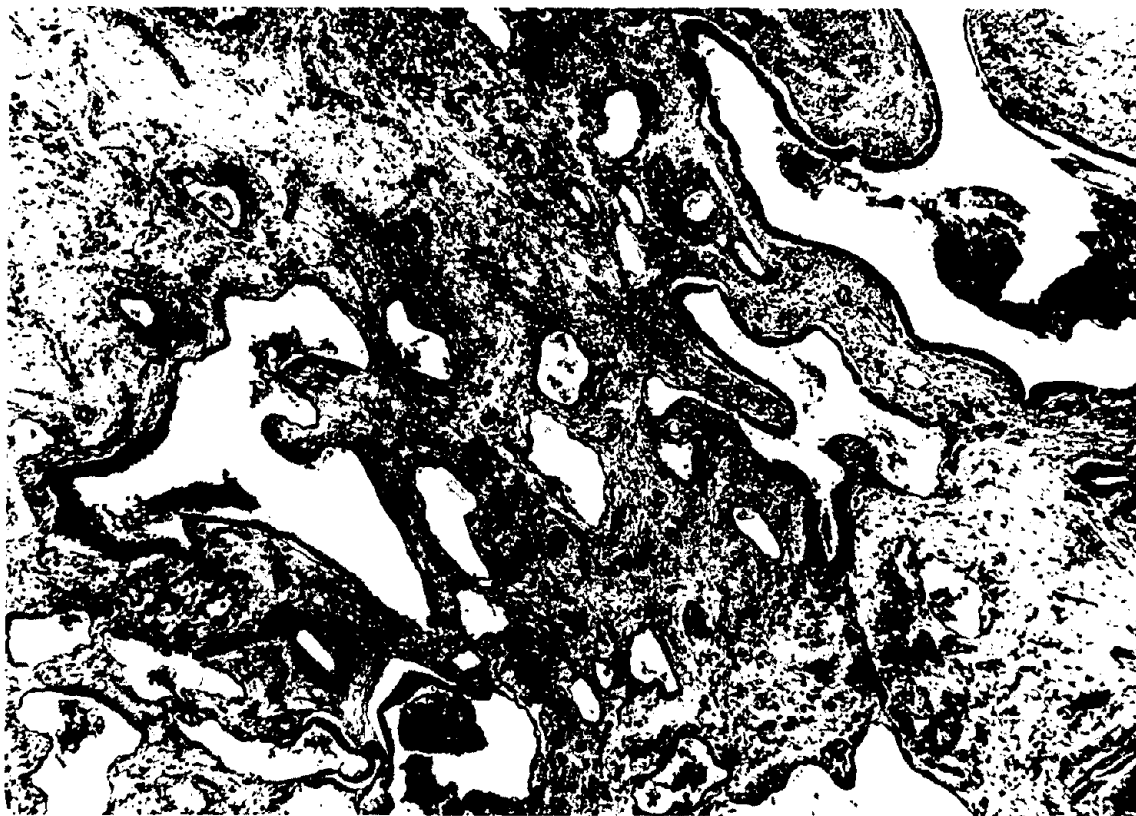


Figure II-16. Section of lung from same case as figure 15 showing interstitial and intraalveolar fibrosis. Hematoxylin and eosin  $\times 40$ .



**Figure II-17. Section of lung showing honeycombing. The pulmonary architecture has been replaced by thick bands of fibrous tissue outlining cystic spaces. There is a moderate chronic inflammatory cell infiltrate of the parenchyma. Hematoxylin and eosin  $\times 40$ .**

showing a preponderance of adenocarcinomas.

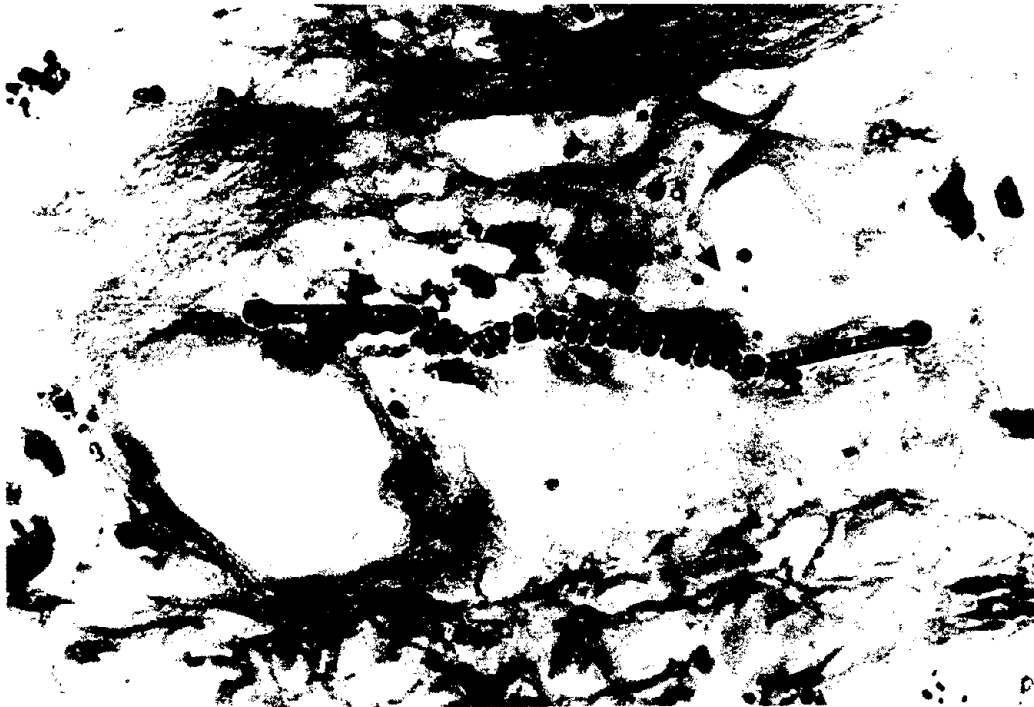
Metaplastic and pre-malignant changes have been observed in the bronchi and within areas of fibrosis in asbestosis (42)(101). It has yet to be determined whether sputum cytology is of value in early detection of carcinoma in asbestos workers (35).

### **Mesothelioma**

Mesothelioma is a rare tumor arising from the mesothelial cells that line the pleural, pericardial, and peritoneal cavities. The first case associated with asbestos exposure was reported by Wyers in 1946 (165). In 1960 this association was firmly established by Wagner and co-workers in a study of individuals exposed to crocidolite asbestos in the Northwest Province of South Africa (152). Since then, cases have been reported from all major industrial countries. Exposure to crocidolite and amosite (45) (125) appear to carry the greatest risk for developing mesothelioma, whereas workers exposed predominantly to chrysotile asbestos appear to have the least risk (18)

(45). The tumor is almost invariably associated with asbestos exposure—a positive history being obtained in 80-90% of cases (13)(151); however, there is no evidence for a dose-response relationship. Although exceedingly rare in the general population, mortality from mesothelioma may approach 10% among some groups of asbestos workers (127).

The tumor occurs in both sexes and has a latency period in excess of 20 years—usually 30 to 40 years. There is no association with cigarette smoking. The tumors are ivory colored and, in typical cases of pleural mesothelioma, encase the lungs in a rubbery mass of tissue. Pleural plaques and asbestosis may also be present, though in the majority of cases mesotheliomas occur in the absence of these lesions. The tumor tends to spread along the interlobar fissures and to invade the subpleural portions of the lungs. Direct invasion of adjacent organs, such as heart, diaphragm, and liver and extension into surgical incisions and aspiration needle tracts are characteristic. Metastases to local lymph nodes and the



**Figure II-18. Asbestos body within an area of fibrosis. The body is composed of a translucent core fiber with a beaded iron-protein coat. An uncoated fiber is also seen (arrow). Hematoxylin and eosin X 600.**

lung are also fairly common. Extrathoracic metastases are relatively rare, and their presence should raise a suspicion as to the authenticity of the tumor.

Microscopically, the tumor can be classified into tubo-papillary, sarcomatous, and mixed types. The tubo-papillary is the most common type and is easily confused with metastatic carcinoma from the lung or elsewhere. Special stains may aid in differentiation in some cases. Mesotheliomas usually contain the mucopolysaccharide, hyaluronic acid, which stains with Hale's colloidal iron and with alcian blue. The specificity of the reaction can be determined by pretreatment of the tissue section with hyaluronidase (16). Hyaluronic acid may also be demonstrated by electrophoresis of tumor tissue (154). Adenocarcinomas usually contain intracytoplasmic mucin droplets rather than hyaluronic acid (16). More recently it has been suggested that the absence of carcinoembryonic antigen (CEA) may be a useful adjunct for diagnosis (153). In the United States and Canada, special panels of

pathologists (mesothelioma panels) exist to provide a diagnostic referral service (50).

### CLINICAL EVALUATION

Clinical evaluation of the asbestos-exposed worker should include a full occupational and environmental history, full medical history, chest radiographs, and spirometry. Evaluation of the occupational and environmental history is especially important. The patient may have had only a few weeks of employment in construction or a shipyard as a summer job years before; yet, it is well documented that such brief exposures may manifest in asbestos related diseases 20 to 30 years later. It is important to assess other occupational exposures, such as coal or hard rock mining, which may produce rounded opacities on radiographic evaluation. Family history is also important. Asbestos insulation workers, as in many trades, tend to work in that trade from generation to generation. Therefore, the possibility of asbestos exposure in the home as a child should not be overlooked. Although a single PA radiograph is recommended for screening for

asbestos related disease in the clinical evaluation, a lateral chest radiograph should also be obtained to evaluate the lung zones behind the heart and provide a baseline for future evaluation. Although impairment is better correlated with radiographic abnormality in asbestosis than in other forms of pneumoconiosis, it is still highly variable. Therefore pulmonary function evaluation is required to assess the nature and extent of lung function abnormality.

**Symptoms and Signs:** Unlike silicosis and coal workers' pneumoconiosis, the asbestos worker may present with dyspnea in the absence of radiographic abnormality. Exertional dyspnea is the most prominent symptom with progression and is the major complaint in asbestosis. A chronic cough which is usually dry, but which may be productive especially among smokers and those working a dusty job, is another common finding. This is consistent with epidemiological studies showing increased bronchitis and airways obstruction especially among smoking asbestos workers. With progression of asbestosis, dyspnea becomes marked and is accompanied by tachypnea.

Pleural plaques or thickening are typically not accompanied by symptoms and may therefore be present years before detection. Some of these patients will report chest tightness or difficulty taking a deep breath. With marked pleural thickening, dyspnea is usually the principal complaint. Asbestos induced pleural effusions are not unusual and may cause pleuritic pain, but pleural pain is often not present even when a friction rub is heard.

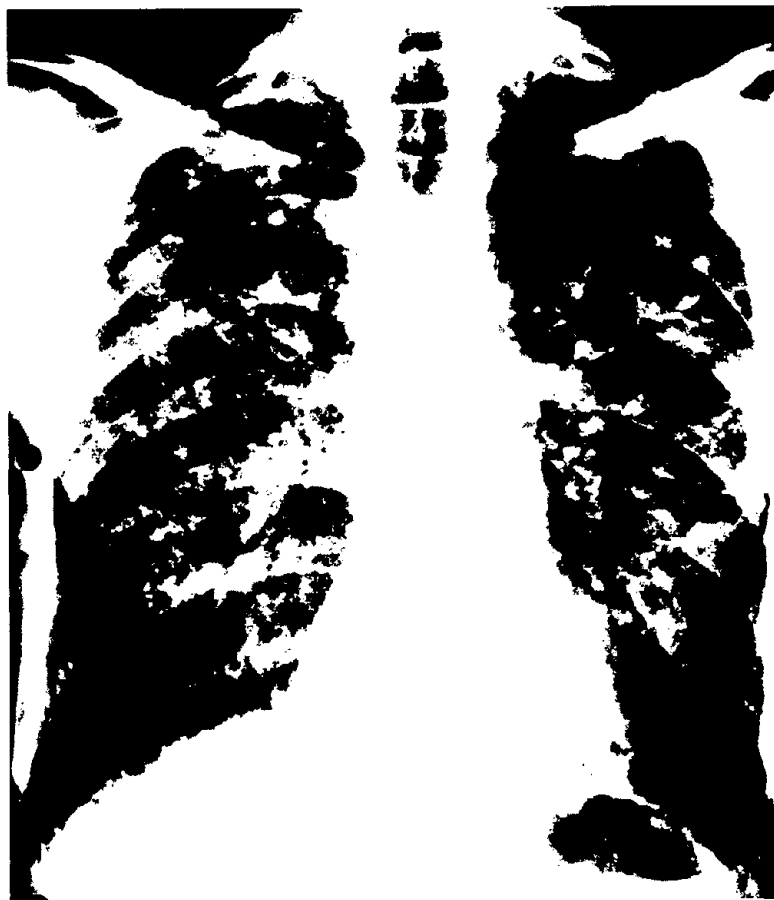
Physical examination is usually not remarkable, especially in early cases of asbestosis. In most cases, the first sign, and often the only sign, is crisp basal crepitations usually best detected anteriorly and laterally at the end of a full inspiration. Clear mid-inspiratory crepitations may be heard over the mid and lower lung zones in more advanced cases of asbestosis. Digital clubbing is found in advanced asbestosis. Cyanosis, like clubbing, is a late sign in those with far advanced disease.

Physical findings in patients with pleural plaques or thickening are few unless the thickening is marked or an effusion is present. In such instances decreased thoracic expansion, dullness to percussion, and diminished breath sounds are found. Pleural friction rubs may also sometimes be detected in patients with pleural involvement.

**Radiographic Findings:** The radiographic findings of asbestosis and asbestos related pleural plaques and thickening are best described through systematic application of the 1980 ILO Classification for interpretation of the pneumoconioses (44). Guidelines for obtaining a technically satisfactory radiograph and for its interpretation are included in the 1980 ILO Classification. Because of the well known variation in interpretation of radiographs from reader to reader, it is recommended that the ILO standard films be used as a guide and that more than one independent reading be obtained (89). This is especially important in evaluation of clinical series and in population studies.

The small irregular opacities of asbestosis are most commonly distributed in the mid and lower lung zones. Their profusion (number of opacities per unit area) is dependent on the degree and length of asbestos exposure and may be quantified into categories (0,1,2,3, by the 1980 ILO Classification). The size and shape of the opacities may be described by using the symbols "s" (irregular opacities less than 1.5 mm in diameter), "t" (irregular opacities 1.5 to 3.0 mm in diameter), or "u" (irregular opacities greater than 3 mm, but less than 10 mm in diameter). Rounded opacities (p,q,r) may also be seen, but if profuse should alert the reader to the possibility of other siliceous dust exposure—this pattern is not uncommon among asbestos miners and asbestos cement manufacturers. With progression, all lung zones may be affected and radiological evidence of honeycombing in the lower zones is not unusual (Figure II-19). Rarely coalescence of opacities may produce large opacities which are ill defined and may be several centimeters in diameter (Figure II-20). Other late manifestations include irregular diaphragmatic, pleural and cardiac borders ("shaggy heart"), often associated with pleural thickening or plaques (Figure II-21).

It is, however, the early cases of asbestosis rather than the advanced cases which are difficult to interpret. It is known that smoking and repeated infections (bronchitis and pneumonia) may produce irregular opacities, especially in older individuals. Morgan et al. have shown that as a consequence, the frequency distributions of small opacities in persons with and without pneumoconiosis may be expected to overlap each other at a low profusion level (90). This obser-



**\*Figure II-19. Advanced asbestosis—profusion 3/3 with all lung zones involved with s/t opacities.**

\*Source: American College of Radiology Teaching Module on Asbestos Related Disease. American College of Radiology, Chevy Chase, Maryland, 1981 NIOSH contract.

vation, together with reader variability, means that caution must be used in ascribing low levels of profusion (0/1,1/0) to asbestos exposure, without consideration of other factors or etiologies—scleroderma, lipoid pneumonia, desquamative interstitial pneumonitis, and sarcoid may all present with basal irregular opacities similar to asbestosis.

Pleural plaques are fibrotic processes which begin below the surfaces of the parietal pleura, are usually smooth or nodular, are often bilateral, and are rarely over 1 cm in thickness. They are most commonly found on the posterolateral or anterior chest walls between the sixth and tenth ribs and in the aponeurotic portion of the diaphragm. Pleural plaques tend to spare the apices and costophrenic angles and, with time, tend to calcify. Plaques vary from small circular or linear opacities to large irregular opacities—

some may encircle the lung. Even without calcification, they are sufficiently characteristic that an asbestos etiology should be presumed whenever they are seen. They greatly assist in the assessment of early parenchymal disease.

The 1980 ILO Classification provides an expanded and complete scheme for codifying pleural changes arising from asbestos exposure (44). The reader is asked to note whether the diaphragm and costophrenic angles are affected. Classification is provided for both diffuse and circumscribed plaques by width (O, A, B, C) and extent (0, 1, 2, 3) evaluated en face on projections. Finally, pleural calcification on the diaphragm, chest wall, or other sites may be specified.

Pleural plaques are often mimicked by the images of small divisions of the external abdominal oblique and the serratus anterior muscles which originate from the external surfaces of the



July 8, '75

**\*Figure II-20. Advanced asbestosis—profusion 2/3 with all lung zones involved with s/t opacities. Large opacities in left mid-zone. Poorly differentiated squamous cell carcinoma of the right hilum.**

\*Source: American College of Radiology Teaching Module on Asbestos Related Disease. American College of Radiology, Chevy Chase, Maryland, 1981 NIOSH contract.

ribs posteriorly and laterally. Unlike most plaques, however, these images are bilaterally symmetrical, occur in rhythmic sequence along the lateral chest walls, are generally smooth, regular, and less opaque than plaques. Oblique radiographs are often useful in differentiating these shadows from plaques or to better define plaques.

**Lung Function:** Lung function testing has been applied to the study of asbestosis since its introduction to clinical medicine in the 1940's. The specific type of lung function test is dictated by the type of investigation. Spirometry has served well as a tool for industrial medical surveillance and for prospective epidemiological studies. Assessment of lung volumes and gas exchange ( $D_{LCO}$  and arterial blood gases) have been useful additional laboratory tests used to evaluate those exposed to asbestos.

Classically, advanced asbestosis has been considered as a disease which restricts lung volumes (especially VC, and to a lesser extent, RV) and produces gas exchange measurements consistent with an "alveolar capillary" block (i.e., decreased  $D_{LCO}$  and in more advanced cases, depressed resting  $Pa_{O_2}$ )(3).  $CO_2$  exchange is usually not affected. In far advanced cases arterial oxygen desaturation is observed; this usually corresponds to central cyanosis and marked dyspnea.

Recent papers on lung function among those with asbestosis have suggested that a mixed restrictive and obstructive pattern and obstructive defect are also commonly found among those with asbestosis. In 1972, Muldoon and Turner-Warwick reported 13 of 60 asbestos workers evaluated at the Brompton Hospital had a pure





**\*Figure II-21. Chronic calcified fibrous pleuritis involving the right chest wall and costophrenic angle.**

\*Source: American College of Radiology Teaching Module on Asbestos-Related Disease. American College of Radiology, Chevy Chase, Maryland, 1981 NIOSH contract.

obstructive ventilatory defect; 3, a mixed pattern; 32, restriction; and 12 were normal (72). In 1975, Fournier-Massey and Becklake reported that among 1,000 Canadian asbestos miners and millers, 12.8% had a restrictive pattern and 12.2% an obstructive pattern (30). Murphy et al. in a study of shipyard workers, found no more obstruction among asbestos workers than matched controls (94). However, Rodriguez-Roisen et al. recently reported an obstructive pattern, defined by reductions in forced expiratory flow at 75% of the vital capacity, in 34 of 40 asbestos workers referred to the Pneumoconiosis Medical Panel and the Brompton Hospital, London (114). Although only 7 of 34 were considered non-smokers, the authors suggest that airways obstruction, particularly affecting small airways, is a common functional abnormality attributable

to asbestos exposure. This view is consistent with pathological observations which show peribronchiolar fibrosis to be an early lesion in asbestosis (see Pathology). The extent and severity of obstructive defects among asbestos workers, however, still needs full epidemiological evaluation with attention to other risk factors, especially smoking.

**Other Medical Tests:** Serological tests of those with asbestosis have shown increased levels of antinuclear factor (ANF) and rheumatoid factor (RF)(142)(147). Others have reported normal levels in mild cases, suggesting that these findings may be the result of nonspecific lung damage (24)(144). However, Gregor et al. have recently reported a series of 119 subjects followed prospectively at the Brompton Hospital and assessed for progression in asbestosis relative to auto-

antibody status (36). Although the numbers were small, there was some suggestion that those who showed a progression over three to seven years had higher antinuclear antibody titers and with greater frequency. These authors suggest that this finding, if confirmed, might indicate a greater degree of inflammation associated with greater alveolar macrophage turnover; this may be an important event in rapid progression among some with asbestosis.

HLA phenotype is another serological test which has been studied in relationship to asbestosis, extent of radiographic profusion, and progression of asbestosis. In a preliminary study, Merchant et al. reported a slight increase in HLA-27 phenotype among men with asbestosis and this was associated with a greater degree of fibrosis (radiographic profusion) (82). However, upon prospective evaluation of the HLA system in asbestosis, Turner-Warwick concluded that HLA phenotype was not of significant importance in the etiology of asbestosis (146).

### PREVENTION

Available epidemiologic data support a linear, no threshold dose-response relationship between asbestos exposure and the risk of lung cancer. Additionally, no threshold has been convincingly demonstrated for nonmalignant respiratory diseases associated with asbestos exposure. Thus, any asbestos exposure carries with it some increased risk of asbestos related diseases. Accordingly, asbestos exposure should be eliminated or reduced to the lowest level possible.

The most effective method for eliminating asbestos related diseases is substitution of less toxic materials or modification of a process or product to eliminate asbestosis. Materials commonly used for substitution include fibrous glass, rock wool, slag wool, and various ceramic and man-made fibers. Asbestos pipe insulation has been satisfactorily replaced with calcium-silicate insulation block. These substitute materials are not totally without risk; thus appropriate work practices and engineering controls are still required.

Appropriately designed and maintained engineering techniques are the control method of choice where asbestos substitutes cannot be used. Processing of asbestos in a wet state has been shown to be an effective control method in many asbestos processing industries, includ-

ing the asbestos textile industry. The most commonly used control measure in asbestos processing plants is local exhaust ventilation whereby liberated dust is collected at the dust source and removed from the breathing zone of workers. Methods of local exhaust ventilation also have been developed for handtools such as saws and drills used in the construction industry.

Appropriate work practices are an important component of any dust control program. These include use of wet methods or high efficiency vacuum cleaners for cleaning of asbestos contaminated areas and proper disposal of asbestos contaminated waste. Showering and changing of work clothes at the end of the work shift are important in eliminating "take-home" exposures. Respiratory protection is appropriate for short-term jobs or operations where controls may be unfeasible; however, use of respirators is not an acceptable substitute for engineering controls.

The combined effects of asbestos exposure and cigarette smoking in increasing the risks of lung cancer and asbestosis are well established. In addition to reducing or eliminating asbestos exposures, asbestos workers should be educated on the multiplicative risks of smoking and asbestos exposures and encouraged not to smoke. Anti-smoking programs are important for asbestos workers.

Various regulations have been promulgated in the United States specifying exposure limits, exposure monitoring requirements and medical surveillance requirements. In 1972, the Occupational Safety and Health Administration promulgated its first exposure standard for asbestos fibers, specifying a limit of five fibers/cc of fibers longer than 5 $\mu$ m (fibers/cc) on an eight hour time-weighted-average basis. This was reduced to two fibers/cc on July 1, 1976. Subsequent reviews of new literature on health hazards of asbestos prompted the National Institute for Occupational Safety and Health to recommend an eight hour exposure limit of 0.1 fiber/cc and elimination of all but essential uses of asbestos.

**Research Priorities:** Although asbestosis is well characterized clinically and has been the subject of a good deal of epidemiological research, a number of research priorities remain:

1. Epidemiological studies are needed to further characterize: potential asbestos risk from exposure in the railroad in-

dustry; tremolite exposure from contaminated vermiculite and talc in the users of these products; the risk (if any) among those working in the crushed stone industry; and to assess the risk of pleural abnormalities in the absence of parenchymal changes.

2. Research is needed to further assess differences in lung cancer and pneumoconiosis risks for various manufacturing and mining populations.
3. Pathological standards developed to characterize asbestosis need to be tested for reliability and validity in a controlled trial.
4. More sensitive and specific tests are needed to assess asbestos lung deposition and injury.
5. Immunological, serological, and bronchial lavage studies of the progression of asbestosis are needed to better characterize the natural history of asbestosis.
6. Experimental animal and clinical trials with promising chemotherapeutic modalities, for both asbestosis and asbestos-associated cancer, should be a high priority.
7. Research must continue on other fibrous materials, such as wollastonite and fine fibrous glass and mineral wool, to document other health effects which may be associated with these fibrous materials.

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## OCCUPATIONALLY INDUCED LUNG CANCER EPIDEMIOLOGY

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### INTRODUCTION

It has been estimated that lung cancer will kill approximately 77,000 men and 28,000 women in the United States during 1981 (16). This accounts for 34% of all types of cancer deaths in males and for 15% in females. It is expected that 122,000 new cases of lung cancer will occur in the United States in 1981. This will account for a total of 22% of deaths in males and 8% of deaths in females. The age-adjusted lung cancer death rates have increased steadily in men from 5 per 100,000 deaths in 1930 to about 70 per 100,000 deaths in 1980. In females, the rate did not climb as steadily: from 2 to 3 per 100,000 deaths in 1930 to about 7 to 8 per 100,000 deaths in the mid-sixties. However, from the mid-sixties to 1980, the rate has increased rapidly to approximately 18 per 100,000 deaths. It has been suggested that the rapid rise in lung cancer among females is because of the increasing number of women in the work force and because many more women have taken up smoking (128).

### ASBESTOS

#### Occupational Exposure—Historical Studies

In 1935, 55 years after the usage of asbestos was introduced on a large-scale basis in industry, suspicion of an association between asbestosis and lung cancer was reported by Lynch and Smith (75) in the United States and by Gloyne (38) in the United Kingdom. About 10 years later, case reports of pleural and peritoneal tumors associated with asbestos began to appear (144)(145)(149). Epidemiologic evidence from Doll showed a tenfold risk of lung cancers in the U.K. asbestos textile workers who had been employed from 1930, that was prior to regulations that were written to help workers improve dust conditions in factories (27). Similar findings were

reported in the United States in 1961. Mesotheliomas were also detected, but this fact was not published until later (81)(119). Possible variations in risk with other types of asbestos fibers were rarely considered in the earlier reports. Since 1964, following the recommendations of the UICC Working Group on Asbestos Cancers (UICC 1965)(136) for new studies, there has been an expansion of epidemiological studies in many parts of the world.

#### Epidemiologic Studies—Lung Cancer

##### *Mixed Fiber Types*

In most industrial processes different fiber types are mixed, so that pure exposures to a single asbestos type are rare. Mortality studies of defined populations of asbestos manufacturing, insulating, and shipyard workers have provided the most concrete evidence concerning the association between bronchial cancer and exposure to asbestos. Reports received from several countries: England (30)(92), Germany (12), the United States (118), the Netherlands (129), and Italy (112) have confirmed this evidence.

Elmes and Simpson (31) have extended their earlier report (30) to include deaths occurring since 1965 through 1975. The mortality trend has shifted from a preponderance of asbestosis and gastrointestinal deaths to malignancies from lung cancer and mesothelioma, (diseases associated with longer latent periods). These authors report that their findings would suggest any standard based "on the prevention of asbestosis, may not provide adequate protection against neoplasia."

A sevenfold excess of lung cancer was found in a group of insulation workers who had been exposed to chrysotile and amosite asbestos, but not crocidolite (121). Enterline and Henderson reported a 4.4 times increased risk of (respiratory cancer) mortality among retired men who had

worked as production or maintenance employees in the asbestos industry and who had been exposed to mixed fibers (32). Among men with mixed fiber exposure (crocidolite and chrysotile) in the asbestos cement industry, the rate was 6.1 times the expected rate. In a British naval dockyard population, Harries showed that there had been an increased rise in mesotheliomas since 1964 (43). However, the full biologic effects of asbestos in shipyard workers would not have been expected to be detected until the 1970's and thereafter (117).

Edge reported that shipyard workers with mixed asbestos exposure and pleural plaques (without evidence of pulmonary fibrosis) had a 2.5 times increased risk of developing carcinoma of the bronchus, when compared with matched controls without plaques (29). In a study of sheet-metal workers with measurable and mixed asbestos exposure, an excess of deaths from malignant neoplasms (24.7% of the deaths for two cohorts, selected for 5 or more years, who worked in the trade; with 19.1% of deaths for a group where 14.5% was expected) was largely attributed to an excess of malignant tumors of the respiratory tract (21). Of the 307 deaths in the first cohort, 32 lung cancer deaths were significantly in excess (1.7 times the expected level).

Weill et al. reported on the mortality experience of a cohort of 5,645 men employed in the production of asbestos cement products and who had at least 20 years since first exposure (146). These workers were exposed largely to chrysotile with some crocidolite and amosite. Among this group, 601 persons were identified as deceased by the Social Security Administration. The vital status of 25% was unknown, and were assumed to be alive, which probably resulted in underestimation of the true risk. Death certificates were obtained for 91% of the known dead. Dust exposures were estimated, using each worker's employment history in conjunction with historical industrial hygiene data.

Weill et al. observed increased respiratory cancer mortality only among those with exposure in excess of 100 mppcf-year, where 23 cases were observed vs. 9.3 expected (146). The unusually low SMR for all causes in the low-exposure groups suggests the possibility of a selection bias and any interpretation of risks at low exposures should be done with caution. Separating the cohort by fiber type exposure, the authors concluded that the addition of crocidolite to chryso-

tile enhanced the risk for respiratory malignancy; however, an excess risk was observed among those not exposed to crocidolite with cumulative exposures in excess of 200 mppcf-months. Both average concentration of exposure and duration of exposure were found to be related to cancer risk.

McDonald and McDonald studied the mortality of 199 workers exposed to crocidolite during gas mask manufacture in Canada from 1939 to 1942 (84). This cohort was followed through 1975, when by this time 56 deaths occurred. Out of these 56 deaths, 4 (7%) were from mesothelioma and 8 (14%) from lung cancer.

### *Chrysotile*

McDonald et al. reported an increased risk of lung cancer among men employed in Quebec chrysotile mines and mills (85)(86). The risk of lung cancer among those workers most heavily exposed was five times greater than those least exposed.

Kogan et al. investigated the cancer mortality among workers in asbestos mining and milling industries between 1948 and 1967 (54). The total cancer mortality rate among workers was 1.6 times higher than that found in the general male population; for female workers the rates were 0.8 times higher for those in mines and 1.1 for those in mills. The lung cancer risk for male miners and millers was twice that of the general male population. For females in mines and mills, the risks were 2.1 and 1.4 times that of the general female population, respectively. For workers over 50 years of age, the risk of lung cancer was greater: for men in mining, 4.0; those in milling, 5.9; for women in mining, 9.5; and those in milling, 39.8 times that found in the general population.

Wagoner et al. reported on the cancer risk among a cohort of workers in a major manufacturing complex utilizing predominantly chrysotile asbestos in textile, friction, and packaging products (143). An excess of respiratory cancer occurred among asbestos workers in each duration-of-employment category down to and including one through nine years. They observed statistically significant standard mortality ratios of 122 for all malignant neoplasms of the respiratory system. The asbestos workers in this study were located in the area of predominantly Amish dutch population with known low frequencies of smoking. The authors, nevertheless,

used the general white male U.S. population as a control group, which most likely resulted in an underestimation of the degree of risk.

Robinson et al. (106) reported an additional 8 years of observation and 385 deaths to the Wagoner et al. (143) study of mortality patterns of workers among one facility manufacturing asbestos textile, friction, and packing exposed predominately to chrysotile. Except for 3 years (during World War II), chrysotile constituted over 99% (per year) of the total quantity of asbestos processed. During those 3 years, amosite was selectively used to a limited extent because of Naval specifications and accounted for approximately 5% of the total asbestos used per year. Crocidolite and amosite (for the other years) accounted for less than 1% of the total usage in very selected areas. Exposures to these two types may have played a role in the etiology of disease; however, due to the overwhelming exposure of the cohort to chrysotile, it is likely that the other exposures played a minor role in the overall mortality patterns. Robinson et al. confirmed the observations of Wagoner et al. that statistically significant excess deaths were due to bronchogenic cancer.

Weiss reported no unusual mortality experience over a 30-year period for a cohort of workers employed in a paper and millboard plant, reported to be using only chrysotile (147). The author concluded the study results were suggestive of a minimal hazard from chrysotile. This conclusion must be viewed in light of the limitations inherent in the study. First, the population studied was small ( $n = 264$ ) and only 66 workers had died at the time of the analyses. Moreover, the unusually low SMR for many of the contrasts in the Weiss et al. paper suggests the possibility of a selective bias greater than usually seen when contrasting industrial populations are contrasted with the general population.

Enterline and Henderson found that retired men who had worked as production or maintenance employees in the asbestos industry, and had been exposed only to chrysotile, and who had reached 65 years of age, had a respiratory cancer risk 2 to 4 times greater than that expected (32). Among men within the asbestos cement industry exposed only to chrysotile, a one- to four-fold excess of respiratory cancer was found.

### **Anthophyllite**

In Finland, anthophyllite mining has been associated with an excess bronchial cancer risk

of 1 to 4 times the overall expected and about double this figure for those with more than 10 years' exposure time (53)(87)(88).

### **Synergism**

There is marked enhancement of the risk of lung carcinoma in those workers exposed to asbestos who smoke cigarettes (11)(25). Hammond and Selikoff interpret the excess lung carcinoma risk from asbestos in nonsmokers to be small (41). No link between cigarette smoking and mesotheliomas has been observed in a prospective study by Hammond and Selikoff (41). A preliminary study on female workers employed between January 1940 and December 1967, in a predominantly chrysotile asbestos textile plant, revealed 7 lung cancer deaths among 580 women when only 0.63 deaths were expected ( $p < 0.01$ ) (64). One lung cancer death was observed in a smoker, two in women of undetermined smoking history, and four in women who "never" smoked cigarettes (as determined from hospital admission charts).

It is important to note that the historic documentation of cigarette consumption patterns is lacking for most retrospective cohort studies done on asbestos workers. It is also important to note that a sizable portion of the general population, the group usually selected for comparison in these studies, are cigarette smokers. Therefore, the risk of lung cancer demonstrated for these industrial groups exposed to asbestos is of such magnitude that it precludes the identification of an independent etiologic role for cigarette smoking.

Hammond et al. have attempted to correct this methodological problem by comparing 12,051 asbestos insulation workers having complete smoking histories to a control population, with no smoking histories (42). Their control population consisted of 73,763 men from the American Cancer Society's prospective cancer prevention study who were similar to the asbestos workers in that they were white males; nonfarmers; had no more than a high school education; a history of occupational exposure to dust fumes, vapors, gases, chemicals, or radiation; and were alive as of January 1, 1967. Non-smoking asbestos workers showed a five times greater risk of dying from lung cancer than their smoking controls. Both smokers and nonsmokers exhibited a fivefold relative risk; however, the attributable risk was greater among the smokers. This higher attributable risk can be accounted for by the large

number of smokers in the asbestos-exposed population and the comparison population.

Liddell et al. has also studied the smoking patterns among asbestos workers through administering questionnaires to living workers or relatives of deceased workers, who died after 1951 (68). The authors report SMR's of 48 and 46 for nonsmokers and ex-smokers, increasing to 206 for heavy smokers. This study is unreliable however, because specific smoking death rates were not used for the calculation of expected lung cancer deaths, and this underestimated the risks among nonsmokers.

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## MESOTHELIOMA

*Ruth Lilis*

### DEFINITION

The primary malignant neoplasm of the pleura—diffuse pleural mesothelioma—has been recognized and accepted as a nosologic entity only during the last 20 years (77), although as early as 1767 Joseph Lieutand (cited by Robertson) reported two cases of probable mesothelioma among 3,000 autopsies, and E. Wagner described the pathology in 1870 (53)(72).

It is not known with certainty when the term “mesothelioma” was first used; one of the early reports indicating a primary and malignant tumor of the pleura and using the term mesothelioma was that by DuBray and Rosson (14).

In 1931, Klemperer and Rabin published a comprehensive description of the distinctive features of diffuse pleural neoplasms and recommended these tumors “should be designated mesothelioma,” since they arise from the surface lining cells of the pleura, the mesothelium (27). The malignant, diffuse pleural mesothelioma arises from the multipotential coelomic mesothelial cell of the pleura. Similarly, malignant tumors originating in the mesothelial cells of the peritoneum are peritoneal mesothelioma.

The definition of pleural mesothelioma thus includes:

- the origin of the tumor in the mesothelial cells of pleura
- the diffuse character of the tumoral growth, often involving a large surface or even the entire pleura of one lung, at the time of diagnosis
- the characteristic rapid growth and extension over the surface of the pleural serosa (closely related to the diffuse character)
- the high degree of malignancy, expressed in rapid growth, local invasiveness (soft tissue and bone structures of chest wall,

underlying lung, adjacent pericardium, regional lymph nodes), and frequent metastases to a variety of organs, including brain, liver, kidney, adrenals, etc. These characteristics of pleural mesothelioma have an integrative expression in the mean survival time after diagnosis, which does not exceed 12 months in most reported series, with or without therapeutic attempts.

The association between malignant “endothelioma of the pleura” (mesothelioma) and asbestos exposure was first reported by Wyers (80). Wagner et al., published a report on 33 cases of diffuse pleural mesothelioma from the North West Cape Province of South Africa; most of these cases had occurred over a four year period, and in all but one, exposure to asbestos (crocidolite) could be established (77). Mesothelioma was not necessarily preceded by asbestosis (interstitial pulmonary fibrosis); the exposure was occupational in some cases, but in others, only environmental (residential) exposure had occurred. The long latency period—a mean of 40 years—between initial asbestos exposure and the development of malignant pleural mesothelioma was another striking characteristic of these cases. The carcinogenic hazard of relatively low levels of asbestos exposure; the possibility that pleural mesothelioma associated with asbestos exposure may develop in the absence of preceding pulmonary interstitial fibrosis; and the long latency period between onset of exposure and development of the malignant mesothelioma, were thus outlined.

### LIST OF CAUSATIVE AGENTS

Asbestos fiber is widely accepted as the causative agent in the vast majority of mesothelioma cases. So far, asbestos is the only fibrous mineral



where epidemiologic data have shown an association between exposure and pleural and peritoneal mesothelioma in man.

Asbestiform minerals are grouped in two major categories: chrysotile, which is a serpentine, and the amphiboles, which include crocidolite, amosite, anthophyllite, and tremolite.

The first large group of malignant pleural mesothelioma cases due to asbestos exposure was related to crocidolite in South Africa (77). This fact, and subsequent reports on mesothelioma cases from Great Britain where crocidolite had been extensively used, contributed to the empirical and one-sided view that crocidolite was the main or even the only type of asbestos with a specific carcinogenic potential resulting in the eventual development of mesothelioma.

The major increase in mesothelioma incidence in the United States—where chrysotile has been and still is the main type of asbestos used—supports a causal association between chrysotile exposure and development of mesothelioma (4)(31)(59)(63)(64). Epidemiologic evidence for worker cohorts has shown chrysotile to be equally as potent as other fiber types insofar as lung cancer is concerned (13)(49)(80). While the number of mesothelioma cases from populations exposed only to chrysotile has been small, an association with chrysotile exposure has been definitively established. Amosite has also been shown to have a similar carcinogenic effect; a significant number of mesothelioma cases have occurred in a cohort of 933 amosite factory workers (62). Experimental studies on rats using inhalation of five types of asbestos fiber resulted in the development of mesothelioma with chrysotile (Canadian), crocidolite, amosite, and anthophyllite (74). Previous experiments using intrapleural administration of amosite, chrysotile, and crocidolite had given similar results, with chrysotile giving the largest number of mesotheliomas, followed by crocidolite and amosite (73). Shabad et al. also reported on the experimental production of pleural mesothelioma in rats, with intrapleural administration of chrysotile (65). Thus, both epidemiologic evidence and experimental confirmation indicate that chrysotile, amosite, and crocidolite asbestos are causative agents for mesothelioma.

Recently another type of fibrous mineral—naturally occurring zeolites (aluminum silicates) of the fibrous variety (erionite, mordenite)—has come under close scrutiny as a potential causative agent

for malignant mesothelioma. The evidence for this association is based on the findings in a rural area of endemic mesothelioma in Turkey, where mineralogic investigations have not found any asbestos minerals, but have identified fibrous zeolites. Although this is still being actively researched and conclusive evidence is not yet resolved, fibrous zeolites are considered highly suspicious at the present time.

Reports on endemic mesothelioma in other parts of the world—such as in a rural area in India—have not yet identified the etiologic agent; the possibility that zeolites may be the causative agent cannot be excluded, since zeolites are known to be present in that area.

Experimental studies using intrapleural application suggest that other fibrous materials, such as fibrous glass, may also induce malignant mesothelioma (68). Epidemiologic evidence for fibrous glass as a causative agent for mesothelioma has not been reported, but fibrous glass has to be included as a suspected causative agent.

### LIST OF OCCUPATIONS AND INDUSTRIES INVOLVED

Occupations and industries at risk to mesothelioma include all of those listed for asbestosis.

All available information indicates that mesothelioma may be the result of low levels and/or relatively short (of the order of several weeks to several months) asbestos exposure. The dose-response relationship for mesothelioma is therefore different than that for asbestosis (which develops with higher exposure levels over longer time periods) or bronchial carcinoma associated with asbestos exposure (which increases in incidence even after short periods of high asbestos exposure levels, but shows a marked increase in incidence with duration of exposure) (58). Since low asbestos exposure levels carry a significant risk of mesothelioma, occupations and industries characterized by relatively low asbestos levels (auto mechanics and brake repair, tapers in dry wall construction, handling of finished asbestos products including asbestos cement), while at relatively low risk for the development of parenchymal interstitial fibrosis (asbestosis), are nevertheless at high risk for mesothelioma.

Equally important is the fact that numerous workers in the various trades which do not simply direct asbestos exposure, such as electricians, painters, welders, carpenters, etc., in shipbuilding or ship repair, in construction, in maintenance

work at chemical plants, and even automobile salesmen supervising repair work, are frequently exposed to asbestos due to their mere presence in work areas where asbestos is being handled. This "bystander" exposure has been repeatedly documented to be responsible for numerous cases of mesothelioma (20)(51). It is therefore important to establish the principle that such indirect exposure carries a significant risk of mesothelioma.

Whitwell et al. found that 83% of mesothelioma cases reviewed contained over 100,000 asbestos fibers per gram of dried lung tissue; in cases of asbestosis the number of asbestos fibers was much higher, exceeding 3,000,000 per gram of dried lung tissue (79).

In shipyard workers, more and more mesothelioma cases have been reported; most of these have occurred in trades other than insulation workers, indicating that the risk is widespread (20)(61). The distribution of trades in private shipyards in the United States in 1943 is presented in Table VIII-24. A list of occupational titles in an Eastern U.S. shipyard in 1975 is given in Table VIII-25.

It is difficult to construct a complete list of all occupations in which asbestos exposure may occur at one time or another. Since short-term asbestos exposure (several weeks to several months) is often responsible for mesothelioma occurring 25, 30, 40, or 50 years later, the occupation/industry involved at the time of the diagnosis of a malignant tumor may differ from the occupation/industry where the exposure actually occurred. Therefore, at any point in time, much higher numbers of individuals are at risk for the development of mesothelioma than those working in industries and occupations known to be associated with asbestos exposure. Recollection of remote past exposures and of specific jobs in which they occurred is a formidable task, but crucial when assessing whether one particular case of mesothelioma is related to past asbestos exposure.

## EPIDEMIOLOGY

The relationships between asbestos exposure and pleural mesothelioma regarding latency period, dose-response characteristics, populations at risk, and incidence of disease have been presented in the section—List of Occupations and Industries Involved, page 672.

Pleural mesothelioma is a rapidly progressing malignant tumor, the resulting disability is

**Table VIII-24**  
PERCENTAGE DISTRIBUTION  
OF TRADES IN PRIVATE SHIPYARDS  
IN THE UNITED STATES, JUNE 1943

<i>Trade</i>	<i>Percentage</i>
Welders	15.3
Shipfitters	11.0
Machinists	8.1
Pipefitters	7.2
Electricians	6.6
Carpenters	6.1
Laborers	5.5
Burners	3.8
Painters	3.1
Sheetmetal workers	3.0
Riggers	2.8
Chippers and caulkers	2.8
Boilermakers	2.3
Crane operators	1.3
Pipe coverers	0.2
All other	21.1

Source: Bureau of Labor Statistics, Bulletin 824, "War-time Employment, Production, and Conditions of Work in Shipyards," 1945.

total, and the condition is usually fatal in one to two years. There are no confounding conditions or risk factors which limit the ability to establish cause-effect relationships.

## ESTIMATE OF POPULATION AT RISK AND PREVALENCE OF DISEASE

The population at risk for developing mesothelioma includes:

- all occupations with direct contact and handling of asbestos.
- employees with other occupations (electricians, welders, painters, carpenters, etc.) who work or have worked—even for short periods—in areas where asbestos has been handled by others.
- family members (household contacts) of asbestos workers who have been exposed to asbestos fibers brought into the household by the worker. Household contamination has been found to result in asbestos exposure of family members of asbestos workers, sufficient in magnitude to induce mesothelioma (1)(2)(5)(32)(41)(46)(55)(56).
- individuals who have resided in the vi-

**Table VIII-25**  
OCCUPATIONAL TITLES IN AN EASTERN U.S. SHIPYARD, 1975

Guard & Watchman	Heat Treater	Power House	Shipfitter
Construction	Tool Grinder	Engineer	Lead Bonder
Mechanic	Tool Room	Molder	Welder
Laborer	Attendant	Foundryman	Burner
Firefighter	Lathe Operator	Foundry Chipper	Rigger
Scrap Material	Miller	Melter	Sheetmetal Mechanic
Sorter	Drill Operator	Coremaker	Joiner
Painter	Grinder	Pipefitter	Carpenter
Painter Cleaner	Machinist	Silver Brazier	Industrial Radiography
Maintenance	Engraver	Pipecoverer	Technician
Painter	Layout	Electrician	Radiological Control
Truck Driver	Machine Rigger	Electronics	Monitor
Fork Lift Operator	Make Ready Man	Technician	Clerk
Warehouseman	Crane Operator	Maintenance	Data Processor
Transportation	Maintenance	Electrician	Secretary
Locomotive	Machinist	Loftsman	Timekeeper
Operator	Dock Crew	Blacksmith	
Toolmaker	Inspector	Furnaceman	

cinity (one mile) of an asbestos plant, shipyard, or other source of asbestos contamination.

The population at risk at any point in time has to include all persons who have been exposed *in the past*. Given the long latency period between asbestos exposure and development of mesothelioma (on the average 35-40 years), individuals who have been exposed (even for short periods of time) during the last 50 years have to be considered potentially at risk.

Contributing to the population size at risk is (1) the fact that short duration of asbestos exposure (several weeks to several months) is sufficient to induce mesothelioma; (2) the high job mobility, especially during World War II; (3) the marked increase in the total amount of asbestos used per year; and (4) the diversification of its uses. The estimate of the population at risk is, for the same reasons, a complex and difficult task.

Attempts to assess the incidence of mesothelioma in populations at risk are also fraught with difficulties; these have multiple sources.

1. The complexity of the diagnostic criteria, which require pathologic confirmation; the most rigorous criteria make the diagnosis dependent on a complete autopsy (for the exclusion of another primary site of the tumor, which might have metastasized to the pleural cavity).

Only a proportion of all deaths are followed by a postmortem examination. This proportion varies with geographic area, with the time period considered, and with other factors.

2. Even when tissue specimens are examined by experienced pathologists, the diagnosis is not always simple; differences of opinion may persist and result in conclusions on the pathologic characteristics such as "possible mesothelioma" or probable mesothelioma."
3. Evaluation of the incidence of mesothelioma from death certificates has been reported, by all those who have investigated this problem, as incomplete, leading to a marked but quantitatively variable underestimate of the number of cases. This problem is compounded by the fact that the coding of causes of death does not provide a separate code for mesothelioma, but includes it with cancer of the lung or pleura.
4. The most reliable data are those based on the cohort approach: asbestos-exposed employees followed for many years, with a comprehensive assessment of causes of death. The long latency period between

onset of asbestos exposure and mesothelioma has resulted in a limited number of studies with a long enough follow-up period to realistically reflect its incidence. In all these cohort studies, most with several reports published over time, it is a rule without exception that the longer the observation period, the higher the incidence of mesothelioma.

Although the most relevant data on mesothelioma risk in asbestos-exposed populations are derived from long-term cohort studies, other studies following different approaches have also revealed the paramount importance of long-term follow-up and completeness of diagnostic means. The most significant information follows.

By 1965, 160 cases of mesothelioma had been recorded in the United Kingdom, 123 from England and Wales, 36 from Northern Ireland, and only one from Scotland (39). When a systematic review of all necropsy and surgical biopsy reports in all hospitals was undertaken, 80 cases of mesothelioma were found to have occurred in Scotland for the years 1950-1967. Many cases were in employees who had had no direct exposure to asbestos but had been employed in the shipbuilding industry, in a wide variety of trades.

The Mesothelioma Register in Great Britain (Employment Medical Inspector's Advisory Service)—with data sources in death certificates, Cancer Bureau registrations, Pneumoconiosis Medical Panels (claims for benefits under the National Insurance Acts), chest physicians, surgeons, pathologists and coroners—had 413 cases reported for 1967-1968; 75% of the confirmed cases with definite asbestos exposure came from shipbuilding, asbestos factories, and insulation work; the other 25% from a variety of occupations (welders, electricians, gas workers, mechanics, chemical workers, etc.). The highest rate/million per year of mesothelioma (confirmed cases) figures were 8.93 and 8.24, both in shipbuilding areas. The incidence of definite mesothelioma in the United Kingdom for the period 1967-1968 was 120 per year. It was concluded that this figure may considerably understate the true incidence.

McDonald and McDonald reviewed evidence published between 1959 and 1976, including cohort studies of asbestos workers; "population studies" (mesothelioma surveys in Canada and the United States describing "case-series

referable to some kind of denominator"); case reports unrelated to any denominator; and mortality statistics, mainly in Canada, the United States, and the United Kingdom (37). Data from the Third U.S. National Cancer Survey (42) was also reviewed. A total of 4,539 cases had been published after 1958. (This figure did not include cases from official mortality statistics and Third U.S. National Cancer Survey.) The incidence of mesothelioma for the period preceding 1958 had been very low: in 1957 Hachberg mentioned 43 cases in 60,042 autopsies over the 40-year period, 1910-1949, i.e., less than 1 case per year and only 0.07% of the autopsies performed (Philadelphia, Baltimore, Minneapolis, New York, and Toronto in North America and Munich, Prague, and Copenhagen in Europe).

The marked increase in the incidence of mesothelioma over the last 20 years is evident when comparing the total number of reported cases (436) for the period 1955-1959, with that of 1,697 cases of mesothelioma for the period 1965-1969 (an almost fourfold increase). Interestingly, 9% of cases were due to neighborhood or household-family exposure.

In the Third National Cancer Survey (1975), a thorough ascertainment was done using hospital records and pathology material, besides death certificates, in selected areas comprising approximately 10% of the population of the United States (deaths in 1971). The annual rate per million for males 45 and over was 11.20 and for females in the same age range, 3.53.

Reports from other countries, such as Germany, Sweden, the Netherlands and Great Britain, indicate much higher rates than those published for Canada by McDonald (10 per million for males and 4 per million for females, over 45-years-old) for some cities and regions, most with large shipyards: Walcheren had a death rate 23.3 times higher than that expected according to the Canadian rates; Wilhelmshaven (21.5 times higher); Plymouth (14.3 times higher); and Rotterdam, Harlem, Hamburg, Malmö, Nantes, and Trieste (with rates 7-8 times higher) (38)(51)(69). These data indicate that annual incidence rates for mesothelioma in geographical areas with shipyards and/or other important asbestos industries or uses are of the order of 200/1 million or higher, for men aged 45 or over.

The most relevant data on the incidence of mesothelioma in exposed populations are derived from cohort studies of occupational groups. But

only studies with long follow-up (30-40 years) can provide comprehensive information, although even these might not include all the cases. It has been estimated, from the relatively limited number of such studies, that between 5% and 11% of all deaths in asbestos-exposed workers are due to mesothelioma (16)(26)(43)(45)(61)(62)(63). In a cohort of 632 asbestos insulation workers observed prospectively from January 1, 1943 to December 31, 1976, 38 out of a total of 478 deaths were due to mesothelioma (see Table VIII-26) (60). The mortality experience of a large cohort of 17,800 asbestos workers in the United States and Canada (Table VIII-27) observed from 1967 to 1977 indicates that 175 out of 2,270 deaths were due to mesothelioma. In a cohort of amosite asbestos factory workers employed from 1941-1945, and observed until 1977, 16 out of 594 deaths were due to mesothelioma (Table VIII-28) (62). In another cohort of 689 asbestos factory workers employed before January 1939, and observed from 1959 through 1975, 26 out of 274 deaths were due to mesothelioma (48)(60). Newhouse reported the mortality experience of workers in an East London asbestos factory, 1931-1970; out of a total of 461 deaths, 35 were due to mesothelioma (43).

The importance of long-term observation is shown in Tables VIII-29, VIII-30, and VIII-31.

Two further problems are: 1) the correct assessment of all those at risk for developing mesothelioma in various occupations, or who have had such exposure even for short periods of time sometime during the last 40-50 years; and 2) quantification of the risk for "bystander" exposure, neighborhood or other types of environmental exposure (buildings, schools, etc.), and household-family exposure.

Although no firm data are as yet available for these types of asbestos exposure, according to the information available on cases occurring after short (several weeks) and relatively low levels of exposure, it has to be assumed that the risk is of the same order of magnitude as that for occupationally-exposed groups.

### **PATHOLOGY, PATHOGENESIS, AND PATHOPHYSIOLOGY**

The pathology of mesothelioma is largely determined by the potential of the mesothelial cells to produce tumors of epithelial, mesenchymal, or most commonly a mixed type. This potential is related to the embryologic origin of the mesothelium, which is derived from coelomic epithelium developed from the mesoderm and

underlined by mesenchymal tissue (27).

The macroscopic features of pleural mesothelioma are those of a gray-white or yellow-gray mass, varying in extent from a part of the lung's surface to a complete, or almost complete, encasement of the lung. The tumor has a rapid growth rate, extending along the serosa, with a tendency to grow along the interlobar fissures. Both the parietal and visceral pleura are involved; often the tumor seems to have originated in the visceral pleura (for example, in the minor fissure).

Two types of mesothelioma can be observed: 1) the scirrhous type, presenting as a hard sheet, with variable thickness often exceeding one inch, rapid encasement and compression of the lung, partial or total obliteration of the pleural cavity, and contraction of the hemithorax; and 2) the encephaloid type, presenting as large tumor masses, often multiple, sometimes with extremely rapid growth (seen on chest x-rays as "scalloping").

Continuous spread—with local invasion of the pericardium, mediastinum, chest wall, diaphragm, and, through it, the liver and peritoneum, or into the contralateral pleura—is frequent. The underlying lung can be invaded directly, into the pulmonary parenchyma immediately underlying the pleura, or by spread into septal and perivascular lymphatics, with lymph node involvement in about 50% of cases. Distant metastases, thought in the past to be rare, are, on the contrary, quite frequent, affecting the brain, liver, kidney, adrenals, thyroid, lung, or other organs in more than 50% of cases. Tumor growth along the needle biopsy track or surgical scar after thoracotomy is common.

Microscopic features are characterized by diversity of appearance, not only from case to case, but also in the same tumor, where both epithelial (or tubulo-papillary) and mesenchymal (or fibrosarcomatous) areas can be observed. According to the microscopic pattern, mesothelioma can be classified into four types: 1) epithelial or tubulo-papillary, with the epithelial cells usually cuboidal or flattened, tending to form tubular and papillary structures, separated by a more or less abundant matrix; 2) mesenchymal or fibrosarcomatous, appearing as a spindle cell sarcoma, but sometimes with extensive areas of acellular collagen; 3) mixed, the most frequent form, containing both epithelial and fibrosarcomatous areas; 4) the undifferentiated type, with polygonal, less often spheroidal cells, with large nuclei and scanty mitotic figures. These cells resemble those of the tubulo-papillary

Table VIII-26

EXPECTED AND OBSERVED DEATHS AMONG 632 NY-NJ ASBESTOS INSULATION WORKERS OBSERVED PROSPECTIVELY JANUARY 1, 1943 - DECEMBER 31, 1976

	Number of Men Man-years of observation	632 13,925
	<i>Deaths 1.1.43-12.31.76</i>	
<i>Cause of death</i>	<i>Expected*</i>	<i>Observed</i>
<i>Total deaths, all causes</i>	328.9	478
<i>Total cancer, all sites</i>	51.0	210
Lung cancer	13.3	93
Pleural mesothelioma	**	11
Peritoneal mesothelioma	**	27
Cancer of esophagus	1.4	1
Cancer of stomach	5.4	19
Cancer of colon - rectum	8.3	23
All other cancer	28.06	36
<i>Asbestosis</i>	**	41
<i>All other causes</i>	262.6	227

\*Expected deaths are based upon age and sex-specific U.S. death rates of the National Center for Health Statistics, 1949-1975 actual rates, 1943-1948 extrapolated from 1949-1955 rates, and 1976 extrapolated from 1967-1975 data.

\*\*These are rare causes of death in the general population.

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Table VIII-27

DEATHS AMONG 17,800 ASBESTOS INSULATION WORKERS IN THE UNITED STATES AND CANADA JANUARY 1, 1967 — JANUARY 1, 1977

	Number of Men Man-years of observation	17,800 166,855	
	<i>Expected*</i>	<i>Observed</i>	<i>Ratio</i>
<i>Total deaths, all causes</i>	1,660.96	2,270	1.37
<i>Total cancer, all sites</i>	319.90	994	3.11
Lung cancer	105.97	485	4.58
Pleural mesothelioma	**	66	—
Peritoneal mesothelioma	**	109	—
Cancer of esophagus	7.01	18	2.57
Cancer of stomach	14.23	22	1.55
Cancer of colon - rectum	37.86	59	1.56
All other cancer	154.83	235	1.52
<i>Asbestosis</i>	**	162	—
<i>All other causes</i>	1,351.06	1,114	0.82

\*Expected deaths are based upon white male age-specific mortality data of the U.S. National Center for Health Statistics for 1967-1975 and extrapolation to 1976.

\*\*These are rare causes of death in the general population.

**Table VIII-28**  
**EXPECTED AND OBSERVED DEATHS**  
**AMONG 933 AMOSITE FACTORY WORKERS EMPLOYED**  
**1941-1945, OBSERVED TO DECEMBER 31, 1977**

	<i>Deaths 1941-1977</i>		
	<i>Expected<sup>(a)</sup></i>	<i>Observed</i>	<i>Ratio</i>
<i>Total deaths</i>	368.62	594	1.61
<i>Cancer, all sites</i>	73.35	195	2.66
Lung cancer	19.16	100	5.22
Pleural mesothelioma	<sup>(b)</sup>	8	—
Peritoneal mesothelioma	<sup>(b)</sup>	8	—
G.I. cancer	21.55	32	1.48
All other cancer	32.64	47	1.44
<i>Asbestosis</i>	<sup>(b)</sup>	30	—
<i>Other noninfectious</i>			
<i>respiratory disease</i>	8.47	19	2.24
<i>All other causes</i>	286.80	350	1.22

<sup>(a)</sup> Expected deaths based upon age-specific death rate data for New Jersey white males in corresponding years. In 4 cases, ages were not known; omitted from calculations. 39 men partially traced and 890 traced to death on December 31, 1977.

<sup>(b)</sup> Death rates not available, but these have been rare causes of death in the general population.

type.

A property of mesothelial cells is the production of acid mucopolysaccharides, especially hyaluronic acid, which stains strongly with colloidal iron, but not with periodic acid Schiff (PAS). This last characteristic is useful in differentiating mesothelioma from adenocarcinoma; the latter usually gives a positive stain with PAS. The hyaluronidase test (digestion of hyaluronic acid by the enzyme) is useful in a limited number of cases, since the tubulopapillary type of the tumor is the only form which consistently produces hyaluronic acid. Therefore a negative hyaluronidase test does not exclude the diagnosis of mesothelioma.

The pathogenesis of mesothelioma is not yet completely understood. Nevertheless, the following facts of major theoretical and practical consequence have been established:

- mesothelioma may result from exposure to crocidolite, chrysotile and/or amosite; the evidence is derived from epidemiologic and experimental animal studies.
- relatively low levels and short duration of exposure can produce mesothelioma.

- while a dose-response relationship may exist, it has not been quantitatively clarified, and therefore available information can only be interpreted to indicate that any asbestos exposure, given a long enough period of follow-up, may induce mesothelioma.
- the hypothesis according to which polycyclic aromatic hydrocarbons adsorbed on asbestos fibers are important in the induction of mesothelioma has not been confirmed, nor has that attributing a similar effect to adsorbed trace metals (19).
- cigarette smoking has no etiologic relationship with mesothelioma.
- in experimental studies, intrapleural administration of asbestos, but also of similarly sized fibers of fibrous glass and fibrous aluminum oxide, resulted in pleural mesothelioma (66)(67)(68). This seems to indicate that fibrous characteristics, rather than the chemical composition, are crucial for this specific carcinogenic effect.
- a special selectivity in the distribution of asbestos fibers, relevant to the problem

Table VIII-29

EXPECTED AND OBSERVED DEATHS AMONG 689 ASBESTOS FACTORY WORKERS,  
EMPLOYED BEFORE JANUARY 1, 1939 DURING THE SEVENTEEN YEARS  
FROM JANUARY 1, 1959 THROUGH DECEMBER 31, 1975

	1959-1964		1965-1970		1971-1975		1959-1975	
	Obs.	Exp.	Obs.	Exp.	Obs.	Exp.	Obs.	Exp.
<i>All causes</i>	59	52.41	123	69.85	92	65.93	274	188.19
<i>Cancer, all sites</i>	21	10.47	45	14.70	33	14.73	99	39.92
Lung cancer	6	2.96	18	4.65	11	4.92	35	12.53
Pleural mesothelioma	1	n.a.	5	n.a.	7	n.a.	14	n.a.
Peritoneal mesothelioma	1	n.a.	6	n.a.	4	n.a.	12	n.a.
Cancer of esophagus, stomach, colon and rectum	4	2.23	5	2.92	3	2.83	15	7.99
Cancer, all other sites	9	5.28	11	7.13	8	6.98	23	19.40
<i>All respiratory disease</i>	14	3.01	10	4.56	18	4.60	42	12.16
Asbestosis	12	n.a.	8	n.a.	15	n.a.	35	n.a.
Other respiratory	2	(b)	2	(b)	3	(b)	7	(b)
<i>All other causes</i>	24	38.93	68	50.59	41	46.60	133	136.11
Person-years of observation	3,962		3,411		2,273		9,646	

(a) Pleural mesothelioma included with cancer of bronchus in calculating ratio since expected rates are based upon "cancer of lung, pleura, bronchus, trachea."

(b) This rate is virtually identical with that of "all respiratory disease."  
n.a.—not available.



**Table VIII-30**  
**MORTALITY EXPERIENCE AMONG 17,800 ASBESTOS INSULATION WORKERS**  
**IN THE UNITED STATES AND CANADA 1967-1977:**  
**OBSERVATIONS IN 2,270 CONSECUTIVE DEATHS**

		Number of men		17,800							
		Man-years		166,855							
		Duration from onset of work exposure (years)									
Cause of death	Total	<10	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45 +	
All causes	2,270	51	85	188	320	388	340	253	203	442	
Cancer, all sites	994	7	17	59	125	193	186	128	95	184	
Lung	485	0	7	29	59	104	112	66	39	69	
Pleural mesothelioma	66	0	0	2	6	15	10	16	4	3	
Peritoneal mesothelioma	109	0	0	3	3	18	22	18	16	29	

**Table VIII-31**  
**EXPECTED AND OBSERVED DEATHS**  
**AMONG 933 AMOSITE ASBESTOS FACTORY WORKERS EMPLOYED 1941-45**  
**OBSERVED TO DECEMBER 31, 1977**

<i>Deaths of lung cancer and mesothelioma</i>						
<i>Time from onset (years)</i>	<i>Man-years</i>	<i>Lung cancer</i>			<i>Mesothelioma</i>	
		<i>Exp.</i>	<i>Obs.</i>	<i>Ratio</i>	<i>Pleural</i>	<i>Peritoneal</i>
<5	4,331	0.95	0	—	0	0
5-9	4,095	1.78	3	—	0	0
10-14	3,784	2.57	13	5.06	0	0
15-19	3,362	3.19	20	6.27	0	0
20-24	2,837	3.49	18	5.16	1	0
25-29	2,250	3.59	25	6.96	2	4
30-34	1,553	3.16	17	5.38	5	3
35+	192	0.41	4	—	0	1
	22,404	19.14	100	5.22	8	8

of mesothelioma induction, has been demonstrated by Roe et al. (54). After subcutaneous injection in mice (experiments with three types of asbestos), wide dissemination from the site of injection and a highly selective distribution were observed; the main sites of asbestos accumulation were the visceral and parietal pleura and the serosal surface in the abdominal cavity.

- the fiber size (cross-sectional diameter and length) seems to be important, since smaller fibers penetrate deeply into the periphery

of the lung and subpleural areas (21)(22)(67)(68)(70)(75).

The evidence for marked effects, including the carcinogenic mesothelioma inducing effect of small fibers (length less than 5  $\mu$ m) has emerged relatively recently (122)(24)(75). This is important in view of the fact that handling or treating asbestos as well as use of asbestos products generates fragmentation (both longitudinally and transversely) of fibers resulting in a larger number of shorter and thinner fibers or even fibrils. Chrysotile is especially prone to undergo such

fragmentation.

## CLINICAL DESCRIPTION

### Symptoms

Chest pain (unilateral) and shortness of breath are the most common presenting symptoms. The chest pain may be diffuse and dull or it may be of the pleuritic type; it often progresses to be severe. Shortness of breath may rapidly progress, especially with the development of a pleural effusion.

Other relatively frequent symptoms are loss of appetite, weight loss, fatigue, and in some cases fever; cough is infrequent.

### Physical Signs

Pleural effusion occurs in the majority of cases, with dullness on percussion and decreased breath sounds. Rapid recurrence after aspiration of pleural fluid is the rule. The pleural fluid may be serous and clear but sometimes is hemorrhagic.

Retraction of the affected hemithorax, and shifting of the mediastinum to the side of the lesion may occur.

### Natural History

Rapid tumor growth—often after pleural biopsy, i.e., needle biopsy or thoracotomy—with subcutaneous tumor nodules may involve the chest wall, the ribs and vertebrae, the mediastinum (sometimes with superior vena cava syndrome), and/or the pericardium with pericardial effusion. Distant metastases to the liver or other intra-abdominal organs, sometimes with ascites, can be clinically detected.

The metastatic spread of mesothelioma is much more frequent than previously thought and has been shown to occur in the majority of cases in which an autopsy was performed; both lymph node metastases and distant hematogenous metastases can be found. Spread of the mesothelioma to the opposite pleural cavity, and also to the peritoneum, is frequent; most often this is the result of a local invasive process, through the mediastinum or through the diaphragm.

The natural history of the disease is that of a rapid downhill course; death occurs in the majority of cases after an interval of months to one or two years. The mean survival from first diagnosis does not exceed 12 months. Although all therapeutic methods have been used, often in combination (surgery, radiotherapy, chemotherapy), no significant difference in survival of pa-

tients with pleural mesothelioma has been consistently achieved.

### Laboratory Investigations

Radiographic changes are characteristically unilateral and progressive. The two main modalities of radiologic changes in pleural mesothelioma are: 1) unilateral pleural effusion; 2) large, nodular, protuberant opacities projecting from the pleura into the pulmonary parenchyma. Most often a combination of these changes is found.

Aspiration of the pleural fluid may be helpful in revealing underlying solid tumoral opacities. Extension of the tumoral growth over the apical pleura and into the mediastinal pleura is frequent. PA chest radiographs should be complemented by oblique views of the chest whenever a suspicion of pleural mesothelioma arises. Other radiographic evidence of asbestos-related parenchymal and/or pleural changes may or may not be present. Pleural plaques or calcifications are a useful marker of past asbestos exposure.

Pulmonary function studies are irrelevant for the diagnosis of mesothelioma.

Pleural fluid aspiration, while often necessary to alleviate respiratory distress, is of limited diagnostic use. Cytology of the pleural effusion is often fraught with the difficulty of distinguishing between mesothelial malignant cells and "atypical" mesothelial cells. The detection of hyaluronic acid in the pleural fluid is useful, although it can be found with other malignant tumors of the pleura; a negative result does not discard the diagnosis (6)(25)(76).

Needle biopsy specimens are insufficient for tissue diagnosis, since tissue specimens so obtained might not include malignant changes (although such changes may well be present in adjacent areas of the pleura) and since there is marked variability of pathologic changes.

Thoracotomy with surgical pleural biopsy, although providing adequate tissue specimens for diagnostic purposes, is often followed by local extension of tumor growth into the chest wall.

### Treatment

There is no effective therapeutic approach, although surgery to reduce the tumor mass (9), radiotherapy (17)(57)(71), chemotherapy, single drugs (7)(18)(29)(30)(40), or combinations of two, three, or four drugs, and all possible combinations of these methods have been attempted (35).

Wanebo et al. reported on 66 cases with

malignant mesothelioma (78). For the epithelial type, pleurectomy combined with irradiation and chemotherapy seemed to be more effective; in the fibrosarcomatous type, surgery resulted in longer survival.

### Prognosis

The disease is fatal, and progression is usually rapid, with marked deterioration over short periods of time. In exceptional cases, longer survival (several years) can occur even in the absence of any therapeutic procedure.

### DIAGNOSTIC CRITERIA

The diagnostic criteria for pleural mesothelioma are:

- a history of asbestos exposure in the past. Occupational exposure (even for short periods) or household or neighborhood exposure has to be actively searched for and can be established in the vast majority of cases if histories are taken by a physician with experience in occupational medicine (11).
- long latency period, usually more than 20 years from onset of exposure, most often between 30 and 40 years.
- clinical symptoms: unilateral chest pain and/or significant increase in dyspnea over a short period of time (weeks or months).
- physical findings: consistent with pleural effusion.
- radiographic abnormalities presenting as pleural effusion or pleural thickening often with large nodular opacities projecting from the pleura. Rapid increase in pleural thickening or the appearance of irregularities of the pleura are highly suspicious. Rapid progression of radiologic changes.
- tissue diagnosis on an adequate specimen (thoracotomy with pleural biopsy). Microscopic findings consistent with the epithelial (tubulopapillary), mesenchymal (fibrosarcomatous), or mixed or undifferentiated type.

The complexities and difficulties of the pathologic diagnosis have been discussed. The finding of hyaluronic acid in the pleural fluid of tissue specimen is useful, but the diagnosis cannot be discarded when the test is negative.

In the differential diagnosis of pleural mesothelioma, the following problems are of practical importance: (a) Benign pleural effusions may occur in a patient with present or past asbestos exposure. The clinical course is usually indicative, since benign pleural effusions tend to resolve spontaneously over several weeks. Nevertheless, such a "benign pleural effusion" has been observed, in some cases, to be a precursor of pleural mesothelioma. (b) Pleural fibrosis is a common finding in persons with present or past asbestos exposure; the prevalence increases with time since onset of exposure. In cases with extensive pleural fibrosis, especially when the width on chest x-ray exceeds 10 mm, the differential diagnosis between pleural fibrosis and pleural mesothelioma may be difficult. The presence of similar pleural changes on previous x-ray films makes the diagnosis of mesothelioma less likely; repeat chest x-ray films after several weeks are necessary when no previous chest x-ray are available. (c) The differential diagnosis between pleural mesothelioma (*primary* malignant tumor originating in the pleura) and secondary involvement of the pleura by a malignant tumor, either lung cancer or another primary malignant tumor with metastatic spread to the pleura, has been given much attention. In the case of lung cancer, sputum cytology and fiber optic bronchoscopy with bronchial biopsy, in addition to the radiologic appearance, contribute to the differential diagnosis. The proportion of cases which remain undecided is small. The possibility of a malignant primary tumor originating in another site, with metastatic spread to the pleura is investigated by the routine clinical work-up. Patients with no other detectable primary tumor but with clinical and radiologic features of mesothelioma have, with a high degree of probability, pleural mesothelioma. The absolute certainty of this differential diagnosis is reached only after postmortem examination.

In reviewing the experience accumulated over the last 20 years, it becomes obvious that pleural mesothelioma has been largely underdiagnosed in the past. This has been established in prospective cohort studies of asbestos-exposed workers (28)(33)(34)(38)(44)(47)(60); in many studies investigating diagnostic accuracy in series of reported mesothelioma cases (15); and in systematic reviews of all pathology material—as in Scotland where 80 undiagnosed cases were discovered (39).

In the 1967-1977 cohort study of 17,800 asbestos insulation workers in the United States and Canada, out of a total of 2,270 consecutive deaths, 60 were recorded on the death certificate as mesothelioma (31 pleural, 29 peritoneal). Review of medical records, including pathology reports, chest x-ray films, postmortem examinations (when available) and independent review of tissue specimens by experienced pathologists resulted in a diagnosis of mesothelioma in 175 cases (66 pleural, 109 peritoneal). The death certificate accuracy was 47% for pleural mesothelioma and 27% for peritoneal mesothelioma (Table VIII-32). In another cohort of 689 asbestos workers, 11 cases of mesothelioma (4 pleural, 7 peritoneal) were recorded on death certificates for the period 1959-1975. Review of medical records and pathology material resulted in a diagnosis of mesothelioma in 26 cases (14 pleural, 12 peritoneal), with the death certificate accuracy only 28% for pleural mesothelioma, and 58% for peritoneal mesothelioma (Table VIII-33).

In the majority of pleural mesothelioma cases it is possible to establish the diagnosis *in-travita*. The greater awareness of population groups with present or past exposure, of the Department of Health, Education and Welfare, of other governmental agencies, and of the medical community are expected to result in earlier diagnosis. This is a prerequisite for future meaningful attempts of therapy.

The requirement of postmortem examination for the definitive diagnosis is necessary for the complete assessment of mesothelioma incidence from an epidemiologic point of view, although it is expected that a higher index of suspicion will substantially reduce the difference between the number of cases diagnosed while alive and those in which the diagnosis is reached only after postmortem examination.

### METHODS OF PREVENTION

The prevention of pleural mesothelioma is dependent on the reduction of exposure to asbestos fiber to the minimum possible level, since this adverse health effect has been specifically associated with low level and short-term exposure. In December 1976, NIOSH, based on a "Reexamination and Update of Information on the Health Effects of Occupational Exposure to Asbestos," recommended to the DHEW and OSHA that the standard be reduced to 0.1 fibers/cm<sup>3</sup>. This was

based on the lowest concentration at which asbestos fibers can be reliably identified by phase contract microscopy.

### RESEARCH NEEDS

Critical problems where research is needed:

1. Determine mechanisms of carcinogenicity (mineral fibers; potential effect of other mineral fibers, such as zeolites, titanite fibers, etc.).
2. Define, to the extent that it is at all possible, the lowest level of asbestos exposure which may result in mesothelioma. This is of paramount importance for the acceptable standard.
3. Establish the role(s) of immune mechanisms in individual susceptibility for mesothelioma.
4. Determine mechanisms of carcinogenicity in peritoneal mesothelioma, including the significance of ingestion of fibers. This is important since water may be polluted with mineral fibers from various sources, and the risk of mesothelioma from such a situation has not yet been assessed.
5. Establish mesothelioma therapy.

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**Table VIII-32**  
**MORTALITY EXPERIENCE AMONG 17,800 ASBESTOS INSULATION WORKERS**  
**IN THE UNITED STATES AND CANADA 1967-1977:**  
**OBSERVATIONS IN 2,270 CONSECUTIVE DEATHS**

<i>Accuracy of death certificate categories</i>					
<i>Cause of death</i>	<i>Expected</i>	<i>Death Certificate</i>		<i>Ascertained</i>	
		<i>Number</i>	<i>o/e</i>	<i>Number</i>	<i>o/e</i>
<i>Cancer, all sites</i>	319.90	888	2.77	994	3.10
Cancer, lung	105.97	403	3.80	485	4.57
Pleural mesothelioma	—	31	—	66	—
Peritoneal mesothelioma	—	29	—	109	—
Cancer, esophagus	7.01	16	2.28	18	2.56
Cancer, stomach	14.23	19	1.34	22	1.55
Cancer, colon	37.86	58	1.50	59	1.56
Cancer, pancreas	17.46	48	2.75	22	1.26
Cancer, liver	7.50	18	2.40	5	0.66
Cancer, brain	10.34	19	1.84	14	1.35
<i>Asbestosis</i>	—	108	—	162	—
<i>Chronic obstructive lung disease</i>	58.58	127	2.17	66	1.13

Death certificate accuracy: Cancer, 89%; lung cancer, 83%; G.I. cancer, 94%; pleural mesothelioma, 47%; peritoneal mesothelioma, 27%.

**Table VIII-33**  
**RELATION BETWEEN DIAGNOSIS OF CAUSE OF DEATH AS RECORDED**  
**ON THE DEATH CERTIFICATE AND AS ASCERTAINED BY REVIEW**  
**OF ALL AVAILABLE INFORMATION, IN 274 DEATHS AMONG 689**  
**ASBESTOS WORKERS OBSERVED JANUARY 1, 1959 - DECEMBER 31, 1975**

	<i>Death certificate</i>	<i>Ascertained</i>
<i>Cancer, all sites</i>	94	99
Cancer of lung	36	35
Pleural mesothelioma	4	14
Peritoneal mesothelioma	7	12
Mesothelioma — unspecified site	7	0
Cancer of esophagus, stomach, colon, and rectum	12	15
All other cancer	28	23
<i>All respiratory disease</i>	43	42
Asbestosis	26	35
Pneumoconiosis	8	0
All respiratory disease	9	7
<i>All other causes</i>	137	133

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# **WORK-RELATED LUNG DISEASE SURVEILLANCE REPORT**

**Division of Respiratory Disease Studies**

**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Public Health Service  
Centers for Disease Control  
National Institute for Occupational Safety and Health**

**October 1991**

**DISCLAIMER**

**Mention of company names or products does not constitute endorsement by the National Institute for Occupational Safety and Health**

## Preface

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The Work-Related Lung Disease Surveillance Report was compiled by the Division of Respiratory Disease Studies, National Institute for Occupational Safety and Health. This report represents a summary of surveillance data for various occupational respiratory diseases. Some data originated from programs administered by the Division, e.g., information provided by the Coal Workers' X-ray Surveillance Program and the National Coal Workers' Autopsy Study. Other data were obtained from publications, reports, and data tapes provided by the National Center for Health Statistics, the Bureau of Labor Statistics, the Mine Safety and Health Administration, the Occupational Safety and Health Administration, the Health Care Financing Administration, and the Social Security Administration.

This report has two major sections: Figures and Tables. Section I contains 21 figures and Section II contains 59 tables. Figures display data from tables containing information best presented in graphical form. A corresponding table is provided for each graph to enable determination of actual numbers. A more detailed listing of the individual tables by disease category can be found at the first part of the Tables section. The Appendix briefly describes each of the major sources of data used in the report and, in some cases, directs the reader to additional documentation.

This first edition of the Work-Related Lung Disease Surveillance Report is a response to numerous requests for information about the extent of lung disease caused by exposures in the workplace. Surveillance information, including that contained in this report, derives from various sources which differ in completeness of reporting, case definitions, and populations of interest. Nevertheless, surveillance information can be of use in establishing priorities for investigation and intervention, as

well as in tracking progress toward the elimination of preventable disease.

Comments and suggestions from users of the report, as well as information about the uses to which it is being put, would be appreciated and will be used to increase the utility of future editions. Comments and suggestions may be sent to:

Work-Related Lung Disease Report  
Epidemiological Investigations Branch  
Division of Respiratory Disease Studies  
National Institute for Occupational Safety  
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# Contents

---

Preface	iii
---------	-----

Acknowledgments	iv
-----------------	----

## Section I - Figures

List of Figures	1
-----------------	---

Asbestosis	2
------------	---

Coal Workers' Pneumoconiosis	4
------------------------------	---

Silicosis	7
-----------	---

Exposure to Cotton Dust	9
-------------------------	---

Pneumonopathy due to Inhalation of other Dust	10
--	----

Hypersensitivity Pneumonitis	10
------------------------------	----

Toxic Agents	11
--------------	----

Dust Diseases of the Lungs	11
----------------------------	----

Compensation	12
--------------	----

## Section II - Tables

List of Detailed Tables	13
-------------------------	----

Asbestosis	16
------------	----

Coal Workers' Pneumoconiosis	27
------------------------------	----

Silicosis	37
-----------	----

Exposure to Cotton Dust	44
-------------------------	----

Pneumonopathy due to Inhalation of other Dust	46
--	----

Hypersensitivity Pneumonitis	50
------------------------------	----

Toxic Agents	54
--------------	----

Occupational Illness	59
----------------------	----

Dust Diseases of the Lungs	65
----------------------------	----

Dust Exposure Data	69
--------------------	----

Compensation	71
--------------	----

## Appendix

Sources and Limitations of Data	75
---------------------------------	----

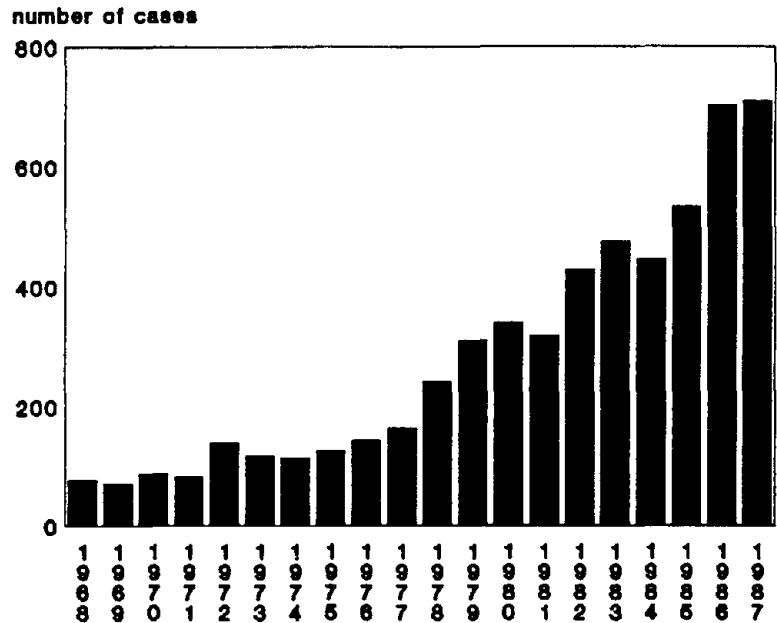
## Asbestosis

Asbestosis includes ICD-8 code 515.2 (asbestosis) and ICD-9 code 501 (asbestosis).

See Appendix for more information about multiple cause of death data.

See Table 6 for data.

**Figure 1. Multiple cause of death listings with any mention of asbestosis in United States residents age 15 and over, from 1968 to 1987**

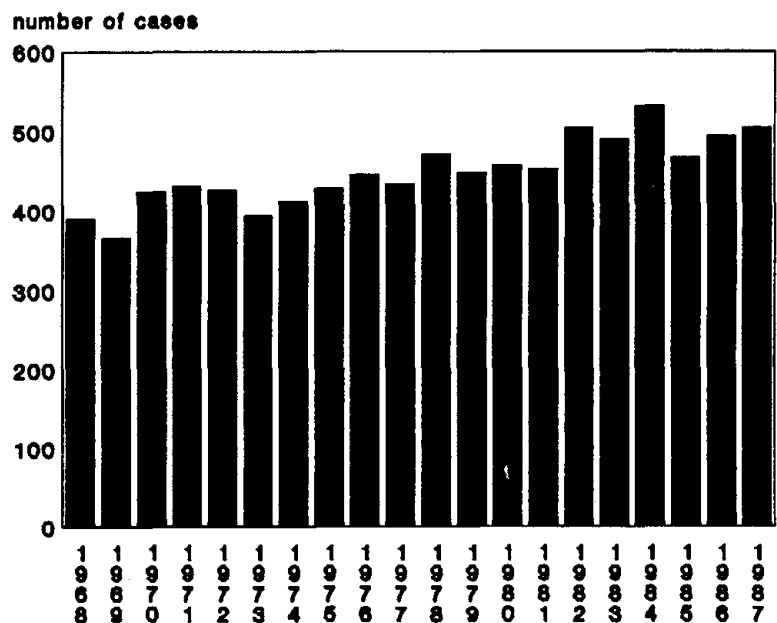


**Figure 2. Multiple cause of death listings with any mention of malignant neoplasm of pleura in United States residents age 15 and over, from 1968 to 1987**

Malignant neoplasm of pleura includes ICD-8 code 163.0 (malignant neoplasm of parietal pleura) and ICD-9 codes 163.0 (malignant neoplasm of parietal pleural), 163.1 (malignant neoplasm of visceral pleura), and 163.9 (malignant neoplasm of pleura, unspecified).

See Appendix for more information about multiple cause of death data.

See Table 7 for data.

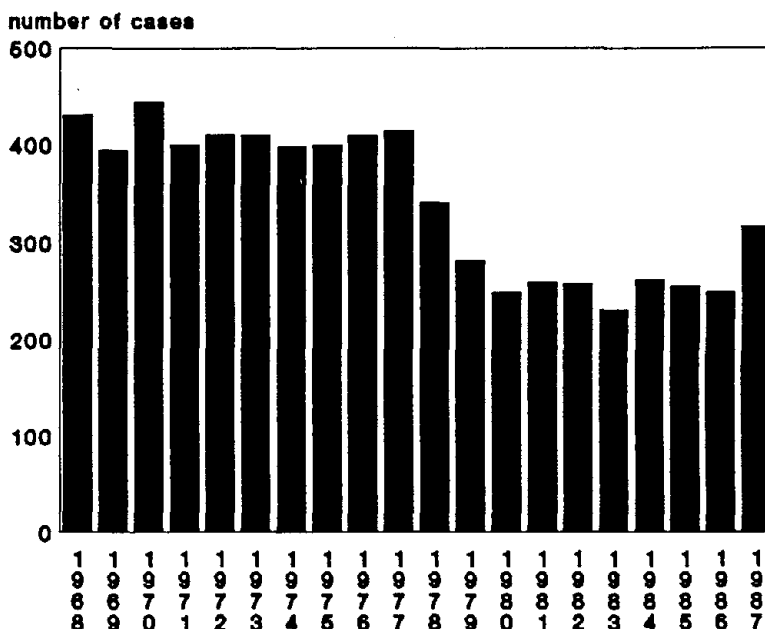


Malignant neoplasm of peritoneum includes ICD-8 code 158.9 (malignant neoplasm of peritoneum, excluding malignant neoplasm of retroperitoneal tissue) and ICD-9 codes 158.8 (malignant neoplasm, specified parts of peritoneum) and 158.9 (malignant neoplasm of peritoneum, unspecified).

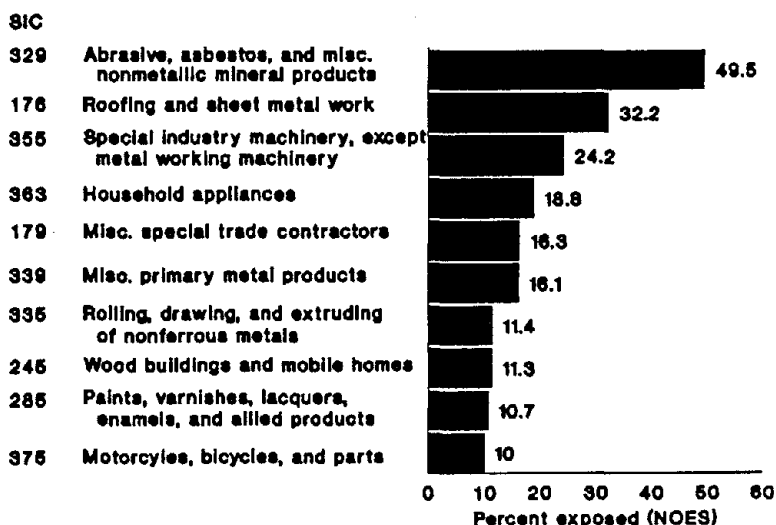
See Appendix for more information about multiple cause of death data.

See Table 8 for data.

**Figure 3. Multiple cause of death listings with any mention of malignant neoplasm of peritoneum in United States residents age 15 and over, from 1968 to 1987**



**Figure 4. Non-mining industries with the highest proportions of workers potentially exposed to asbestos dust, 1986**



Estimates of the proportions of workers potentially exposed to asbestos are based on data from the National Occupational Exposure Survey (NOES).

See Appendix for more information about NOES.

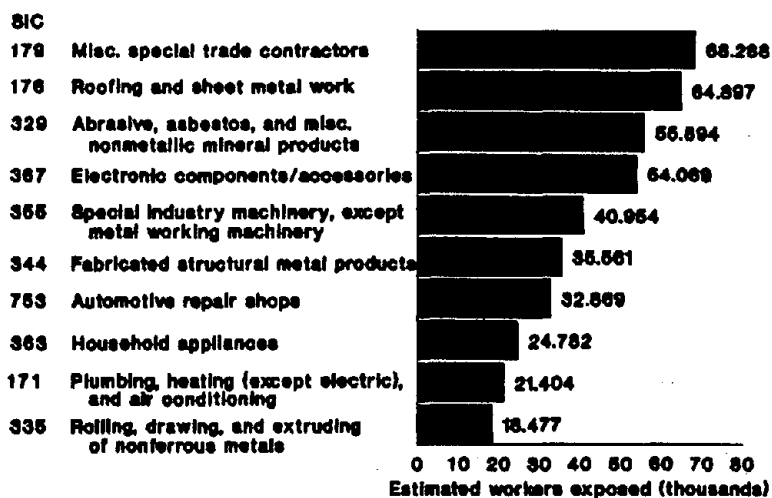
See Table 2 for data.

Estimates of the numbers of workers potentially exposed to asbestos are based on data from the 1986 County Business Patterns and the National Occupational Exposure Survey (NOES). SIC 13 (Oil and gas extraction) which was not calculated from NOES information, would have an estimated 40,824 workers exposed to asbestos based on an assumption that 10% of the oil and gas extraction work force is exposed to asbestosis.

See Appendix for more information about NOES and County Business Patterns.

See Table 3 for data.

**Figure 5. Non-mining Industries with the largest numbers of workers potentially exposed to asbestos dust, 1986**





**Table 1. Estimated numbers of workers with potential exposure to asbestos dust, by state, 1986**

State	Workers with Potential Exposure	
	Estimate # 1	Estimate # 2
Alabama.....	10,306	11,484
Alaska.....	722	1,298
Arizona.....	11,249	12,425
Arkansas.....	6,528	6,953
California.....	76,882	80,448
Colorado.....	7,420	9,794
Connecticut.....	14,045	14,140
District of Columbia.....	807	811
Delaware.....	1,961	1,965
Florida.....	29,715	30,746
Georgia.....	18,198	18,978
Hawaii.....	1,477	1,496
Idaho.....	1,577	1,893
Illinois.....	37,523	39,928
Indiana.....	20,748	21,562
Iowa.....	7,451	7,623
Kansas.....	6,755	8,147
Kentucky.....	10,679	14,475
Louisiana.....	9,344	15,396
Maine.....	3,064	3,080
Maryland.....	11,239	11,509
Massachusetts.....	23,847	24,010
Michigan.....	27,725	28,790
Minnesota.....	10,989	11,578
Mississippi.....	6,743	7,444
Missouri.....	13,753	14,320
Montana.....	990	1,537
Nebraska.....	3,067	3,200
Nevada.....	2,206	2,663
New Hampshire.....	4,633	4,662
New Jersey.....	24,234	24,461
New Mexico.....	2,703	4,473
New York.....	43,104	43,683
North Carolina.....	18,780	19,098
North Dakota.....	936	1,453
Ohio.....	39,745	41,916
Oklahoma.....	6,449	10,561
Oregon.....	6,039	6,224
Pennsylvania.....	36,987	40,296
Rhode Island.....	4,286	4,305
South Carolina.....	10,391	10,550
South Dakota.....	1,251	1,496
Tennessee.....	15,440	16,135
Texas.....	44,031	61,023
Utah.....	4,194	5,000
Vermont.....	1,884	1,955
Virginia.....	17,140	19,067
Washington.....	10,062	10,332
West Virginia.....	3,035	7,054
Wisconsin.....	15,566	15,704
Wyoming.....	829	2,891
TOTAL.....	688,729	760,032

NOTE: Both Estimate # 1 and Estimate # 2 are based on 1986 County Business Patterns and National Occupational Exposure Survey (NOES) estimates of the numbers of workers potentially exposed to asbestos. The differences between the two estimates are for establishments engaged in mining (SICs 10-14). The only NOES estimate used for SICs 10-14 is 0.49% of workers in SIC 1389. Estimate # 1 assumes 0.49% of workers in SIC 138 have potential exposure, but all other workers in SICs 10-14 have 0% potential exposure. Estimate # 2 assumes 10% of all workers in SICs 10-14 (and also SIC 138) have potential exposure to asbestos.

**Table 2. Non-mining industries with the highest proportions of workers potentially exposed to asbestos dust, 1986**

SIC	Description	Percent exposed (NOES)	Estimated workers exposed (1986)
329	Abrasive, asbestos, and miscellaneous nonmetallic mineral products.....	49.5	55,894
176	Roofing and sheet metal work.....	32.2	64,897
355	Special industry machinery, except metalworking machinery.....	24.2	40,954
363	Household appliances.....	18.8	24,782
179	Miscellaneous special trade contractors.....	16.3	68,288
339	Miscellaneous primary metal products.....	16.1	4,563
335	Rolling, drawing, and extruding of nonferrous metals.....	11.4	18,477
245	Wood buildings and mobile homes.....	11.3	7,340
285	Paints, varnishes, lacquers, enamels, and allied products.....	10.7	5,979
375	Motorcycles, bicycles, and parts.....	10.0	1,228

NOTE: Estimates of the proportion of workers potentially exposed to asbestos dust are based on data from the 1986 County Business Patterns and the National Occupational Exposure Survey (NOES).

**Table 3. Non-mining industries with the largest numbers of workers potentially exposed to asbestos dust, 1986**

SIC	Description	Percent exposed (NOES)	Estimated workers exposed (1986)
179	Miscellaneous special trade contractors.....	16.3	68,288
176	Roofing and sheet metal work.....	32.2	64,897
329	Abrasive, asbestos, and miscellaneous nonmetallic mineral products.....	49.5	55,894
367	Electronic components and accessories.....	9.7	54,069
355	Special industry machinery, except metalworking machinery.....	24.2	40,954
344	Fabricated structural metal products.....	9.0	35,561
753	Automotive repair shops.....	7.6	32,869
363	Household appliances.....	18.8	24,782
171	Plumbing, heating (except electric), and air conditioning.....	3.7	21,404
335	Rolling, drawing, and extruding of nonferrous metals.....	11.4	18,477

NOTE: Estimates of the number of workers potentially exposed to asbestos dust are based on data from the 1986 County Business Patterns and the National Occupational Exposure Survey (NOES).

**Table 4. Medicare hospitalizations with any mention of asbestosis, by state, from 1984 to 1989**

State	Number of hospitalizations					
	1984	1985	1986	1987	1988	1989
Alabama.....	60	69	111	136	138	181
Alaska.....	-	3	7	6	-	5
Arizona.....	66	32	51	88	47	64
Arkansas.....	26	12	34	21	53	14
California.....	607	578	587	766	866	884
Colorado.....	16	32	21	45	24	41
Connecticut.....	48	66	64	64	71	117
Delaware.....	30	34	41	52	52	52
District of Columbia.....	11	1	4	11	13	3
Florida.....	199	255	294	319	402	482
Georgia.....	54	40	48	90	84	115
Hawaii.....	17	19	11	12	17	22
Idaho.....	25	28	24	30	11	35
Illinois.....	96	111	200	156	138	220
Indiana.....	16	11	10	24	25	55
Iowa.....	15	8	27	24	22	34
Kansas.....	5	26	20	38	23	31
Kentucky.....	19	26	25	50	34	55
Louisiana.....	94	84	112	89	127	184
Maine.....	81	69	81	103	83	81
Maryland.....	63	96	143	156	175	198
Massachusetts.....	68	158	289	385	346	369
Michigan.....	43	34	36	42	51	71
Minnesota.....	22	47	25	49	70	78
Mississippi.....	70	57	124	150	186	199
Missouri.....	64	65	58	69	98	105
Montana.....	4	13	8	12	21	28
Nebraska.....	14	11	16	35	28	33
Nevada.....	22	25	14	17	30	13
New Hampshire.....	23	37	27	48	49	36
New Jersey.....	326	497	726	612	712	760
New Mexico.....	6	13	10	13	16	27
New York.....	84	193	288	349	401	437
North Carolina.....	52	42	67	110	157	161
North Dakota.....	21	-	-	1	-	9
Ohio.....	38	62	124	145	140	206
Oklahoma.....	15	16	22	15	25	12
Oregon.....	92	107	63	103	108	72
Pennsylvania.....	439	520	512	626	632	826
Rhode Island.....	17	15	8	12	16	22
South Carolina.....	31	55	83	83	96	79
South Dakota.....	1	7	3	6	1	6
Tennessee.....	42	68	95	77	107	76
Texas.....	150	214	213	318	359	529
Utah.....	13	10	18	17	10	10
Vermont.....	3	3	4	5	5	10
Virginia.....	122	114	154	227	301	281
Washington.....	210	160	234	250	265	272
West Virginia.....	32	67	49	99	99	91
Wisconsin.....	34	38	30	45	53	62
Wyoming.....	3	3	10	1	9	-
<b>TOTAL.....</b>	<b>3,626</b>	<b>4,252</b>	<b>5,226</b>	<b>6,203</b>	<b>6,801</b>	<b>7,758</b>

NOTE: Asbestosis = ICD-9CM code 501.

SOURCE: Medicare Provider Analysis and Review (MEDPAR), Office of Statistics and Data Management, Bureau of Data Management and Strategy, Health Care Financing Administration.

- indicates quantity zero.

**Table 5. Asbestosis cases reported to state workers' compensation agencies, by state, from 1980 to 1987**

State	Number of cases							
	1980	1981	1982	1983	1984	1985	1986	1987
Alabama.....								
Alaska.....		-	-	-	1	2	3	-
Arizona.....	-	-	1	-	-	-	1	-
Arkansas.....	1	-	-	-	-	-	1	-
California.....	11	10	7	25	8	8	28	22
Colorado.....	3	-	-	5	1	5	5	11
Connecticut....								
Delaware.....	2	2	5	6	8	10	15	4
District of Columbia.....								
Florida.....								
Georgia.....								
Hawaii.....	-	-	1	-	1	15	18	29
Idaho.....	-	-						
Illinois.....								
Indiana.....	1	1	1	-	-	1	-	-
Iowa.....	-	3	-	-	1	1	3	1
Kansas.....								
Kentucky.....	3	8	19	3	4	4	5	5
Louisiana.....						-	-	8
Maine.....	-	-	8					15
Maryland.....	1	2	4	1	2	3	-	1
Massachusetts..	-							
Michigan.....	-	1	-	1	2	2	1	2
Minnesota.....	6	15	6	12	8			
Mississippi.....	1	26	32	163	4	47	32	240
Missouri.....	2	2	1	2	1	1	3	3
Montana.....	-	-	-	-	-			
Nebraska.....	1	-	-	1	-	2	4	3
Nevada.....								
New Hampshire..								
New Jersey.....	2							
New Mexico.....	-	-	-	-	-	-	-	-
New York.....	29	16	22	22	22	34	33	48
North Carolina..	8	10	8	19	17	3	4	10
North Dakota...								
Ohio.....	12	34	44	44	45	86	461	1,099
Oklahoma.....							-	
Oregon.....	-	-	1	4	6	5	-	3
Pennsylvania...								
Rhode Island...								
South Carolina..								
South Dakota...								
Tennessee.....	3	2	16	2	2	9	8	50
Texas.....								
Utah.....	1	1	-	2	2	2		
Vermont.....	2	-	-	-	-			
Virginia.....	5		15	21	18	12	2	2
Washington.....	36	42	33	62	51	47	38	28
West Virginia..								
Wisconsin.....	4	6	9	5	7	13	15	21
Wyoming.....	-	-	-	-	-	-	-	-

NOTE: Asbestos = SDS code 283. Statistics for Arkansas, Delaware, New York, and North Carolina are for closed cases. Statistics for other states are for cases that occurred or were received during the year.

SOURCE: Bureau of Labor Statistics Supplementary Data System.

- indicates quantity zero. Empty space indicates information not available.

**Table 6. Multiple cause of death listings with any mention of asbestosis in United States residents age 15 and over, from 1968 to 1987**

Year	Number of cases
1968.....	77
1969.....	71
1970.....	87
1971.....	83
1972.....	138
1973.....	117
1974.....	114
1975.....	126
1976.....	143
1977.....	163
1978.....	240
1979.....	309
1980.....	339
1981.....	318
1982.....	428
1983.....	476
1984.....	445
1985.....	534
1986.....	702
1987.....	710

NOTE: Asbestosis = ICD-8 code 515.2 and ICD-9 code 501.

SOURCE: Tabulations are based on National Center for Health Statistics multiple cause of death data tapes, 1968-87.



# **NIOSH MANUAL OF ANALYTICAL METHODS**

## **THIRD EDITION**

Peter M. Eller, Ph.D., CIH  
Editor

## **VOLUME 1**

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Public Health Service  
Centers for Disease Control  
National Institute for Occupational Safety and Health  
Division of Physical Sciences and Engineering  
Cincinnati, Ohio

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DHHS (NIOSH) Publication No. 84-100

FORMULA: various	FIBERS
	METHOD: 7400
M.W.: various	ISSUED: 2/15/84
	REVISION #3: 5/15/89
OSHA: 0.2 asbestos fiber ( $\geq 5 \mu\text{m}$ long)/cc; 1 asbestos fiber/cc/30 minute excursion [1]	PROPERTIES: solid, fibrous
MSHA: 2 asbestos fibers ( $> 5 \mu\text{m}$ long)/cc [2]	
NIOSH: carcinogen; control to lowest level possible [3]; 3 glass fibers ( $> 10 \mu\text{m} \times < 3.5 \mu\text{m}$ )/cc [4]	
ACGIH: 0.2 crocidolite; 0.5 amosite; 2 chrysotile and other asbestos, fibers/cc [5]	

SYNONYMS: actinolite [CAS #13768-00-8] or ferroactinolite; cummingtonite-grunerite (amosite) [CAS #12172-73-5]; anthophyllite [CAS #17068-78-9]; chrysotile [CAS #12001-29-5] or serpentine; crocidolite [CAS #12001-28-4] or riebeckite; tremolite [CAS #14567-73-8]; amphibole asbestos; fibrous glass.

SAMPLING	MEASUREMENT
SAMPLER: FILTER (0.45- to 1.2- $\mu\text{m}$ cellulose ester membrane, 25-mm diameter; conductive cowl on cassette)	!TECHNIQUE: LIGHT MICROSCOPY, PHASE CONTRAST ! !ANALYTE: fibers (manual count) ! !SAMPLE PREPARATION: acetone/triacetin "hot block" method [6] !
FLOW RATE*: 0.5 to 16 L/min	!
VOL-MIN*: 400 L @ 0.1 fiber/cc -MAX*: (step 4, sampling)	!COUNTING RULES: Described in previous version ! of this method as A rules [1,7] !
*Adjust to give 100 to 1300 fibers/ $\text{mm}^2$	!
SHIPMENT: routine (pack to reduce shock)	!EQUIPMENT: 1. Positive phase-contrast microscope ! 2. Walton-Beckett graticule ! (100- $\mu\text{m}$ field of view) Type G-22 ! 3. phase-shift test slide (HSE/NPL) !
SAMPLE STABILITY: stable	!
FIELD BLANKS: 10% of samples	!CALIBRATION: HSE/NPL test slide !
ACCURACY	!RANGE: 100 to 1300 fibers/ $\text{mm}^2$ filter area !
RANGE STUDIED: 80 to 100 fibers counted	!ESTIMATED LOD: 7 fibers/ $\text{mm}^2$ filter area !
BIAS: see EVALUATION OF METHOD	!PRECISION: 0.10 to 0.12 [7]; see EVALUATION OF ! METHOD !
OVERALL PRECISION ( $s_p$ ): 0.115 to 0.13 [7]	!

APPLICABILITY: The quantitative working range is 0.04 to 0.5 fiber/cc for a 1000-L air sample. The LOD depends on sample volume and quantity of interfering dust, and is  $< 0.01$  fiber/cc for atmospheres free of interferences. The method gives an index of airborne fibers. It is primarily used for estimating asbestos concentrations, though PCM does not differentiate between asbestos and other fibers. Use this method in conjunction with electron microscopy (e.g., Method 7402) for assistance in identification of fibers. Fiber  $< \text{ca. } 0.25 \mu\text{m}$  diameter will not be detected by this method [8]. This method may be used for other materials such as fibrous glass by using alternate counting rules (see Appendix C).

INTERFERENCES: Any other airborne fiber may interfere since all particles meeting the counting criteria are counted. Chain-like particles may appear fibrous. High levels of non-fibrous dust particles may obscure fibers in the field of view and increase the detection limit.

OTHER METHODS: This method introduces changes for improved sensitivity and reproducibility. It replaces P&CAM 239 [7,9] and NIOSH Method 7400, Revision #2 (dated 8/15/87).

## FIBERS

METHOD: 7400

## REAGENTS:

1. Acetone.\*
2. Triacetin (glycerol triacetate), reagent grade.

\*See SPECIAL PRECAUTIONS.

## EQUIPMENT:

1. Sampler: field monitor, 25-mm, three-piece cassette with ca. 50-mm electrically-conductive extension cowl and cellulose ester filter, 0.45- to 1.2- $\mu$ m pore size, and backup pad.  
NOTE 1: Analyze representative filters for fiber background before use. Discard the filter lot if mean is  $\geq 5$  fibers per 100 graticule fields. These are defined as laboratory blanks. Manufacturer-provided quality assurance checks on filter blanks are normally adequate as long as field blanks are analyzed as described below.  
NOTE 2: The electrically-conductive extension cowl reduces electrostatic effects. Ground the cowl when possible during sampling [10].  
NOTE 3: Use 0.8- $\mu$ m pore size filters for personal sampling. The 0.45- $\mu$ m filters are recommended for sampling when performing TEM analysis on the same samples. However, their higher pressure drop precludes their use with personal sampling pumps.
2. Sampling pump, 0.5 to 16 L/min (see step 4 for flow rate), with flexible connecting tubing.
3. Microscope, positive phase (dark) contrast, with green or blue filter, adjustable field iris, 8 to 10X eye-piece, and 40 to 45X phase objective (total magnification ca. 400X); numerical aperture = 0.65 to 0.75.
4. Slides, glass, frosted-end, pre-cleaned, 25- x 75-mm.
5. Cover slips, 22- x 22-mm, No. 1-1/2, unless otherwise specified by microscope manufacturer.
6. Lacquer or nail polish.
7. Knife, #10 surgical steel, curved blade.
8. Tweezers.
9. Heated aluminum block for clearing filters on glass slides (see ref. [6] for specifications or see manufacturer's instructions for equivalent devices).
10. Micropipets, 5- $\mu$ L and 100- to 500- $\mu$ L.
11. Graticule, Walton-Beckett type, 100- $\mu$ m diameter circular field (area = 0.00785 mm<sup>2</sup>) at specimen plane (Type G-22). Available from PTR Optics Ltd., 145 Newton Street, Waltham, MA 02154 [phone (617) 891-6000] and McCrone Accessories and Components, 850 Pasquinelli Drive, Westmont, IL 60559 [phone (312) 887-7100].  
NOTE: The graticule is custom-made for each microscope. (See Appendix A for the custom-ordering procedure).
12. HSE/NPL phase contrast test slide, Mark II. Available from PTR Optics Ltd. (address above).
13. Telescope, ocular phase-ring centering.
14. Stage micrometer (0.01-mm divisions).
15. Wire, multi-stranded, 22-gauge.
16. Tape, shrink- or adhesive-.

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**SPECIAL PRECAUTIONS:** Acetone is extremely flammable. Take precautions not to ignite it. Heating of acetone in volumes greater than 1 mL must be done in a ventilated laboratory fume hood using a flameless, spark-free heat source.

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## SAMPLING:

1. Calibrate each personal sampling pump with a representative sampler in line.
2. For personal sampling, fasten sampler to the worker's lapel near the worker's mouth. Remove top cover from cowl extension ("open-face") and orient face down. Wrap joint between cowl and monitor body with tape to help hold the cassette together, keep the joint free of dust, and provide a marking surface to identify the cassette.

NOTE: If possible, ground the cassette to remove any surface charge, using a wire held in contact (e.g., with a hose clamp) with the conductive cowl and an earth ground such as a cold-water pipe.

3. Submit at least two field blanks (or 10% of the total samples, whichever is greater) for each set of samples. Handle field blanks in the same fashion as other samplers. Open field blank cassettes at the same time as other cassettes just prior to sampling. Store top covers and cassettes in a clean area with the top covers from the sampling cassettes during the sampling period.
4. Sample at 0.5 L/min or greater [1]. Adjust sampling flow rate,  $Q$  (L/min), and time,  $t$  (min), to produce a fiber density,  $E$ , of 100 to 1300 fibers/mm<sup>2</sup> ( $3.85 \cdot 10^4$  to  $5 \cdot 10^5$  fibers per 25-mm filter with effective collection area  $A_c = 385 \text{ mm}^2$ ) for optimum accuracy. These variables are related to the action level (one-half the current standard),  $L$  (fibers/cc), of the fibrous aerosol being sampled by:

$$t = \frac{A_c \cdot E}{Q \cdot L \cdot 10^3}, \text{ min.}$$

NOTE 1: The purpose of adjusting sampling times is to obtain optimum fiber loading on the filter. A sampling rate of 1 to 4 L/min for 8 hrs is appropriate in atmospheres containing ca. 0.1 fiber/cc in the absence of significant amounts of non-asbestos dust. Dusty atmospheres require smaller sample volumes ( $\leq 400$  L) to obtain countable samples. In such cases take short, consecutive samples and average the results over the total collection time. For documenting episodic exposures, use high flow rates (7 to 16 L/min) over shorter sampling times. In relatively clean atmospheres, where targeted fiber concentrations are much less than 0.1 fiber/cc, use larger sample volumes (3000 to 10000 L) to achieve quantifiable loadings. Take care, however, not to overload the filter with background dust. If  $\geq 50\%$  of the filter surface is covered with particles, the filter may be too overloaded to count and will bias the measured fiber concentration.

NOTE 2: OSHA regulations specify a maximum sampling rate of 2.5 L/min [1].

NOTE 3: OSHA regulations specify a minimum sampling volume of 48 L for an excursion measurement [1].

5. At the end of sampling, replace top cover and end plugs.
6. Ship samples with conductive cowl attached in a rigid container with packing material to prevent jostling or damage.

NOTE: Do not use untreated polystyrene foam in shipping container because electrostatic forces may cause fiber loss from sample filter.

## SAMPLE PREPARATION:

NOTE 1: The object is to produce samples with a smooth (non-grainy) background in a medium with refractive index of  $\leq 1.46$ . This method collapses the filter for easier focusing and produces relatively permanent mounts which are useful for quality control and interlaboratory comparison. The aluminum "hot block" or similar flash vaporization techniques may be used outside the laboratory [6]. Other mounting techniques meeting the above criteria may also be used (e.g., the laboratory fume hood procedure for generating acetone vapor as described in Method 7400 - revision of 5/15/85, or the non-permanent field mounting technique used in P&CAM 239 [3,7,9,12]). A videotape of the mounting procedure is available from the NIOSH Publication Office [13].

- NOTE 2: Excessive water in the acetone may slow the clearing of the filters, causing material to be washed off the surface of the filter. Also, filters that have been exposed to high humidities prior to clearing may have a grainy background.
7. Ensure that the glass slides and cover slips are free of dust and fibers.
  8. Adjust the rheostat to heat the "hot block" to ca. 70 °C [6].
 

NOTE: If the "hot block" is not used in a fume hood, it must rest on a ceramic plate and be isolated from any surface susceptible to heat damage.
  9. Mount a wedge cut from the sample filter on a clean glass slide.
    - a. Cut wedges of ca. 25% of the filter area with a curved-blade knife using a rocking motion to prevent tearing. Place wedge, dust side up, on slide.
 

NOTE: Static electricity will usually keep the wedge on the slide.
    - b. Insert slide with wedge into the receiving slot at the base of "hot block". Immediately place tip of a micropipet containing ca. 250 µL acetone (use the minimum volume needed to consistently clear the filter sections) into the inlet port of the PTFE cap on top of the "hot block" and inject the acetone into the vaporization chamber with a slow, steady pressure on the plunger button while holding pipet firmly in place. After waiting 3 to 5 sec for the filter to clear, remove pipet and slide from their ports.
 

CAUTION: Although the volume of acetone used is small, use safety precautions. Work in a well-ventilated area (e.g., laboratory fume hood). Take care not to ignite the acetone. Continuous, frequent use of this device in an unventilated space may produce explosive acetone vapor concentrations.
    - c. Using the 5-µL micropipet, immediately place 3.0 to 3.5 µL triacetin on the wedge. Gently lower a clean cover slip onto the wedge at a slight angle to reduce bubble formation. Avoid excess pressure and movement of the cover glass.
 

NOTE: If too many bubbles form or the amount of triacetin is insufficient, the cover slip may become detached within a few hours. If excessive triacetin remains at the edge of the filter under the cover slip, fiber migration may occur.
    - d. Glue the edges of the cover slip to the slide using lacquer or nail polish [14]. Counting may proceed immediately after clearing and mounting are completed.
 

NOTE: If clearing is slow, warm the slide on a hotplate (surface temperature 50 °C) for up to 15 min to hasten clearing. Heat carefully to prevent gas bubble formation.

#### CALIBRATION AND QUALITY CONTROL:

10. Microscope adjustments. Follow the manufacturer's instructions. At least once daily use the telescope ocular (or Bertrand lens, for some microscopes) supplied by the manufacturer to ensure that the phase rings (annular diaphragm and phase-shifting elements) are concentric. With each microscope, keep a logbook in which to record the dates of calibrations and major servicings.
  - a. Each time a sample is examined, do the following:
    - (1) Adjust the light source for even illumination across the field of view at the condenser iris. Use Kohler illumination, if available. With some microscopes, the illumination may have to be set up with bright field optics rather than phase contrast optics.
    - (2) Focus on the particulate material to be examined.
    - (3) Make sure that the field iris is in focus, centered on the sample, and open only enough to fully illuminate the field of view.
  - b. Check the phase-shift detection limit of the microscope periodically for each analyst/microscope combination:
    - (1) Center the HSE/NPL phase-contrast test slide under the phase objective.
    - (2) Bring the blocks of grooved lines into focus in the graticule area.
 

NOTE: The slide contains seven blocks of grooves (ca. 20 grooves per block) in descending order of visibility. For asbestos counting the microscope optics must completely resolve the grooved lines in block 3 although they may appear somewhat faint, and the grooved lines in blocks 6 and 7 must be invisible

when centered in the graticule area. Blocks 4 or 5 must be at least partially visible but may vary slightly in visibility between microscopes. A microscope which fails to meet these requirements has resolution either too low or too high for fiber counting.

- (3) If image quality deteriorates, clean the microscope optics. If the problem persists, consult the microscope manufacturer.

11. Document the laboratory's precision for each counter for replicate fiber counts.
  - a. Maintain as part of the laboratory quality assurance program a set of reference slides to be used on a daily basis [15]. These slides should consist of filter preparations including a range of loadings and background dust levels from a variety of sources including both field and PAT samples. The Quality Assurance Officer should maintain custody of the reference slides and should supply each counter with a minimum of one reference slide per workday. Change the labels on the reference slides periodically so that the counter does not become familiar with the samples.
  - b. From blind repeat counts on reference slides, estimate the laboratory intra- and intercounter  $s_r$  (step 21). Obtain separate values of relative standard deviation for each sample matrix analyzed in each of the following ranges: 5 to 20 fibers in 100 graticule fields, >20 to 50 fibers in 100 graticule fields, >50 to 100 fibers in 100 graticule fields, and 100 fibers in less than 100 graticule fields. Maintain control charts for each of these data files.

NOTE: Certain sample matrices (e.g., asbestos cement) have been shown to give poor precision [16]

12. Prepare and count field blanks along with the field samples. Report counts on each field blank.

NOTE 1: The identity of blank filters should be unknown to the counter until all counts have been completed.

NOTE 2: If a field blank yields greater than 7 fibers per 100 graticule fields, report possible contamination of the samples.

13. Perform blind recounts by the same counter on 10% of filters counted (slides relabeled by a person other than the counter). Use the following test to determine whether a pair of counts by the same counter on the same filter should be rejected because of possible bias: Discard the sample if the absolute value of the difference between the square roots of the two counts (in fiber/mm<sup>2</sup>) exceeds 2.8 (X)  $s_r$ , where X = the average of the square roots of the two fiber counts (in fiber/mm<sup>2</sup>) and  $s_r$  = one-half the intracounter relative standard deviation for the appropriate count range (in fibers) determined from step 11. For more complete discussions see reference [15].

NOTE 1: Since fiber counting is the measurement of randomly placed fibers which may be described by a Poisson distribution, a square root transformation of the fiber count data will result in approximately normally distributed data [15].

NOTE 2: If a pair of counts is rejected by this test, recount the remaining samples in the set and test the new counts against the first counts. Discard all rejected paired counts. It is not necessary to use this statistic on blank counts.

14. The analyst is a critical part of this analytical procedure. Care must be taken to provide a non-stressful and comfortable environment for fiber counting. An ergonomically designed chair should be used, with the microscope eyepiece situated at a comfortable height for viewing. External lighting should be set at a level similar to the illumination level in the microscope to reduce eye fatigue. In addition, counters should take 10 to 20 minute breaks from the microscope every one or two hours to limit fatigue [17]. During these breaks, both eye and upper back/neck exercises should be performed to relieve strain.

15. All laboratories engaged in asbestos counting should participate in a proficiency testing program such as the AIHA-NIOSH Proficiency Analytical Testing (PAT) Program or the AIHA Asbestos Analyst Registry and routinely exchange field samples with other laboratories to compare performance of counters.

NOTE: OSHA requires that each analyst performing this method take the NIOSH direct training course #582 or equivalent [1]. Instructors of equivalent courses should have attended the NIOSH #582 course at NIOSH within three years of presenting an equivalent course.

## MEASUREMENT:

16. Center the slide on the stage of the calibrated microscope under the objective lens. Focus the microscope on the plane of the filter.
17. Adjust the microscope (Step 10).

NOTE: Calibration with the HSE/NPL test slide determines the minimum detectable fiber diameter (ca. 0.25  $\mu\text{m}$ ) [8].

18. Counting rules: (same as P&CAM 239 rules [3,7,9]; see APPENDIX B).
  - a. Count only fibers longer than 5  $\mu\text{m}$ . Measure length of curved fibers along the curve.
  - b. Count only fibers with a length-to-width ratio equal to or greater than 3:1.
  - c. For fibers which cross the boundary of the graticule field:
    - (1) Count any fiber longer than 5  $\mu\text{m}$  which lies entirely within the graticule area.
    - (2) Count as 1/2 fiber any fiber with only one end lying within the graticule area, provided that the fiber meets the criteria of rules a and b above.
    - (3) Do not count any fiber which crosses the graticule boundary more than once.
    - (4) Reject and do not count all other fibers.
  - d. Count bundles of fibers as one fiber unless individual fibers can be identified by observing both ends of a fiber.
  - e. Count enough graticule fields to yield 100 fibers. Count a minimum of 20 fields. Stop at 100 graticule fields regardless of count.
19. Start counting from the tip of the filter wedge and progress along a radial line to the outer edge. Shift up or down on the filter, and continue in the reverse direction. Select graticule fields randomly by looking away from the eyepiece briefly while advancing the mechanical stage. Ensure that, as a minimum, each analysis covers one radial line from the filter center to the outer edge of the filter. When an agglomerate covers ca. 1/6 or more of the graticule field, reject the graticule field and select another. Do not report rejected graticule fields in the total number counted.

NOTE 1: When counting a graticule field, continuously scan a range of focal planes by moving the fine focus knob to detect very fine fibers which have become embedded in the filter. The small-diameter fibers will be very faint but are an important contribution to the total count. A minimum counting time of 15 seconds per field is appropriate for accurate counting.

NOTE 2: This method does not allow for differentiation of fibers based on morphology. Although some experienced counters are capable of selectively counting only fibers which appear to be asbestiform, there is presently no accepted method for ensuring uniformity of judgment between laboratories. It is, therefore, incumbent upon all laboratories using this method to report total fiber counts. If serious contamination from non-asbestos fibers occurs in samples, other techniques such as transmission electron microscopy must be used to identify the asbestos fiber fraction present in the sample (see NIOSH Method 7402). In some cases (i.e., for fibers with diameters  $>1 \mu\text{m}$ ), polarized light microscopy techniques may be used to identify and eliminate interfering non-crystalline fibers [18].

NOTE 3: Under certain conditions, electrostatic charge may affect the sampling of fibers. These electrostatic effects are most likely to occur when the relative humidity is low (below 20%), and when sampling is performed near the source of aerosol. The result is that deposition of fibers on the filter is reduced, especially near the edge of the filter. If such a pattern is noted during fiber counting, choose fields as close to the center of the filter as possible [10].

## CALCULATIONS AND REPORTING OF RESULTS:

20. Calculate and report fiber density on the filter,  $E$  (fibers/ $\text{mm}^2$ ), by dividing the average fiber count per graticule field,  $F/n_f$ , minus the mean field blank count per graticule field,  $B/n_b$ , by the graticule field area,  $A_f$  (approx. 0.00785  $\text{mm}^2$ ):

$$E = \frac{\left( \frac{F}{n_f} - \frac{B}{n_b} \right)}{A_f}, \text{ fibers/mm}^2.$$

NOTE: Fiber counts above 1300 fibers/mm<sup>2</sup> and fiber counts from samples with >50% of filter area covered with particulate should be reported as "uncountable" or "probably biased."

21. Calculate and report the concentration, C (fibers/cc), of fibers in the air volume sampled, V (L), using the effective collection area of the filter, A<sub>c</sub> (approx. 385 mm<sup>2</sup> for a 25-mm filter):

$$C = \frac{(E)(A_c)}{V \cdot 10^3}$$

NOTE: Periodically check and adjust the value of A<sub>c</sub>, if necessary.

22. Report intralaboratory and interlaboratory relative standard deviations (Step 11) with each set of results.

NOTE: Precision depends on the total number of fibers counted [7,19]. Relative standard deviation is documented in references [7,18,19,20] for fiber counts up to 100 fibers in 100 graticule fields. Comparability of interlaboratory results is discussed below. As a first approximation, use 213% above and 49% below the count as the upper and lower confidence limits for fiber counts greater than 20 (Fig. 1).

#### EVALUATION OF METHOD:

A. This method is a revision of P&CAM 239 [3,7,9]. A summary of the revisions is as follows:

##### 1. Sampling:

The change from a 37-mm to a 25-mm filter improves sensitivity for similar air volumes. The change in flow rates allows for 2-m<sup>3</sup> full-shift samples to be taken, providing that the filter is not overloaded with non-fibrous particulates. The collection efficiency of the sampler is not a function of flow rate in the range 0.5 to 16 L/min [11].

##### 2. Sample Preparation Technique:

The acetone vapor-triacetin preparation technique is a faster, more permanent mounting technique than the dimethyl phthalate/diethyl oxalate method of P&CAM 239 [6,8,9]. The aluminum "hot block" technique minimizes the amount of acetone needed to prepare each sample.

##### 3. Measurement:

- The Walton-Beckett graticule standardizes the area observed [21,22,23].
- The HSE/NPL test slide standardizes microscope optics for sensitivity to fiber diameter [8,21].
- Because of past inaccuracies associated with low fiber counts, the minimum recommended loading has been increased to 100 fibers/mm<sup>2</sup> filter area (80 fibers total count). Lower levels generally result in an overestimate of the fiber count when compared to results in the recommended analytical range [25]. The recommended loadings should yield intracounter  $s_r$  in the range of 0.10 to 0.17 [7,24,26].

#### B. Interlaboratory comparability:

An international collaborative study involved 16 laboratories using prepared slides from the asbestos cement, milling, mining, textile, and friction material industries [16]. The relative standard deviations ( $s_r$ ) varied with sample type and laboratory. The ranges were:

	$s_r$		
	<u>Intralaboratory</u>	<u>Interlaboratory</u>	<u>Overall</u>
AIA (NIOSH Rules)*	0.12 to 0.40	0.27 to 0.85	0.46

\*Under AIA rules, only fibers having a diameter less than 3 μm are counted and fibers attached to particles larger than 3 μm are not counted. NIOSH Rules are otherwise similar to the AIA rules.



A NIOSH study was conducted using field samples of asbestos [24]. This study indicated intralaboratory  $s_r$  in the range 0.17 to 0.25 and an interlaboratory  $s_r$  of 0.45. This agrees well with other recent studies [16,19,21].

At this time, there is no independent means for assessing the overall accuracy of this method. One measure of reliability is to estimate how well the count for a single sample agrees with the mean count from a large number of laboratories. The following discussion indicates how this estimation can be carried out based on measurements of the interlaboratory variability, as well as showing how the results of this method relate to the theoretically attainable counting precision and to measured intra- and interlaboratory  $s_r$ . (NOTE: The following discussion does not include bias estimates and should not be taken to indicate that lightly loaded samples are as accurate as properly loaded ones).

Theoretically, the process of counting randomly-distributed (Poisson) fibers on a filter surface will give an  $s_r$  that depends on the number,  $N$ , of fibers counted:

$$s_r = 1/(N)^{1/2} \quad (1)$$

Thus  $s_r$  is 0.1 for 100 fibers and 0.32 for 10 fibers counted. The actual  $s_r$  found in a number of studies is greater than these theoretical numbers [16,19,20,21].

An additional component of variability comes primarily from subjective interlaboratory differences. In a study of ten counters in a continuing sample exchange program, Ogden [18] found this subjective component of intralaboratory  $s_r$  to be approximately 0.2 and estimated the overall  $s_r$  by the term:

$$\frac{(N + (0.2 \cdot N)^2)^{1/2}}{N} \quad (2)$$

Ogden found that the 90% confidence interval of the individual intralaboratory counts in relation to the means were  $+2 s_r$  and  $-1.5 s_r$ . In this program, one sample out of ten was a quality control sample. For laboratories not engaged in an intensive quality assurance program, the subjective component of variability can be higher.

In a study of field sample results in 46 laboratories, the Asbestos Information Association also found that the variability had both a constant component and one that depended on the fiber count [21]. These results gave a subjective interlaboratory component of  $s_r$  (on the same basis as Ogden's) for field samples of ca. 0.45. A similar value was obtained for 12 laboratories analyzing a set of 24 field samples [24]. This value falls slightly above the range of  $s_r$  (0.25 to 0.42 for 1984-85) found for 80 reference laboratories in the NIOSH Proficiency Analytical Testing (PAT) program for laboratory-generated samples [20].

A number of factors influence  $s_r$  for a given laboratory, such as that laboratory's actual counting performance and the type of samples being analyzed. In the absence of other information, such as from an interlaboratory quality assurance program using field samples, the value for the subjective component of variability is estimated as 0.45. It is hoped that laboratories will carry out the recommended interlaboratory quality assurance programs to improve their performance and thus reduce the  $s_r$ .

The above relative standard deviations apply when the population mean has been determined. It is more useful, however, for laboratories to estimate the 90% confidence interval on the mean count from a single sample fiber count (Figure 1). These curves assume similar shapes of the count distribution for interlaboratory and intralaboratory results [19].

For example, if a sample yields a count of 24 fibers, Figure 1 indicates that the mean inter-laboratory count will fall within the range of 227% above and 52% below that value 90% of the time. We can apply these percentages directly to the air concentrations as well. If, for instance, this sample (24 fibers counted) represented a 500-L volume, then the measured concentration is 0.02 fibers/mL (assuming 100 fields counted, 25-mm filter, 0.00785 mm<sup>2</sup> field counting area). If this same sample were counted by a group of laboratories, there is a 90% probability that the mean would fall between 0.01 and 0.08 fiber/mL. These limits should be reported in any comparison of results between laboratories.

Note that the  $s_r$  of 0.45 used to derive Figure 1 is used as an estimate for a random group of laboratories. If several laboratories belonging to a quality assurance group can show that their interlaboratory  $s_r$  is smaller, then it is more correct to use that smaller  $s_r$ . However, the estimated  $s_r$  of 0.45 is to be used in the absence of such information. Note also that it has been found that  $s_r$  can be higher for certain types of samples, such as asbestos cement [16].

Quite often the estimated airborne concentration from an asbestos analysis is used to compare to a regulatory standard. For instance, if one is trying to show compliance with an 0.5 fiber/mL standard using a single sample on which 100 fibers have been counted, then Figure 1 indicates that the 0.5 fiber/mL standard must be 213% higher than the measured air concentration. This indicates that if one measures a fiber concentration of 0.16 fiber/mL (100 fibers counted), then the mean fiber count by a group of laboratories (of which the compliance laboratory might be one) has a 95% chance of being less than 0.5 fibers/mL; i.e.,  $0.16 + 2.13 \times 0.16 = 0.5$ .

It can be seen from Figure 1 that the Poisson component of the variability is not very important unless the number of fibers counted is small. Therefore, a further approximation is to simply use +213% and -49% as the upper and lower confidence values of the mean for a 100-fiber count.

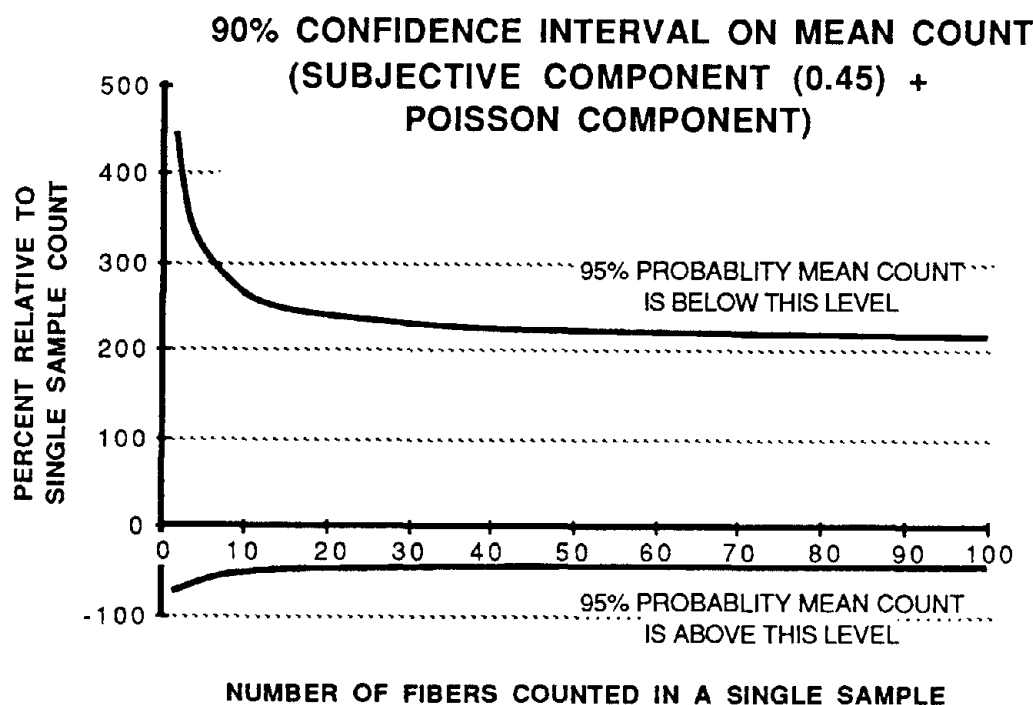


Figure 1. Inter-laboratory Precision of Fiber Counts

The curves in Figure 1 are defined by the following equations

$$UCL = \frac{2x + 2.25 + \sqrt{(2.25 + 2x)^2 - 4(1 - 2.25s_r^2)x^2}}{2(1 - 2.25s_r^2)} \quad (3)$$

$$LCL = \frac{2x + 4 - \sqrt{(4 + 2x)^2 - 4(1 - 4s_r^2)x^2}}{2(1 - 4s_r^2)} \quad (4)$$

where  $s_r$  = subjective inter-laboratory relative standard deviation, which is close to the total inter-laboratory  $s_r$  when approximately 100 fibers are counted.

$x$  = total fibers counted on sample

LCL = lower 95% confidence limit

UCL = upper 95% confidence limit.

Note that the range between these two limits represents 90% of the total range.

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#### APPENDIX A: CALIBRATION OF THE WALTON-BECKETT GRATICULE

Before ordering the Walton-Beckett graticule, the following calibration must be done to obtain a counting area (D) 100  $\mu\text{m}$  in diameter at the image plane. The diameter,  $d_c$  (mm), of the circular counting area and the disc diameter must be specified when ordering the graticule.

1. Insert any available graticule into the eyepiece and focus so that the graticule lines are sharp and clear.
2. Set the appropriate interpupillary distance and, if applicable, reset the binocular head adjustment so that the magnification remains constant.
3. Install the 40 to 45X phase objective.
4. Place a stage micrometer on the microscope object stage and focus the microscope on the graduated lines.
5. Measure the magnified grid length of the graticule,  $L_o$  ( $\mu\text{m}$ ), using the stage micrometer.
6. Remove the graticule from the microscope and measure its actual grid length,  $L_a$  (mm). This can best be accomplished by using a stage fitted with verniers.
7. Calculate the circle diameter,  $d_c$  (mm), for the Walton-Beckett graticule:

$$d_c = \frac{L_a}{L_o} \times D.$$

Example: If  $L_o = 112 \mu\text{m}$ ,  $L_a = 4.5 \text{ mm}$  and  $D = 100 \mu\text{m}$ , then  $d_c = 4.02 \text{ mm}$ .

8. Check the field diameter,  $D$  (acceptable range  $100 \mu\text{m} \pm 2 \mu\text{m}$ ) with a stage micrometer upon receipt of the graticule from the manufacturer. Determine field area (acceptable range  $0.00754$  to  $0.00817 \text{ mm}^2$ ).

#### APPENDIX B: EXAMPLES OF COUNTING RULES

Figure 2 shows a Walton-Beckett graticule as seen through the microscope. The rules will be discussed as they apply to the labeled objects in the figure.

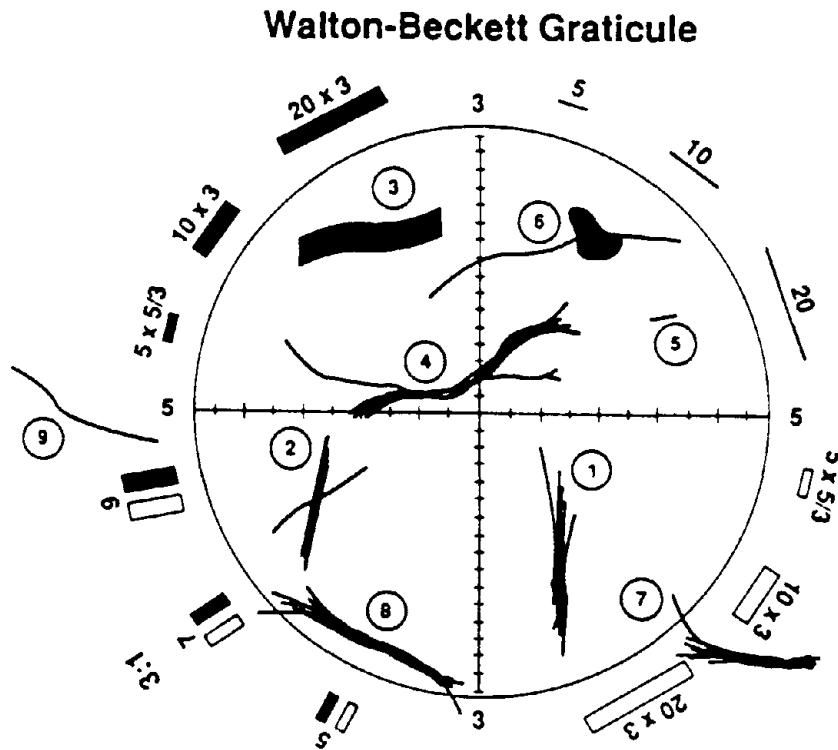


Figure 2. Walton-Beckett graticule with fibers.

METHOD: 7400

FIBERS

FIBER COUNT		DISCUSSION
Object	Count	
1	1 fiber	Optically observable asbestos fibers are actually bundles of fine fibrils. If the fibrils seem to be from the same bundle the object is counted as a single fiber. Note, however, that all objects meeting length and aspect ratio criteria are counted whether or not they appear to be asbestos.
2	2 fiber	If fibers meeting the length and aspect ratio criteria (length $>5\ \mu\text{m}$ and length-to-width ratio $>3$ to 1) overlap, but do not seem to be part of the same bundle, they are counted as separate fibers.
3	1 fiber	Although the object has a relatively large diameter ( $>3\ \mu\text{m}$ ), it is counted as fiber under the rules. There is no upper limit on the fiber diameter in the counting rules. Note that fiber width is measured at the widest compact section of the object.
4	1 fiber	Although long fine fibrils may extend from the body of a fiber, these fibrils are considered part of the fiber if they seem to have originally been part of the bundle.
5	Do not count	If the object is $\leq 5\ \mu\text{m}$ long, it is not counted.
6	1 fiber	A fiber partially obscured by a particle is counted as one fiber. If the fiber ends emanating from a particle do not seem to be from the same fiber and each end meets the length and aspect ratio criteria, they are counted as separate fibers.
7	1/2 fiber	A fiber which crosses into the graticule area one time is counted as 1/2 fiber.
8	Do not count	Ignore fibers that cross the graticule boundary more than once.
9	Do not count	Ignore fibers that lie outside the graticule boundary.

#### Appendix C. ALTERNATE COUNTING RULES

Other counting rules may be more appropriate for measurement of specific non-asbestos fiber types, such as fibrous glass. These include the "B" rules (from NIOSH Method 7400, Revision #2, dated 8/15/87), the World Health Organization reference method for man-made mineral fiber [27], and the NIOSH fibrous glass criteria document method [4]. The upper diameter limit in these methods prevents measurements of non-respirable fibers. It is important to note that the aspect ratio limits included in these methods vary. NIOSH recommends the use of the 3:1 aspect ratio in counting fibers.

It is emphasized that hybridization of different sets of counting rules is not permitted. Report specifically which set of counting rules are used with the analytical results.

## FIBERS

METHOD: 7400

## "B" Counting Rules:

1. Count only ends of fibers. Each fiber must be longer than 5  $\mu\text{m}$  and less than 3  $\mu\text{m}$  diameter.
2. Count only ends of fibers with a length-to-width ratio equal to or greater than 5:1.
3. Count each fiber end which falls within the graticule area as one end, provided that the fiber meets rules b.1 and b.2. Add split ends to the count as appropriate if the split fiber segment also meets the criteria of rules 1 and 2 above.
4. Count visibly free ends which meet rules 1 and 2 above when the fiber appears to be attached to another particle, regardless of the size of the other particle. Count the end of a fiber obscured by another particle if the particle covering the fiber end is less than 3  $\mu\text{m}$  in diameter.
5. Count free ends of fibers emanating from large clumps and bundles up to a maximum of 10 ends (5 fibers), provided that each segment meets rules 1 and 2 above.
6. Count enough graticule fields to yield 200 ends. Count a minimum of 20 graticule fields. Stop at 100 graticule fields, regardless of count.
7. Divide total end count by 2 to yield fiber count.

FORMULA: various	ASBESTOS FIBERS
	METHOD: 7402
M.W.: various	ISSUED: 8/15/87
	REVISION #1: 5/15/89
OSHA: 0.2 asbestos fiber (>5 µm long)/cc 1 asbestos fiber/cc/30 minute excursion [1]	PROPERTIES: solid, fibrous
MSHA: 2 asbestos fibers (>5 µm long)/cc [2]	
NIOSH: carcinogen; control to lowest level possible [3]	
ACGIH: 0.2 crocidolite; 0.5 amosite; 2 chrysotile and other asbestos, fibers/cc [4]	

SYNONYMS: actinolite [CAS #13768-00-8] or ferroactinolite; cummingtonite-grunerite (amosite) [CAS #12172-73-5]; anthophyllite [CAS #17068-78-9]; chrysotile [CAS #12001-29-5] or serpentine; crocidolite [CAS #12001-28-4] or riebeckite; tremolite [CAS #14567-73-8]; amphibole asbestos.

SAMPLING	MEASUREMENT
SAMPLER: FILTER (0.45- to 1.2-µm cellulose ester membrane, 25-mm diameter; conductive cassette)	! TECHNIQUE: MICROSCOPY, TRANSMISSION ELECTRON ! (TEM) !
FLOW RATE*: 0.5 to 16 L/min	! ANALYTE: asbestos fibers !
VOL-MIN*: 400 L @ 0.1 fiber/cc -MAX*: (step 4, sampling) *Adjust for 100 to 1300 fibers/mm <sup>2</sup>	! SAMPLE PREPARATION: modified Jaffe wick !
SHIPMENT: routine (pack to reduce shock)	! EQUIPMENT: transmission electron microscope; ! energy dispersive X-ray system (EDX) ! analyzer !
SAMPLE STABILITY: stable	! CALIBRATION: qualitative electron diffraction; ! calibration of TEM magnification ! and EDX system !
FIELD BLANKS: 10% of samples	! RANGE: 100 to 1300 fibers/mm <sup>2</sup> filter ! area [5] !
ACCURACY	! ESTIMATED LOD: 1 confirmed asbestos fiber above ! 95% of expected mean blank value !
RANGE STUDIED: 80 to 100 fibers counted [1000-L samples]	! PRECISION: 0.28 when 65% of fibers are asbestos; ! 0.20 when adjusted fiber count is ! applied to PCM count [6]. !
BIAS: not determined	!
OVERALL PRECISION (s <sub>r</sub> ): EVALUATION OF METHOD	!

APPLICABILITY: The quantitative working range is 0.04 to 0.5 fiber/cc for a 1000-L air sample. The LOD depends on sample volume and quantity of interfering dust, and is <0.01 fiber/cc for atmospheres free of interferences. This method is used to determine asbestos fibers in the optically visible range and is intended to complement the results obtained by phase contrast microscopy (Method 7400).

INTERFERENCES: Other amphibole particles that have aspect ratios greater than 3:1 and elemental compositions similar to the asbestos minerals may interfere in the TEM analysis. Some non-amphibole minerals may give electron diffraction patterns similar to amphiboles. High concentrations of background dust interfere with fiber identification.

OTHER METHODS: This revises Method 7402 (8/15/87). This method is designed for use with Method 7400 (phase contrast microscopy).



## ASBESTOS FIBERS

METHOD: 7402

## REAGENTS:

1. Acetone. See SPECIAL PRECAUTIONS.

## EQUIPMENT:

1. Sampler: field monitor, 25-mm, three-piece cassette with ca. 50-mm electrically-conductive extension cowl, cellulose ester membrane filter, 0.45- to 1.2- $\mu$ m pore size, and backup pad.  
NOTE 1: Analyze representative filters for fiber background before use. Discard the filter lot if mean count is  $>5$  fibers/100 fields. These are defined as laboratory blanks.  
NOTE 2: Use an electrically-conductive extension cowl to reduce electrostatic effects on fiber sampling and during sample shipment. Ground the cowl when possible during sampling.  
NOTE 3: 0.8- $\mu$ m pore size filters are recommended for personal sampling. 0.45- $\mu$ m filters are recommended for sampling when performing TEM analysis on the samples because the particles deposit closer to the filter surface. However, the higher pressure drop through these filters normally preclude their use with personal sampling pumps.
2. Personal sampling pump, 0.5 to 16 L/min, with flexible connecting tubing.
3. Microscope, transmission electron, operated at ca. 100 kV, with electron diffraction and energy-dispersive X-ray capabilities, and having a fluorescent screen with inscribed or overlaid calibrated scale (Step 15).  
NOTE: The scale is most efficient if it consists of a series of lines inscribed on the screen or partial circles every 2 cm distant from the center.
4. Diffraction grating replica with known number of lines/mm.
5. Slides, glass, pre-cleaned, 25- x 75-mm.
6. Knife, #10 surgical steel, curved-blade.
7. Tweezers.
8. Grids, 200-mesh TEM copper, (optional: carbon-coated).
9. Petri dishes, 15-mm depth. The top and bottom of the petri dish must fit snugly together. To assure a tight fit, grind the top and bottom pieces together with an abrasive such as carborundum to produce a ground-glass contact surface.
10. Foam, clean polyurethane, spongy, 12-mm thick.
11. Filters, Whatman No. 1 qualitative paper or equivalent, or lens paper.
12. Vacuum evaporator.
13. Cork borer, No. 5 (8-mm).
14. Pen, waterproof, marking.
15. Reinforcement, page, gummed.
16. Asbestos standard bulk materials for reference; e.g. SRM #1866, available from the National Institute of Standards and Technology.
17. Carbon rods, sharpened to 1 mm x 8 mm.
18. Microscope, light, phase contrast (PCM), with Walton-Beckett graticule (see method 7400).
19. Grounding wire, 22-gauge, multi-strand.
20. Tape, shrink- or adhesive-.

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**SPECIAL PRECAUTIONS:** Acetone is extremely flammable (flash point = 0 °F). Take precautions not to ignite it. Heating of acetone must be done in a fume hood using a flameless, spark-free heat source.

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## SAMPLING:

1. Calibrate each personal sampling pump with a representative sampler in line [7].
2. For personal sampling, fasten sampler to worker's lapel near worker's mouth. Remove the top cover from cowl extension ("open-face") and orient sampler face down. Wrap joint between extender and monitor body with tape to help hold the cassette together and provide a marking surface to identify the cassette. Where possible, especially at low %RH, attach sampler to electrical ground to reduce electrostatic effects during sampling.

3. Submit at least two field blanks (or 10% of the total samples, whichever is greater) for each set of samples. Remove top covers from the field blank cassettes and store top covers and cassettes in a clean area (e.g., closed bag or box) during sampling. Replace top covers when sampling is completed.
4. Sample at 0.5 to 16 L/min [8]. Adjust sampling rate,  $Q$  (L/min), and time,  $t$  (min), to produce fiber density,  $E$ , of 100 to 1300 fibers/mm<sup>2</sup> [ $3.85 \cdot 10^4$  to  $5 \cdot 10^5$  fibers per 25-mm filter with effective collection area ( $A_c = 385$  mm<sup>2</sup>)] for optimum accuracy. Do not exceed ca. 0.5 mg total dust loading on the filter. These variables are related to the action level (one-half the current standard),  $L$  (fibers/cc), of the fibrous aerosol being sampled by:

$$t = \frac{A_c \cdot E}{Q \cdot L \cdot 10^3}, \text{ min.}$$

NOTE: The purpose of adjusting sampling times is to obtain optimum fiber loading on the filter. A sampling rate of 1 to 4 L/min for 8 hrs (700 to 2800 L) is appropriate in atmospheres containing ca. 0.1 fiber/cc in the absence of significant amounts of non-asbestos dust. Dusty atmospheres require smaller sample volumes ( $\leq 400$  L) to obtain countable samples. In such cases take short, consecutive samples and average the results over the total collection time. For documenting episodic exposures, use high rates (7 to 16 L/min) over shorter sampling times. In relatively clean atmospheres, where targeted fiber concentrations are much less than 0.1 fiber/cc, use larger sample volumes (3000 to 10000 L) to achieve quantifiable loadings. Take care, however, not to overload the filter with background dust [8].

5. At the end of sampling, replace top cover and small end caps.
6. Ship samples upright with conductive cowl attached in a rigid container with packing material to prevent jostling or damage.

NOTE: Do not use untreated polystyrene foam in the shipping container because electrostatic forces may cause fiber loss from sample filter.

#### SAMPLE PREPARATION:

7. Remove three circular sections from any quadrant of each sample and blank filter using a cork borer [9]. The use of three grid preparations reduces the effect of local variations in dust deposit on the filter.
8. Affix the circular filter sections to a clean glass slide with a gummed page reinforcement. Label the slide with a waterproof marking pen.  
NOTE: Up to eight filter sections may be attached to the same slide.
9. Place the slide in a petri dish which contains several paper filters soaked with 2 to 3 mL acetone. Cover the dish. Wait 2 to 4 min for the sample filter(s) to fuse and clear.  
NOTE: The "hot block" clearing technique [10] of Method 7400 or the DMF clearing technique [11] may be used instead of steps 8 and 9.
10. Transfer the slide to a rotating stage inside the bell jar of a vacuum evaporator. Evaporate a 1- by 5-mm section of a graphite rod onto the cleared filter(s). Remove the slide to a clean, dry, covered petri dish [9].
11. Prepare a second petri dish as a Jaffe wick washer with the wicking substrate prepared from filter or lens paper placed on top of a 12-mm thick disk of clean, spongy polyurethane foam [12]. Cut a V-notch on the edge of the foam and filter paper. Use the V-notch as a reservoir for adding solvent.  
NOTE: The wicking substrate should be thin enough to fit into the petri dish without touching the lid.
12. Place the TEM grids face up on the filter or lens paper. Label the grids by marking with a pencil on the filter paper or by putting registration marks on the petri dish halves and marking with a waterproof marker on the dish lid. In a fume hood, fill the dish with acetone until the wicking substrate is saturated.  
NOTE: The level of acetone should be just high enough to saturate the filter paper without creating puddles.

13. Remove about a quarter section of the carbon-coated filter from the glass slide using a surgical knife and tweezers. Carefully place the excised filter, carbon side down, on the appropriately-labeled grid in the acetone-saturated petri dish. When all filter sections have been transferred, slowly add more solvent to the wedge-shaped trough to raise the acetone level as high as possible without disturbing the sample preparations. Cover the petri dish. Elevate one side of the petri dish by placing a slide under it (allowing drops of condensed acetone to form near the edge rather than in the center where they would drip onto the grid preparation).

#### CALIBRATION AND QUALITY CONTROL:

14. Determine the TEM magnification on the fluorescent screen:
  - a. Define a field of view on the fluorescent screen either by markings or physical boundaries.  
NOTE: The field of view must be measurable or previously inscribed with a scale or concentric circles (all scales should be metric) [12].
  - b. Insert a diffraction grating replica into the specimen holder and place into the microscope. Orient the replica so that the grating lines fall perpendicular to the scale on the TEM fluorescent screen. Ensure that goniometer stage tilt is zero.
  - c. Adjust microscope magnification to 10,000X. Measure the distance (mm) between the same relative positions (e.g., between left edges) of two widely-separated lines on the grating replica. Count the number of spaces between the lines.  
NOTE: On most microscopes the magnification is substantially constant only within the central 8- to 10-cm diameter region of the fluorescent screen.
  - d. Calculate the true magnification (M) on the fluorescent screen:

$$M = \frac{X \cdot G}{Y}$$

where: X = total distance (mm) between the two grating lines;  
 G = calibration constant of the grating replica (lines/mm);  
 Y = number of grating replica spaces counted

- e. After calibration, note the apparent sizes of 0.25 and 5.0  $\mu\text{m}$  on the fluorescent screen. (These dimensions are the boundary limits for counting asbestos fibers by phase contrast microscopy.)
15. Measure 20 grid openings at random on a 200-mesh copper grid by placing a grid on a glass slide and examining it under the PCM. Use the Walton-Beckett graticule to measure the grid opening dimensions. Calculate an average graticule field dimension from the data and use this number to calculate the graticule field area for an average grid opening.  
NOTE: A grid opening is considered as one graticule field.
16. Obtain reference selected area electron diffraction (SAED) or microdiffraction patterns from standard asbestos materials prepared for TEM analysis.  
NOTE: This is a visual reference technique. No quantitative SAED analysis is required [12]. Microdiffraction may produce clearer patterns on very small fibers or fibers partially obscured by other material.
  - a. Set the specimen holder at zero tilt.
  - b. Center a fiber, focus, and center the smallest field-limiting aperture on the fiber. Use a 20-cm camera length and 10X binocular head. Obtain a diffraction pattern. Photograph each distinctive pattern and keep the photo for comparison to unknowns.  
NOTE: Not all fibers will present diffraction patterns. The objective lens current may need adjustment to give optimum pattern visibility. There are many more amphiboles which give diffraction patterns similar to the analytes named on p.7402-1. Some, but not all, of these can be eliminated by chemical separations. Also, some non-amphiboles (e.g., pyroxenes, some talc fibers) may interfere.

17. Acquire energy-dispersive X-ray (EDX) spectra on approximately 5 fibers having diameters between 0.25 and 0.5  $\mu\text{m}$  of each asbestos variety obtained from standard reference materials [12].

NOTE: The sample may require tilting to obtain adequate signal. Use same tilt angle for all spectra.

- Prepare TEM grids of all asbestos varieties.
- Use acquisition times (at least 100 sec) sufficient to show a silicon peak at least 75% of the monitor screen height at a vertical scale of  $\geq 500$  counts per channel.
- Estimate the elemental peak heights visually as follows:
  - Normalize all peaks to silicon (assigned an arbitrary value of 10).
  - Visually interpret all other peaks present and assign values relative to the silicon peak.
  - Determine an elemental profile for the fiber using the elements Na, Mg, Si, Ca, and Fe. Example: 0-4-10-3-<1 [12].

NOTE: In fibers other than asbestos, determination of Al, K, Ti, S, P, and F may also be required for fiber characterization.

- Determine a typical range of profiles for each asbestos variety and record the profiles for comparison to unknowns.

#### MEASUREMENT:

18. Perform a diffraction pattern inspection on all sample fibers counted under the TEM, using the procedures given in step 17. Assign the diffraction pattern to one of the following structures:

- chrysotile;
- amphibole;
- ambiguous;
- none.

NOTE: There are some crystalline substances which exhibit diffraction patterns similar to those of asbestos fibers. Many of these, (brucite, halloysite, etc.) can be eliminated from consideration by chemistry. There are, however, several minerals (e.g., pyroxenes, massive amphiboles, and talc fibers) which are chemically similar to asbestos and can be considered interferences. The presence of these substances may warrant the use of more powerful diffraction pattern analysis before positive identification can be made. If interferences are suspected, morphology can play an important role in making positive identification.

19. Obtain EDX spectra in either the TEM or STEM modes from fibers on field samples using the procedure of step 18. Using the diffraction pattern and EDX spectrum, classify the fiber:
- For a chrysotile structure, obtain EDX spectra on the first five fibers and one out of ten thereafter. Label the range profiles from 0-5-10-0-0 to 0-10-10-0-0 as "chrysotile."
  - For an amphibole structure, obtain EDX spectra on the first 10 fibers and one out of ten thereafter. Label profiles ca. 0-2-10-0-7 as "possible amosite"; profiles ca. 1-1-10-0-6 as "possible crocidolite"; profiles ca. 0-4-10-3-<1 as "possible tremolite"; and profiles ca. 0-3-10-0-1 as "possible anthophyllite."

NOTE: The range of profiles for the amphiboles will vary up to  $\pm 1$  unit for each of the elements present according to the relative detector efficiency of the spectrometer.

- For an ambiguous structure, obtain EDX spectra on all fibers. Label profiles similar to the chrysotile profile as "possible chrysotile." Label profiles similar to the various amphiboles as "possible amphiboles." Label all others as "unknown" or "non-asbestos."

20. Counting and Sizing:

- Insert the sample grid into the specimen grid holder and scan the grid at zero tilt at low magnification (ca. 300 to 500X). Ensure that the carbon film is intact and unbroken over ca. 75% of the grid openings.

- b. In order to determine how the grids should be sampled, estimate the number of fibers per grid opening during a low-magnification scan (500 to 1000X). This will allow the analyst to cover most of the area of the grids during the fiber count and analysis. Use the following rules when picking grid openings to count [12,13]:

- (1) Light loading (<5 fibers per grid opening): count total of 40 grid openings.
- (2) Moderate loading (5 to 25 fibers per grid opening): count minimum of 40 grid openings or 100 fibers.
- (3) Heavy loading (>25 fibers per opening): count a minimum of 100 fibers and at least 6 grid openings.

Note that these grid openings should be selected approximately equally among the three grid preparations and as randomly as possible from each grid.

- c. Count only grid openings that have the carbon film intact. At 500 to 1000X magnification, begin counting at one end of the grid and systematically traverse the grid by rows, reversing direction at row ends. Select the number of fields per traverse based on the loading indicated in the initial scan. Count at least 2 field blanks per sample set to document possible contamination of the samples. Count fibers using the following rules:

- (1) Count all particles with diameter greater than  $0.25\ \mu\text{m}$  that meet the definition of a fiber (aspect ratio  $\geq 3:1$ , longer than  $5\ \mu\text{m}$ ). Use the guideline of counting all fibers that would have been counted under phase contrast light microscopy (Method 7400). Use higher magnification (10000X) to determine fiber dimensions and countability under the acceptance criteria. Analyze a minimum of 10% of the fibers, and at least 3 asbestos fibers, by EDX and SAED to confirm the presence of asbestos. Fibers of similar morphology under high magnification can be identified as asbestos without SAED. Particles which are of questionable morphology should be analyzed by SAED and EDX to aid in identification.

- (2) Count fibers which are partially obscured by the grid as half fibers.

NOTE: If a fiber is partially obscured by the grid bar at the edge of the field of view, count it as a half fiber only if more than  $2.5\ \mu\text{m}$  of fiber is visible.

- (3) Size each fiber as it is counted and record the diameter and length:

- (a) Move the fiber to the center of the screen. Read the length of the fiber directly from the scale on the screen.

NOTE 1: Data can be recorded directly off the screen in mm and later converted to  $\mu\text{m}$  by computer.

NOTE 2: For fibers which extend beyond the field of view, the fiber must be moved and superimposed upon the scale until its entire length has been measured.

- (b) When a fiber has been sized, return to the lower magnification and continue the traverse of the grid area to the next fiber.

- d. Record the following fiber counts:

- (1)  $f_s$ ,  $f_b$  = number of asbestos fibers in the grid openings analyzed on the sample filter and corresponding field blank, respectively.
- (2)  $F_s$ ,  $F_b$  = number of fibers, regardless of identification, in the grid openings analyzed on the sample filter and corresponding field blank, respectively.

#### CALCULATIONS:

21. Calculate and report the fraction of optically visible asbestos fibers on the filter,  $(f_s - f_b)/(F_s - F_b)$ . Apply this fraction to fiber counts obtained by PCM on the same filter or on other filters for which the TEM sample is representative. The final result is an asbestos fiber count. The type of asbestos present should also be reported.
22. As an integral part of the report, give the model and manufacturer of the TEM as well as the model and manufacturer of the EDX system.

METHOD: 7402

ASBESTOS FIBERS

## EVALUATION OF METHOD:

The TEM method, using the direct count of asbestos fibers, has been shown to have a precision of 0.275 ( $s_p$ ) in an evaluation of mixed amosite and wollastonite fibers. The estimate of the asbestos fraction, however, had a precision of 0.11 ( $s_p$ ). When this fraction was applied to the PCM count, the overall precision of the combined analysis was 0.20 [6].

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- [13] Steel, E. B. and J. A. Small. "Accuracy of Transmission Electron Microscopy for the Analysis of Asbestos in Ambient Environments," Anal. Chem., 57, 209-213 (1985).

METHOD REVISED BY: Paul A. Baron, Ph.D.; NIOSH/DPSE.



FORMULA:  $\text{Mg}_3\text{Si}_2\text{O}_5(\text{OH})_4$

## CHRYSOTILE ASBESTOS

M.W.: ca. 283

METHOD: 9000

ISSUED: 2/15/84

EPA Standard (Bulk): 1% by weight

PROPERTIES: solid, fibrous mineral; conversion to Forsterite at 580 °C; attacked by acids; loses water above 300 °C

SYNONYMS: Chrysotile; CAS #12001-29-5.

SAMPLING	MEASUREMENT
BULK SAMPLE: 1 to 10 grams	! TECHNIQUE: X-RAY POWDER DIFFRACTION
SHIPMENT: seal securely to prevent escape of asbestos	! ANALYTE: chrysotile
SAMPLE STABILITY: indefinitely	! PREPARATION: grind under liquid $\text{N}_2$ ; wet-sieve through 10- $\mu\text{m}$ sieve
BLANKS: none required	! DEPOSIT: 5 mg dust on 0.45- $\mu\text{m}$ Ag membrane filter
ACCURACY	! XRD: Cu target X-ray tube
RANGE STUDIED: 1 to 100% in talc [3]	! Optimize for intensity; 1° slit
BIAS: negligible if standards and samples are matched in particle size [3]	! Integrated intensity with background subtraction
OVERALL PRECISION ( $s_p$ ): unknown; depends on matrix and concentration	! CALIBRATION: suspensions of asbestos in 2-propanol
	! RANGE: 1 to 100% (w/w) asbestos
	! ESTIMATED LOD: 0.2% asbestos in talc and calcite; 0.4% in heavy X-ray absorbers such as $\text{Fe}_2\text{O}_3$
	! PRECISION ( $s_p$ ): 0.07 (5 to 100%); 0.10 (at 3%); 0.125 (at 1%)

APPLICABILITY: Analysis of percent chrysotile asbestos in bulk samples.

INTERFERENCES: Antigorite (massive serpentine), Chlorite, Kaolinite, Bementite, and Brushite. X-ray fluorescence and absorption is a problem with some elements; fluorescence can be circumvented with a diffracted beam monochromator, and absorption is corrected for in this method.

OTHER METHODS: This is P&CAM 309 [2] applied to bulk samples only, since the sensitivity is not adequate for personal air samples. The EPA Test Method for the determination of asbestos in bulk insulation samples is similar to this one [4]. Method 7400 is an optical counting procedure for airborne fibers in personal samples.



**CHRYSTILE ASBESTOS (bulk)****METHOD: 9000****REAGENTS:**

1. Chrysotile\*, available from:  
Analytical Reference Minerals,  
Measurements Research Branch,  
DPSE, NIOSH, 4676 Columbia Parkway,  
Cincinnati, OH 45226; or  
UICC Asbestos Reference Sample Set,  
UICC MRC Pneumoconiosis Unit  
Llandough Hospital, Penarth,  
Glamorgan, CF6 1XW, UK.2.
2. 2-Propanol.\*
3. Desiccant.
4. Glue or tape for securing Ag  
filters to XRD holders.

\*See Special Precautions.

**EQUIPMENT:**

1. Vials, plastic (for bulk sample).
2. Freezer mill, liquid N<sub>2</sub>-cooled, (Spex Model 6700 or equivalent), grinding vials (Spex 6701), extractor (Spex 6704).
3. Ultrasonic bath.
4. Sieve, 10- $\mu$ m, for wet-sieving.
5. Filters, polycarbonate, 1.0- $\mu$ m, 37-mm (Nuclepore or equivalent).
6. Filtration apparatus and side-arm vacuum flask with 25- and 37-mm filter holders.
7. Oven, drying, 110 °C.
8. Analytical balance, readable to 0.01 mg.
9. Beaker, Griffin, 50-mL, with watchglass cover.
10. Filters, silver membrane, 25-mm diameter, 0.45- $\mu$ m pore size (Selas Flotronics, Dresher, PA 19006, or equivalent).
11. Desiccator.
12. Bottles, glass, 1-L, with ground glass stoppers.
13. Wash bottle, polyethylene.
14. Magnetic stirrer.
15. X-ray powder diffractometer with copper target X-ray tube and scintillation detector.
16. Reference specimen (mica, Arkansas stone or other stable standard) for data normalization.
17. Volumetric pipettes and flasks.

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**SPECIAL PRECAUTIONS:** Asbestos, a human carcinogen, should be handled in a hood [1].

2-Propanol is flammable.

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**SAMPLING:**

1. Place several grams of the dust to be analyzed in a plastic vial, seal the vial securely and ship in a padded carton.

**SAMPLE PREPARATION:**

2. Place ca. 0.5 g of sample dust in a grinding vial and grind in a liquid nitrogen-cooled mill for 2 to 10 min.
3. Wet sieve the ground dust using a 10- $\mu$ m sieve and 2-propanol. Place the dust on the sieve and place the sieve directly in an ultrasonic bath or in a wide dish in the bath. Use enough 2-propanol to cover the dust (put water in the bath if a dish is used to contain the 2-propanol). Apply ultrasonic power to sieve the dust.  
NOTE: It may take some time to obtain several mg of dust. Heating of the 2-propanol is likely and cooling periods may be required.
4. Recover the sieved sample dust from the 2-propanol by filtering the suspension through a non-fibrous filter (polycarbonate) or by driving off the 2-propanol on a hot plate. Dry the sieved sample in 110 °C oven for 4 hrs or more.

## METHOD: 9000

## CHYSOTILE ASBESTOS (bulk)

5. Weigh out ca. 5 mg of the sieved material onto a small square of tared weighing paper. Record the actual weight,  $W$ , to the nearest 0.01 mg. Transfer the dust to a 50-mL beaker, washing the weighing paper with several mL of 2-propanol. Add 10 to 15 mL 2-propanol to the beaker.
6. Cover the beaker with a watchglass. Agitate in an ultrasonic bath at least 3 min until all agglomerated particles are dispersed. Wash the underside of the watchglass with 2-propanol, collecting the washings in the beaker.
7. Place a silver filter in the filtration apparatus. Attach the funnel securely over the entire filter circumference. With no vacuum, pour 2 to 3 mL 2-propanol onto the filter. Pour the sample suspension from the beaker into the funnel and apply vacuum. During filtration, rinse the beaker several times and add rinsings to the funnel.  
NOTE: Control the filtration rate to keep the liquid level in the funnel near the top during rinsing. Do not wash the walls or add 2-propanol to the funnel when the liquid level is lower than 4 cm above the filter. Leave the vacuum on after filtration for sufficient time to produce a dry filter.
8. Remove the filter with forceps and attach it to the sample holder for XRD analysis.

## CALIBRATION AND QUALITY CONTROL

9. Prepare and analyze working standard filters:
  - a. Prepare two suspensions of chrysotile asbestos in 2-propanol by weighing 10 and 100 mg of the dry powder to the nearest 0.01 mg. Quantitatively transfer each to a 1-L glass-stoppered bottle using 1.00 L 2-propanol.  
NOTE: Depending on the particle size of the standard, it may need to be ground and wet-sieved (step 3). Dry the standards in a 110 °C oven for 4 hrs or more. Store in a dessicator.
  - b. Suspend the powder in the 2-propanol with an ultrasonic probe or bath for 20 min. Immediately move the flask to a magnetic stirrer with thermally-insulated top and add a stirring bar to the suspension. Cool the solution to room temperature before withdrawing aliquots.
  - c. Mount a filter on the filtration apparatus. Place several mL 2-propanol on the filter surface. Turn off the stirrer and shake vigorously by hand. Within a few seconds of setting the bottle down, remove the lid and withdraw an aliquot from the center of the 10- or 100-mg/L suspension. Do not adjust the volume in the pipet by expelling part of the suspension. If more than the desired aliquot is withdrawn, return all of the suspension to the bottle, rinse and dry the pipet, and take a new aliquot. Transfer the aliquot from the pipet to the filter. Keep the tip of the pipet near the surface but not submerged in the delivered suspension.
  - d. Rinse the pipet with several mL 2-propanol, draining the rinse into the funnel. Repeat the rinse several more times. Prepare working standard filters, in triplicate, by this technique, at e.g., 0, 20, 30, 50 100, 200 and 500  $\mu\text{g}$ .
  - e. Apply vacuum and rapidly filter the suspension. Leave vacuum on until filter is dry. Do not wash down the sides of the funnel after the deposit is in place since this will rearrange the material on the filter. Transfer the filter to the sample holder.
  - f. Analyze by XRD (step 12). The XRD intensities (12.d.) are designated  $I_X^0$  and are then normalized (12.e.) to obtain  $\hat{I}_X^0$ . The intensities for standards greater than 200  $\mu\text{g}$  should be corrected for matrix absorption (12.f. and 13).
  - g. Prepare a calibration graph by plotting  $\hat{I}_X^0$ , as a function of  $\mu\text{g}$  of each standard.  
NOTE: Poor repeatability (greater than 10% above 0.04 mg chrysotile) at any given level indicates that new standards should be made. The data should lie along a straight line. It is preferable to use a weighted least squares with  $1/\sigma^2$  weighting, where  $\sigma^2$  is the variance of the data at a given loading.

## CHRYSTILE ASBESTOS (bulk)

METHOD: 9000

- h. Determine the slope,  $m$ , of the calibration curve in counts/ $\mu\text{g}$ . The intercept on the abscissa should be within  $\pm 5 \mu\text{g}$  of zero.

NOTE: A large intercept indicates an error in determining the background, i.e., an incorrect baseline has been calculated or interference by another phase.

10. Select six silver membrane filters as media blanks (to test for sample self-absorption) randomly from the same box of filters to be used for depositing the samples. Mount each of the media blanks on the filtration apparatus and apply vacuum to draw 5 to 10 mL of 2-propanol through the filter. Remove, let dry and mount on sample holders. Determine the net normalized count for the silver peak,  $\hat{I}_{Ag}$ , for each media blank (step 12). Obtain an average value for the six media blanks.

## MEASUREMENT:

11. Obtain a qualitative X-ray diffraction scan (e.g., 10 to 80 degrees 2-theta) of the sample to determine the presence of chrysotile and interferences. The expected diffraction peaks are as follows:

Mineral	Peak (2-Theta Degrees)	
	Primary	Secondary
Chrysotile	12.08	24.38
Silver	38.12	44.28

12. Mount the filter (sample, standard or blank) in the XRD instrument and:
- Determine the net intensity,  $I_r$ , of the reference specimen before each filter is scanned. Select a convenient normalization scale factor,  $N$ , which is approximately equivalent to the net count for the reference specimen peak, and use this value of  $N$  for all analyses.
  - Measure the diffraction peak area of a chrysotile peak that is free of interference. Scan times should be long, e.g., 15 min.
  - Measure the background on each side of the peak for one-half the time used for peak scanning. The sum of these two counts is the average background. Determine the position of the background for each sample.
  - Calculate the net intensity,  $I_x$  (the difference between the peak integrated count and the total background count).
  - Calculate and record the normalized intensity,  $\hat{I}_x$ , for the sample peak on each sample and standard:

$$\hat{I}_x = \frac{I_x \cdot N}{I_r}$$

NOTE: Normalizing to the reference specimen intensity compensates for long-term drift in X-ray tube intensity. If intensity measurements are stable, the reference specimen may be run less frequently; net intensities should be normalized to the most recently measured reference intensity.

- f. Determine the net count,  $I_{Ag}$ , of an interference-free silver peak on the sample filter following the same procedure. Use a short scan time for the silver peak (e.g., 5% of scan time for analyte peaks) throughout the method.

## METHOD: 9000

## CHYSOTILE ASBESTOS (bulk)

## CALCULATIONS:

13. Calculate the percentage of chrysotile in the bulk dust sample:

$$C = \frac{(\hat{I}_x \cdot f(T) - b)}{m \times W}, \%$$

where:

$\hat{I}_x$  = normalized intensity for sample peak

b = intercept of calibration graph ( $\hat{I}_x^0$  vs. W)

m = slope of calibration graph (counts/ $\mu$ g)

$f(T) = \frac{-R \ln T}{1 - TR}$  = absorption correction factor (Table 1)

$R = \sin(\theta_{Ag}) / \sin(\theta_x)$

$T = \hat{I}_{Ag} / (\text{average } \hat{I}_{Ag}^0) = \text{transmittance of sample}$

$\hat{I}_{Ag}$  = normalized silver peak intensity from sample

average  $\hat{I}_{Ag}^0$  = normalized silver peak intensity from media blanks (average of six values)

W = mass of deposited sample in  $\mu$ g.

NOTE: For a more detailed discussion of the absorption correction procedure, see references [5] to [8].

## EVALUATION OF METHOD:

This method is based on the work of B.A. Lange in developing P&CAM 309 [2,3]. Samples in the range of 1 to 100% chrysotile in talc were studied to establish the feasibility of an XRD method for airborne asbestos. Analytical precision was as follows:

% Chrysotile in Talc	$s_r$ (%)
100	6.9
10	4.7
7	9.8
5	8.2
3	10.1
1	12.5

This work also showed that bias of results after absorption corrections are made is essentially negligible.

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CHRYSTILE ASBESTOS (bulk)METHOD: 9000

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METHOD REVISED BY: M. T. Abell, NIOSH/DPSE.

## METHOD: 9000

## CHYSOTILE ASBESTOS (bulk)

Table 1. Absorption correction factor as a function of transmittance for some chrysotile-silver peak combinations.

Transmittance T	Chrysotile Silver	f(T)		Transmittance T	f(T)	
		12.08 38.12	24.38 38.12		12.08 38.12	24.38 38.12
1.00		1.0000	1.0000	0.69	1.6839	1.3142
0.99		1.0157	1.0078	0.68	1.7151	1.3277
0.98		1.0317	1.0157	0.67	1.7470	1.3414
0.97		1.0480	1.0237	0.66	1.7797	1.3555
0.96		1.0647	1.0319	0.65	1.8132	1.3698
0.95		1.0817	1.0402	0.64	1.8475	1.3845
0.94		1.0991	1.0486	0.63	1.8827	1.3995
0.93		1.1168	1.0572	0.62	1.9188	1.4148
0.92		1.1350	1.0659	0.61	1.9558	1.4305
0.91		1.1535	1.0747	0.60	1.9938	1.4465
0.90		1.1724	1.0837	0.59	2.0328	1.4629
0.89		1.1917	1.0928	0.58	2.0728	1.4797
0.88		1.2114	1.1021	0.57	2.1139	1.4969
0.87		1.2316	1.1115	0.56	2.1560	1.5145
0.86		1.2522	1.1212	0.55	2.1993	1.5325
0.85		1.2733	1.1309	0.54	2.2438	1.5510
0.84		1.2948	1.1409	0.53	2.2895	1.5700
0.83		1.3168	1.1510	0.52	2.3365	1.5895
0.82		1.3394	1.1613	0.51	2.3848	1.6095
0.81		1.3624	1.1718	0.50	2.4344	1.6300
0.80		1.3859	1.1825	0.49	2.4855	1.6510
0.79		1.4100	1.1933	0.48	2.5380	1.6727
0.78		1.4346	1.2044	0.47	2.5921	1.6950
0.77		1.4598	1.2157	0.46	2.6478	1.7179
0.76		1.4856	1.2272	0.45	2.7051	1.7414
0.75		1.5120	1.2389	0.44	2.7642	1.7657
0.74		1.5390	1.2508	0.43	2.8251	1.7907
0.73		1.5666	1.2630	0.42	2.8879	1.8165
0.72		1.5949	1.2754	0.41	2.9526	1.8431
0.71		1.6239	1.2881	0.40	3.0195	1.8705
0.70		1.6536	1.3010			



FORMULA: various	ASBESTOS (bulk)
	METHOD: 9002
M.W.: various	ISSUED: 5/15/89

EPA Standard (Bulk): 1%                      PROPERTIES: solid, fibrous, crystalline, anisotropic

SYNONYMS: actinolite [CAS #13768-00-8], or ferroactinolite; cummingtonite-grunerite (amosite) [CAS #12172-73-5]; anthophyllite [CAS #17068-78-9]; chrysotile [CAS #12001-29-5] or serpentine; crocidolite [CAS #12001-28-4] or riebeckite; tremolite [CAS #14567-73-8]; amphibole asbestos.

SAMPLING	MEASUREMENT
BULK SAMPLE: 1 to 10 grams	! !TECHNIQUE: MICROSCOPY, STEREO AND POLARIZED ! LIGHT, WITH DISPERSION STAINING
SHIPMENT: seal securely to prevent escape of asbestos	! !ANALYTE: actinolite asbestos, amosite, ! anthophyllite asbestos, chrysotile,
SAMPLE STABILITY: stable	! crocidolite, tremolite asbestos !
BLANKS: none required	!EQUIPMENT: microscope, polarized light: 100-400X ! dispersion staining objective, ! stereo microscope: 10-45X !
	!RANGE: 1% to 100% asbestos !
ACCURACY	!
RANGE STUDIED: <1% to 100% asbestos	!ESTIMATED LOD: <1% asbestos [1] !
BIAS: not determined	!PRECISION: not determined !
PRECISION: not determined	! !

APPLICABILITY: This method is useful for the qualitative identification of asbestos and the semi-quantitative determination of asbestos content of bulk samples, expressed as a percent of projected area. The method measures percent asbestos as perceived by the analyst in comparison to standard area projections, photos, and drawings, or trained experience. The method is not applicable to samples containing large amounts of fine fibers below the resolution of the light microscope.

INTERFERENCES: Other fibers with optical properties similar to the asbestos minerals may give positive interferences. Optical properties of asbestos may be obscured by coating on the fibers. Fibers finer than the resolving power of the microscope (ca. 0.3  $\mu$ m) will not be detected. Heat and acid treatment may alter the index of refraction of asbestos and change its color.

OTHER METHODS: This method (originally designated as method 7403) is designed for use with NIOSH Methods 7400 (phase contrast microscopy) and 7402 (electron microscopy/EDS). The method is similar to the EPA bulk asbestos method [1].



ASBESTOS (bulk)

METHOD: 9002

**REAGENTS:**

1. Refractive index (RI) liquids for Dispersion Staining: high-dispersion (HD) series, 1.550, 1.605, 1.620.
2. Refractive index liquids: 1.670, 1.680, and 1.700.
3. Asbestos reference samples such as SRM #1866, available from the National Institute of Standards and Technology.\*
4. Distilled Water (optional).
5. Concentrated HCl: ACS reagent grade (optional).

\*See SPECIAL PRECAUTIONS

**EQUIPMENT:**

1. Sample containers: screw-top plastic vials of 10- to 50-mL capacity.
2. Microscope, polarized light, with polarizer, analyzer, port for retardation plate, 360° graduated rotating stage, substage condenser with iris, lamp, lamp iris, and:
  - a. Objective lenses: 10X, 20x, and 40X or near equivalent.
  - b. Ocular lense: 10X minimum.
  - c. Eyepiece reticle: crosshair.
  - d. Dispersion staining objective lens or equivalent.
  - e. Compensator plate: ca. 550 nm  $\pm$  20 nm, retardation: "first order red" compensator.
3. Microscope slides: 75 mm x 25 mm.
4. Cover slips: 22 mm x 22 mm.
5. Ventilated hood or negative pressure glove box.
6. Mortar and pestle: agate or porcelain.
7. Stereomicroscope, ca. 10 to 45X.
8. Light source: incandescent or fluorescent.
9. Tweezers, dissecting needles, spatulas, probes, and scalpels.
10. Glassine paper or clean glass plate.
11. Low-speed hand drill with coarse burr bit (optional).

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**SPECIAL PRECAUTIONS:** Asbestos, a human carcinogen, should be handled only in an exhaust hood (equipped with a HEPA filter). [2] Precautions should be taken when collecting unknown samples, which may be asbestos, to preclude exposure to the person collecting the sample and minimize the disruption to the parent material [3]. Disposal of asbestos-containing materials should follow EPA Guidelines [4].

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**SAMPLING:**

1. Place 1 to 10 g of the material to be analyzed in a sample container.  
 NOTE: For large samples (i.e., whole ceiling tiles) that are fairly homogenous, a representative small portion should be submitted for analysis. Sample size should be adjusted to ensure that it is representative of the parent material.
2. Make sure that sample containers are taped so they will not open in transit.
3. Ship the samples in a rigid container with sufficient packing material to prevent damage or sample loss.

**SAMPLE PREPARATION:**

4. Visually examine samples in the container and with a low-magnification stereomicroscope in a hood. (If necessary, a sample may be carefully removed from the container and placed on glassine transfer paper or clean glass plate for examination). Break off a portion of the sample and examine the edges for emergent fibers. Note the homogeneity of the sample. Some hard tiles can be broken, and the edges examined for emergent fibers. If fibers are found, make an estimate of the amount and type of fibers present, confirm fiber type (steps 6 through 13) and quantify (steps 14 through 16).

5. In a hood, open sample container and with tweezers remove small, representative portions of the sample.
  - a. If there are obvious separable layers, sample and analyze each layer separately.
  - b. If the sample appears to be slightly inhomogeneous, mix it in the sample container with tweezers or a spatula before taking the portion for analysis. Alternatively, take small representative portions of each type of material and place on a glass slide.
  - c. On hard tiles that may have thin, inseparable layers, use a scalpel to cut through all the layers for a representative sample. Then cut it into smaller pieces after placing RI liquid on it before trying to reduce the thickness. Alternatively, use a low-speed hand drill equipped with a burr bit to remove material from hard tiles. Avoid excessive heating of the sample which may alter the optical properties of the material.

NOTE: This type of sample often requires ashing or other specialized preparation.
  - d. If the sample has large, hard particles, grind it in a mortar. Do not grind so fine that fiber characteristics are destroyed.
  - e. If necessary, treat a portion of the sample in a hood with an appropriate solvent to remove binders, tars, and other interfering materials which may be present in the sample. Make corrections for the non-asbestos material removed by this process.

NOTE: Other methods of sample preparation such as acid and sodium metaphosphate treatment and ashing are not normally necessary. However, if needed, use as described in Reference [1].
6. After placing a few drops of RI liquid on the slide, put a small portion of sample in the liquid. Tease apart with a needle or smash small clumps with the flat end of a spatula or probe, producing a uniform thickness of particles so that better estimates of projected area percentages can be made. Mix the fibers and particles on the slide so that they are as homogeneous as possible.

NOTE: An even dispersion of sample should cover the entire area under the cover slip. Some practice will be necessary to judge the right amount of material to place on the slide. Too little sample may not give sufficient information and too much sample cannot be easily analyzed.

#### CALIBRATION AND QUALITY CONTROL:

7. Check for contamination of microscope slides, cover slips and refractive index liquids once per day of operation. Record results in a separate logbook.
8. Verify the refractive indices of the refractive index liquids used once per week of operation. Record these checks in a separate logbook.
9. Follow the manufacturer's instructions for illumination, condenser alignment and other microscope adjustments. Perform these adjustments prior to each sample set.
10. Determine percent of each identified asbestos species by comparison to standard projections (Figure 1) [1]. If no fibers are detected in a homogeneous sample, examine at least two additional preparations before concluding that no asbestos is present.
11. If it appears that the preparation technique might not be able to produce a homogeneous or representative sample on the slide, prepare a duplicate slide and average the results. Occasionally, when the duplicate results vary greatly, it will be necessary to prepare additional replicate slides and average all the replicate results. Prepare duplicate slides of at least 10% of the samples analyzed. Average the results for reporting.
12. Analyze about 5% blind samples of known asbestos content.
13. Laboratories performing this analytical method should participate in the National Voluntary Laboratory Accreditation Program [5] or a similar interlaboratory quality control program. Each analyst should have completed formal training in polarized light microscopy and its application to crystalline materials. In lieu of formal training, laboratory training in asbestos bulk analysis under the direction of a trained asbestos bulk analyst may be substituted. Due to the subjective nature of the method, frequent practice is essential in order to remain proficient in estimating projected area percentages.

## QUALITATIVE ASSESSMENT:

14. Scan the slide to identify any asbestos minerals using the optical properties of morphology, refractive indices, color, pleochroism, birefringence, extinction characteristics, sign of elongation, and dispersion staining characteristics.

NOTE: Identification of asbestos using polarized light microscopy is unlike most other analytical methods. The quality of the results is dependent on the skill and judgment of the analyst. This method does not lend itself easily to a step-wise approach. Various procedures devised by different analysts may yield equivalent results. The following step-wise procedure repeatedly utilizes the sample preparation procedure previously outlined.

- a. Prepare a slide using 1.550 HD RI liquid. Adjust the polarizing filter such that the polars are partially crossed, with ca. 15° offset. Scan the preparation, examining the morphology for the presence of fibers. If no fibers are found, scan the additional preparations. If no fibers are found in any of the preparations, report that the sample does not contain asbestos, and stop the analysis at this point.
- b. If fibers are found, adjust the polarizing filter such that the polars are fully crossed. If all of the fibers are isotropic (disappear at all angles of rotation) then those fibers are not asbestos. Fibrous glass and mineral wool, which are common components of suspect samples, are isotropic. If only isotropic fibers are found in the additional preparations, report no asbestos fibers detected, and stop the analysis.
- c. If anisotropic fibers are found, rotate the stage to determine the angle of extinction. Except for tremolite-actinolite asbestos which has oblique extinction at 10-20°, the other forms of asbestos exhibit parallel extinction. Tremolite may show both parallel and oblique extinction.
- d. Insert the first-order red compensator plate in the microscope and determine the sign of elongation. All forms of asbestos have a positive sign of elongation except for crocidolite. If the sign of elongation observed is negative, go to step "g".  
NOTE: To determine the direction of the sign of elongation on a particular microscope configuration, examine a known chrysotile sample and note the direction (NE-SW or NW-SE) of the blue coloration. Chrysotile has a positive sign of elongation.
- e. Remove the first-order red compensator and uncross the polarizer. Examine under plane polarized light for blue and gold-brown Becke colors at the fiber-oil interface (i.e., index of refraction match). Becke colors are not always evident. Examine fiber morphology for twisted, wavy bundles of fibers which are characteristic of chrysotile. Twisted, ribbon-like morphology with cellular internal features may indicate cellulose fibers. It may be necessary to cross the polars partially in order to see the fibers if the index of refraction is an exact match at 1.550. If the fibers appear to have higher index of refraction, go to step "h", otherwise continue.
- f. Identification of chrysotile. Insert the dispersion staining objective. Observing dispersion staining colors of blue and blue-magenta confirms chrysotile. Cellulose, which is a common interfering fiber at the 1.550 index of refraction, will not exhibit these dispersion staining colors. If chrysotile is found, go to step 15 for quantitative estimation.
- g. Identification of crocidolite. Prepare a slide in 1.700 RI liquid. Examine under plane-polarized light (uncrossed polars); check for morphology of crocidolite. Fibers will be straight, with rigid appearance, and may appear blue or purple-blue. Crocidolite is pleochroic, i.e., it will appear to change its color (blue or gray) as it is rotated through plane polarized light. Insert the dispersion staining objective. The central stop dispersion staining colors are red magenta and blue magenta, however these colors are sometimes difficult to impossible to see because of the opacity of the dark blue fibers. If observations above indicate crocidolite, go to step 15 for quantitative estimation.

- h. Identification of amosite. Prepare a slide in 1.680 RI liquid. Observe the fiber morphology for amosite characteristics: straight fibers and fiber bundles with broom-like or splayed ends. If the morphology matches amosite, examine the fibers using the dispersion staining objective. Blue and pale blue colors indicate the cummingtonite form of amosite, and gold and blue colors indicate the grunerite form of amosite. If amosite is confirmed by this test, go to step 15 for quantitative estimation, otherwise continue.
- i. Identification of anthophyllite-tremolite-actinolite. Prepare a slide in 1.605 HD RI liquid. Examine morphology for comparison to anthophyllite-tremolite-actinolite asbestos. The refractive indices for these forms of asbestos vary naturally within the species. Anthophyllite can be distinguished from actinolite and tremolite by its nearly parallel extinction. Actinolite has a light to dark green color under plane-polarized light and exhibits some pleochroism. For all three, fibers will be straight, single fibers possibly with some larger composite fibers. Cleavage fragments may also be present. Examine using the central stop dispersion staining objective. Anthophyllite will exhibit central stop colors of blue and gold/gold-magenta; tremolite will exhibit pale blue and yellow; and actinolite will exhibit magenta and golden-yellow colors.
- NOTE: In this refractive index range, wollastonite is a common interfering mineral with similar morphology including the presence of cleavage fragments. It has both positive and negative sign of elongation, parallel extinction, and central stop dispersion staining colors of pale yellow and pale yellow to magenta. If further confirmation of wollastonite versus anthophyllite is needed, go to step "j". If any of the above forms of asbestos was confirmed above, go to step 15 for quantitative estimation. If none of the tests above confirmed asbestos fibers, examine the additional preparations and if the same result occurs, report the absence of asbestos in this sample.
- j. Wash a small portion of the sample in a drop of concentrated hydrochloric acid on a slide. Place the slide, with cover slip in place, on a warm hot plate until dry. By capillary action, place 1.620 RI liquid under the cover slip and examine the slide. Wollastonite fibers will have a "cross-hatched" appearance across the length of the fibers and will not show central stop dispersion colors. Anthophyllite and tremolite will still show their original dispersion colors.

NOTE: There are alternative analysis procedures to the step-wise approach outlined above which will yield equivalent results. Some of these alternatives are:

- i. Perform the initial scan for the presence of asbestos using crossed polars as well as the first-order red compensator. This allows for simultaneous viewing of birefringent and amorphous materials as well as determining their sign of elongation. Some fibers which are covered with mortar may best be observed using this configuration.
- ii. Some analysts prefer to mount their first preparation in a RI liquid different than any asbestos materials and conduct their initial examination under plane-polarized light.
- iii. If alternative RI liquids are used from those specified, dispersion staining colors observed will also change. Refer to an appropriate reference for the specific colors associated with asbestos in the RI liquids actually used.

#### QUANTITATIVE ASSESSMENT:

15. Estimate the content of the asbestos type present in the sample using the 1.550 RI preparation. Express the estimate as an area percent of all material present, taking into account the loading and distribution of all sample material on the slide. Use Figure 1 as an aid in arriving at your estimate. If additional unidentified fibers are present in the sample, continue with the qualitative measurement (step 14).

NOTE: Point-counting techniques to determine percentages of the asbestos minerals are not generally recommended. The point-counting method only produces accurate quantitative

data when the material on the slide is homogeneous and has a uniform thickness, which is difficult to obtain [6]. The point-counting technique is, recommended by the EPA to determine the amount of asbestos in bulk [1]; however in the more recent Asbestos Hazard Emergency Response Act (AHERA) regulations, asbestos quantification may be performed by a point-counting or equivalent estimation method [7].

16. Make a quantitative estimate of the asbestos content of the sample from the appropriate combination of the estimates from both the gross and microscopic examinations. If asbestos fibers are identified, report the material as "asbestos-containing". Asbestos content should be reported as a range of percent content. The range reported should be indicative of the analyst's precision in estimating asbestos content. For greater quantities use Figure 1 in arriving at your estimate.

#### EVALUATION OF METHOD:

The method is compiled from standard techniques used in mineralogy [8-13], and from standard laboratory procedures for bulk asbestos analysis which have been utilized for several years. These techniques have been successfully applied to the analysis of EPA Bulk Sample Analysis Quality Assurance Program samples for more than 8 years [1,5]. However, no formal evaluation of this method, as written, has been performed.

#### REFERENCES:

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Figure 1. Percent estimate comparator.

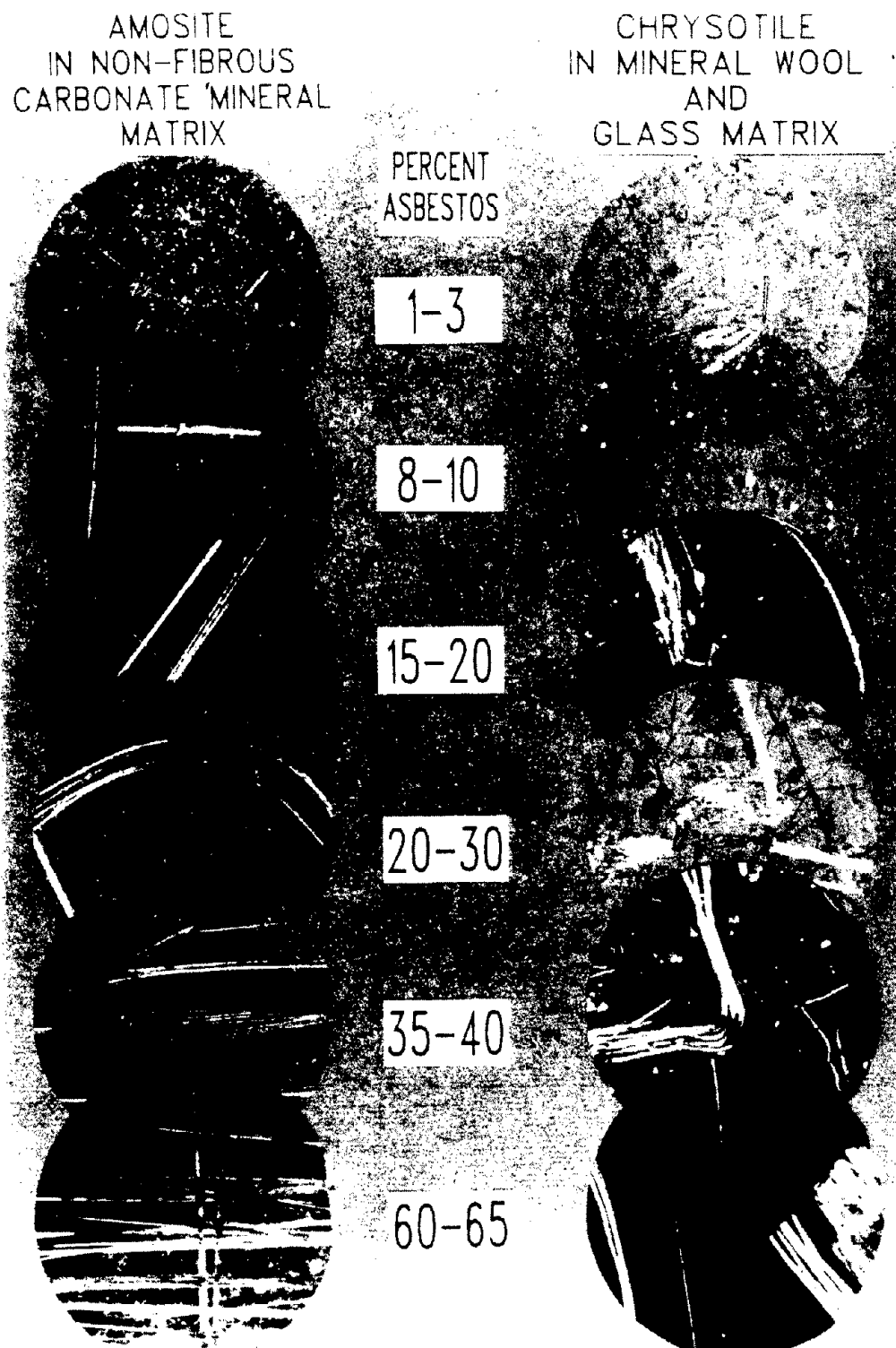


Table 1. Optical Properties of Asbestos Fibers

Mineral	Morphology and Color	Refractive Index (Approximate Values)		Birefringence
		$\perp$ to Elongation	$\parallel$ to Elongation	
Chrysotile	Wavy fibers with kinks. Splayed ends on larger bundles. Colorless to light brown upon being heated. Nonpleochroic. Aspect ratio typically >10:1.	1.54	1.55	0.002 - 0.014
Cummingtonite- Grunerite (Amosite)	Straight fibers and fiber bundles. Bundle ends appear broom-like or splayed. Colorless to brown upon heating. May be weakly pleochroic. Aspect ratio typically >10:1.	1.67	1.70	0.02 - 0.03
Crocidolite (Riebeckite)	Straight fibers and fiber bundles. Longer fibers show curvature. Splayed ends on bundles. Characteristic blue color. Pleochroic. Aspect ratio typically >10:1.	1.71	1.70	0.014 - 0.016 Interference colors may be masked by blue color.
Anthophyllite	Straight fibers and fiber bundles. Cleavage fragments may be present. Colorless to light brown. Nonpleochroic to weakly pleochroic. Aspect ratio generally <10:1.	1.61	1.63	.019 - .024
Tremolite- Actinolite	Straight and curved fibers. Cleavage fragments common. Large fiber bundles show splayed ends. Tremolite is colorless. Actinolite is green and weakly to moderately pleochroic. Aspect ratio generally <10:1.	1.60 - 1.62 (tremolite)	1.62 - 1.64 (tremolite)	0.02 - 0.03
		1.62 - 1.67 (actinolite)	1.64 - 1.68 (actinolite)	

Table 1. Optical Properties of Asbestos Fibers (Continued)

Mineral	Extinction	Sign of Elongation	<u>Central Stop Dispersion Staining Colors</u>		
			RI Liquid	$\perp$ to Vibration	$\parallel$ to Vibration
Chrysotile	Parallel to fiber length	+(length slow)	1.550 <sup>HD</sup>	Blue	Blue-magenta
Cummingtonite- Grunerite (Amosite)	Parallel to fiber length	+(length slow)	1.670	Red magenta to blue	Yellow
Cummingtonite Grunerite			Fibers subjected to high temperatures will not dispersion-stain. 1.680 1.680	pale blue blue	blue gold
Crocidolite (Riebeckite)	Parallel to fiber length	-(length fast)	1.700  1.680	Red magenta  yellow	Blue-magenta  pale yellow
Anthophyllite	Parallel to fiber length	+(length slow)	1.605 <sup>HD</sup>  1.620 <sup>HD</sup>	Blue  Blue-green	Gold to gold-magenta  Golden-yellow
Tremolite- Actinolite	Oblique - 10 to 20° for fragments. Some composite fibers show $\parallel$ extinction.	+(length slow)	1.605 <sup>HD</sup>	Pale blue (tremolite)  Yellow (actinolite)	Yellow (tremolite)  Pale yellow (actinolite)

HD = high-dispersion RI liquid series.





Testimony of the  
National Institute for Occupational Safety and Health  
on the  
Occupational Safety and Health Administration's  
Proposed Rule on  
Occupational Exposure to Asbestos, Tremolite, Anthophyllite,  
and Actinolite

29 CFR Parts 1910 and 1926  
Docket No. H-033-e

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Washington, D.C.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Public Health Service  
Centers for Disease Control

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I am Richard A. Lemen, Assistant Director of the National Institute for Occupational Safety and Health (NIOSH). With me today are senior staff from NIOSH. Our purpose for appearing at this hearing is to provide testimony to the Occupational Safety and Health Administration (OSHA) regarding the changes proposed to its existing regulation for occupational exposure to asbestos, tremolite, anthophyllite, and actinolite in general [29 CFR 1910] and construction industries [29 CFR 1926].

## I. BACKGROUND

Several issues regarding the regulation of asbestos were remanded to OSHA by the U.S. Court of Appeals. Four of these issues would require new OSHA rulemaking: (1) the establishment of operation-specific permissible exposure limits (PELs), (2) the extension of requirements for reporting and information transfer, (3) the expansion of the competent person<sup>1</sup> requirement to all construction operations, and (4) clarification of the exemption for small-scale, short-duration operations. Specifically, OSHA has proposed the following:

- that a PEL of 0.1 fiber/cc (100,000 fibers/m<sup>3</sup>) for asbestos be adopted for all general and construction industry workers
- that operation-specific work practices and controls be mandated to reduce exposures
- that additional requirements be established for communicating asbestos hazards among building owners, employers, and workers
- that OSHA be notified before the commencement of any removal, demolition, or renovation operation with potential exposures to asbestos
- that all construction operations with potential exposure to asbestos be overseen by a competent person
- that the definition of a small-scale, short-duration operation be more specific
- that the exemptions from the use of negative-pressure enclosures be made more explicit

NIOSH supports OSHA in proposing changes to the existing asbestos standard to minimize the risk of asbestos-related diseases in workers. The current standard is insufficient to protect the health of workers exposed to asbestos and the proposed changes represent a prudent and necessary approach to accomplish this goal.

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<sup>1</sup>A competent person is defined in the current asbestos construction standard [29 CFR 1926.58(b)] as "...one who is capable of identifying existing asbestos...hazards in the workplace and has the authority to take prompt corrective measures to eliminate them...".

## II. NIOSH STATEMENT OF POLICY

On June 21, 1984, and May 9, 1990, NIOSH testified at the OSHA public hearings on occupational exposure to asbestos and presented supporting evidence that there is no safe airborne concentration of fibers for any asbestos mineral [NIOSH 1984; NIOSH 1990a]. NIOSH stated that not even the lowest fiber exposure limit could assure workers of absolute protection from exposure-related cancer. This conclusion was consistent with previous positions taken by NIOSH in the 1976 criteria document on asbestos [NIOSH 1976] and the joint NIOSH/OSHA report of 1980 [NIOSH/OSHA 1980]. In its 1984 testimony, NIOSH urged that the goal be to eliminate exposures to asbestos fibers or, where they cannot be eliminated, to limit them to the lowest possible concentration [NIOSH 1984].

NIOSH concluded that (1) for regulatory purposes, phase contrast microscopy (PCM) was still the most practical technique for assessing exposures to asbestos fibers when using the criteria in NIOSH Analytical Method 7400 and (2) no distinction should be made between airborne exposures to asbestos fibers and their nonasbestiform analogs when they meet the criteria of a fiber as defined on a microscopic level [NIOSH 1990a]. NIOSH also recognized that PCM lacks specificity when asbestos and other fibers occur in the same environment, and that PCM cannot detect fibers with diameters less than approximately 0.25 micrometer. NIOSH further stated that it might be necessary to analyze air samples by electron microscopy where both electron diffraction and microchemical analysis can be used to help identify the type and concentration of asbestos fibers.

## III. NIOSH RESPONSE TO THE REGULATORY RESPONSES PROPOSED BY OSHA

### A. 29 CFR Parts 1910 and 1926 - Reduction of the Asbestos PEL to 0.1 Fiber/cc

#### 1. Exposure Limit for Asbestos

NIOSH supports OSHA's proposed rule to reduce the PEL for asbestos from 0.2 to 0.1 fiber/cc for all workers. Lowering the time-weighted average (TWA) PEL from the current standard of 0.2 to 0.1 fiber/cc will substantially reduce the health risk; however, even at this concentration, the resulting number of cancer deaths estimated by OSHA would still be excessive. If exposures are reduced to 0.1 fiber/cc, OSHA estimates that the mortality risk would be 3.4 deaths per 1,000 workers for a lifetime of exposure to asbestos, and 2.3 deaths per 1,000 for 20 years of exposure. Although the ultimate goal is to eliminate all possible exposures to asbestos, such a recommendation is not currently feasible in all workplaces. Where fiber exposures cannot be eliminated, exposures must be controlled to concentrations below 0.1 fiber/cc.

## 2. Methods for Sampling and Analysis of Airborne Fibers

Using NIOSH analytical Method 7400, NIOSH has been able to measure airborne fiber concentrations lower than 0.1 fiber/cc in some occupational environments (Note: NIOSH will provide OSHA with these data at a later date). However, unlike most industrial and construction work sites, these environments had minimal nonasbestos contamination to interfere with the counting of fibers. The limits of fiber detection and quantitation using Method 7400 depend on the sample volume and the quantity of interfering dust. The limit of detection is 0.01 fiber/cc in a 1,000-liter air sample from environments free of interferences. The quantitative working range of the method is 0.04 to 0.5 fiber/cc in a 1,000-liter air sample.

A significant consideration in establishing a PEL should be the lowest concentration that can be accurately measured using currently available analytical techniques. NIOSH has concluded that (1) for regulatory purposes, PCM is the most practical technique for assessing asbestos fiber exposures when using the criteria given in NIOSH analytical Method 7400, and (2) concentrations below 0.1 fiber/cc can be accurately measured in certain occupational environments. NIOSH recognizes that mixed-fiber exposures may occur in the workplace and that fibers may need to be identified. In such cases, Method 7400 can be supplemented with electron microscopy as described in Method 7402, using electron diffraction and microchemical analysis to improve the specificity of the fiber determinations [NIOSH 1990a].

In NIOSH's judgment, the establishment of a PEL or action level below 0.1 fiber/cc for most industrial or construction work sites would be difficult at this time. However, any detectable concentration of asbestos found in the workplace warrants further evaluation and, if necessary, the implementation of measures to reduce exposures.

## 3. Use of TWAs to Measure Exposures

OSHA has proposed that the 0.1-fiber/cc PEL for asbestos be based on an 8-hour TWA concentration. Although TWAs are the generally acceptable method for measuring exposures, we recommend the use of alternative approaches for evaluating airborne exposures to asbestos. In fact, an alternative approach may be necessary to measure exposures more precisely for short-duration tasks. As stated in our testimony to OSHA in 1984 [NIOSH 1984], one way to ascertain the effectiveness of controls is to restrict the period over which workplace concentrations can be averaged. For example, personal sampling pumps with a flow rate of 4 liters/min are available and would allow a sampling time of 100 min to achieve the minimum 400-liter sample. This sampling strategy will ensure that exposures are being limited to the lowest fiber concentration that can be ascertained by Method 7400.

NIOSH is presently reviewing the OSHA analytical method ID 160 (Ex. 1-129) and will provide comments to OSHA at a later date.

**B. 29 CFR 1910.1001(f)--Use of Engineering Controls, Good Work Practices, and Personal Protective Equipment**

**1. Methods of Compliance**

NIOSH strongly recommends that whenever possible workers and employers eliminate asbestos exposures completely through engineering controls (including substitution), and good work practices rather than protect themselves with personal protective equipment, including respiratory protection.

In our previous comments and testimony to the OSHA docket on the proposed rule for methods of compliance, NIOSH stated its strong support to the existing OSHA policy for controlling exposures to hazardous agents [NIOSH 1989a; NIOSH 1990b]. This policy states that, whenever feasible, engineering controls and work practices should be used to prevent exposures, and that personal protective equipment, including respiratory protection, should be used only when engineering controls are not feasible. Because of the many deficiencies of respiratory protection in reducing worker exposure to airborne asbestos, NIOSH continues to recommend that respiratory protection be used only when engineering controls are not feasible for reducing exposures to below the PEL. In comments to the OSHA docket on methods of compliance, NIOSH discussed several deficiencies regarding respiratory protection, including uncertainty in the level of protection provided by respirators, the physiological and psychological burden to the respirator wearer, the difficulty of implementing and enforcing a respirator user program, safety concerns for the respirator wearer, and numerous other deficiencies.

**2. Respiratory Protection**

Because asbestos is a known human carcinogen, NIOSH recommends that only the most protective respirators be worn when exposures exceed the NIOSH recommended exposure limit (REL) of 0.1 fiber/cc. These respirators are specified as follows in the NIOSH Respirator Decision Logic (RDL) [NIOSH 1987]:

- Any self-contained respirator equipped with a full facepiece and operated in a pressure-demand or other positive pressure mode, or;
- Any supplied-air respirator equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode in combination with an auxiliary, self-contained breathing apparatus operated in a pressure-demand or other positive-pressure mode.

Where exposure concentrations have been documented to be below 0.1 fiber/cc, or where certain small scale, short-duration tasks must be performed (e.g., maintenance and repair), or where the most protective respirators cannot be feasibly used, the most protective air-purifying respirators (APRs) could be permitted. The most protective APRs are the tight-fitting, high efficiency, powered air-purifying respirators (PAPRs) and the non-powered, full facepiece, high

efficiency respirator. These respirators have assigned protection factors (APF) of 50. The loose-fitting PAPR has an APF of 25 and is less protective [NIOSH 1987]. Among air-purifying respirators, the PAPR class of respirator has the distinct advantage of using the battery-powered blower to provide a filtered airstream to the wearer. Thus, unlike the wearers of nonpowered, air-purifying respirators, the wearers of PAPRs have less breathing resistance which results in less physiological burden to the respirator wearer. The airstream of the PAPR also provides a cooling effect in warm temperatures.

**C. 29 CFR 1910.1001(f)(1)(x)—Use of Engineering Controls and Work Practices for Brake and Clutch Repair and Service**

**1. Recommended Controls**

NIOSH supports OSHA in requiring the use of specific controls (e.g., enclosures and high-efficiency particulate air [HEPA] filter vacuum systems) during automotive brake and clutch repairs. As documented by NIOSH [Ex. 1-113], the proper use of these engineering controls and work practices can control airborne asbestos concentrations below 0.1 fiber/cc. When citing the NIOSH report, Control of Asbestos Exposure During Brake Drum Service (Ex. 1-113) in the proposed rule, OSHA incorrectly cited some of the results. Specifically, the proposed rule states the following (page 29725):

"Also it must be demonstrated that the 'equivalent' method results in exposures which are equal or less than the exposures resulting from the use of Method A, the Enclosed Cylinder/HEPA Vacuum System Method, as set forth in Ex. 1-112."

In our report (Ex. 1-112) the Enclosed Cylinder/HEPA Vacuum System Method is Method B, and the Glove Box/Vacuum Method (described on page 29724 of the proposed rule) is Method A.

In addition, the Enclosed Cylinder/HEPA Vacuum System Method was cited as a primary engineering control method, and the Glove Box/HEPA Vacuum Enclosure Method and the HEPA-Filter Equipped Vacuum Cleaner are listed as equivalent methods. The NIOSH report (Ex. 1-112) states that when using transmission electron microscopy (TEM), the arithmetic mean concentration for personal samples was 0.044 fiber/cc with the Enclosed Cylinder/HEPA Vacuum System Method; 0.021 fiber/cc with the Glove Box/HEPA Vacuum Enclosure Method (Method A); and 0.022 fiber/cc with the HEPA-Filter Equipped Vacuum Cleaner Method. Statistically, no significant difference occurred in asbestos exposures among these three engineering control methods. Currently, OSHA specifies, as mandatory, three of the five NIOSH control methods that were shown to be effective in the NIOSH study, while the other two methods, the Glove Box/HEPA Vacuum Enclosure and HEPA-Filter Equipped Vacuum Cleaner, are listed as equivalent methods. NIOSH recommends that OSHA specify, as mandatory, all



five control methods specified in the NIOSH study. The data from this study suggest no objective basis for differentiating the relative effectiveness between the five methods in controlling exposures below 0.1 fiber/cc.

## **2. Documentation of the Effectiveness of Engineering Controls**

As part of the recordkeeping and documentation requirements proposed by OSHA in Appendix F to 29 CFR 1910.1001, NIOSH recommends that OSHA also require employers to perform periodic maintenance inspections of engineering controls and to maintain a written log of their effectiveness and any actions taken to ensure their proper functioning.

## **3. Respiratory Protection**

Where exposures have been documented to be below 0.1 fiber/cc, or where the most protective respirators cannot be feasibly used, the most protective APRs could be permitted. The most protective APRs are the tight-fitting, high-efficiency, powered air-purifying respirators (PAPRs) and the nonpowered, full facepiece, high-efficiency respirator. These respirators have APFs of 50.

### **D. 29 CFR 1910.1001(f)(1)(xi)—Required Use of Specific Work Practices During Maintenance of Vinyl Asbestos Floor Tile**

NIOSH agrees that OSHA should require specific work practices to minimize the release of asbestos fibers during the routine maintenance of vinyl asbestos floor tile (VAT). In a study conducted by the Los Angeles Unified School District [LAUSD 1990] to assess airborne exposures to asbestos during the stripping and buffing of VAT with rotary-powered equipment, fibers were released by all methods employed. Fiber concentrations were below 0.1 fiber/cc when analyzed by PCM, but TEM revealed that concentrations often exceeded 70 structures\*/mm<sup>2</sup>.

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\*Structures are a microscopic bundle, cluster, fiber, or matrix that may contain asbestos [Public Law 99-519, 1986].

The results from this study suggest that the following work practices would help to minimize fiber exposures during the routine cleaning of VAT:

- Using chemical stripping products that do not require the use of power equipment to loosen and remove wax
- Refraining from buffing floors routinely
- Using only low-abrasion pads on powered equipment operating at 190 rpm or less when buffing is necessary
- Encouraging the routine damp-mopping of floors to remove excess dust and decrease the need for stripping
- At a minimum, workers should be required to wear the most protective air-purifying respirators as specified in III.C.3.

The proposed OSHA rule requires employers to inform their workers about the risk of exposure while cleaning VAT. NIOSH recommends that this section (1) clearly define the employer's responsibility in this area, and (2) specifically direct the employer to instruct workers about the proper cleaning of VAT according to the requirements of this section. This section should also provide for the revision of these work practices as safer methods of VAT cleaning are developed.

**E. 29 CFR 1910.1001(k)(7)--Housekeeping**

NIOSH endorses the proposed change in housekeeping requirements for primary and secondary asbestos operations. This change would require that the premises be cleaned at least once per shift with a HEPA vacuum and that wet methods be used where feasible. NIOSH agrees with this provision and its intent to prevent the reentrainment of asbestos into the air.

**F. 29 CFR 1926.58(b)--Definitions of Small-Scale, Short-Duration Operations**

Comments were requested concerning the definition of small-scale, short-duration operations in which workers may be exposed to asbestos. OSHA has proposed that such operations be exempted from the required establishment of full-size, negative-pressure enclosures. NIOSH recognizes that certain small-scale jobs may not permit the establishment of negative-pressure enclosures or other types of engineering controls; however, the Institute recommends that such controls be used whenever feasible. This recommendation is based on our concern that short-term, intermittent exposures to asbestos may occur and may (1) pose a health risk to workers and (2) contaminate other work areas if the operation is improperly performed. NIOSH recommends that the following provisions be incorporated into OSHA's proposed rule for small-scale, short-duration operations:

1. Include a maximum surface area or volume limit for the repair or removal of asbestos on pipes.
2. Require the use of respiratory protection for all workers because of the potential for accidental exposure to asbestos. At a minimum, workers should be required to wear the most protective air-purifying respirator as specified in III.C.3.
3. Require workers to wear appropriate protective equipment. At a minimum, workers should wear disposable clothing and be afforded access to shower facilities.
4. Require a competent person to be present at each work site to ensure that all possible engineering controls are in place and working properly and that appropriate work practices and protective equipment are being used to minimize exposures.

**G. 29 CFR 1926.58(e)(6)(iii)—Exemption of Roofing Operations from the Requirement for Negative-Pressure Enclosures**

OSHA has also asked for comments about the proposal to exempt asbestos roofing removal from the requirement to provide negative-pressure enclosures. Studies have shown that airborne exposures to asbestos may occur during the removal of asbestos roofing [NIOSH 1985; Millar 1989; Mueller 1989], and that such operations may pose a health risk to workers. On the basis of these data, NIOSH recommends that all workers potentially exposed to asbestos during the removal of asbestos-containing roofing material be required to wear, at a minimum, the most protective air-purifying respirator as specified in III.C.3. In addition, workers should be required to wear protective equipment (including disposable clothing at a minimum) and should have access to shower facilities. NIOSH also recommends incorporating the following provisions into the proposed rule as a prudent approach to minimizing the release of asbestos fibers during the removal of asbestos roofing:

1. Cut or remove asbestos-roofing material by hand whenever possible.
2. Equip all powered tools with a HEPA filter vacuum system or, on flat roofs, a misting device.
3. Use a HEPA filter vacuum to collect all loose dust left by the sawing operation.
4. Double-bag all waste (i.e., wrap it in two layers of 6-mil polyethylene), or place it in sealed containers; use a hoist or crane to lower all bags, wrapped material, and drums to the ground.
5. Isolate all roof-level air intake and discharge sources, or shut down all mechanical systems and seal off all outside vents with two layers of 6-mil polyethylene.

#### **H. 29 CFR 1926.58(d)—Communication Among Employers and Owners**

OSHA has proposed that project managers or building owners be required to provide employers and workers with information about the presence, location, and quantity of asbestos-containing materials on a prospective job site, as cited in 29 CFR 1926.58(d). In addition, project and building owners must maintain records of all information provided pursuant to this section or otherwise concerning the presence, location, and quantity of asbestos-containing materials in the building. NIOSH supports the OSHA provisions for notification and recordkeeping and recommends that these provisions be applied to all operations with the potential for asbestos exposure.

#### **I. 29 CFR 1926.58(e)(6)—Regulated Areas for Asbestos Removal, Maintenance, Demolition, and Renovation Operations**

##### **1. Negative-Pressure Enclosures**

OSHA has proposed that negative-pressure enclosures be established in all regulated areas before commencing removal, demolition, maintenance, or renovation operations, except where negative-pressure enclosures are not feasible. In theory, such a requirement should effectively prevent contamination of personnel and areas outside of a properly designed, constructed, and maintained enclosure. However, NIOSH has observed the release of asbestos fibers from improperly designed, constructed, or maintained negative-pressure enclosures [NIOSH 1989b], and we are not aware of any studies evaluating their effectiveness or delineating important parameters such as minimum pressure differential, minimum air flow, or maximum volumes feasible for various barrier materials. NIOSH therefore recommends that the following design requirements be incorporated into the proposed rule for negative-pressure enclosures:

- Design air-flow patterns within the enclosure to move airborne particles away from the worker.
- Provide adequate filtration and filter maintenance to prevent asbestos from escaping in the air discharged from the enclosure.
- Take precautionary measures to prevent back-flow of airborne asbestos if the vacuum device fails.

When negative-pressure enclosures are used, precautionary measures are needed to ensure that workers who are constructing or working inside the enclosures are wearing the appropriate protective clothing and respirators. In addition, adhesives commonly used during such construction may expose workers to high concentrations of methylene chloride [Fleeger and Lee 1988] and may therefore require the use of proper ventilation and respiratory equipment.

NIOSH is in the process of evaluating the effectiveness of negative-pressure glove bags and boxes in controlling exposures to airborne asbestos. Reports of these studies will be forwarded to OSHA upon their completion.

## **2. Glove Bags**

NIOSH has found that airborne fibers are released in the workplace when glove bags are used to remove asbestos pipe lagging [NIOSH 1990c]. Although the reasons for these releases were not determined, the study indicated that glove bags did not control asbestos exposures as anticipated. Thus NIOSH strongly supports OSHA in requiring that negative-pressure enclosures be used in conjunction with glove bags. Furthermore, NIOSH recommends that OSHA require the use of respiratory protection whenever glove bags are used [EPA/NIOSH 1986]. At a minimum, NIOSH recommends that workers should be required to wear the most protective air-purifying respirator as specified in III.C.3.

## **J. 29 CFR 1926.58(o)–Competent Person**

OSHA has proposed that a competent person be present at all work sites where workers are removing, demolishing, or renovating asbestos-containing materials. This person would be responsible for ensuring compliance with all requirements in the proposed standard (e.g., those concerning engineering controls, respiratory protection, notification, etc.). The competent person would also be required to receive appropriate training relevant to these provisions. The requirements for such training could be met through the completion of a course conducted by a U.S. Environmental Protection Agency (EPA) training center certified by EPA or through the completion of an equivalent course.

NIOSH recommends that a competent person be present at all sites where workers are demolishing, renovating, or removing asbestos materials where the potential exists for exposure. Training and certification through completion of an approved OSHA course should be required for all competent persons. Courses designed to meet the requirements of EPA's Asbestos Containing Materials in Schools Standard [40 CFR part 763] (particularly those developed for asbestos abatement supervisors and operations and maintenance personnel) provide only the minimum curricula needed for a competent person. An appropriate training curriculum for qualifying persons should cover the following subjects:

1. Identification of possible hazards at the work site, including asbestos-containing materials
2. Proper handling methods for asbestos-containing materials, including appropriate engineering and work practice controls specified in the standard
3. Reporting, recordkeeping, and record transfer requirements specified in the standard

4. Requirements for personal air monitoring and actions to be based on monitoring results
5. The selection of appropriate protective equipment (including respirators) and the feasibility of using them at various asbestos-related operations

All training should include both classroom time and hands-on experience. Course work should cover many of the possible work activities that OSHA defines for a regulated asbestos area, and it should include instruction about the requirements for small-scale, short-duration operations.

NIOSH also recommends that the successful completion of an approved training course be required for all workers who routinely handle asbestos-containing materials. At a minimum, such a training course should meet the requirements specified in EPA's Model Accreditation Plan for training asbestos abatement workers.

If OSHA adopts the requirements for mandatory training and certification of competent persons and workers, a model curriculum should be developed. NIOSH would assist OSHA in developing a training curriculum and would help reconcile any differences with the training requirements in EPA's rule on asbestos-containing materials in schools [52 FR 41826 (1987)].

**K. 29 CFR 1926.58(p)--Notification to OSHA and Method of Notification**

NIOSH supports OSHA's requirement for the employer to provide the OSHA area office with written notification within 10 working days of any planned demolition, renovation, removal or other related activity that would disturb asbestos-containing materials. This requirement should (1) help OSHA target and set priorities for inspections, and (2) heighten the employer's awareness of the applicable requirements of the standard. If OSHA adopts the NIOSH recommendation that an approved training curriculum be used for competent persons and other workers engaged in demolishing, renovating, or removing asbestos-containing materials, the notification to OSHA should acknowledge worker certification of this training.

Given the complexity of some demolition and renovation activities, asbestos-containing materials may not be identified until after the work has been initiated. Disturbance of these materials could pose a significant health risk to workers and require appropriate engineering controls, work practices and personal protective equipment. Therefore, the notification to OSHA should include (1) a description of procedures to be used if unexpected amounts of asbestos-containing materials are discovered at the work site, (2) a statement about the availability of a competent person, and (3) verification that involvement will be limited to workers certified in handling asbestos-containing materials. A written description of a contingency plan would enable the OSHA area office to evaluate whether the employer is prepared to handle such a situation adequately.

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Statement By

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Before the

Subcommittee on Toxic Substances, Environmental  
Oversight, Research and Development  
Committee on Environment and Public Works

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Public Health Service  
Centers for Disease Control

April 26, 1990

I am pleased to testify on the science and the health effects caused by exposures to asbestos. I am currently Assistant Director of the National Institute for Occupational Safety and Health (NIOSH). I have been involved with the study of occupational exposures to asbestos since 1970. I am also the primary author of the NIOSH recommended standard for occupational exposure to asbestos, the asbestos monograph published by the International Agency for Research on Cancer, and numerous articles on asbestos in the scientific literature. I have attached a copy of my complete curriculum vitae.

Asbestos is a generic term referring to a group of naturally occurring fibrous minerals that are commercially prized for their thermal and insulative properties, in addition to their flexibility, durability and tensile strength. Because of these characteristics, asbestos is highly persistent in the human body once inhaled or ingested.

Based on studies of workers who were heavily and regularly exposed to asbestos before general government regulation of the workplace, we know that asbestos causes specific diseases such as asbestosis, an irreversible and progressively disabling lung disease which impairs breathing, and mesothelioma, an invariably fatal cancer of the lining of the chest, pericardium, or abdominal cavity. Asbestos is one of the leading causes of lung cancer in non-smokers. Asbestos exposure for smokers increases the risk of lung cancer approximately 55 times that of those who are not exposed to asbestos and who do not smoke. Asbestos is also associated with an increased risk of gastrointestinal and other cancers.

The conclusion drawn by many experts, in this and other countries, and best summarized in the 1987 Supplement of the World Health Organization's International Agency for Research on Cancer, is that " . . . occupational exposure to chrysotile, amosite and anthophyllite asbestos and to mixtures containing crocidolite results in an increased risk of lung cancer, as does exposure to minerals containing tremolite and actinolite and to tremolitic material mixed with anthophyllite and small amounts of chrysotile. Mesotheliomas have been observed after occupational exposure to crocidolite, amosite, tremolitic material and chrysotile asbestos. Gastrointestinal cancers occurred at an increased incidence in groups occupationally exposed to crocidolite, amosite, chrysotile or mixed fibres containing crocidolite, although not all studies are consistent in this respect. An excess of laryngeal cancer has also been observed in some groups of exposed workers. No clear excess of cancer has been associated with the presence of asbestos fibres in drinking water. Mesotheliomas have occurred in individuals living in the neighbourhood of asbestos factories and mines, and in people living with asbestos workers."

Recent reports have appeared in the scientific literature to suggest that different forms of asbestos are not equally pathogenic (Mossman and Gee, 1989 and Mossman et al., 1990). However, there is a great deal of uncertainty associated with these findings and equally important contradictory evidence. Results from research involving animal bioassays present a strong case that there is no safe form of asbestos. Wagner et al. (1979), then with the United Kingdom's Medical Research Council, have shown that a commercial grade, predominantly short-fiber Canadian chrysotile (a purportedly less hazardous form of asbestos), and an ingredient used primarily in paint and in plastic tile fillers, induces mesotheliomas when injected intrapleurally into rats, and induces primary lung neoplasms in rats exposed by inhalation. Not only has chrysotile been found to be as potent as crocidolite and other amphiboles in inducing mesotheliomas when injected intrapleurally (Wagner et al., 1973), it has been found equally potent in inducing pulmonary neoplasms through inhalation

exposures (Wagner et al., 1974). Chrysotile also appears to be more potently fibrogenic and carcinogenic than amphiboles, in relation to the quantity of dust deposited and retained in the lungs of rats (Wagner et al., 1974).

There is the hypothesis that chrysotile is less hazardous because of its chemical and biological reactivity. In fact chrysotile fibers are much more chemically and biologically reactive than amphibole fibers (Davis et al., 1978; Davis et al., 1986a; and Davis et al., 1986b). In contact with body tissues, chrysotile fibers lose their structural elements and divide into smaller fibrils, making their recognition difficult by the usual analytical methods. In fact, many of the fibers are removed from the lungs to other organs in the body and up through the bronchi. These findings also support the hypothesis that chrysotile fibers cause cellular injury, fibrosis and lung cancer. These fibers are less readily detected in the tissue after the damage is done. The concentration of dust in the lungs of rats exposed to Canadian chrysotile (Wagner et al., 1974) was only 1.8% to 2.2% of the dust concentration in the lungs of animals exposed to amphiboles, after 24 months of inhalation exposure. Yet the lung tumor incidences and degree of pulmonary fibrosis were similar among groups of rats exposed to different forms of asbestos.

At this time, there is no compelling evidence to justify different public health policy for different asbestos fiber types. The reason for higher incidence of lung cancer and mesothelioma in workers exposed to amphiboles is probably related to higher concentrations of respirable fibers during their exposures (NIOSH, 1979). Furthermore, most commercially exploited deposits of chrysotile are contaminated with some type of the amphibole form of asbestos (Bartlett, 1988 and Campbell, 1988).

Other international expert groups have reached similar conclusions regarding the uncertainty of the hypothesis that some forms of asbestos may be less hazardous. In a recently released document from an expert panel convened by the World Health Organization in 1989, the panel concluded: "it is difficult to substantiate this difference [in pathogenicity] firmly after standardization for exposure levels, type of industry, duration of employment, etc." This conclusion agrees with the findings of the 1984 report of the Canadian Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. The Commission recommended that textile manufacturing using a form of asbestos purported to be less hazardous (chrysotile) be banned, and concluded that "all fiber types can cause all asbestos-related diseases."

The Centers for Disease Control (CDC) through NIOSH has recently submitted to Occupational Safety and Health Administration (OSHA) on April 8, 1990 a reiteration of its previous testimony of June 21, 1984, that "... there is no safe concentration for exposure to asbestos." Not even the lowest exposure limit for asbestos could assure all workers absolute protection from exposure-related cancer. OSHA projects that at the current occupational standard for asbestos of 0.2 fibers/cc over a working lifetime, 67 cancers for every 1,000 exposed workers can be expected to develop (OSHA, 1986). In the April 8, 1990 submittal to OSHA, CDC through NIOSH also reaffirmed its position that there is no scientific basis for differentiating between types of asbestos fibers for regulatory purposes. The scientific evidence to date suggests that fiber morphology (size and shape) is the most critical factor in the pathogenicity of the material and as such the most prudent public health policy is to regulate asbestos based upon its morphology and not on its mineralogic source.

I would be happy to answer any questions the subcommittee may have.



## **PART II**

### **NIOSH BIBLIOGRAPHY ON ASBESTOS**



## NIOSH BIBLIOGRAPHY - ASBESTOS

**NUMBERED PUBLICATIONS** document the results of NIOSH research. Included in this category are Criteria Documents, Current Intelligence Bulletins, Alerts, Health and Safety Guides, technical reports of scientific investigations, compilations of data, work-related booklets, symposium and conference proceedings, and NIOSH administrative and management reports.

**Building Air Quality: A Guide for Building Owners and Facility Managers, 1991.** (Joint NIOSH-EPA Publication)

NIOSH PUB NO: 91-114. 253 pp.

Available from NIOSH No Charge

GPO NO: 055-000-00390-4 \$24.00

**Control of Asbestos Exposure During Brake Drum Service, 1989.**

NIOSH PUB NO: 89-121. 79 pp.

Available from NIOSH No Charge

NTIS NO: PB 90-168501 \$19.00

**Criteria for a Recommended Standard: Occupational Exposure to Asbestos, 1972.**

(Revised: See next entry.)

NIOSH PUB NO: 72-10267. 129 pp.

NTIS NO: PB 209510 \$26.00

**Criteria for a Recommended Standard: Occupational Exposure to Asbestos (Revised), 1976.**

NIOSH PUB NO: 77-169. 100 pp.

Available from NIOSH No Charge

NTIS NO: PB 273965 \$19.00

(An abstract of this document is contained on pages 9-20 in Part I.)

**Current Intelligence Bulletin 5 - Asbestos Exposure During Servicing of Motor Vehicle Brake and Clutch Assemblies, 1975.**

NIOSH PUB NO: 78-127. 125 pp. (This publication is a compendium of Current Intelligence Bulletins 1-18.)

Available from NIOSH No Charge

NTIS NO: PB 83-105080 \$26.00

**Estimates of Pulmonary and Gastrointestinal Deposition for Occupational Fiber Exposures, 1979.**

NIOSH PUB NO: 79-135. 84 pp.

NTIS NO: PB 80-149644 \$19.00

**An Evaluation of Glove Bag Containment in Asbestos Removal, 1990.**

NIOSH PUB NO: 90-119. 131 pp.

Available from NIOSH

No Charge

GPO NO: 017-033-00451-0

\$ 7.00

NTIS NO: PB 91-188995

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**An Evaluation of Vacuum Equipment for Collection of Asbestos Waste, 1980.**

NIOSH PUB NO: 80-137. 77 pp.

NTIS NO: PB 82-150236

\$19.00

**A Guide to Respiratory Protection for the Asbestos Abatement Industry, 1986.** (Joint NIOSH-EPA Publication)

EPA PUB NO: 560-OPTS-86-001. 173 pp.

NTIS PB 87-157574 \$26.00

**Good Practices Manual for Insulation Installers, 1977.**

NIOSH PUB NO: 77-188. 40 pp.

NTIS NO: PB 83-178822 \$17.00

**NIOSH Certified Equipment List as of December 1991.**

NIOSH PUB NO: 92-101. 576 pp.

Available from NIOSH

No Charge

GPO NO: 017-033-00457-9

To be announced

**NIOSH Guide to Industrial Respiratory Protection, 1987.**

NIOSH PUB NO: 87-116

Available from NIOSH

No Charge

NTIS NO: PB 88-188347

\$32.00

**NIOSH Manual of Analytical Methods - Third Edition, 1984, and Supplements, 1985, 1987, 1989, 1990.**

GPO NO: 917-011-00000-1

\$32.00

(Copies of the NIOSH analytical methods for asbestos are contained on pages 117-157 in Part I.)

**Occupational Diseases: A Guide to Their Recognition (Revised), 1977.**

NIOSH PUB NO: 77-181. 619 pp.

Available from NIOSH

No Charge

NTIS NO: PB 83-129528

\$66.00

**Occupational Exposure to Talc Containing Asbestos, 1980.**

NIOSH PUB NO: 80-115. 117 pp.

NTIS NO: PB 80-193352

\$26.00



**Occupational Respiratory Diseases, 1986.**

NIOSH PUB NO: 86-102. 825 pp.

Available from NIOSH No Charge

GPO NO: 017-033-00425-1 \$47.00

NTIS NO: PB 87-205662 80.00

(Copies of the sections on asbestosis, lung cancer, and mesothelioma are contained on pages 29-101 in Part I.)

**Occupational Safety and Health Guidelines for Chemical Hazards - Supplement I, 1988.**

NIOSH PUB NO: 88-118. 214 pp.

Available from NIOSH No Charge

GPO NO: 017-033-00439-1 \$11.00

NTIS NO: PB 89-203129 35.00

(A copy of the Guideline for asbestos is contained on pages 3-7 in Part I.)

**Proceedings of a Roundtable Discussion on Thermal Analysis Techniques, 1976.**

NIOSH PUB NO: 76-173. 260 pp.

NTIS NO: PB 266511 \$35.00

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NIOSH PUB NO: 90-108. 1608 pp.

NTIS NO: PB 91-188821 (Part I) \$80.00

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**Recommended Industrial Ventilation Guidelines, 1976.**

NIOSH PUB NO: 76-162. 333 pp.

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NTIS NO: PB 274750 \$19.00

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**USPHS/NIOSH Membrane Filter Method for Evaluating Airborne Asbestos Fibers, 1979.**

NIOSH PUB NO: 79-127. 89 pp.

NTIS NO: PB 297731 \$19.00

**Work Related Lung Disease Surveillance Report, 1991.**

NIOSH PUB NO: 91-113. 84 pp.

Available from NIOSH No Charge

NTIS NO: PB 92-136266 \$19.00

(Excerpts on asbestos from this report are contained on pages 103-115 in Part I.)

**Workplace Exposure to Asbestos - Review and Recommendations, 1980.**

NIOSH PUB NO: 81-103. 39 pp.

Available from NIOSH No Charge

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(An abstract of this publication is contained on pages 21-28 in Part I.)

*TESTIMONY consists of both written comments and oral testimony presented before Congressional committees and at regulatory hearings convened by either the Department of Labor (DOL), Occupational Safety and Health Administration (OSHA) or the Environmental Protection Agency (EPA). The following list of NIOSH testimony on asbestos is arranged in reverse chronological order.*

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NTIS NO: PB 92-139088 \$17.00

(A copy of this testimony is contained on pages 159-173 in Part I.)

NIOSH [1991]. Post-hearing comments to DOL on OSHA's proposed rule on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 6 pp.  
NTIS NO: PB 92-136043 \$12.50

NIOSH [1990]. Comments by R.W. Niemeier to DOL, post-hearing comments to the Occupational Safety and Health Administration's proposed rule on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 22 pp.  
NTIS NO: PB 91-212514 \$17.00

NIOSH [1990]. Testimony by R.A. Lemen to DOL on the Occupational Safety and Health Administration's notice of proposed rulemaking on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 17 pp.  
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NIOSH [1990]. Statement by R.A. Lemen before the Subcommittee on Toxic Substances, Environmental Oversight, Research and Development, Committee on Environment and Public Works. 6 pp.  
 NTIS NO: PB 91-132852 \$12.50  
 (A copy of this statement is contained on pages 175-177 in Part I.)

NIOSH [1990]. Comments by R.A. Lemen to DOL on the Occupational Safety and Health Administration's notice of proposed rulemaking on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 15 pp.  
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**MISCELLANEOUS REPORTS** include documents that do not fit into one of the preceding five sections.

**Application of Neural Network Technology to Fiber Image Analysis, Final Report: Analytical Methods for Asbestos Fibers, December 17, 1990.** 43 pp.  
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**Asbestos Related Disease - A Community Epidemic in the Making, 1971.** 6 pp.

NTIS NO: PB 90-155896 \$12.50

**Caution: Asbestos Dust . . . is Hazardous to Your Health, 1973.** 16 pp.

NTIS NO: PB 88-204649 \$17.00

**Chrysotile Asbestos in Industry, February 1978.** 20 pp.

NTIS NO: PB 90-155953 \$17.00

**Control Technology (CT) Reports - Asbestos.**

CT reports represent the results of field study surveys that evaluate health hazard control systems at individual worksites. A list of CT reports related to asbestos, along with ordering information, is available at no charge from NIOSH.

**Epidemiology of Asbestos Related Diseases, July 1981.** 47 pp.

NTIS NO: PB 90-155904 \$17.00

**An Evaluation of Phase Contrast Microscopes for Asbestos Counting, March 1974.** 11 pp.

NTIS NO: PB 90-130022 \$17.00

**Health Hazard Evaluations (HHE) - Asbestos.** A typical HHE involves studying a workplace, such as a particular department in a factory, industrial plant, or other worksite. The study is done by NIOSH in response to concerns expressed by employees, employee representatives, or employers, to find out whether there is a health hazard to employees caused by exposure to hazardous materials (chemical or biological contaminants) in the workplace. NIOSH also evaluates other potentially hazardous working conditions, such as exposures to heat, noise, radiation, or musculoskeletal stresses. A list of HHE's related to asbestos, along with ordering information, is available at no charge from NIOSH.

**Industrywide Study (IWS) Reports - Asbestos.**

IWS reports represent the results of industrial hygiene field studies at individual worksites that assess whether specific occupational exposures of particular workers are associated with adverse health effects. A list of IWS reports related to asbestos, along with ordering information, is available at no charge from NIOSH.

**Mining Surveillance: Potentially Toxic Occupational Exposures, May 1979.** 19 pp.

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**National Occupational Health Survey of Mining -  
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NTIS NO: PB 90-163676 \$19.00**

**Occupational Health Study of the Asbestos Products  
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**Proceedings of the First NCI/EPA/NIOSH Collaborative  
Workshop: Progress on Joint Environmental and  
Occupational Cancer Studies, Rockville, MD,  
*May 6-8, 1980*. 863 pp.  
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**Report on Testing of the Automated Fiber Counter,  
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**A Review of the Scientific Basis for EPA's School  
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State Health Officials, *October 1984*. 22 pp.  
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**Testing of Hair Dryers for Asbestos Emissions,  
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