ASBESTOS BIBLIOGRAPHY

(Revised)

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

INTRODUCTION

This publication is a compendium of NIOSH research and recommendations on asbestos. It updates and supersedes the NIOSH document *Asbestos Publications* dated June 1992.

This publication is divided into three Parts:

- Part I consists of full or partial text of selected NIOSH documents on asbestos. These documents provide an overview of NIOSH research on the health hazards of asbestos and NIOSH recommendations on workplace exposure to asbestos.

- Part II contains a comprehensive bibliography of NIOSH documents on asbestos. It is divided into two sections: (A) NIOSH-authored documents (which include numbered publications, testimony, journal articles, and miscellaneous reports) and (B) NIOSH-funded documents (which include grant and contract reports). Each document citation includes the title and year of publication and bibliographic or ordering information (see below).

- Part III contains summary asbestos information from other Federal agencies.

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4. Copies of journal articles, book chapters, and proceedings may be obtained from public or university libraries using the bibliographic information shown in the citation.

PART I

FULL OR PARTIAL TEXT 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 Recommendations may be superseded by new developments in these fields; therefore, readers are advised to regard these recommendations as general guidelines.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) $3MgO\text{-}2SiO_2\text{-}2H2O$;
(3) $(FeMg)SiO_3$;
(4) $NaFe(SiO_3)_2\text{-}FeSiO_3\text{-}H_2O$;
(5) $Ca_2Mg_5Si_8O_{22}(OH)_2$;
(6) $(MgFe)_7Si_8O_{22}(OH)_2$;
(7) $CaO\text{-}3(MgFe)O\text{-}4SiO_2$

• **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 (μ)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®) 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 li nits for asbestos

	Exposu limits mg
OSHA PEL TWA	(
Action level	
NIOSH REL TWA (Ca)†	
ACGIH TLV® TWA (Ala)§	
Chrysotile	
Amosite	
Crocidolite	
Other forms	2.0

* Fibers greater than 5 μm 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 mo: itoring, medical screening, and morbidity and mortality data. his analysis may provide information about the relatedness adverse health effects and occupational exposure that cai ot be discerned from results in individual workers. Sensit vity, 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.**

BIBLIOGRAPHY

- American Conference of Governmental Industrial Hygienists: "Asbestos," *Documentation of the Threshold Limit Values and Biological Exposure Indices* (5th ed.), Cincinnati, 1986.
- American Conference of Governmental Industrial Hygienists: *TLVs® Threshold Limit Values and Biological Exposure Indices for 1987-88*, Cincinnati, 1987.
- American Lung Association of San Diego and Imperial Counties: "Taking the Occupational History," *Annals of Internal Medicine*, 99:641-651, November 1983.
- Clayton, G.D., and Clayton, F.E. (eds.): *Toxicology*, Vol. IIB of *Patty's Industrial Hygiene and Toxicology* (3rd rev. ed.), John Wiley & Sons, Inc., New York, 1981.
- *Code of Federal Regulations*, U.S. Department of Labor, Occupational Safety and Health Administration, 29 CFR 1910.1001, OSHA 2206, revised July 1, 1986.
- *Code of Federal Regulations*, U.S. Department of Transportation, 49 CFR 172.101, Transportation 49, revised October 1, 1982.
- Goldman, R.H., and Peters, J.M.: "The Occupational and Environmental Health History," *Journal of the American Medical Association*, 246:2831-2836, 1981.
- Halperin, W.E., Ratcliffe, J., Frazier, T.M., Wilson, L., Becker, S.P., and Shulte, P.A.: "Medical Screening in the Workplace: Proposed Principles," *Journal of Occupational Medicine*, 28(8): 547-552, 1986.
- Hankinson, J.L.: "Pulmonary Function Testing in the Screening of Workers: Guidelines for Instrumentation, Performance, and Interpretation," *Journal of Occupational Medicine*, 28(10):1081-1092, 1986.
- Hawley, G.G.: *The Condensed Chemical Dictionary* (10th ed.), Litton Educational Publishing, Inc., New York, 1981.
- International Agency for Research on Cancer: *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man, Some Inorganic and Organometallic Compounds*, Vol. 2, Lyon, France, 1973.
- International Agency for Research on Cancer: *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man, Asbestos*, Vol. 14, Lyon, France, 1977.
- Leidel, N.A., Busch, K.A., and Lynch, J.R.: *Occupational Exposure Sampling Strategy Manual*, U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, DHEW (NIOSH) Publication No. 77-173, Cincinnati, 1977.
- Lemen, R.A., Director, Division of Standards Development and Technology Transfer, National Institute for Occupational Safety and Health, U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control: *Congressional Testimony before the Subcommittee on Public Buildings and Grounds, Committee on Public Works and Transportation, U.S. House of Representatives*, March 21, 1984.
- Levy, B.S., and Wegman, D.H. (eds.): *Occupational Health: Recognizing and Preventing Work-Related Disease*, Little, Brown and Company, Boston, 1983.
- Mark, H.F., Othmer, D.F., Overberger, C.G., Seaborg, G.T., Grayson, M., and Eckroth, D. (eds.): *Kirk-Othmer Encyclopedia of Chemical Technology* (3rd ed.), John Wiley & Sons, Inc., New York, 1978.
- National Institute for Occupational Safety and Health, U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control: *Criteria for a Recommended Standard. . . .Occupational Exposure to Asbestos*, HSM Publication No. (NIOSH) 72-10267, 1972.
- National Institute for Occupational Safety and Health, U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control: "Current Intelligence Bulletin 5, Asbestos Exposure During Servicing of Motor Vehicle Brake and Clutch Assemblies," *Current Intelligence Bulletin Reprints—Bulletins 1 thru 18*, DHEW (NIOSH) Publication No. 78-127, 1978.
- National Institute for Occupational Safety and Health, U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control: *Occupational*

Diseases—A Guide to their Recognition (rev. ed. 2nd printing), DHEW (NIOSH) Publication No. 77-181, 1978.
• National Institute for Occupational Safety and Health, U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control: *Revised Recommended Asbestos Standard*, DHEW (NIOSH) Publication No. 77-169, 1976.
• National Institute for Occupational Safety and Health, U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control: *NIOSH Manual of Analytical Methods* (3rd ed., Vol. 1), Eller, P.M. (ed.), DHHS (NIOSH) Publication No. 84-100, Cincinnati, 1984.
• National Institute for Occupational Safety and Health, U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control: *NIOSH Testimony to DOL, Statement of the National Institute for Occupational Safety and Health, The Public Hearing on Occupational Exposure to Asbestos*, Public Docket No. H-033C, June 21, 1984.
• National Institute for Occupational Safety and Health, U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control: *Registry of Toxic Effects of Chemical Substances* (Microfiche Edition), Sweet, D.V., and Lewis, R.J. (eds.), Cincinnati, April 1985.
• National Institute for Occupational Safety and Health, U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control: *Workplace Exposure to Asbestos, Review and Recommendations*, NIOSH-OSHA Asbestos Work Group, DHHS (NIOSH) Publication No. 81-103, 1980.
• Proctor, N.H., and Hughes, J.P.: *Chemical Hazards of the Workplace*, J.B. Lippincott Company, Philadelphia, 1978.
• Rom, W.N. (ed.): *Environmental and Occupational Medicine*, Little, Brown and Company, Boston, 1983.
• Rothstein, M.A.: *Medical Screening of Workers*, Bureau of National Affairs, Washington, DC, 1984.
• Rutstein, D.D., Mullan, R.J., Frazier, T.M., Halperin, W.E., Melius, J.M., and Sestito, J.P.: "Sentinel Health Events (Occupational): A Basis for Physician Recognition and Public Health Surveillance," *American Journal of Public Health*, 73:1054-1062, 1983.
• Scientific Assembly on Environmental and Occupational Health: "Evaluation of Impairment/Disability Secondary to Respiratory Disease," *American Review of Respiratory Diseases*, 126: 945-951, 1982.
• Scientific Assembly on Environmental and Occupational Health: "Proposed Guidelines for Case Evaluation (of Asbestos-Associated Diseases) Based on Assessment of Evidence Available March 1983," *American Thoracic Society News*, Fall 1983.
• Scientific Assembly on Environmental and Occupational Health: "Surveillance for Respiratory Hazards in the Occupational Setting," *American Review of Respiratory Diseases*, 126:952-956, 1982.

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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REVISED RECOMMENDED
ASBESTOS STANDARD

Table of Contents

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 (μm) 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 μm/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 μm/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.

Edward J. Baier

for John F. Finklea, M.D.
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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 μm 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 chrysostile, 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 μm 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 μm in length/m^3 (0.1 fibers/cc), on an 8-hour-TWA basis with peak concentrations not exceeding 500,000 fibers >5 μm in length/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 μm in length/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 μm in length/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

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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 * 2,000,000 fibers per cubic meter greater than 5 μm in length (2Mf/m^3). Since the 1972 promulgation of this 2,000,000 f/m^3 standard, OSHA, in 1975, proposed lowering the standard to 500,000 f/m^3; NIOSH, in 1976, recommended lowering the standard to 100,000 f/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.

*Effective January 19, 1989, the OSHA Permissible Exposure Limit (PEL) was changed to 0.2 f/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 noncommercial 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 μm 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 (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 μm 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.

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ASBESTOSIS

John M. Dement
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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 comsumption 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
Total	552,100	24,700	3,500	2,700

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 of 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

Author(s)	Date	Study Population	Fiber Type	Study Design	Summary of Important Findings
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 gastro-intestinal 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)

Author(s)	Date	Study Population	Fiber Type	Study Design	Summary of Important Findings
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 broncogenic 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)

Author(s)	Date	Study Population	Fiber Type	Study Design	Summary of Important Findings
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 × yrs. Among those exposed to greater than 100 mppcf × 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 × 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 × 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, nonexposed 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 POPULATONS

Author(s)	Date	Study Population	Fiber Type	Study Design	Summary of Important Findings
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
Kilviluoto et al.	1960, 1965, 1979	Persons in Central Finland	Anthophyllite 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 nonsmokers. Same trends seen in FEV_1. Obstructive impairment seen in high exposure group. Few trends in diffusing capacity.

Table II-12

SUMMARY OF RESPIRATORY MORBIDITY STUDIES OF ASBESTOS EXPOSED POPULATONS (Continued)

Author(s)	Date	Study Population	Fiber Type	Study Design	Summary of Important Findings
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 nonsmokers 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 rales 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 POPULATONS (Continued)

Author(s)	Date	Study Population	Fiber Type	Study Design	Summary of Important Findings
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_1, FEF_{25-75} and FEV_1/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_1 reduced in those with x-ray changes.
Weiss and Theodas	1978	98 workers age 40 or over in two plants	Chrosotile 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 POPULATONS (Continued)

Author(s)	Date	Study Population	Fiber Type	Study Design	Summary of Important Findings
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_1 and FVC reduced in association with dust and fiber exposures.

Table II-12

SUMMARY OF RESPIRATORY MORBIDITY STUDIES OF ASBESTOS EXPOSED POPULATONS (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 ≥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_1 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_1, 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_1 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_1 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 ≥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 experience 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 11-11). 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 11-12). 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 II-11. Diaphragmatic pleura of 68-year-old ex-construction worker. Numerous dome shaped and flattened, ivory colored plaques are seen over both hemidiaphragms.

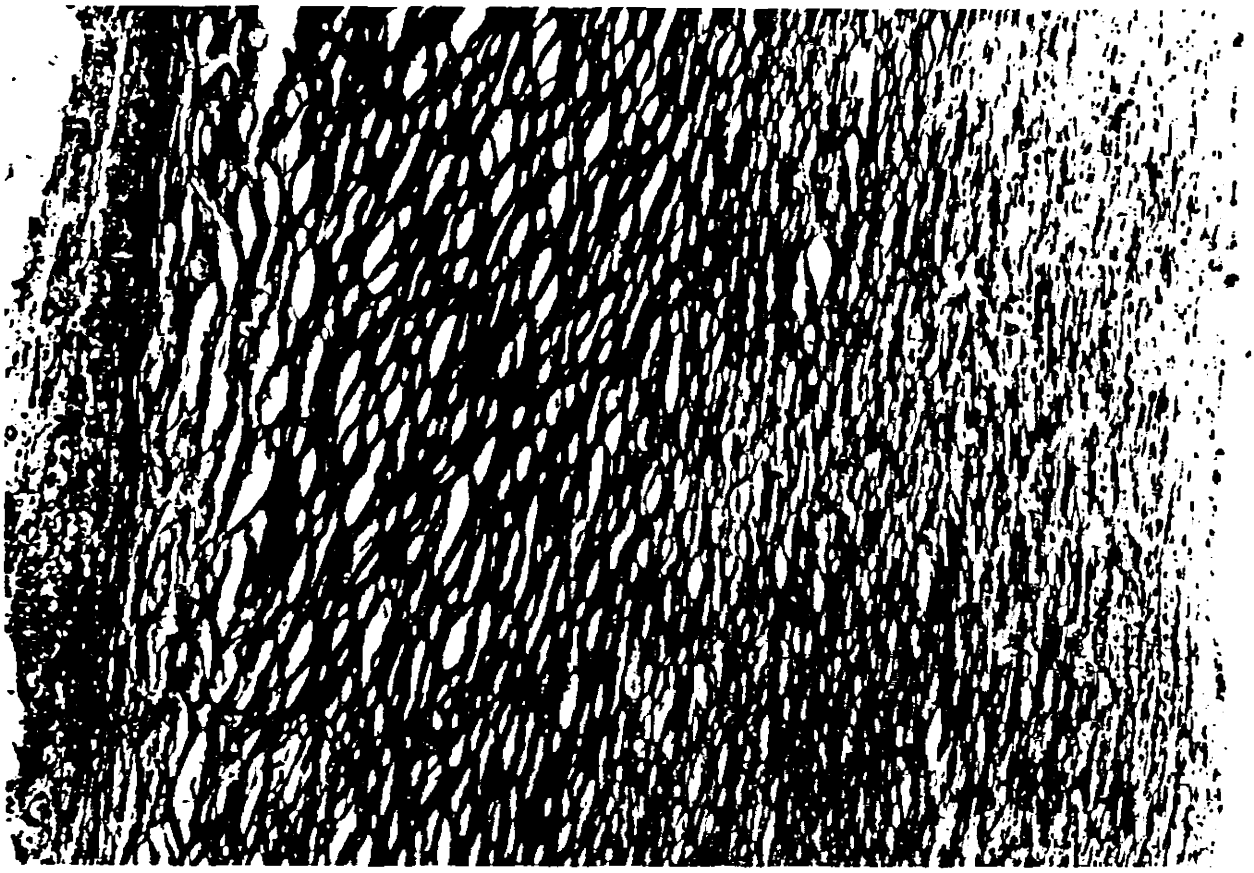

Figure II-12. Histological section of pleural plaque. The plaque is composed of acellular bundles of collagen fibers arranged in a "basket weave" pattern. Hematoxylin and eosin × 64.

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 5μm 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 μm in length) to be

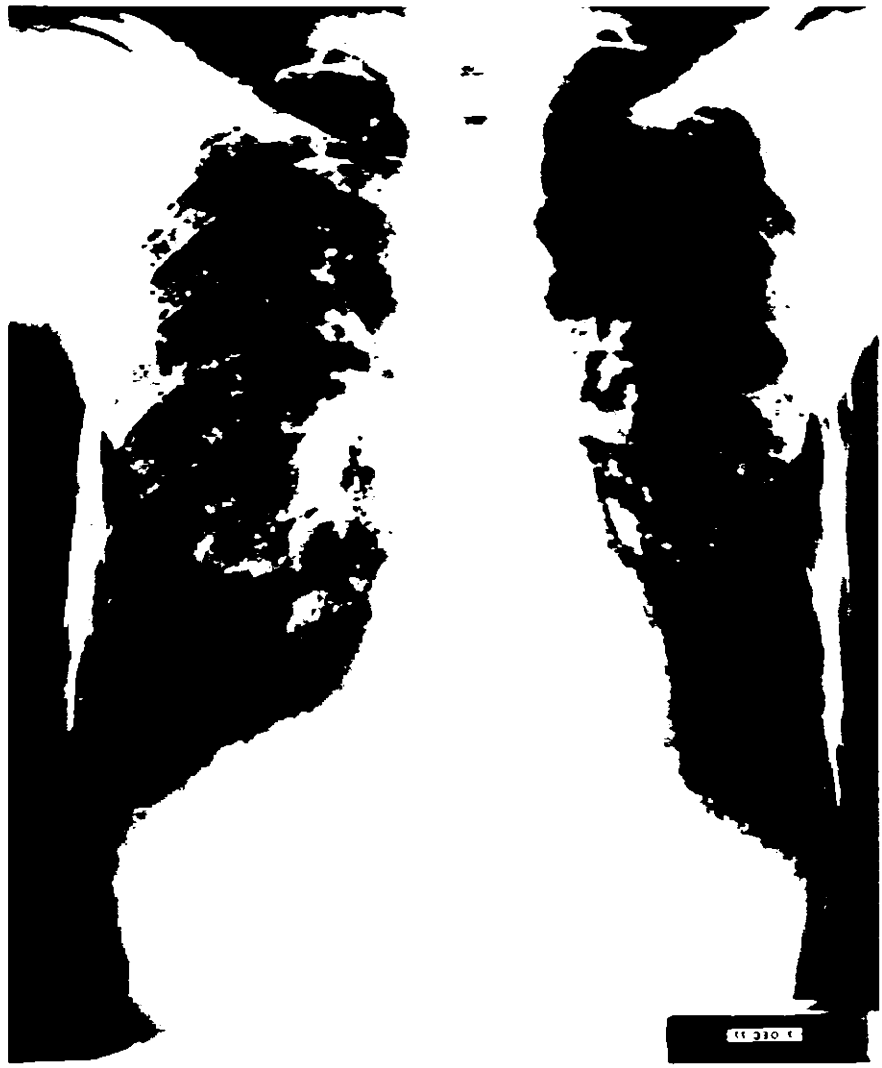

Figure II-13 (B). Roentgenogram showing marked interstitial disease with honeycombing which is most severe in the mid zones.

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

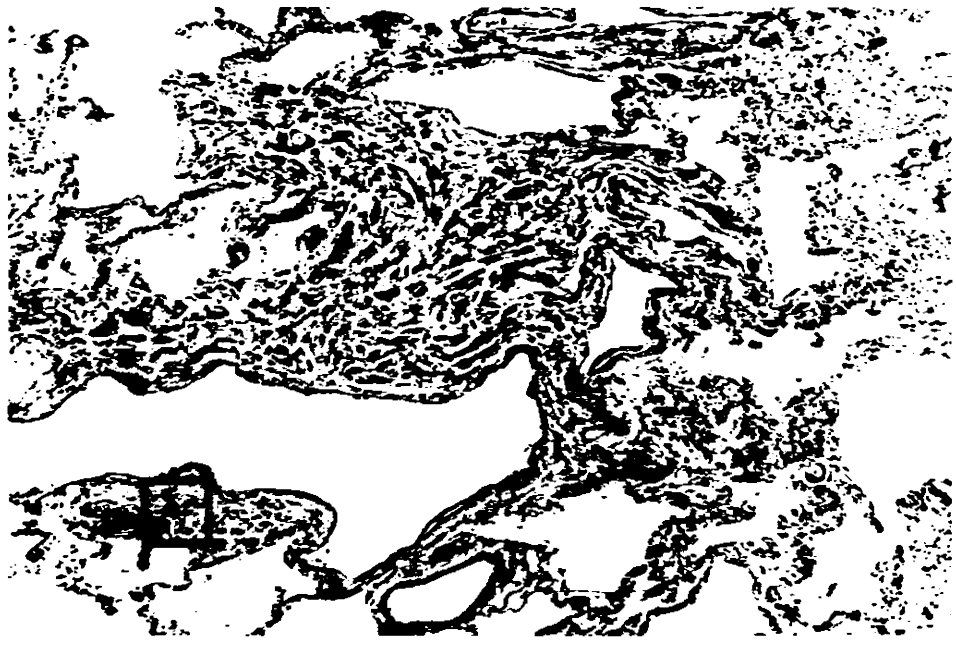

Figure II-14. Section of lung from 68-year-old asbestos insulation worker showing the histological features of mild asbestosis. The lesion is characterized by peribronchiolar fibrosis in which there are numerous asbestos bodies. Inset shows an asbestos body. Hematoxylin and eosin × 100.

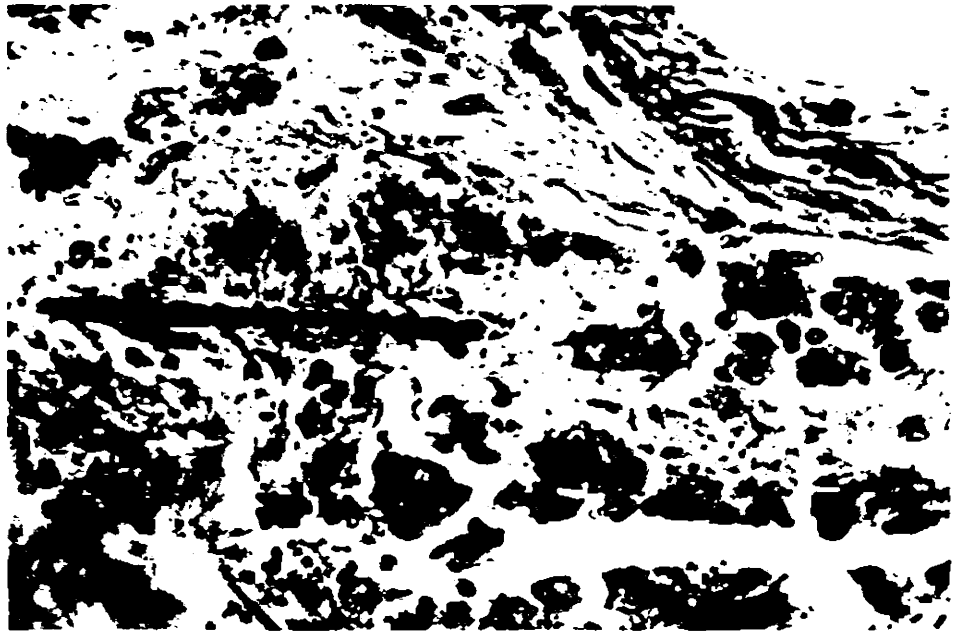

Figure II-14 (Inset).

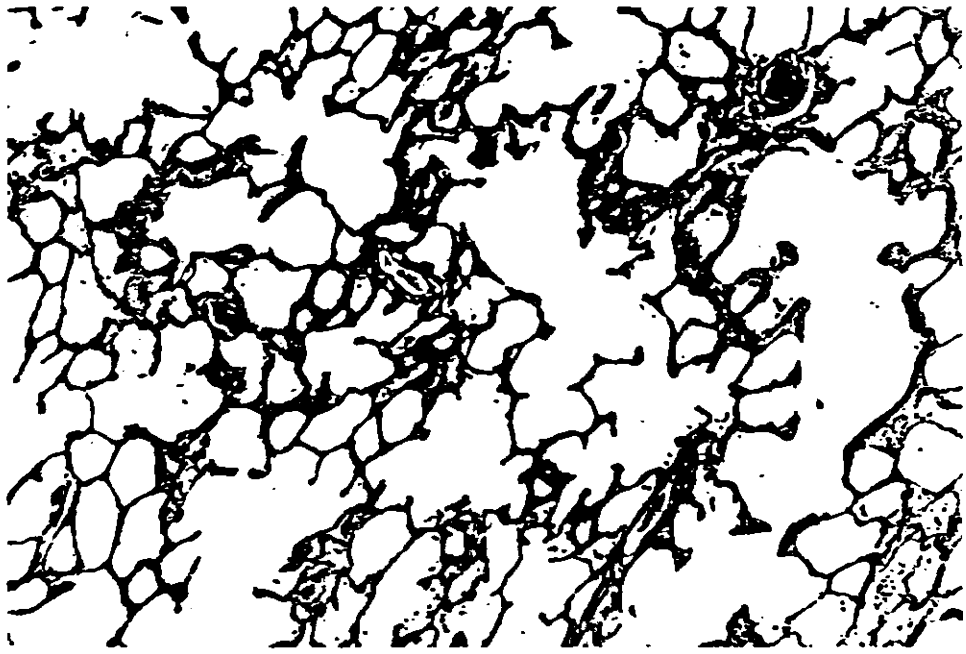

Figure II-15. Section of lung from 48-year-old worker in an asbestos textile mill showing diffuse interstitial and peribronchiolar fibrosis. Hematoxylin and eosin × 40.

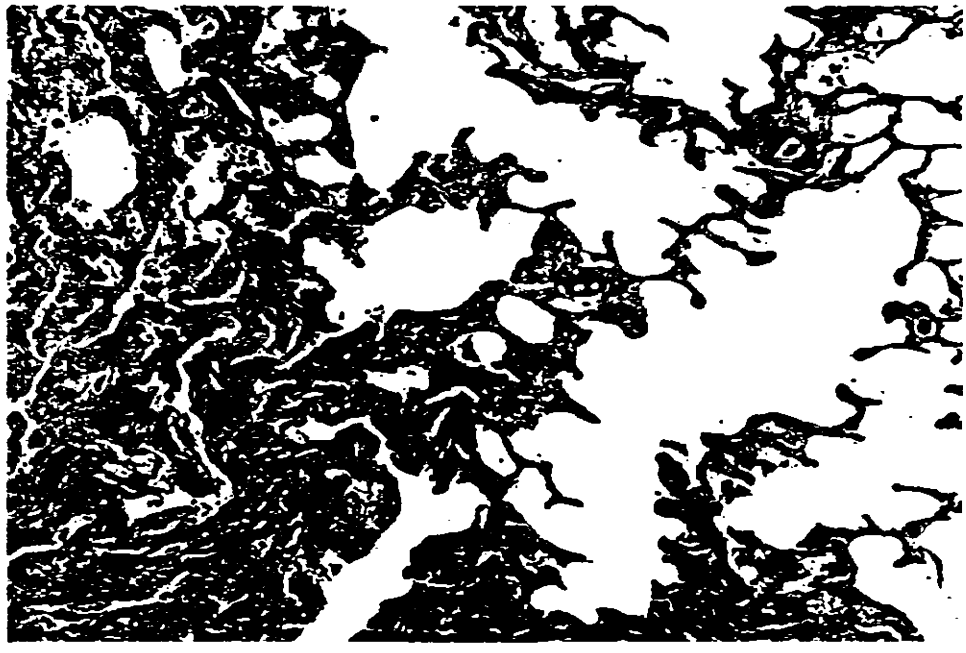

Figure II-16. Section of lung from same case as figure 15 showing interstitial and intraalveolar fibrosis. Hematoxylin and eosin × 40.

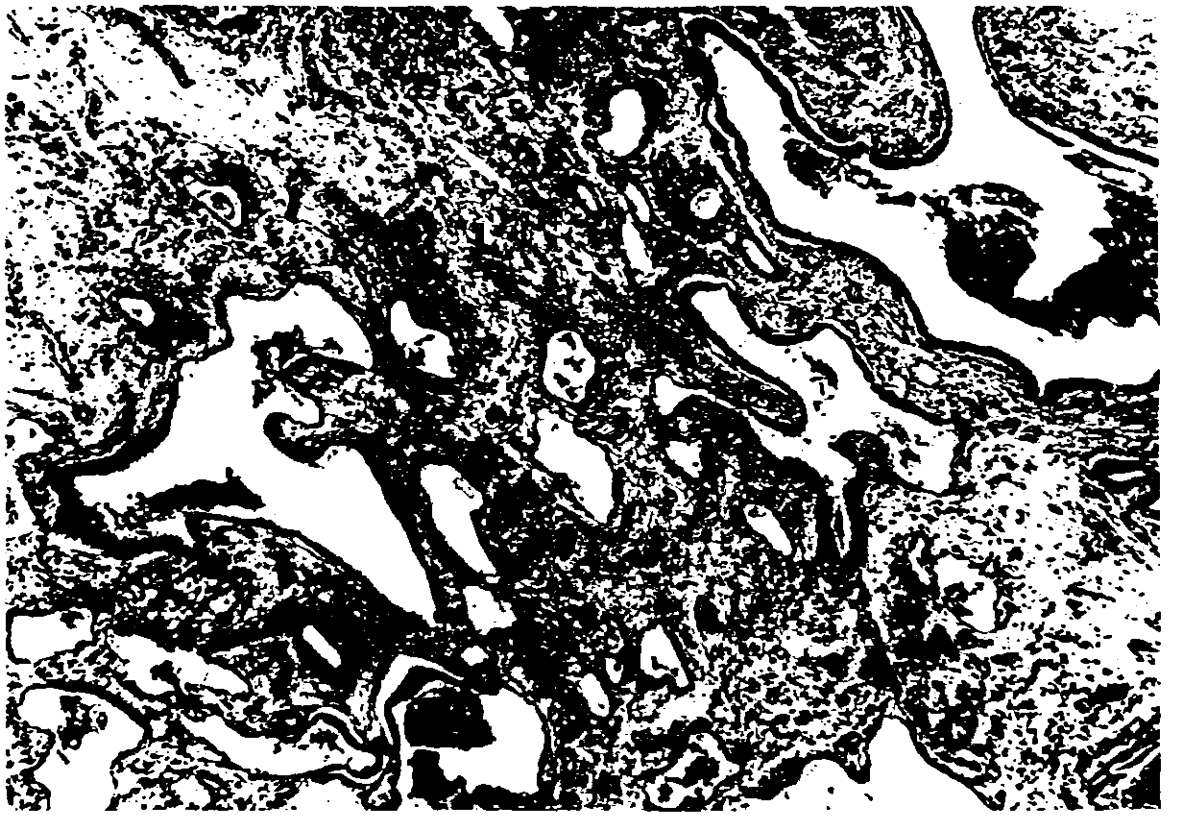

Figure II-17. Section of lung showing honeycombing. The pulmonary architecture has been replaced by thick bands of fibrous tissue outlining cystic spaces. There is a moderate chronic inflammatory cell infiltrate of the parenchyma. Hematoxylin and eosin × 40.

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

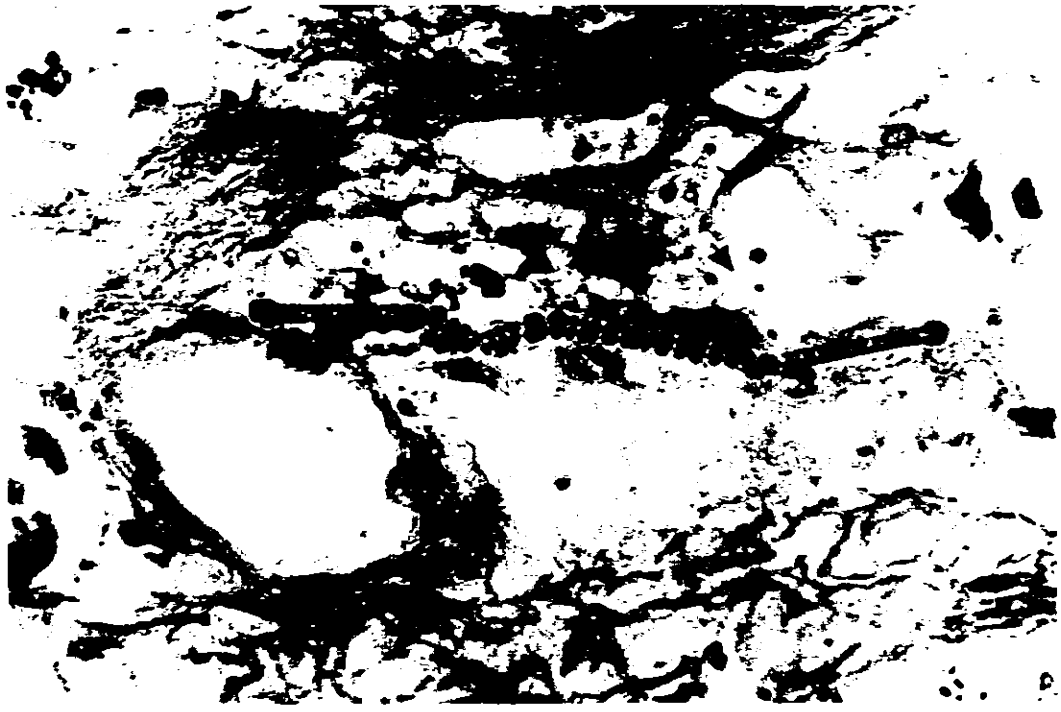

Figure II-18. Asbestos body within an area of fibrosis. The body is composed of a translucent core fiber with a beaded iron-protein coat. An uncoated fiber is also seen (arrow). Hematoxylin and eosin X 600.

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 metastic 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-

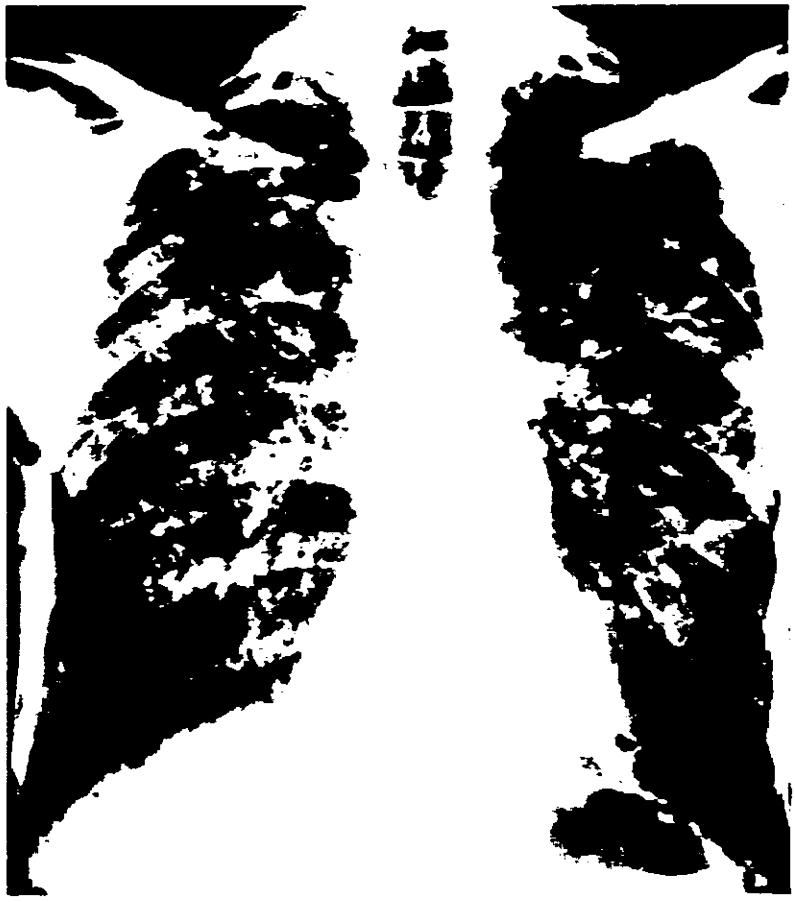

***Figure II-19. Advanced asbestosis—profusion 3/3 with all lung zones involved with s/t opacities.**

*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

***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_{O2})(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, Rodriquez-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 nonsmokers, 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, including 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 5μm (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.

REFERENCES

1. Anderson, H. A., Lilis, R., Daum, S. M., and Selikoff, I. J.: Asbestosis among household contacts of asbestos factory workers. Ann NY Acad Sci *330*:387-399, 1979.
2. Asbestos Dust: Technological feasibility assessment and economic impact analysis of the proposed federal occupational standard. Research Triangle Institute, Contract No. J-9-F-6-0225, U.S. Department of Labor, September, 1978.
3. Becklake, M. R.: Asbestos-related diseases of the lung and other organs: their epidemiology and implications for clinical practice. Am Rev Respir Dis *114*:187-227, 1976.
4. Becklake, M. R., Foumier-Massey, G., Rossiter, C. E., and McDonald, J. C.: Lung function in chrysotile asbestos mine and mill workers of Quebec. Arch Environ Health *24*(6):401-409, 1972.
5. Becklake, M. R., Liddell, F. D. K., Manfreda, J., and McDonald, J. C.: Radiological changes after withdrawal from asbestos exposure. Br J Ind Med *36*:23-28, 1979.
6. Berry, G., Gilson, J. C., Holmes, S., Lewinsohn, H. C., and Roach, S. A.: Asbestosis: A study of dose-response relationships in an asbestos textile factory. Br J Ind Med *36*:98-112, 1979.
7. Boutin, C., Viallat, J. R., and Bellenfant, M.: Radiological features in chrysotile asbestos mine and mill workers in Corsica. In: *Biological Effects of Mineral Fibres,* J. C. Wagner (Ed.), International Agency for Research on Cancer, Scientific Publication No. 30, 507-510, 1980.
8. British Occupational Hygiene Society: Hygiene standards for chrysotile asbestos dust. Ann Occup Hyg *11*:47, 1968.
9. Brown, D. P., Dement, J. M., and Wagoner, J. K.: Mortality patterns among miners and millers occupationally exposed to asbestiform talc. In: *Dusts and Disease,* R. A. Lemen and J. M. Dement (Eds.), Park Forest South, Illinois: Pathotox Publishers, Inc., 317-324, 1979.
10. Churg, A. and Warnock, M. L.: Asbestos and other ferruginous bodies. Their formation and significance. Ann J Path *102*:447-456, 1981.
11. Churg, A., Warnock, M. L., and Green, N.: Analysis of the cores of ferruginous (asbestos) bodies from the general population. II. True asbestos bodies and pseudo-asbestos bodies. Lab Invest *40*:31-38, 1979.
12. Clifton, R. A.: Asbestos: Mineral commodity profiles. U.S. Department of Interior, July, 1979.
13. Cochrane, J. C. and Webster, I.: Mesothelioma in relation to asbestos fibre exposure. South African Med J *12*:279-281, 1978.
14. Cooke, W. E.: Fibrosis of the lungs due to the inhalation of asbestos dust. Br Med J *2*:147, 1924.
15. Corrin, B. and Price, A. B.: Electron

microscopic studies in desquamative interstitial pneumonia associated with asbestos. Thorax *27:*324-331, 1972.
16. Craighead, J. E., Abraham, J. L., Churg, A., Green, F. H. Y., Kleinerman, J., Pratt, P. C., Seemayer, T. A., Vallyathan, V., and Weill, H.: The pathology of asbestos associated diseases of the lungs and pleural cavities. Arch Pathol Lab Med *106:*543-596, 1982.
17. Daley, A. R., Zupko, A. J., and Hebb, J. L.: Technological feasibility and economic impact of OSHA proposed revision to the Asbestos Standard. Weston Environmental Consultants- Designers, March, 1976.
18. Dement, J. M., Harris, R. L., Symons, M. J., and Shy, C.: Estimates of dose-response for respiratory cancer among chrysotile asbestos textile workers. In: *Proceedings of the 5th International Conference on Inhaled Particles and Vapours*, BOHS, 1980.
19. Dement, J. M. and Zumwalde, R. D.: Occupational exposures to talcs containing asbestiform minerals. In: *Dust and Disease*. R. Lemen and J. M. Dement (Eds.). Park Forest South, Illinois: Pathotox Publishers, 278-305, 1979.
20. Doll, R.: Mortality from lung cancer in asbestos workers. Br J Ind Med *12:*81-86, 1955.
21. Donnelly, J. A.: Pulmonary asbestosis. Am J Pub Health *23:*1275-1281, 1933.
22. Donnelly, J. A.: Pulmonary asbestosis: Incidence and prognosis. J Ind Hyg *18:*222-278, 1936.
23. Dreessen, W. C., DallaValle, J. M., Edwards, T. J., Miller, J. W., Sayers, R. R., Eason, H. F., and Price, M. F.: A study of asbestosis in the asbestos textile industry. Public Health Bulletin 217, 1938.
24. Edge, J. R.: Asbestos related disease in Barrow-in-Furness. Environ Res *11:* 244-247, 1976.
25. Edge, J. R.: Incidence of bronchial carcinoma in shipyard workers with pleural plaques. Ann NY Acad Sci *330:*289-294, 1979.
26. Egbert, D. S. and Geiger, A. J.: Pulmonary asbestosis and carcinoma. Report of a case with necropsy findings. Am Rev of Tuberculosis *34:*143-146, 1936.
27. Elmes, P. C. and Simpson, M. J. C.: Insulation workers in Belfast. A further study of mortality due to asbestos exposure (1940-1975). Br J Ind Med *34*(3):174-180, 1977.
28. Elmes, P. C. and Simpson, M. J. C.: Insulation workers in Belfast, III, mortality 1940-1966. Br J Ind Med *28:*226-236, 1971.
29. Estimates of the fraction of cancer in the United States related to occupational exposures. Report of National Cancer Institute, NIEHS, NIOSH Work Group, Sept. 15, 1978.
30. Fournier-Massey, G. C. and Becklake, M. R.: Pulmonary function profiles in Quebec asbestos workers. Bull Physiopath Resp *11:*429-445, 1975.
31. Gamble, J., Fellner, W., and DiMeo, M. J.: Respiratory morbidity among miners and millers of asbestiform talc. In: *Dusts and Disease*, R. A. Lemen and J. M. Dement (Eds.). Park Forest South, Illinois: Pathotox Publishers, Inc., 307-316, 1979.
32. Gillam, J. D., Dement, J. M., Lemen, R. A., Wagoner, J. K., Archer, V. E., and Blejer, H. P.: Mortality patterns among hard rock gold miners exposed to an asbestiform mineral. Ann NY Acad Sci *271:*336-344, 1976.
33. Gloyne, S. R.: A case of oat cell carcinoma of the lung occurring in asbestosis. Tubercle *18:*100-101, 1936.
34. Gloyne, S. R.: Two cases of squamous carcinoma of the lung occurring in asbestosis. Tubercle *17:*5-10, 1935.
35. Greenberg, S. D., Hurst, G. A., Christianson, S. C., Matlage, W. J., Hurst, I. J., and Marbry, L. C.: Pulmonary cytopathology of former asbestos workers. Am J Clin Pathol *66:*815-822, 1976.
36. Gregor, A., Parkes, R. W., duBois, R., and Turner-Warwick, M.: Radiographic progression of asbestosis: Preliminary Report. Ann NY Acad Sci *330:*147-156, 1979.
37. Gross, P., DeTreville, T. P., Cralley, L. J., and Davis, J. M. C.: Pulmonary fer-

ruginous bodies: Development in response to filamentous dusts and a method of isolation and concentration. Arch Pathol *85:*539-546, 1968.
38. Hammond, E. C., Selikoff, I. J., and Seidman, H.: Asbestos, exposure, cigarette smoking, and death rates. Ann NY Acad Sci *330:*473-490, 1979.
39. Harington, J.S.: Asbestos and mesothelioma in man. Nature (London) *232:* 54-55, 1971.
40. Hourihane, D. O'B., Lessof, L., and Richardson, P. C.: Hyaline and calcified pleural plaques as an index of exposure to asbestos. A study of radiological and pathological features of 100 cases with a consideration of epidemiology. Br Med J *1:*1069-1074, 1966.
41. Hourihane, D. O'B. and McCaughey, W. T. E.: Pathological aspects of asbestosis. Postgrad Med J *42:*613-622, 1966.
42. Hueper, W. C.: *Recent Results in Cancer Research.* Berlin: Springer Verlag, p. 44, 1956.
43. International Agency for Research on Cancer: IARC Monographs on the evaluation of carcinogenic risk of chemicals to man. Asbestos *14*, 1977.
44. International Labour Office. ILO-1980 International Classification of Radiographs of the Pneumoconioses, Occupational Safety and Health Series XX, International Labour Office, Geneva, Switzerland.
45. Jones, J. S. P., Pooley, F. D., Clark, N. J., Owen, W. G., Roberts, G. H., Smith, P., Wagner, J. C., and Berry G.: The pathology and mineral content of lungs in cases of mesothelioma in the United Kingdom in 1976. In: *Biological Effects of Mineral Fibers,* Vol. 1. J. C. Wagner, Ed., World Health Organization, IARC Scientific Publication No. 30, pp. 187-198, 1980.
46. Jones, J. S. P., Pooley, F. D., and Smith, P. G.: Factory populations exposed to crocidolite, asbestos—A continuing survey. International Agency for Research on Cancer, Inserm Symposium Series 52. IARC Scientific Publication No. 13, 117, 1976.
47. Jones, J. S. P., Smith, P. G., Pooley, F. D., et al.: The consequences of exposure to asbestos dust in a wartime gas-mask factory. In: Biological Effects of Mineral Fibres. J. C. Wagner (Ed.). International Agency for Research on Cancer, Scientific Publication No. 30, 637-653, 1980.
48. Jones, R. N., Diem, J. E., Glindmeyer, H., Weill, H., and Gilson, J. C.: Progression of asbestos radiographic abnormalities: Relationships to estimates of dust exposure and annual decline in lung function. In: *Biological Effects of Mineral Fibres*, J. C. Wagner, Ed., International Agency for Research on Cancer, Scientific Publication No. 30, 537-543, 1980.
49. Kannerstein, M. and Churg, J.: Pathology of carcinoma of the lung associated with asbestos exposure. Cancer *30:*14-21, 1972.
50. Kannerstein, M., Churg, J., and McCaughey, W. T. E.: Functions of mesothelioma panels. Ann NY Acad Sci *330:*433-439, 1979.
51. Kilviluoto, R.: Pleural calcification as a roentgenologic sign of non-occupational endemic anthophyllite-asbestos. ACTA Radiologica (Supplement) *194:*1-67, 1960.
52. Kleinfeld, M., Giel, C. P., Majeraonowski, J. F., and Messite, J.: Talc pneumoconiosis: a report of six patients with post mortem findings. Ind Hyg Rev. *6:* 5-29, 1964.
53. Kleinfeld, M., Messite, J., and Kooyman, O.: Mortality among talc miners and millers in New York State. Arch. Environ. Health *14:*663-672, 1967.
54. Kleinfeld, M., Messite, J., Kooyman, O., and Shapiro, J.: Pulmonary ventilatory function in talcosis of the lung. Ind Hyg Rev *7:*14-23, 1965.
55. Kleinfeld, M., Messite, J., and Langer, A. J.: A study of workers exposed to asbestiform minerals in commercial talc manufacture. Env Res *6:*132-143, 1973.
56. Kleinfeld, M. J., Messite, J., Shapiro, J., Kooyman, O., and Swencicki, R.: Lung function in talc workers: a comparative physiologic study of workers exposed to fibrous and granular talc dusts. Ind Hyg Rev *7:*3-13, 1965.
57. Kleinfeld, M., Messite, J., Swencicki, R., and Sarfaly, J.: Lung function changes in talc pneumoconiosis. JOM *7:*12-17,

1965.
58. Kleinfeld, M., Messite, J., and Zaki, H.: Mortality experiences among talc workers. A follow-up study. JOM *16:*345-349, 1974.
59. Knox, J. F., Doll, R. S., and Hill, I. D.: Cohort analysis of changes in incidence of bronchial carcinoma in a textile asbestos factory. Ann NY Acad Sci *132:*526-535, 1965.
60. Knox, J. F., Holmes, S., Doll, R., and Hill, I. D.: Mortality from lung cancer and other causes among workers in an asbestos textile factory. Br J Ind Med *25:*293-303, 1968.
61. Kogan, F. M., Guselnikova, N. A., and Gulevskaya, H. R.: Cancer mortality rate among workers in the asbestos industry in the Urals. Gig i Sanit *37*(7):29, 1972.
62. Kuhn, C. and Kuo, T. T.: Cytoplasmic hyalin in asbestosis. A reaction of injured alveolar epithelium. Arch Pathol *95:*190-194, 1973.
63. Kuschner, M. and Wright, G.: The effects of intratracheal instillation of glass fiber of varying sizes in guinea pigs. In Proceedings of a Symposium: Occupational Exposure to Fibrous Glass. U.S. Department of Health, Education, and Welfare, National Institute for Occupational Safety and Health, HEW Publication No. (NIOSH) 76-151, 1976.
64. Langer, A. M., Maggiore, C. M., Nicholson, W. J., Rohl, A. N., Rubin, I. B., and Selikoff, I. J.: The contamination of Lake Superior with amphibole gangue minerals. Ann NY Acad Sci *330:*549-572, 1979.
65. LeBouffant, L.: Investigations and analysis of asbestos fibers and accompanying minerals in biological materials. Environ Health Persp *9:*149-153, 1974.
66. Lee, D. H. and Selikoff, I. J.: Historical background to the asbestos problem. Environ Res *18:*300-314, 1979.
67. Lewinsohn, H. C.: The medical surveillance of asbestos workers. Roy Soc Health J *92*(2):69-77, 1972.
68. Liddell, D., Eyssen, G., Thomas, D., and McDonald, C.: Radiological changes over 20 years in relation to chrysotile exposure in Quebec. In: *Inhaled Particles IV*, W. H. Walton (ed.). Oxford: Pergamon Press, 799-812, 1977.
69. Liddell, F. D. K. and McDonald, J. C.: Radiological findings as predictors of mortality in Quebec asbestos workers. Br J Ind Med *37:*257-267, 1980.
70. Lynch, K. M. and Smith, W. A.: Pulmonary asbestosis I. Report of bronchial carcinoma and epithelial metaplasia. Am J Cancer *36:*567-572, 1931.
71. Lynch, K. M. and Smith, W. A.: Pulmonary asbestosis III. Carcinoma of the lung in asbesto-silicosis. Am J Cancer *14:*56-64, 1935.
72. Maldoon, B. C. and Turner-Warwick, M.: Lung function studies in asbestos workers. Br J Dis Chest *66:*121-132, 1972.
73. Mancuso, T. F. and Coulter, E. J.: Methodology in industrial health studies. The cohort approach, with special reference to an asbestos company. Arch Environ Health *6:*210-226, 1963.
74. Martinischnig, K. M., Newell, D. J., Barnsley, W. C., Cowan, W. K., Feinmann, E. L., and Oliver, E.: Unsuspected exposure to asbestos and bronchogenic carcinoma. Br Med J *1*(6063):746-749, 1977.
75. McDonald, A. D. and McDonald, J. C.: Mesothelioma after crocidolite exposure during gas mask manufacture. Environ Res *17:*340, 1978.
76. McDonald, J. C., Becklake, M. R., Gibbs, G. W., McDonald, A. D., and Rossiter, C. E.: The health of chrysotile asbestos mine and mill workers of Quebec. Arch Environ Health *28(2):*61-68, 1974.
77. McDonald, J. C., Becklake, M. R., Fournier-Massey, G., and Rossiter, C. E.: Respiratory symptoms in chrysotile asbestos mine and mill workers of Quebec. Arch Environ Health *24*(5):358-363, 1972.
78. McDonald, J. C., Gibbs, G. W., Liddell, F. V. D., and McDonald, A. D.: Mortality after long exposure to cummingtonite grunerite. Am Rev Respir Dis *118:*271-277, 1978.
79. McDonald, J. C. and Liddell, F. D. K.: Mortality in Canadian miners and millers exposed to chrysotile. Ann NY Acad Sci

330:1-9, 1979.
80. McDonald, J. C., Liddell, F. D. K., Gibbs, G. W., Eyssen, G. E., and McDonald, A. D.: Dust exposure and mortality in chrysotile mining, 1910-75. Br J Ind Med *37*:11-24, 1980.
81. McDonald, J. C., McDonald, A. D., Gibbs, G. W., Siemiatycki, J., and Rossiter, C. E.: Mortality in the chrysotile asbestos mines and mills of Quebec. Arch Environ Health *22*:677-686, 1971.
82. Merchant, J. A., Klonda, P. T., Soutar, C. A., Parkes, W. R., Lawler, S. D., and Turner-Warwick, M.: The HLA system in asbestos workers. Br Med J *189*:91, 1975.
83. Merewether, E. R. A.: In: Annual Report of the Chief Inspector of Factories for the year 1947. London: Her Majesty's Stationery Office. pp 56-58, January, 1947.
84. Merewether, E. R. A. and Price, C. W.: Report on the effects of asbestos dust on the lungs and dust suppression in the asbestos industry. I. Occurrence of pulmonary fibrosis and other pulmonary affections in asbestos workers. II. Processes giving rise to dust and methods for its suppression. London: Her Majesty's Stationery Office, 1930.
85. Meurman, L. O.: Pleural fibrocalcific plaques and asbestos exposure. Environ Res *2*:30-46, 1968.
86. Meurman, L. O., Kilviluoto, R., and Hakama, M.: Combined effects of asbestos exposure and tobacco smoking on Finnish anthophyllite miners and millers. Ann NY Acad Sci *330*:491-495, 1979.
87. Meurman, L. O., Kilviluoto, R., and Hakama, M.: Mortality and morbidity among the working population of anthophyllite asbestos miners in Finland. Br J Ind Med *31*(2):105-112, 1974.
88. Mills, R. G.: Pulmonary asbestosis: Report of a case. Minn Med J *13*:495-499, 1930.
89. Morgan, R. H.: Proficiency examination of physicians for classifying pneumoconiosis chest films. Am J Roentgenol *132*:803-808, 1979.
90. Morgan, R. H., Donner, M. W., Gayler, B. W., et al.: Decision processes and observer errors in the diagnosis of pneumoconiosis by chest roentgenography. Am J Roentgenol *117*: 757-764, 1973.
91. Morgan, W. K. C.: Rheumatoid pneumoconiosis in association with asbestosis. Thorax *19*:433-435, 1964.
92. Murphy, R. L., Ferris, B., and Burgess, W. A.: Effects of low concentrations of asbestos. Clinical, environmental, radiologic and epidemiologic observations in shipyard pipe coverers and controls. N Eng J Med *285*(23):1271-1278, 1971.
93. Murphy, R. L. H., Gaensler, E. A., Ferris, B. G., et al.: Diagnosis of asbestosis. Observations from a longitudinal survey of shipyard pipe coverers. Am J Med *65*(3):488-498, 1978.
94. Murphy, R. L. H., Gaensler, E. A., Redding, R. A., Belleau, R., Kellan, P. J., Smith, A. A., and Ferris, B. D., Jr.: Low exposure to asbestos. Gas exchange in ship pipe covers and controls. Arch Environ Hlth *25*:253-264, 1972.
95. Murray, M.: As reported by Lee, D. H. K. and Selikoff, I. J.: Historical background to the asbestos problem. Environ Res *18*:300-314, 1979.
96. Newhouse, M. L.: A study of the mortality of workers in an asbestos factory. Br J Ind Med *26*:294-301, 1969.
97. Newhouse, M. L.: Cancer among workers in the asbestos textile industry. In: Proceedings of the Conference on Biological Effects of Asbestos. P. Bogovski, J. C. Gibson, V. Timbrell, and J. C. Wagner, (Eds.). International Agency for Research on Cancer, 203-208, 1973.
98. Newhouse, M. L., Berry, G., Wagner, J. C., and Turok, M. E.: A study of the mortality of female asbestos workers. Br J Ind Med *29*(2):134-141, 1972.
99. Nicholson, W. J., Selikoff, I. J., Seidman, H., Lilis, R., and Formby, P.: Longterm mortality of chrysotile miners and millers in the Thetford Mines, Quebec. Ann NY Acad Sci *330*:11-21, 1979.
100. Niewoehner, D. F., Kleinerman, J., and Rice, D. B.: Pathologic changes in the peripheral airways of young cigarette smokers. N Eng J Med *291*:755-758, 1974.

101. Nishimura, M. and Sera, Y.: Lung cancer occurring with pneumoconiosis and histological study of the bronchial epithelium. Kinkishuo-Byoin Nat Sanatorium, Iryo, Japan, *23*(2):194-203, 1969.
102. Nordmann, M.: The occupational basis for asbestosis in asbestos workers. J Ind Hyg *20:*184, 1938.
103. Noro, L.: Occupational and non-occupational asbestosis in Finland. Am Ind Hyg Assoc J *29:*195-201, 1968.
104. Oliver, T.: Clinical aspects of pulmonary asbestosis. Br Med J *2:*1026, 1927.
105. Pancoast, H. K., Miller, T. C., and Landis, H. R. M.: A roentgenologic study of the effects of dust inhalation upon the lungs. Trans Assoc of Am Physicians *32:*97-108, 1917.
106. Pancoast, H. K. and Pendergrass, F. P.: Pneumoconiosis: A Roentgenological Study. New York: Paul B Hoeber Inc., 1926.
107. Pano, F. W., Patton, J. R., and Hobbs, A. A.: Pneumoconiosis in the talc industry. Am J Roent and Rad Ther *47:*507-524, 1942.
108. Peto, J.: Dose-response relationships for asbestos-related disease: Implication for hygiene standards, Part II, Mortality. Ann NY Acad Sci *330:*195-204, 1979.
109. Peto, J., Doll, R., Howard, S. V., Kinlen, L. J., and Lewinsohn, H. C.: Mortality study among workers in an English asbestos factory. Br J Ind Med *34*(3):169-173, 1977.
110. Pott, F. and Friedrichs, K. H.: Tumors in rats after intraperitoneal injection of fibrous dust. Naturwissenschaften *59:*318, 1972 (Ger.).
111. Pott, F., Friedrichs, K. H., and Huth, F.: Ergebnisse aus tierversuchens zur Kanzerogenen Wirkung Faserformiger Stauke undihre deuting in himblick auf die tumorentstehung beim menschen. Zbl Bakt Hyg I Abt Orig B *162:*467-505, 1976.
112. Roberts, G. F.: The pathology of parietal pleural plaques. J Clin Pathol *24:* 348-353, 1971.
113. Robinson, C. F., Lemen, R. A., and Wagoner, J. K.: Mortality patterns, 1940-1975, among workers employed in an asbestos textile fiction and packing products manufacturing facility. In: *Dusts and Diseases*, R. A. Lemen and J. M. Dement (Eds.), Park Forest South Illinois: Pathotox Publishers, Inc., 131-143, 1979.
114. Rodriguez-Roisen, R., Merchant, J. A., Cochrane, G. M., Hickey, B. P. H., Turner-Warwick, M., and Clarke, T. T.: Maximal expiratory flow volume curves in workers exposed to asbestos. Respiration *39:*158-165, 1980.
115. Rohl, A. N., Langer, A. M. and Selikoff, I. J.: Environmental asbestos pollution related to use of quarried serpentine rock. Science *196:*1319-1322, 1977.
116. Rubino, G. F., Newhouse, M., Murray, G., Scansetti, G., Piolatto, G., and Aresini, G.: Radiologic changes after cessation of exposure among chrysotile asbestos miners in Italy. Ann NY Acad Sci *330:*157-161, 1979.
117. Rubino, G. F., Piolatto, G., Newhouse, M. L., et al.: Mortality of chrysotile asbestos workers at the Balangero Mine, Northern Italy. Br J Ind Med *36:*187, 1979.
118. Seidman, H. R., Lilis, R., and Selikoff, I. J.: Short-term asbestos exposure and delayed cancer risk. In: Third International Symposium on Detection and Prevention of Cancer. H. E. Niebergo (Ed.), New York: Marcel Dekker, Inc., 943-960, 1977.
119. Seidman, H., Selikoff, I. J., and Hammond, E. C.: Short-term asbestos exposure and long-term observation. Ann NY Acad Sci *330:*61-89, 1979.
120. Seiler, H. E. and Gilmour, M. D.: A case of pulmonary asbestosis. Br Med J *1:*1112, 1931.
121. Selikoff, I. J.: Clinical survey of chrysotile asbestos miners and millers in Baie Verte, Newfoundland-1976, Report to the National Institute of Environmental Health Sciences, December 22, 1977.
122. Selikoff, I. J.: The occurrence of pleural calcification among asbestos insulation workers. Ann NY Acad Sci *132:*351-367, 1965.
123. Selikoff, I. J., Churg, J., and Hammond, E. C.: Asbestos exposure and neoplasia. JAMA *188*(1):142-146, 1964.
124. Selikoff, I. J., Churg, J., and Hammond,

E. C.: The occurrence of asbestos among insulation workers in the United States. Ann NY Acad Sci *132*:139-155, 1965.
125. Selikoff, I. J., Hammond, E. C., and Churg, J.: Carcinogenicity of amosite asbestos. Arch Environ Health *25*(3):183-186, 1972.
126. Selikoff, I. J., Hammond, E. C., and Seidman, H.: Cancer risk of insulation workers in the United States. In: Proceeding of the Conference on Biological Effects of Asbestos. P. Bogovoki, J. C. Gilson, V. Timbrell, and J. C. Wagner (Eds.). International Agency for Research on Cancer, 209-216, 1973.
127. Selikoff, I. J., Hammond, E. C., and Seidman, H.: Mortality experience of insulation workers in the United States and Canada, 1943-1976. Ann NY Acad Sci *330*:91-116, 1979.
128. Selikoff, I. J. and Lee, D. H.: *Asbestos and Disease*. New York: Academic Press, Inc., 1978.
129. Selikoff, I. J., Seidman, H., and Hammond, E. C.: Mortality effect of cigarette smoking among amosite asbestos factory workers. J Natl Cancer Inst *65*(3):507-513, 1980.
130. Sheers, G.: Asbestos-associated disease in employees of Devonport Dockyard. Ann NY Acad Sci *330*:281-287, 1979.
131. Schull, J. R.: Asbestosis. A roentgenologic review of 71 cases. Radiology *27*:279-292, 1936.
132. Siegal, W., Smith, A. R., and Greenburg, L.: The dust hazard in tremolite mining, including roentgenological findings in talc workers. Am J Roentgenology and Radium Ther *49*:11-29, 1942.
133. Siegal, W., Smith, A. R., and Greenburg, L.: Study of talc miners and millers. Industrial Bull *22*:3-12, 1943.
134. Simson, F. W.: Pulmonary asbestosis in Africa. Br Med J *1*:885, 1928.
135. Simson, F. W. and Strachan, A. S.: Asbestos bodies in sputum; a study of specimens from 50 workers in asbestos mill. J Path and Bact *34*:1-4, 1931.
136. Sluis-Cremer, G. K.: The relationship between asbestosis and bronchial cancer. Chest *78*:380-381, 1980.
137. Smith, M. J., and Naylor, B.: A method for extracting ferruginous bodies from sputum and pulmonary tissue. Am J Clin Pathol *58*:250-255, 1972.
138. Spencer, H.: The pneumoconioses and other occupational lung diseases. In: *Pathology of the Lung*, Vol. 1, 1977. New York: Pergamon Press, pp. 371-462, 1977.
139. Stanton, M. F., Layard, M., Miller, M., and Kent, E.: Carcinogenicity of fibrous glass: Pleural response in the rat in relation to fiber dimension. J Natl Cancer Inst *58*:587-603, 1977.
140. Stewart, I. M., Putscher, R. E., Hermecki, H. J., and Shimps, R. J.: Asbestos fibers in discharges from selected mining and milling activities. Final Report. EPA Contract No. 68-01-2690, January, 1977.
141. Stewart, M. J.: The immediate diagnosis of pulmonary asbestosis at necropsy. Br Med J *2*:509, 1928.
142. Stansfield, D. and Edge, J. R.: Circulatory rheumatoid factor and antinuclear antibodies in shipyard asbestos workers with pleural plaques. Br J Dis Chest *68*:166-170, 1974.
143. Suzuki, Y. and Churg, J.: Structure and development of the asbestos body. Am J Pathol *55*:79-107, 1969.
144. Taivanen, O., Salmirolli, M., G'Molnar, G.: Pulmonary asbestosis and autoimmunity. Br Med J *1*:691-692, 1976.
145. Thompson, J. G.: The pathogenesis of pleural plaques. In: *Pneumoconiosis, Proceedings of the International Conference, Johannesburg, 1969*, H. A. Shapiro, Ed., Cape Town: Oxford University Press, pp. 138-141, 1969.
146. Turner-Warwick, M.: HLA phenotypes in asbestos workers. Br J Dis Chest *73*:243-244, 1979.
147. Turner-Warwick, M. and Parkes, W. R.: Circulatory rheumatoid and antinuclear factors in asbestos workers. Br Med J *111*:492-495, 1970.
148. U.S. Public Health Service, National Institute for Occupational Safety and Health. Criteria for a Recommended Standard: Occupational Exposure to Asbestos. Publication No. HSM 72-10267, 1972.
149. U.S. Public Health Service, National Institute for Occupational Safety and Health. Workplace exposure to

Asbestos: Review and Recommendations. Publication No. DHHS (NIOSH) 81-103, 1980.
150. Vallyathan, N. V., Green, F. H. Y., and Craighead, J. E.: Recent advances in the study of mineral pneumoconiosis. Pathol Annual *15:*77-104, 1980.
151. Wagner, J. C., Gilson, J. C., Berry, G., and Timbrell, V.: Epidemiology of asbestos cancers. Br Med Bull *27:*71-76, 1971.
152. Wagner, J. C., Sleggs, C. A., and Marchand, P.: Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. Br J Ind Med *17:*260-271, 1960.
153. Wang, N. S., Haung, S. N., and Gold, P.: Absence of carcinoembryonic antigen-like material in mesothelioma. An immunohistochemical differentiation from other lung cancer. Cancer *44:*937-943, 1979.
154. Waxler, B., Eisenstein, R., and Battifora, H.: Electrophoresis of tissue glycosaminoglycans as an aid in the diagnosis of mesotheliomas. Cancer *44:*221-227, 1979.
155. Webster, I.: Malignancy in relation to crocidolite and amosite. In: Proceedings of the Conference on Biological Effects of Asbestos. P. Bogovski, J. C. Gilson, V. Timbrell, J. C. Wagner (Eds.). International Agency for Research on Cancer, 195-198, 1973.
156. Weill, H., Hughes, J., and Waggenpack, C.: Influence of dose and fiber type on respiratory malignancy risks in asbestos cement manufacturing. Am Rev Respir Dis *120:*345-354, 1979.
157. Weill, H., Waggenpack, C., Bailey, W., Ziskind, M., and Rossiter C.: Radiographic and physiologic patterns among workers engaged in manufacture of asbestos cement products. A Preliminary Report. JOM *15*(3):248-252, 1973.
158. Weill, H., Ziskind, M. M., and Waggenpack, C.: Lung function consequences of dust exposure in asbestos cement manufacturing plants. Arch Environ Health *30*(2):88-97, 1975.
159. Weiss, W.: Cigarette smoking, asbestos, and pulmonary fibrosis. Am Rev Respir Dis *104:*223-227, 1971.
160. Weiss, W.: Mortality of a cohort exposed to chrysotile asbestos. JOM *19*(11):737-740, 1977.
161. Weiss, W. and Theodas, P. A.: Pleuropulmonary disease among asbestos workers in relation to smoking and type of exposure. JOM *20:*341-345, 1978.
162. Whitwell, F., Newhouse, M. L., and Bennett, D. R.: A study of the histological cell types of lung cancer in workers suffering from asbestosis in the United Kingdom. Br J Ind Med *31:*298-303, 1974.
163. Whitwell, F., Scott, J., and Grimshaw, M.: Relationship between occupations and asbestos-fibre content of the lungs in patients with pleural mesothelioma, lung cancer, and other diseases. Thorax *32:*377-386, 1977.
164. Wood, W. B. and Gloyne, S. R.: Pulmonary asbestosis. Lancet *1:*445, 1930.
165. Wyers, H.: Thesis presented to the University of Glasgow for the degree of Doctor of Medicine, 1946.

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 sheetmetal 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 chrysotile 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. 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.

REFERENCES

1. Agricola, A.C.: 1557, "De Re Metallica," Basel. Cited by Hueper, 1942, and as translated by Hoover, H.C. and Hoover, L.C. Dover Publications, New York, 1950.
2. Alwens, W. and Jonas, W.: [Chrome Lung Cancer] ACTA Union Internationale Centre Cancer *3:*103-114, 1938.
3. Archer, V.E., Gillam, J.D., and Wagoner, J.K.: Respiratory disease mortality among uranium miners. N.Y. Acad. Sci. *271:*280-293, 1976.
4. Archer, V.E., Wagoner, J.K., and Lundin, F.E.: Uranium mining and cigarette smoking effects in man. JOM *15:*204-211, 1973.
5. Archer, V.W., Saccomanno, G., and Jones, H.H.: Frequency of different histologic types of bronchogenic carcinoma as related to radiation exposure. Cancer *34:*2056-2060, 1974.
6. Arnstein, A.: Sozialhygienische Untersuchungen uber die Bengleute in den Schneeberger Kobalt-Gruben, insbesodere uber des Vorkommen des sogenannten "Schneeberger Lungenkrebses" Osterreich Sanitatswesen Wien Arbeit., a.d. Geb. I. Soz. Med. Beihefte. *5:*64-83, 1913.
7. Axelson, O. and Sundell, L.: Mining, lung cancer and smoking. Scand. J. Environ. Health *4:*46-52, 1978.
8. Baetjer, A.M.: Pulmonary carcinoma in chromate workers I. A review of the literature and report of cases. Arch. Ind. Hyg. Occup. Med. *2:*485-504, 1950.
9. Baetjer, A.M., Levin, M.L., Lillenfeld, A.: Analysis of mortality experience of Allied Chemical Plant-2000 Rase Street, Baltimore, MD. Unpublished report submitted to Allied Chemical Corporation, Morristown, N.J., July 16, 1974.
10. Beebe, Gilbert W., Ph.D.: Lung cancer in World War I veterans: Possible relation to mustard-gas injury and 1918 influenza epidemic. J. Nat Cancer Inst. *25:*1231-1252, 1960.
11. Berry, G., Newhouse, M.L., and Turok, M.: Combined effect of asbestos ex-

posure and smoking on mortality from lung cancer in factory workers. Lancet *2:*476, 1972.
12. Bohlig, H., Dabbert, A.F., Palguen, P., Hain, E., and Hinz, I.: Epidemiology of malignant mesothelioma in Hamburg. Environ. Res. *3:*365, 1970.
13. Boucot, K.R., Weiss, W., Seidman, H., et al.: The Philadelphia Pulmonary Neoplasm Research Project: Basic Risk Factors of Lung Cancer in Older Men. Am J. Epidemiol. *95:*4-16, 1972.
14. Bourasset, A. and Galland, G.: Cancer des voies respiratoires et exposition aux sels de nickel. Arch. Malad. Prof. *27:*227-229, 1966.
15. Brinton et al. Morbidity and mortality experience among chromate workers. Public Health Rep. *67:*835-47, 1952.
16. Cancer Statistics. *Ca-A Cancer Journal for Clinicians.* Published by the American Cancer Society. Jan/Feb *31*(1):13-28, 1981.
17. Case, R.A.M. and Lea, A.J.: Mustard gas poisoning, chronic bronchitis, and lung cancer. Brit. J. Prev. Soc. Med. *9:*62-72, 1955.
18. Chief Inspector of Factories: *Annual Report of the Chief Inspector of Factories for the Year* 1932, London, HMSO, p. 103, 1933.
19. Chief, Inspector of Factories: *Annual Report of the Chief Inspector of Factories for the Year* 1950, London, HMSO, p, 145, 1952.
20. Committee of Medical and Biologic Effect of Environmental Pollutants (CMBE-FEP). Nickel, Washington, DC., National Academy of Sciences, 1975.
21. Cooper, C.W.: Study of sheet metal workers. Final Contract Report, Contract No. HSM-099-71-55, National Institute for Occupational Safety and Health, 1975.
22. Costa, M. and Mollenhourer, H.H.: Carcinogenic activity of particulate nickel compounds is proportional to their cellular uptake. Science *209:*515-517, 1980.
23. de Villiers, A.J. and Windish, J.P.: Lung cancer in a fluospar mining community I. radiation, dust and mortality experience. Br. J. Ind. Med. *21:*94-109, 1964.
24. Discher, D.P., Breitenstein, B.D., and Schweid, A. I.: Sputum cytology among aluminum potroom workers. Ann. N.Y. Acad. Sci. *271:*239-242, 1976.
25. Doll, R.: The age distribution of cancer: Implications for model of carcinogenesis. J. Roy. Statist. Soc. *A134:*133, 1971.
26. Doll, R.: Cancer of the lung and nose in nickel workers. Br J. Ind. Med. *15:* 217-223, 1958.
27. Doll, R.: Mortality from lung cancer in asbestos workers. Br. J. Ind. Med. *12:*81, 1955.
28. Doll, R., Morgan, L.G., and Speizer, F.E.: Cancers of the lung and nasal sinuses in nickel workers. Br. J. Cancer *24:* 623-632, 1970.
29. Edge, J.R.: Asbestos related disease in Barrow-in-Furness. Environ. Res. *11:*244, 1976.
30. Elmes, P.C. and Simpson, J.C.: Insulation workers in Belfast. 3. Mortality. 1940-66. Br. J. Ind. Med.*28:*226, 1971.
31. Elmes, P.C. and Simpson, M.J.C.: Insulation workers in Belfast. A further study of mortality due to asbestos exposure 1040-75. Br. J. Ind. Med. *34:*174, 1977.
32. Enterline, P.E. and Henderson, V.: Type of asbestos and respiratory cancers in the asbestos industry. Arch. Environ. Health *27:*312, 1973.
33. Fern, V.H.: Arsenic as a teratogenic agent. Environ. Health Persp. *19:*191-196, 1977.
34. Figueroa, W.G., Raszowski, R., and Weiss, W.: Lung cancer in chloromethyl methyl ether workers. New Engl. J. Med *228:* 1096-1097, 1973.
35. Frost, J.V., et al.: Pulmonary cytologic alterations in toxic environmental inhalation. Human Pathology *4:*521-536, 1973.
36. Gafafar, W.M., et al.: Health of workers in chromate producing industry: a study. U.S. Public Health Service, Division of Occupational Health, Publication No. 192, Washington, DC., U.S. Public Health Service.
37. Garcus, J.L., Reese, W.H., Rutter, H.A.: Induction of lung adenomas in newborn mice by bis (chloromethyl) ether. Toxic. Appl. Pharmacol. *15:*92-96, 1969.
38. Gloyne, S.R.: Two cases of squamous carcinoma of the lung occurring in asbes-

tosis. Tubercle *17:*5, 1935.
39. Gottlieb, L.S. and Hussen, L.A.: Lung cancer among Navajo uranium miners. Unpublished, 1980.
40. Ham, J.A.: Report of the Royal Commission on the Health and Safety of Workers in Mines. E. Ministry of the Attorney General, Province of Ontario, Toronto, Canada, 1976.
41. Hammond, E.C. and Selikogg, I. J.: Relation of cigarette smoking to risk of death of asbestos-associated disease among insulation workers in the United States. In: Proceedings of the Conference on Biological Effects of Asbestos. P. Bogovski, J.C. Gilson. V. Trimbrell, and J.C. Wagner, Eds., IARC, Lyon, p. 302.
42. Hammond, E.C., Selikoff, I. J., and Seidman, H.: Asbestos exposure, cigarette smoking and death rates. Ann. N.Y. Acad. Sci. *330:*473, 1979.
43. Harries, P.G.: Experience with asbestos disease and its control in Great Britain's naval dockyards. Environ. Res. *11:*261, 1976.
44. Harting, F.H. and Hesse, W.: Der Lungenkrebs, die Bergkrankheit in den Schneeberger Gruben. Viertelj. F. gerichtl. Med. u. offen. Sanitats. *30:*296-308, *Ibid 31:*102-132, and 313-337, 1879.
45. Hill, A.B. and Faning, E.L: Studies in the incidence of cancer in a factory handling inorganic compounds of arsenic. I. Mortality experience in the factory. Br.J. Ind. Med. *5:*1-6, 1948.
46. Hood, R.D., Thacker, G.T. and Patterson, B.L.: Effects in the mouse and rat of prenatal exposure to arsenic. Environ. Health Persp. *19:*219-222, 1977.
47. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man, Vol. 2, Some Inorganic and Organometallic Compounds. Lyon, pp. 100-125, 1973.
48. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Vol. 23, Lyon, pp. 143-204, 1980.
49. IARC Working Group, Nickel and Nickel Compounds, 1975.
50. Infante, P.F., Wagoner, J.K. and Sprince, N.L.: Mortality patterns from lung cancer and non-neoplastic respiratory disease among white males in the Beryllium Case Registry. Environ. Res. *21:*35-43, 1980.
51. Jorgensen, J.S.: A study of mortality from lung cancer among miners in Liruna 1950-1970. Scand. J. Work, Environ. Health *10:*126-133, 1973.
52. Kennaway, E.L. and Kennaway, N.M.: A further study of the incidence of cancer of the lung and larynx. Br J. Cancer *1:*260-298, 1947.
53. Kiviluoto, R.: Pleural calcification as a roentgenologic sign of nonoccupational endemic anthophyllite-asbestos. Acta Radiol. (suppl.) *195:*1, 1960.
54. Kogan, F.M., Guselnikova, N.A., and Gulevskaya, M.R.: The cancer mortality rate among workers in the asbestos industry of the Urals. Gig. Sanit. *37:*29, 1972.
55. Konetzke, G.W.: Berufliche Krebserkrankungen durch arsen and nickel sowei deren Verbindugen. Dtsch. Ges. Wesen. *29:*1334, 1974.
56. Konetzke, G.W.: Die kanzerogene Wirkung von Arsen und Nickel. Arch. Geschulwulstforsch *44:*16-22, 1974.
57. Konstantinov, F.G. and Kuz'minykh, A.I., Tarry substances and 3,4-benzopyrene in the air of electrolytic shops of aluminum works and their carcinogenic significance. Hyg. and Sanit. *36:*368-373, 1971.
58. Kreyberg, L.: Histological lung cancer types. A morphological and biological correlation. Acata Pathologica et Microbiologica Scandinavica, Supplement No. 157, 1962.
59. Kreyberg, L.: Lung cancer in workers in a nickel refinery. Br. J. Ind. Med. *35:* 109-116, 1978.
60. Kuratsune, M., Tokundime, S., Shirakusa, T., Yoshida, M., Tokamitsu, Y., Hayano, T., and Seita, M.: Occupational lung cancer among copper smelters. Int J. Cancer *13:*552-558, 1974.
61. Laskin, S., Drew, R.T., Cappielo, et al.: Inhalation carcinogenicity of alpha haloethers. Presented at the British Occupational Hygiene Society Fourth International Symposium, 1971.
62. Laskin, S., Kuschner, J., Drew, R.T., et al.: Tumors of the respiratory tract in-

duced by inhalation of bis (chloromethyl) ether. Arch, Environ. Health *23:*135-136, 1971.
63. Lee, A.M. and Fraumeni, J.J., Jr.: Arsenic and respiratory cancer in man: an occupational study. J. Natl. Cancer Inst. *42:*1045-52, 1969.
64. Lemen, R.A.: Lung cancer in female asbestos workers. Letter to Vernon Rose, Director, Division of Criteria Documentation and Standards Development, 1976.
65. Lemen, R.A., Johnson, W.M., Wagoner, J.W., Archer, V.E., and Saccomanno, G.: Cytologic observations and cancer incidence following exposure to BCME. Ann. N.Y. Acad. Sci. *271:*71-89, 1976.
66. Lerer, T.J., Redmond, C.K., Breslin, P.P., Salvin, L., and Rush, H.W.: Long-term mortality study of steelworkers VII: Mortality patterns among crane operators. JOM *16:*608-614, 1974.
67. Lessard, R. et al.: Lung cancer in New Caledonia—a nickel smelting island. J. Occup. Med *20:*12, 1978.
68. Liddell, F.D.K., McDonald, J.C., and Thomas, D.C.: Methods of cohort analysis: appraisal by application to asbestos mining. J. Roy. Statist. Soc *A140:*469, 1977.
69. Litvinov, N.W., Goldberg, M.S., and Kimina, S.N.: Morbidity and mortality in man caused by pulmonary cancer and its relation to the pollution of the atmosphere in the areas of aluminum plants. Acta Unionis Internationalis Contra *Cancrum 19:*742-745, 1963.
70. Lloyd, J.W.: Long-term mortality study of steelworkers V. Respiratory cancer in coke plant workers. JOM *13:*53-68, 1971.
71. Lloyd, J.W. and Ciocco, A.: Long-term mortality study of steelworkers: I. Methodology. JOM *11:*299-310, 1969.
72. Lloyd, J.W., Lundin, F.E., Redmond, C.K., and Gieser, P.B.: Long-term mortality study of steelworkers IV. Mortality by work area. JOM *12:*151-157, 1970.
73. Loken, A.C.: Lung cancer in nickel workers. Tidsskr. Norske Laegefor. *70:* 376-378, 1950.
74. Lundin, F.E., Wagoner, J.K., and Archer, V.E.: Radon daughter exposure and respiratory cancer quantitative and temporal aspects. NIOSH-NIEHS Joint Monograph No. 1, Springfield, VA., NTIS, 1971.
75. Lynch, K.M. and Smith, W.A.: Pulmonary asbestosis: Carcinoma of the lung in asbestos-silicosis. AM. J. Cancer *24:*56, 1935.
76. Mabuchi, K., Lillienfeld, A.M., and Snell, L.M.: Cancer and occupational exposure to arsenic: a study of pesticide workers. Preventive Medicine *9:*51-77, 1980.
77. Machle, W. and Gregorius, F.: Cancer of the respiratory system in the United States chromate producing industry. Publ. Health Rep. (Wash.) *63:* 1114-1127, 1948.
78. Mancuso, T.F.: Consideration of chromium as an industrial carcinogen. In: Proceedings of the International Conference on Heavy Metals in the Environment, T.C. Hutchinson, Ed. Toronto, 1975, Toronto, Institute for Environmental Studies, pp. 343-356.
79. Mancuso, T.F.: Mortality study of beryllium industry workers, occupational lung cancer. Environ. Res. *21:*48-55, 1980.
80. Mancuso. T.F.: Occupational lung cancer among beryllium workers. In: *Dusts and Disease.* R.A. Lemen and J.M. Dement, Eds. Pathotox Publishers, Park Forest, Ill, 1979.
81. Mancuso, T.F. and Coulter, E.J.: Methodology in industrial health studies. The cohort approach with special reference to an asbestos company. Arch. Environ. Health *6:*36, 1963.
82. Mastromatteo, E.: Nickel: a review of its occupational health aspects. JOM *9:*127-136, 1967.
83. Mazumdar, S., Lerer, T., and Redmond, C.K.: Long-term mortality study of steelworkers IX: Mortality patterns among sheet and tin mill workers. JOM *17:*751-755, 1975.
84. McDonald, A.D. and McDonald, J.C.: Mesothelioma after crocidolite exposure during gas mask manufacturing. Environ. Res. *17:*340, 1978.
85. McDonald, A.D., Magner, D., and Eyssen, G.: Primary malignant mesothelial tumors in Canada, 1960-1968. A pathologic review by the mesothelioma panel of the Canadian Tumor Reference Cen-

tre. Cancer, *31:*869, 1973.
86. McDonald, J.C., Becklake, M.R., Gibbs, G.W., McDonald, A.D., and Rossiter, C.E.: The health of chrysotile asbestos mine and mill workers of Quebec. Arch Environ. Health *28:*61, 1974.
87. Meurman, L.: Asbestos bodies and pleural plaques in a Finnish series of autopsy cases. Acta. Pathol. Microbiol. Scand. (Suppl.) *181:*1, 1966.
88. Meurman, L., Kiviluoto, R., and Hakama, M.: Mortality and morbidity among the working population of anthophylite asbestos miners in Finland. Br. J. Ind. Med. *31:*105, 1974.
89. Milham, S., Jr.: Cancer mortality patterns associated with exposure to metals. Ann. N.Y. Acad. Sci. *271:*243-249, 1976.
90. Milham, S., Jr. and Strong, T.: Human arsenic exposure in relation to a copper smelter. Environ. Res. *7:*172-182, 1974.
91. Morgan, J.G.: Some observations on the incidence of respiratory cancer in nickel workers. Br. J. Ind Med. *15:*224-234, 1958.
92. Newhouse, M.L. and Thompson, H.: Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. Br. J. Ind. Med *22:*261, 1966.
93. Newman, J., et al.: Histologic types of bronchogenic carcinoma among members of copper-mining and smelting communities. Ann. N.Y. Acad. Sci. *271:*260-268, 1976.
94. Ott, M. G., Holder, B. B., and Gordon, H. L.: Respiratory cancer and occupational exposure to arsenials. Arch. Environ. Health *29:*250-255, 1974.
95. Pederson, L. E., Hogetveit, A. C., and Anderson, A.: Cancer of respiratory organs among workers at a nickel refinery in Norway. Int. J. Cancer *12:*32-41, 1973.
96. Peller, S.: Lung cancer among miner workers in Joachimsthal. Human Biol. *11:*130-143, 1939.
97. Pinto, S. S. and Bennett, B. M.: Effect of arsenic trioxide exposure on mortality. Arch. Environ. Health *7:*583-591, 1963.
98. Pinto, S. S., Henderson, V., and Enterline, P. E.: Mortality experience of arsenic-exposed workers. Arch. Environ. Health *33:*325-332, 1978.
99. Pirchan, A. and Sill, H.: Cancer of the lung in the miners of Jachymov. Am J. Cancer *16:*681-722, 1932.
100. Redmond, C. K.: Comparative cause-specific mortality patterns by work area within the steel industry. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, NIOSH, HEW, Publication No. (NIOSH) 75-157, 1975.
101. Redmond, C. K., Ciocco, A., Lloyd, J. W., and Rush, H. W.: Long-term mortality study of steelworkers VI. Mortality from malignant neoplasms among coke oven workers. JOM *14:*621-629, 1972.
102. Redmond, C. K., Gustin, J., and Kamon, E.: Long-term mortality experience of steelworkers VIII: Mortality patterns of open hearth steelworkers (A preliminary report). JOM *17:*40-43, 1975.
103. Redmond, C. K., Smith, E. M., Lloyd, J. W., and Rush, H. W.: Long-term mortality study of steelworkers: III. Follow-up, JOM *11:*513-521, 1969.
104. Renard, K. G., St. C.: Respiratory cancer mortality in an iron ore mine in northern Sweden. Ambio. *2:*67-69, 1974.
105. Rencher, A. C. and Carter, M. W.: A retrospective epidemiological study of Kennecott's Utah Smelter-Phase II Mortality. Unpublished report submitted to the Kennecott Copper Corporation, New York, NY, April 1971.
106. Robinson, C. F., Lemen, R. A., and Wagoner, J. K.: Mortality patterns, 1940-1975 among workers employed in an asbestos textile friction and packing products manufacturing facilities. In: *Dusts and Disease*. R. A. Lemen and J. M. Dement, Eds. Pathotox Publishers, Park Forest, IL, 1979.
107. Robinson, H.: Long-term mortality study of steelworkers: II. Mortality by level of income in whites and non-whites. JOM *11:*411-416, 1969.
108. Rockette, H. E. and Redmond, C. K.: Long-term mortality of steelworkers X: Mortality patterns among masons. In Press.
109. Rockstroh, H. Von: Sur aetiologie des bronchialkrebses in arsenverarbeitensken. Nickelhitten. Arch. Geschwalstfo.

*14:*151-162, 1958.
110. Roth, F.: Arsenic-liver-tumors (haemangioendothelioma) (Ger). Z. Krebsforsch *61:*468-503, 1975.
111. Roth, F.: Late consequences of chronic arsenicism in Moselle vine-dressers. (Ger) Dtsch. Med. Wochenscher *82:*211-217, 1957.
112. Rubino, G. F., Scanetti, G., Conna, A., and Palestro, G.: Epidemiology of pleural mesothelioma in North-Western Italy (Piedmont). Br. J. Ind. Med. *29:*436, 1972.
113. Saccommano, et al: Histologic types of lung cancer among Uranium miners. Cancer *27*(5):15-523, 1971.
114. Sakabe, H.: Lung cancer due to exposure to bis(chloromethyl)ether. Ind. Health *11:*145, 1973.
115. Saknyn, A. V. and Shabynina, N. K.: Epidemiology of malignant neoplasms at nickel smelters. Gig. Trud. Prof. Zabol. *17:*25-29, 1973.
116. Saknyn, A. V. and Shabynina, N. K.: Some statistical data on carcinogenous hazards for workers engaged in the production of nickel from oxidized ores. Gig. Trud. Prof. Zabol. *14:*10-13, 1970.
117. Selikoff, I. J.: Asbestos disease in the United States 1918-1975. Paper presented at the Conference on Asbestos Disease, Rouen, France, October 27, 1975.
118. Selikoff, I. J.: Partnership for presention — the insulation industry hygiene research program. Ind. Med. *39:*4, 1970.
119. Selikoff, I. J., Churg, J., and Hammond, E. C.: Asbestos exposure and neoplasia. J. Am. Med. Assoc. *188:*22, 1964.
120. Selikoff, I. J., Hammond, C. and Churg, J.: Asbestos exposure, smoking and neoplasia. JAMA *204:*106-112, 1960.
121. Selikoff, I. J., Hammond, E. C., and Seidman, H.: Cancer risk of insulation workers in the United States. In: Proceedings of the Conference of Biological Effects of Asbestos, P. Bogovski, J. C. Gilson, V. Timbrell, and J. C. Wagner, Eds. IARC, Lyon, p. 209, 1973.
122. Sevc, J., Kunz, E., and Placek, V.: Lung cancer in uranium miners and long-term exposure to radon daughter products. Health Phys. *30:*433, 1976.
123. Sevc, J. and Placek, V.: Lung cancer risk in relation to long-term exposure to radon daughters. Second European Congress on Radiation Protection, Budapest, Hungary, 1977.
124. Sevc, J., Placek, V., and Jerubek, J.: Lung cancer risk in relation to long-term radiation exposure in uranium mines. Fourth Conference on Radiation Hygiene, CSSR, 1971.
125. Shuler, P. J., and Bierbaum, P. J.: Environmental surveys of aluminum reduction plants. U.S. Department of Health, Education, and Welfare. NIOSH Publication No. (NIOSH) 74-101, 1974.
126. Snihs, J. O.: The approach to radon problems in non-uranium mines in Sweden. Proceedings of the Third International Congress of the International Radiation Protection Association, September 9-14, 1973.
127. Snihs, J. O.: The approach to radon problems in non-uranium mines in Sweden. In: Proceedings of the Third International Congress of the International Radiation Protection Association, pp. 900-911, 1974.
128. Stellman, S. D. and Stellman, J. M.: *Ca-A Cancer Journal for Clinicians.* Published by the American Cancer Society. Jan/Feb *31*(1):29-43, 1981.
129. Stumphius, J.: Epidemiology of mesothelioma on Waicheren Island. Br. J. Ind. Med. *28:*59, 1971.
130. Sunderman, F. W., Jr.: The current status of nickel carcinogenesis Ann. Clin. Lab. Sci. *3:*156-180, 1973.
131. Tatarskaya, A. A.: Cancer of the respiratory tract in people engaged in nickel industry. Vop. Onkol. *13:*58-60, 1967.
132. Tatarskaya, A. A.: Occupational cancer of upper respiratory passages in the nickel industry. Gig. Trud. Prof. Zabol. *9:* 22-25, 1965.
133. Theiss, A. M., Hay, W., and Zeller, H.: Toxicology of bis(chloromethyl)ether—suspicion carcinogenicity in man. Zentralbl. Arbeismed., *23:*97-102, 1973.
134. Tourine, R. and Ramboud, G.: Les cancers bronchiques primtifs a localisation double unilaterale. J. Fr. Med. Chirurg. Thorac. *22:*757-767, 1968.
135. Tsuchiya, K.: The relation of occupation

to cancer, especially cancer of the lung. Cancer *18:*136-144, 1965.
136. UICC Working Group on Asbestos Cancers. Arch. Environ. Health *11:* 221, 1965.
137. Virtue, J. A.: The relationship between the refining of nickel and cancer of the nasal cavity. Canad. J. Otolaryng. *1:*37-42, 1972.
138. Wada, S., et al.: Mustard gas as a cause of respiratory neoplasia in man. Lancet *1:*1161-1163, 1968.
139. Wagoner, J. K., Archer, V. E., Carroll, B. E., Holaday, D. A., and Lawrence, P. A.: Cancer mortality patterns among U.S. uranium miners and millers, 1950 through 1962. J. Nat. Cancer Inst. *32:*787-801, 1964.
140. Wagoner, J. K., Archer, V. E., and Gillam, J. D.: Mortality of American Indian uranium miners. Proceedings XI International Cancer Congress (Bucalossi, P., Veronesi, U. and Cascinelli, N., Eds.). Vol. 3, pp. 102-107, Excerpta Medica International Congress Services No. 351. Excerpta Medica, Amsterdam, 1975.
141. Wagoner, J. K., Archer, V. E., Lundin, F. E., Jr., Holaday, D. A., and Lloyd, J. W.: Radiation as a cause of lung cancer among uranium miners. New Eng. J. Med *273:*181-188, 1965.
142. Wagoner, J. K., Infante, P. F., and Bayliss, D. L.: Beryllium: An etiologic agent in the induction of lung cancer, nonneoplastic respiratory disease and heart disease among industrially exposed workers. Environ. Res. *21:*15-34, 1980.
143. Wagoner, J. K., Johnson, W. M., and Lemen, R. A.: Malignant and nonmalignant respiratory disease mortality patterns among asbestos production workers. Congressional Record, Senate Proceedings and Debates, 93rd Congress, First Session, U.S. Govt. Printing Office, Vol. 119, Part 6, pp. 7828-7830, 1973.
144. Wedler, H. W.: Asbestose und lungenkrebs. Deut. Med. Wochenschr. *69:*574, 1943.
145. Wedler, H. W.: Asbestose und lungenkrebs bei asbestose. Deut. Arch. Klin. Med. *191:*189, 1943.
146. Weill, H., Hughes, J., and Waggenspack, C.: Influence of dose and fiber type on respiratory malignancy risk in asbestos cement manufacturing. Am. Rev. Resp. Dis. *120:*345, 1979.
147. Weiss, W.: Mortality of a cohort exposed to chrysotile asbestos. JOM *19:*737, 1977.
148. Wicks, M. J., Archer, V. E., Auerbach, O., and Kuschner, M.: Arsenic exposure in a copper smelter as related to histological type of lung cancer. Am. J. Indus. Med., Vol. 2, pp. 25-31, 1981.
149. Wyers, H.: That legislative measures have proved generally effective in the control of asbestosis. Glasgow, M. E. Thesis University of Glasgow, UK, 1946.
150. Yamada, A.: On late injuries following occupational inhalation of mustard gas with special reference to carcinoma of respiratory tract. Acta. Pathol. Jap. *13:*131-155, 1963.
151. Znamenskii, S. L. W.: Occupational bronchogenic cancers in workers extracting, isolating and reprocessing nickel ore. Vop. Onkol *9:*130, 1963.

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 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.

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

Trade	*Percentage*
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, "Wartime Employment, Production, and Conditions of Work in Shipyards," 1945.

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

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 statisitics 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, Malmo, 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 controlateral 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	**632**	
Man-years of observation	**13,925**	
	Deaths 1.1.43-12.31.76	
Cause of death	*Expected**	*Observed*
Total deaths, all causes	328.9	478
Total cancer, all sites	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
Asbestosis	**	41
All other causes	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.

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	**17,800**		
Man-years of observation	**166,855**		
	*Expected**	*Observed*	*Ratio*
Total deaths, all causes	1,660.96	2,270	1.37
Total cancer, all sites	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
Asbestosis	**	162	—
All other causes	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

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

[a] 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.

[b] 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 indicated 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.	Obs./Exp.
All causes	59	52.41	123	69.85	92	65.93	274	188.19	1.46
Cancer, all sites	21	10.47	45	14.70	33	14.73	99	39.92	2.47
Lung cancer	6	2.96	18	4.65	11	4.92	35	12.53	3.91[a]
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	1.88
Cancer, all other sites	9	5.28	11	7.13	8	6.98	23	19.40	1.19
All respiratory disease	14	3.01	10	4.56	18	4.60	42	12.16	3.45
Asbestosis	12	n.a.	8	n.a.	15	n.a.	35	n.a.	—
Other respiratory	2	(b)	2	(b)	3	(b)	7	(b)	—
All other causes	24	38.93	68	50.59	41	46.60	133	136.11	0.98
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

		Deaths of lung cancer and mesothelioma				
Time from onset (years)	*Man-years*	*Lung cancer*			*Mesothelioma*	
		Exp.	*Obs.*	*Ratio*	*Pleural*	*Peritoneal*
<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 um) 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 gen erates 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 patients 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 fibrosacromatous 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 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 intravitam. 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^3. 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.

REFERENCES

1. Anderson, H., et al: Household-contact asbestos neoplastic risk. Ann NY Acad Sci *271:*311, 1976.
2. Ashcoft, T. and Heppleston, A.G.: Mesothelioma and asbestos on Tyneside—a pathological and social study. In: Pneumoconiosis—Proceedings of an International Conference, Johannesburg, 1959, H.A. Shapiro, ed., Oxford University Press, Cape Town, pp. 177-179, 1970.
3. Baris, Y.I.: Pleural mesotheliomas and asbestos pleurisies due to environmental asbestos exposure in Turkey: an analysis of 120 cases. Hacettepe Bull Med Surg *8:*165, 1975.
4. Bignon, J., et al.: Topographic distribution of asbestos fibers in human lung in relation with occupational and non-occupational exposure. Inhaled Particles and Vapours, Proc Int Symp, 4th (in press).
5. Bittersohl, G., and Ose, H.: Zur Epidemio-

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

	Accuracy of death certificate categories				
		Death Certificate		*Ascertained*	
Cause of death	*Expected*	*Number*	*o/e*	*Number*	*o/e*
Cancer, all sites	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
Asbestosis	—	108	—	162	—
Chronic obstructive lung disease	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

	Death certificate	*Ascertained*
Cancer, all sites	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
All respiratory disease	43	42
Asbestosis	26	35
Pneumoconiosis	8	0
All respiratory disease	9	7
All other causes	137	133

logie des Pleuralmesothelioms., Z Gesamte Hyg, *17:*861-864, 1971.
6. Boersma, A., Degand, P., and Havez, R.: Diffuse mesothelioma: Biochemical stages in the diagnosis, detection and measurement of hyaluronic acid in the pleural fluid. In: Biological Effects of Asbestos, IARC Sci Publ, No. 8, p. 65, IARC, Lyon, 1973.
7. Bonadonna, G., et al.: Monochemioterapia con adriamicina in varie neoplasie in fase avanzata dell adulto e del bambino. Tumori *60:*373, 1974.
8. Bruckman, L., Rubino, R.A., and Christine, B.: Asbestos and mesothelioma incidence in Connecticut. J Air Pollut Control Assoc *27:*121-126, 1977.
9. Butchart, E.G., et al.: Pleuropneumonectomy in the management of diffuse malignant mesothelioma of the pleura. Experience with 29 patients. Thorax *31:*15, 1976.
10. Califano, J.A., Jr.: Statement of Secretary Joseph A. Califano, Jr., U.S. Department of Health, Education, and Welfare, April 26, 1978.
11. Cochrane, J.C. and Webster, I.: Mesothelioma in relation to asbestos fibre exposure. A review of 70 serial cases. S.A. Medical Journal, *54:*279-281, 1978.
12. Davis, J.M.G.: Electron-microscope studies of asbestosis in man and animals. Ann NY Acad Sci *132:*98, 1965.
13. Dement, J.M., Harris, R.L., Symons, M.J. and Shy, C.: Estimates of dose-response for respiratory cancer among chrysotile asbestos textile workers. In: Proceedings of the 5th International Conference on Inhaled Particles and Vapours, BOTTS, 1980.
14. Dubray, E.S. and Rosson, F.B.: Primary mesothelioma of the pleura. A clinical and pathologic contribution to pleural malignancy, with a report of case. Arch Intern Med *26:*715, 1920.
15. Ducic, S.: L'exactitude des causes de deces - une comparison avec les diagnostics a l'autopsie dans une serie de mesotheliomes et autres tumeurs malignes du poumon. Can J Public Health *62:*395-402, 1971.
16. Elmes, P.C. and Simpson, M.J.C.: Insulation workers in Belfast. 3. Mortality 1940-1966. Br J Ind Med *28:*226-236, 1971.
17. Eschwege, F. and Schlienger, M.: La radiotherapie des mesotheliomes pleuraux malins. J Radiol Electrol Med Nucl *54:* 255, 1973.
18. Gerner, R.E. and Moore, G.E.: Chemotherapy of malignant mesothelioma. Oncology *30:*152, 1974.
19. Harington, J.S., Allison, A.C., and Badami, D.V.: Mineral fibers: chemical, physiochemical and biological properties. Adv Pharmacol Chemother *12:*291, 1974.
20. Harries, P.G.: Experience with asbestos disease and its control in Great Britain's naval dockyards. Environ Res *11:*261, 1976.
21. Harris, R.L.: A model for deposition of microscopic fibers in the human respiratory system. Thesis, University of North Carolina, Chapel Hill, 1972.
22. Harris, R.L. and Fraser, D.A.: A model for deposition of fibers in the human respiratory system. Am Ind Hyg Assoc J *37:*73, 1976.
23. Harris, R.L., Timbrell, V., and Berry, G.: The influence of fiber shape in lung deposition—mathematical estimates. Inhaled Part. Vap., Proc. Int. Symp. 4th (in press).
24. Holt, P.F., Mills, J., and Young, D.K.: The early effects of chrysotile asbestos dust on the rat lung. J Pathol Bacteriol *87:*15, 1964.
25. Kannerstein, M., Churg, J., and Magner, D.: Histochemical studies in the diagnosis of mesothelioma. In: Biological Effects of Asbestos, P. Bogovski et al., eds. IARC Sci Publ No 8, p. 62, IARC, Lyon, 1973.
26. Kleinfeld, M., Messite, J., and Kooyman, O.: Mortality experience in a group of asbestos workers. Arch Environ Health *15:*177-180, 1967.
27. Klemperer, P. and Rabin, C.B.: Primary neoplasms of the pleura. Arch Pathol *11:*385-412, 1931.
28. Knox, J.G., Holmes, S., Doll, R., and Hill, I.D.: Mortality from lung cancer and other causes among workers in an asbestos textile factory. Br J Ind Med *25:* 293-303, 1968.

29. Kucuksu, N., Ezdinli, E., and Cehreli, C.: Chemotherapy of mesothelioma. Cancer Res 16, Abstr 31, 1975.
30. Kucuksu, N., Thomas, W., and Ezdinli, E.Z.: Chemotherapy of malignant diffuse mesothelioma. Cancer *37:*1265, 1976.
31. Langer, A.M., et al.: Inorganic fibers, including chrysotile, in lungs at autopsy: Preliminary report. Inhaled Particles 3, Proc. Int. Symp. 3rd, p. 683, 1971.
32. Lieben, J. and Pistawka, H.: Mesothelioma and asbestos exposure. Arch Environ Health *14:*559, 1967.
33. Mancuso, T.F.: Discussion on asbestos and neoplasia: Epidemiology. Ann NY Acad Sci *132:*589-594, 1965.
34. Mancuso, T.F. and Coulter, E.J.: Methodology in industrial health studies: the cohort approach with special reference to an asbestos company. Arch Environ Health *6:*210-226, 1963.
35. Martini, N., Bains, M.S., and Beattie, E.J.: Indications for pleurectomy in malignant effusion. Cancer *35:*734, 1975.
36. McDonald, J.C., Liddell, F.D.K., Gibbs, G.W., Eyssen, G.E., and McDonald, A.D.: Dust exposure and mortality in chrysotile minings, 1910-75. Br J Ind Med *37:*11-24, 1980.
37. McDonald, J.C. and McDonald, A.D.: Epidemiology of mesotherlioma from estimated incidence. Prev Med *6:*426-446, 1977.
38. McDonald, J.C., McDonald, A.D., Gibbs, G.W., Siemiatycki, J., and Rossiter, C.E.: Mortality in the chrysotile asbestos mines and mills of Quebec. Arch Environ Health *22:*677-686, 1971.
39. McEwen, J., Finlayson, A., Mair, A., and Gibson, A.M.: Mesothelioma in Scotland. Br Med J *IV:*575-578, 1970.
40. McGowan, L., Bunnag, B., and Arias, L.F.: Mesothelioma of the abdomen in women. Monitoring of therapy by peritoneal fluid study. Gynecol Oncol *3:*10,1975.
41. Milne, J.: Fifteen cases of pleural mesothelioma associated with occupational exposure to asbestos in Victoria. Med J Aust. *II:*669-673, 1969.
42. National Cancer Institute. Third National Cancer Survey—Incidence Data. NCI Monograph 41, March 1975.
43. Newhouse, M.L.: Asbestos in the work place and the community. Ann Occup Hyg *16:*97, 1973.
44. Newhouse, M.L.: Cancer among workers in the asbestos textile industry. In: Biological Effects of Asbestos, Lyon, pp. 203-208, 1972.
45. Newhouse, M.L. and Berry, G.: Predictions of mortality from mesothelial tumours in asbestos factory workers. Br J Ind Med *33:*147, 1976.
46. Newhouse, M.L. and Thompson, H.: Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. Br J Ind Med *22:*261, 1965.
47. Newhouse, M.L., Berry, G., Wagner, J.C., and Turok, M.E.: A study of the mortality of female asbestos workers. Br J Ind Med *29:*134-141, 1972.
48. Nicholson, W.J., Selikoff, I.J., Hamond, E.C., and Seidman, H.: Mortality experience of asbestos factory workers; effect of differing intensities of asbestos exposure. Environ Res (in press).
49. Nicholson, W.J., Selikoff, I.J., Seidman, H., Lilis, R. and Formby, P.: Long-term mortality of chrysotile miners and millers in the Thetford Mines, Quebec. Ann NY Acad Sci *330:*11-21, 1979.
50. Peto, J., Doll, R., Howard, S.V., Kinlen, I.J., and Lewinsohn, A.C.: A mortality study among workers in an English asbestos factory. Br J Ind Med *34:*169-173, 1977.
51. Planteydt, H.T.: Mesothelioma and asbestos bodies in the sputum of workers in a shipyard. Poumon Coeur *5:*545, 1968.
52. Rall, D.P. (Chairman). Asbestos. IARC monographs on the evaluation of the carcinogenic risk of chemicals to man, 14, International Agency for Research on Cancer, Lyon, 1977.
53. Robertson, H.E.: Endothelioma of the pleura. J Cancer Res *8:*317-375, 1924.
54. Roe, F.J.C., et al.: The pathological effects of subcutaneous injections of asbestos fibers in mice; migration of fibers to submesothelial tissues and induction of mesotheliomata. Int J Cancer *2:*628, 1967.
55. Rubino, G.F., et al.: Epidemiology of pleu-

ral mesothelioma in Canada. Br J Ind Med *29:*436, 1972.
56. Rusby, M.L.: Pleural manifestations following the inhalation of asbestos in relation to malignant change. J R Nav Med Serv *54:*142, 1968.
57. Schlienger, M., et al.: Mesotheliomes pleuraux malins. Bull Cancer *56:*265, 1969.
58. Seidman, H., Lilis, R., and Selikoff, I.J.: Short-term asbestos exposure and delayed cancer risk. In: Prevention and Detection of Cancer. H.E. Nieburgs, ed., Marcel Dekker, Inc., New York, *1:*943-960, 1976.
59. Selikoff, I.J. The occurrence of pleural calcification among asbestos insulation workers. Ann NY Acad Sci *132:*351, 1965.
60. Selikoff, I.J., et al.: Mortality experience of insulation workers in the United States and Canada 1943-76. Ann NY Acad Sci, *330:*91-116, 1979.
61. Selilkoff, I.J. and Hammond, E.C.: Asbestos-associated disease in United States shipyards. Ca-A Cancer Journal for Clinicians *28:*87-99, 1978.
62. Selilkoff, I.J., Hammond, E.C., and Churg, J.: Carcinogenicity of amosite asbestos. Arch Environ Health *25:*183, 1972.
63. Selikoff, I.J., Hammond, E.C., and Churg, J.: Neoplasia risk associated with occupational exposure to airborne inorganic fibers. Oncology *5:*55, 1970.
64. Selikoff, I.J., Hammond, E.C., and Churg, J.: Mortality experience of asbestos insulation workers, 1943-1968. Pneumoconiosis, Proc Int Conf, 3rd, p. 180, 1970.
65. Shabad, L.M., et al.: Experimental studies on asbestos carcinogenicity. J Natl Cancer Inst *52:*1175, 1974.
66. Stanton, M.F.: Carcinogenicity of fibrous glass: Pleural response in the rat in relation to fiber dimensions. J Nat Cancer Inst *58:*587, 1977.
67. Stanton, M.F.: Some etiological considerations of fiber carcinogenesis. In: Biological Effects of Asbestos, P. Bogovski et al., eds. IARC Sci Publ No 8, IARC, Lyon, p. 289, 1973.
68. Stanton, M.F. and Wrench, C.: Mechanisms of mesothelioma induction with asbestos and fibrous glass. J Natl Cancer Inst *48:*797, 1972.
69. Stumphius.: Epidemiology of mesothelioma on Walcheren Island. Br J Ind Med *28:*59-66, 1971.
70. Timbrell, V.: The inhalation of fibrous dusts. Ann NY Acad Sci *132:*255, 1965.
71. Voss, A.C., Wollgens, P., and Untucht, H.J.: Das Pleuramesotheliom aus strahlentherapeutischer Sicht. Strahlentherapie *148:*329, 1974.
72. Wagner, E.: Das tuberkelahnliche Lymphadenom. Arch Heilk *11:*495-525, 1870.
73. Wagner, J.C. and Berry, G.: Mesotheliomas in rats following inoculation with asbestos. Br J Cancer *23:*567, 1969.
74. Wagner, J. C., Berry, G., Skidmore, J.W., and Timbrell, V.: The effects of the inhalation of asbestos in rats. Br J Cancer *29:*252, 1974.
75. Wagner, J.C., Berry, G., and Timbrel, V.: Mesotheliomata in rats after inoculation with asbestos and other materials. Br J Cancer *28:*173, 1973.
76. Wagner, J.C., Munday, D.E., and Harington, J.S.: Histochemical demonstration of hyaluronic acid in pleural mesotheliomas. J Pathol Bacteriol *84:* 73-77, 1962.
77. Wagner, J.C., Sleggs, C.A., and Marchand, P. Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. Br J Ind Med *17:*260-271, 1960.
78. Wanebo, H.J., Marini, N., Melamed, M.R., Hilaris, B., and Beattie, E.J.: Pleural mesothelioma. Cancer *38:*2481, 1976.
79. Whitwell, F., et al.: Relationship between occupations and asbestos fiber content of the lungs in patients with pleural mesothelioma, lung cancer and other disease. Thorax *32:*377, 1977.
80. Wyers. H.: Asbestosis. Postgrad Med J *25:* 631-638, 1949.

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, chryostile 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.

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Reprint

Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis

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ABSTRACT

Objectives. This article examines the credibility and policy implications of the "amphibole hypothesis," which postulates that (1) the mesotheliomas observed among workers exposed to chrysotile asbestos may be explained by confounding exposures to amphiboles, and (2) chrysotile may have lower carcinogenic potency than amphiboles.

Methods. A critical review was conducted of the lung burden, epidemiologic, toxicologic, and mechanistic studies that provide the basis for the amphibole hypothesis.

Results. Mechanistic and lung burden studies do not provide convincing evidence for the amphibole hypothesis. Toxicologic and epidemiologic studies provide strong evidence that chrysotile is associated with an increased risk of lung cancer and mesothelioma. Chrysotile may be less potent than some amphiboles for inducing mesotheliomas, but there is little evidence to indicate lower lung cancer risk.

Conclusions. Given the evidence of a significant lung cancer risk, the lack of conclusive evidence for the amphibole hypothesis, and the fact that workers are generally exposed to a mixture of fibers, we conclude that it is prudent to treat chrysotile with virtually the same level of concern as the amphibole forms of asbestos. (*Am J Public Health.* 1996;86:179–186)

Introduction

Chrysotile is the predominant type of asbestos produced and consumed in the world today, and it accounted for over 98.5% of US asbestos consumption in 1992.[1] Although asbestos consumption has declined in North America and Europe, sales in other countries (e.g., Southeast Asia, South America, and Eastern Europe) have increased primarily due to the use of asbestos-based construction materials.[2]

Chrysotile is a serpentine (curly) form of asbestos that is distinguished from other amphibole forms of asbestos (i.e., crocidolite, amosite, tremolite). It has been hypothesized that (1) the mesothelioma risk observed among workers exposed to chrysotile asbestos may be explained by the relatively low concentrations (<1%) of tremolite fibers in commercial chrysotile asbestos fibers and (2) that chrysotile asbestos may be less potent than amphiboles in the induction of asbestosis and lung cancer. This has been dubbed the amphibole hypothesis.[3] It has even been suggested that exposure to chrysotile asbestos in the absence of tremolite may present little or no carcinogenic hazard.[4]

The arguments advanced to support the amphibole hypothesis have been primarily based on pathologic studies of burdens of asbestos fibers in human lungs and on toxicologic, mechanistic, and epidemiologic studies. This article presents a critical review of these arguments and of the literature on the carcinogenic hazards associated with exposure to chrysotile asbestos and considers the implications of these findings for the development of occupational health policies.

Lung Burden Studies

The development of methods that involve electron diffraction and energy dispersive analysis of x-rays (EDAX)[5] has made possible the measurement of the amounts of different fiber types in the lung. The results from lung burden studies have provided the primary basis for the advancement of the amphibole hypothesis.

Case studies of individuals who have worked in industries using or producing chrysotile asbestos revealed an unexpectedly high proportion of amphibole (primarily tremolite) fibers, considering the relatively low percentage of amphibole fibers in commercial chrysotile asbestos.[6] In one of the earliest studies, Pooley observed a greater number of amphibole fibers than chrysotile fibers in 7 of 22 patients with asbestosis who had worked in the Canadian chrysotile mining industry.[7] Rowlands et al. also reported a nearly equal concentration of tremolite fibers and chrysotile fibers in the lungs of 47 workers employed as miners or millers in Quebec.[8] Similarly, in population-based studies the percentage of chrysotile fibers found in the lungs has been surprisingly low considering the fact that chrysotile is the major source of exposure for the general population.[9]

Most case–control studies that evaluated the potential relationship between

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Editor's Note. See related annotation by Cullen (p 158) in this issue.

TABLE 1—Summary of Epidemiological Cohort Studies of Workers Exposed to Predominantly Chrysotile Asbestos

Study	Industry	Lung Cancer Deaths		Mesothelioma Cases	
		Observed	Expected	Observed	Deaths, %
Acheson et al.[27]	Gas masks	6	4.8[a]	1	0.6
Cheng and Kong[28]	Textiles, friction materials, and cement	21	6.7*	0	0
Dement et al.[29]	Textiles	126	64.0*	2	0.2
Finkelstein[30]	Electrical conduit pipe	6	3.7	1	1.0
Finkelstein[31]	Automotive	11	7.9	1–2[b]	1.0–1.9
Hughes et al.[32,c]	Cement manufacturing	70	53.2	1	...
Huilan and Zhiming[33]	8 asbestos factories	65	15.6[a]*	2	0.4
McDonald et al.[34]	Friction products	73	49.1*	0	0
McDonald et al.[35,36,d]	Mining and milling	518	389.7*	28	0.4
Piolatto et al.[37]	Mining	22	19.9	2	0.5
Shiqu et al.[38]	Mining	6	...	3	4.5
Weiss[39]	Paper and millboard	4	4.3	0	0
Total		922[e]	618.9	41.0	0.3

Note. SMR = the standardized mortality ratio, which is the ratio between the observed and expected.

[a]The expected number is for cancer of the lung and pleura combined.

[b]One or two cases of mesothelioma were reported. Only one was included in the totals.

[c]Results are for workers exposed only to chrysotile from one of two plants studied. The total number of deaths was not reported; thus, the percentage of mesothelioma deaths could not be estimated.

[d]Observed and expected numbers exclude observations from the asbestos factory.

[e]The Shiqu et al. study was not included in the total number of lung cancer cases because expected numbers were not reported.[67]

*Significantly different from the observed number, $P < .05$ (two tailed).

mesothelioma risk and lung concentrations of the different fiber types of asbestos demonstrated a clear relationship with amphibole lung burdens but failed to find a relationship with lung chrysotile concentrations.[10–14] McDonald et al. reported an association between mesothelioma and lung concentrations of long (≥ 8 μm) chrysotile fibers in univariate analyses but not in multivariate analysis, which controlled for the other fiber types.[15] Rogers et al. reported a significant association between mesothelioma risk and lung concentrations of short chrysotile fibers (<10 μm) in multivariate models and a significant trend for lung concentrations among mesothelioma case and control subjects who had only chrysotile detected in their lungs.[16]

The interpretation of the results from the studies of lung burden is complicated by differences in the respiratory clearance rates of the different forms of asbestos. Experimental studies demonstrated that chrysotile fibers are cleared far more rapidly from the lungs than are amphibole fibers.[17–19] The retention half-life of chrysotile in human lungs is unknown, but a half-life of 90 days has been reported in experimental studies of baboons.[20] If the half-life for chrysotile is similar for humans and baboons, then clearly the vast majority of the dose received in early years would not be reflected in the lung burdens measured at the time of autopsy. This is of particular concern for mesothelioma, which has been estimated to have a latency period of at least 20 years.[21] For example, assuming a 90-day half-life and first-order kinetics, only approximately $1/(8 \times 10^{22})$ of the dose received 20 years earlier would be predicted to be present in the lungs at the time of the autopsy. Hence, lung burdens of chrysotile may be a poor measure of the integrated exposures to chrysotile.

The high degree of correlation between the lung concentrations of the different fiber types, which has been noted by several investigators, further complicates the interpretation of the lung burden analyses.[15,16,23] Churg reported that the correlation coefficient between the numbers of chrysotile and crocidolite fibers in lungs of asbestosis patients was .88 ($P < .05$).[23] Rowlands et al. reported a stronger correlation between cumulative asbestos exposure and lung fiber counts for tremolite than between cumulative asbestos exposure and lung burdens of chrysotile in their study of Quebec miners and millers.[8] The high degree of correlation might explain the negative findings in some of the case-control studies if amphibole exposures are simply acting as a surrogate for integrated lifetime chrysotile exposure in these studies. As Churg et al. suggested, "It may be true that the tremolite serves as a better measure of past chrysotile than the chrysotile itself."[19]

Finally, studies of fiber counts in extrapulmonary sites raise serious questions about the validity of using lung burden studies for assessing mesothelioma risk. Several investigators reported cases in which short chrysotile fibers were the predominant fiber found in the pleura, pleural plaques, or pleural fibrotic tissue when amphiboles were the predominant fiber found in the lung.[22,24–26] These results suggest that chrysotile may be preferentially translocated to the pleura and that the fiber counts found in the lung may not accurately reflect the concentrations found at the site for mesothelioma induction.

Epidemiologic Studies

Lung Cancer

There have been 12 retrospective cohort mortality studies of workers who were predominantly exposed to chrysotile asbestos fibers. Results for mortality from lung cancer (and mesothelioma) from the most recent updates of these cohorts are summarized in Table 1. Mortality from lung cancer was greater than expected in nearly all of the studies. Combining the results from these studies, there were 928 observed and 618.9 expected lung cancer deaths, resulting in a pooled standardized mortality ratio for lung cancer of 1.50 (95% confidence interval [CI] = 1.40, 1.60). The observed excesses of lung cancer mortality did not appear to be explained by differences in cigarette smoking habits in the studies that had information on tobacco consumption.[28,33,35,36,40,41] Collectively, these studies provide strong evidence that exposure to chrysotile asbestos is associated with an excess risk of lung cancer.

There is little, if any, evidence to suggest that the excess in lung cancer mortality observed in these cohorts may be attributable to tremolite contamination. In fact, this hypothesis is strongly contradicted by the fact that the lung cancer response in the studies of populations with relatively pure chrysotile exposures is similar to that in studies of cohorts with amphibole or mixed exposures. Estimates of the increase in excess relative risk per unit of exposure (i.e., potency) for lung cancer based on cohort studies by industry and fiber type are presented in Table 2. Variations in risk according to

industry type appear to be far more remarkable than variations according to fiber type. The potencies for lung cancer risk are similar among the cohorts with pure chrysotile and mixed exposures in the textile industry and are generally higher than the potencies observed among workers in the mining or asbestos products industries. The studies of asbestos products industry workers all show very low potencies, with the lowest unit risks observed among friction product workers. One study of cement workers, which provided separate analyses for workers exposed to chrysotile asbestos and workers exposed to a mix of chrysotile and crocidolite fibers, produced remarkably similar potency estimates for these two groups.[32] Among the studies of miners, lung cancer potency was substantially lower among workers in the Quebec mining industry who were exposed to chrysotile ores than among crocidolite or tremolite miners.

It has been suggested that the high lung cancer mortality observed among South Carolina textile workers might be explained by exposure to mineral oils.[47] However, Dement et al. demonstrated in case–control analyses that the risk of lung cancer observed in this cohort is unrelated to mineral oil exposure.[29,48] In addition, studies of workers exposed to mineral oils have generally not demonstrated an excess of lung cancer.[49] There is evidence that asbestos fibers in the textile industry were considerably longer than the fibers measured in chrysotile mining and milling and other industries.[50] Thus, differences in fiber dimensions would appear to be a more likely explanation than mineral oil exposures for the higher lung cancer rates observed in textile workers.

TABLE 2—Estimates of Asbestos Potency for Lung Cancer from Studies with Individual Exposure Estimates, by Industry and Fiber Type

Study	Industry	Fiber Type	Excess Relative Risk per Fiber/cc × Yr
Dement et al.[29]	Textiles	Chrysotile	0.031
McDonald et al.[12]	Mainly textiles	Chrysotile, amosite, crocidolite	0.017[a]
Peto et al.[42]	Textiles	Chrysotile, crocidolite	0.015[b]
McDonald et al.[43]	Mining	Tremolite	0.013
de Klerk et al.[44]	Mining and milling	Crocidolite	0.010
McDonald et al.[36]	Mining and milling	Chrysotile	0.0006[a,c]
Henderson and Enterline[45]	Asbestos products	Chrysotile, amosite, crocidolite	0.002[a]
Hughes et al.[32]	Cement products	Chrysotile,[a] chrysotile,[b] and crocidolite	0.0071,[a] 0.0076[b]
Berry and Newhouse et al.[46]	Friction products	Chrysotile	0.00058
McDonald et al.[34]	Friction products	Chrysotile	0.00053[a]

[a]A conversion factor of three fibers per cubic centimeter being equivalent to 1 million particles per cubic foot was assumed.
[b]Data are based on results for workers employed after 1951.
[c]Slope was estimated by fitting a linear relative risk Poisson regression model to the standardized mortality ratio results reported by McDonald et al.[36]

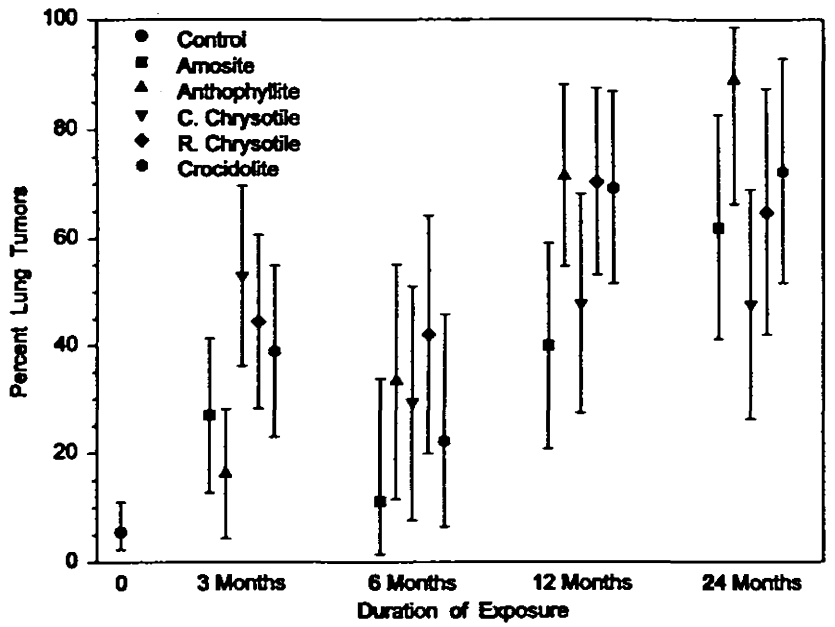
Mesothelioma

A total of 45 cases of mesothelioma (primarily pleural) were reported in the epidemiologic studies of workers who were predominantly exposed to chrysotile asbestos (Table 1). Although it has generally not been possible to estimate expected numbers of mesothelioma deaths, the percentage of deaths due to mesothelioma may be estimated and compared with background percentages. This percentage is 0.3% for all studies combined. In contrast, the percentage of deaths due to pleural malignancies (most of which are mesotheliomas) was only 0.02% in the United States in 1988.[51]

Although the evidence of excess mortality of mesothelioma among workers exposed to commercial chrysotile is compelling, the critical issue is whether this excess may be attributable to trace contamination by tremolite. All of the asbestos workers studied (Table 1) are likely to have potential exposures to tremolite, although in minute concentrations compared with their chrysotile exposures.

In a few studies the percentage of tremolite is known and varies. Contrasting the results from these studies provides some information on the plausibility of the amphibole hypothesis. Two cases of mesothelioma have been reported among chrysotile asbestos miners and millers in Zimbabwe, where the chrysotile ores are believed to be free of tremolite contamination.[52] Begin et al. noted that although exposure to tremolite may be as much as 7.5 times higher in Thetford than in Asbestos, the incidence of mesothelioma in these two Quebec mining towns was proportional to the size of their work forces.[53] He suggested that this fact may indicate that tremolite contamination may not be a determinant of mesothelioma risk in Quebec. In the most recent update of the study of Quebec miners and millers, McDonald et al.[36] presented separate exposure–response analyses for workers at the Thetford and Asbestos mines and mills. There is no indication in their findings that these two facilities exhibit a different exposure–response relationship for mesothelioma. On the other hand, McDonald and McDonald[54] recently reported that the average concentration of tremolite fibers in the lungs of miners was higher in one area of the Thetford mine, which also demonstrated a stronger association with mesothelioma risk than another area of the mine.

Informative comparisons may also be made between the proportion of deaths from mesothelioma observed in the South Carolina textile workers study and that observed in the Quebec miners and millers study. Based on lung burden studies, Sebastien et al. estimated that the proportion of tremolite in dust was probably 2.5 times higher in the Thetford mines of Quebec than in the Charleston textile facility.[47] The percentage of deaths due to mesothelioma in the most recent reports was one half as high in the South Carolina textile workers (0.2%) as it was among Quebec miners and millers (0.4%) (Table 1). However, in making this comparison one needs to consider the fact that the incidence of mesothelioma is known to increase exponentially with follow-up time,[55] and 72% of the Quebec miners and millers had died,[36] compared with 42% of the workers in the South Carolina study,[29] in the most recent updates of these cohorts. In the previous



Note. Data are from Wagner et al.[17]; approximate 95% confidence intervals for a binomial outcome have been added. C = Canadian; R = Rhodesian.

FIGURE 1—Lung tumors in rats exposed to 10 mg/m³ concentrations of asbestos for 3, 6, 12, or 24 months.

update of the Quebec miners and millers study, the percentage that had died was 41% and the percentage of deaths due to mesothelioma was 0.2%, which is nearly identical to the percentage of deaths from mesothelioma in the most recent update of the South Carolina textile workers.[35] The fact that these percentages are so similar is even more remarkable when it is recognized that the fiber exposure levels were approximately ten times higher in the Quebec miners and millers than in the South Carolina textile workers.[47] Thus, comparison of the mesothelioma results from the study of Quebec miners and millers with those from the study of South Carolina textile workers does not provide support for the hypothesis that tremolite exposure explains the mesothelioma excess observed in these studies.

In contrast to the evidence for lung cancer, there is epidemiologic evidence indicating that exposure to chrysotile may be less potent than exposure to some amphiboles with regards to the induction of mesothelioma. Hughes and Weill estimated that the risk of mesothelioma was approximately five times lower among workers exposed to chrysotile fibers than among workers with mixed fiber exposure.[56] The percentage of deaths due to mesothelioma among South African asbestos miners was recently reported to be 4.7% among those exposed to crocidolite, which is substantially greater than the percentage of deaths due to mesothelioma observed in either the Quebec miners (0.4%) or the South Carolina textile workers (0.2%) exposed to predominantly chrysotile fibers.[57] The percentage of deaths due to mesothelioma was only slightly higher among South African miners exposed to amosite (0.6%) than among the chrysotile-exposed cohorts.[57] McDonald et al.[43] reported that the percentage of deaths due to mesothelioma was 2.4% among vermiculite miners who were predominantly exposed to tremolite fibers, which is approximately six times higher than the percentage (0.4%) reported in the study of Quebec miners and millers.[36] It must be recognized that the usefulness of these comparisons is limited by our inability to control for potential differences in exposure concentrations, fiber size distributions, and length of observation and are thus difficult to interpret. Nonetheless, the differences in mesothelioma response observed among chrysotile- and amphibole (primarily crocidolite)-exposed workers are so striking that alternative explanations for these differences appear unlikely.

Toxicologic Studies

Lung Cancer

Toxicologic studies demonstrated that all forms of asbestos can induce lung cancers in experimental animals. For example, the lung tumor response to 3- to 24-month exposures to Union International Contre le Cancer reference amosite, anthophyllite, Canadian chrysotile, Rhodesian chrysotile, and crocidolite is shown in Figure 1.[17] The overlapping 95% confidence intervals suggest that there is no significant difference in potency among the five types of asbestos (i.e., the amphiboles are not systematically more or less potent than the chrysotiles).

Davis and co-workers also compared the carcinogenic potencies of chrysotile and amphibole asbestos by exposing rats to 10 mg of amosite, crocidolite, and Zimbabwe chrysotile per m^3 for 1 year. These investigators found that chrysotile actually produced more lung tumors than the other forms of asbestos.[58] These results obviously differ from those of Wagner et al.[17] and may point to the need to consider differences in fiber length when comparing the potencies of different types of asbestos. Davis et al. noted that 5% of the chrysotile in their study consisted of fibers greater than 20 μm in length vs 0.5% of the fibers for the amosite and crocidolite exposures.[58] Other studies by Davis et al. showed that long-fiber samples of amosite[59] and chrysotile[60] are considerably more active than short-fiber samples in inducing lung tumors.

Davis et al. also showed that tremolite,[61] crocidolite,[58] and long-fiber chrysotile[60] produce similar numbers of lung tumors. Figure 2 represents lung tumors due to amosite, crocidolite, chrysotile, or tremolite from the 1-year inhalation studies of Davis et al. and Davis and Jones, plotted against the exposure concentration in units of fiber count.[58-61] Inspection of Figure 2 suggests that the tumor incidence is strongly related to the concentration of fibers 5 μm or greater in length, regardless of which type of asbestos is involved.

More recently, Coffin et al.[62] reported the results from studies of rats exposed via intratracheal instillation of chrysotile or crocidolite. Although these investigators focused primarily on mesotheliomas, it is worth noting that (summed across all dose groups) intratracheal instillation of chrysotile asbestos produced

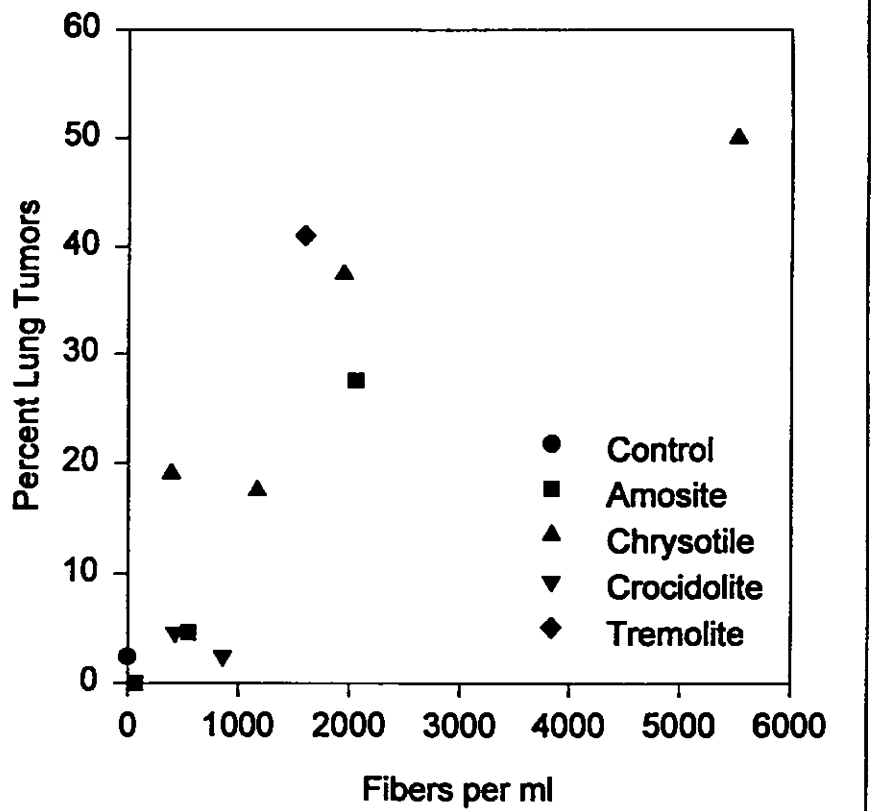
lung carcinomas in 18.3% of the animals tested vs 4.6% for crocidolite.[62]

Overall, the toxicologic data suggest that chrysotile asbestos is at least as potent, if not more so, as the amphibole forms in the induction of lung tumors on a per-milligram basis. The data shown in Figure 2 further suggest that the carcinogenic potencies of the various types are similar when the dosage is measured in terms of the number of fibers greater than 5 μm in length, as is customary in epidemiologic studies.

Mesothelioma

Rats exposed to asbestos by inhalation also develop mesotheliomas, albeit at a low incidence. Wagner et al.[17] exposed rats to 10 mg/m³ of Union International Contre le Cancer reference asbestos[63] for periods of 1 day to 2 years; the mesothelioma yields were amosite, 0.7%; anthophyllite, 1.4%; crocidolite, 2.8%; and Canadian chrysotile, 2.9%. No mesotheliomas were observed in control animals or animals exposed to chrysotile from Zimbabwe.[17] Similarly, Davis et al. and Davis and Jones reported small numbers of mesotheliomas in response to 1-year inhalation exposures to amosite, crocidolite, Canadian chrysotile, and Zimbabwe chrysotile.[58-60] The highest mesothelioma incidence in these studies, 7.5%, was produced by exposure to long-fiber chrysotile.[60] Although the low incidence rates and small numbers of animals make quantitative comparisons uncertain, it cannot be said that these studies provide convincing support for the amphibole hypothesis.

The mesothelioma-inducing potential of asbestos fibers that reach pleural surfaces has also been examined via implantation studies. Union International Contre le Cancer reference amosite, anthophyllite, crocidolite, Canadian chrysotile, and Zimbabwe chrysotile all produced mesotheliomas in rats after intrapleural inoculation.[64] Extensive studies by Stanton and co-workers suggest that all long, thin, durable fibers have the potential to induce mesotheliomas after surgical implantation and that fiber dimensions have much more influence on mesothelioma yield than any differences that may exist between types of asbestos.[65] However, it is certainly possible that different types of asbestos fibers may have differing probabilities of reaching pleural surfaces when inhaled into the lungs. Overall, the implantation studies suggest that chrysotile asbestos does have the potential to induce mesothelioma, but



Note. Data are from Davis et al. and Davis and Jones.[58-61] Controls are the pooled control animals from all four studies.

FIGURE 2—Lung tumors in rats exposed to 10 mg/m³ concentrations of crocidolite, amosite, chrysotile, or tremolite for 1 year.

these studies do not resolve the question of whether or not chrysotile is less potent in this regard than the amphibole forms.

Coffin et al. recently reported that both chrysotile and crocidolite produce mesotheliomas when administered intratracheally.[62] No consistent dose–response relationship was observed in these experiments, but (summing across all dose groups) chrysotile asbestos produced mesotheliomas in 9.5% of the animals vs 5.1% for crocidolite. This suggests that chrysotile may have greater mesothelioma-inducing potential than crocidolite on a per-milligram basis. However, the chrysotile preparation used in this experiment contained more fibers per milligram than the crocidolite preparation, as well as a larger proportion of long fibers. If the experimental exposures are expressed on the basis of the number of fibers greater than 5 μm in length, it appears that crocidolite produced nearly 12 times more mesotheliomas per fiber than chrysotile. It should be noted that the fiber preparations in the Coffin et al. experiments consisted primarily of short fibers, with median fiber lengths on the order of 1 μm for both chrysotile and crocidolite. If short fibers do in fact have some mesothelioma-inducing potential, the attribution of all mesotheliomas to the small fraction of the fibers that were greater than 5 μm in length may lead to an exaggerated estimate of the difference in potency of crocidolite vs chrysotile. In addition, reliance on the quantitative responses in this study should probably be limited due to the lack of dose–response. Nevertheless, these data do provide some support for the hypothesis that chrysotile may have lower mesothelioma-inducing potential than the amphibole forms of asbestos.

Mechanistic Studies

It has been hypothesized that the cytotoxic, genotoxic, and proliferative effects of asbestos are in part mediated by the production of reactive oxygen species released by alveolar macrophages in response to engulfment of long fibers and

that this process may be catalyzed by iron on the fiber surface. Furthermore, it has been suggested that the needle-like configuration, durability, and increased iron content of crocidolite render it more pathogenic than either amosite or chrysotile.[66] Experimental support for this hypothesis is primarily derived from in vitro studies, which suggest that iron could potentially act as a source of free radicals, an inhibitor of tumoricidal defense mechanisms, and a nutrient for unrestricted tumor cell replication.[67] However, comparison of the carcinogenic potencies of fibers in the rat in vivo does not support the hypothesis that carcinogenic potency is related to iron content. As discussed above, Wagner et al.[17] observed similar numbers of tumors in rats with crocidolite, amosite, and chrysotile, even though these fibers have an elemental iron content of 40%, 28%, and less than 1%, respectively.[67] The nonasbestos mineral erionite does not include iron as a constituent[68] but is nonetheless a potent mesothelioma inducer in rats.[69] Silicon carbide "whiskers," with an iron content of essentially zero, induce pleural tumors in rats after intrapleural implantation.[65] Therefore, no obvious correlation between iron content and carcinogenicity is apparent in the rat.

Summary

Our review of both the toxicologic and epidemiologic literature strongly supports the view that occupational exposure to chrysotile asbestos is associated with an increased risk of both lung cancer and mesothelioma. The hypothesis that these observations may be attributable to trace amounts (<1%) of tremolite contamination may seem to be primarily of academic interest, because chrysotile exposures in workers and the public are also contaminated with tremolite. However, the percentage of tremolite has been reported to range from 0.5% to 6.9% in one analysis of eight commercial chrysotile asbestos samples,[6] and it has been suggested that chrysotile from Zimbabwe[70] and other countries may be free of contamination by amphiboles. Hence, the amphibole hypothesis may be of some public health relevance.

In our view, the currently available scientific literature does not provide persuasive evidence for the hypothesis that tremolite contamination explains the mesothelioma excesses observed in the studies of chrysotile-exposed workers. The primary evidence for this hypothesis comes from pathologic studies in which lung burdens were measured. However, interpretation of these studies is hampered by the fact that chrysotile lung burdens are a poor reflection of integrated exposures and the fact that chrysotile exposure is highly correlated with lung burden of the amphiboles (e.g., tremolite). In addition, the pattern of asbestos fiber deposition in the lung does not appear to be consistent with the pattern of deposition in the target tissue (i.e., pleura). The previously reviewed empirical data from toxicologic studies and comparisons of mesothelioma mortality and lung cancer mortality between epidemiologic studies with differing levels of tremolite contamination do not provide support for this hypothesis. Mechanistic arguments that have been made to support the amphibole hypothesis, which are based on in vitro studies of iron content, appear to be contradicted by the lack of correlation between iron content and carcinogenic potency observed in experimental studies.

Whether chrysotile asbestos is less potent than the amphibole forms of asbestos is a question that has not yet been fully resolved. There is currently very little toxicologic evidence to support this hypothesis. There is evidence from epidemiologic studies that chrysotile may be less potent for mesothelioma induction than crocidolite. The proportion of deaths due to mesothelioma are strikingly lower in chrysotile-exposed miners and millers than in crocidolite miners. There is absolutely no epidemiologic or toxicologic evidence to support the argument that chrysotile asbestos is any less potent than other forms of asbestos for inducing lung cancer.

It should be recognized that comparisons of the potency of the different forms of asbestos are severely limited by uncontrolled differences in the bivariate distribution of fiber length and diameter (i.e., fiber dimensions). Experimental studies clearly demonstrated that fiber dimensions are a critical component of the carcinogenic potency of fibers.[65] This concern applies to most of the toxicologic studies in which exposure is determined on an equal mass basis and is particularly pertinent to the epidemiologic investigations. Historic exposures in most of the epidemiologic investigations were based on impinger samples that assessed the number of fibers, and conversion factors were applied to estimate the number of fibers longer than 5 μm. Concerns have been raised about the accuracy of these conversion factors and the potential impact of associated errors on the assessment of risk.[71] The current Occupational Safety and Health Administration (OSHA) method counts asbestos fibers that are longer than 5 μm and that have a length-to-diameter ratio of at least 3 to 1. This method implicitly assumes that fibers less than 5 μm in length are not carcinogenic and that all fibers greater than 5 μm in length are of equal carcinogenic potency. These assumptions are clearly inconsistent with the experimental data and most likely result in substantial misclassification of exposure in the epidemiologic studies.

Policy Implications

The American Conference of Governmental Industrial Hygienists and several countries (e.g., the United Kingdom) have adopted less restrictive standards for chrysotile asbestos than for the other forms of asbestos.[72] In our view, the currently available scientific evidence does not provide sufficient support for developing separate standards for the different forms of asbestos. As this article documents, the scientific evidence for the amphibole hypothesis is still tenuous. Furthermore, the fact remains that in practice workers in this country and other countries are not exposed to pure chrysotile, but rather to a mixture of chrysotile, tremolite, and other forms of asbestos. Thus, it is highly impractical to consider setting separate standards for the different forms of asbestos. Finally, even if one accepts the argument that chrysotile asbestos does not induce mesothelioma (which we do not), the risk of lung cancer (and asbestosis) can not be dismissed, and chrysotile appears to be just as potent a lung carcinogen as the other forms of asbestos. It is noteworthy that the risk of lung cancer is of greater concern than the risk of mesothelioma because in most studies there are at least two excess lung cancers for every mesothelioma observed (see Table 1). There is also the additional concern of asbestosis risk, which was not considered in this article but clearly adds to the risk associated with chrysotile exposure.

Therefore, given the clear evidence of a lung cancer risk, the lack of compelling evidence for the amphibole hypothesis, and the fact that workers are generally exposed to mixture of fiber types, we believe that it is prudent policy to treat chrysotile asbestos with virtually the same level of concern as the amphibole forms of asbestos. This view is consistent with the

past National Institute for Occupational Safety and Health Administration recommendation and the recently revised OSHA standard to limit occupational exposures for all forms of asbestos to 0.1 fiber/cc. □

References

1. Pigg BJ. The uses of chrysotile. *Ann Occup Hyg.* 1994;38:453–458.
2. Lemen RA, Bingham E. A case study in avoiding a deadly legacy in developing countries. *Toxicol Ind Health.* 1994;10(1/2): 59–87.
3. Mossman BT, Bignon J, Corn M, Seaton A, Gee JBL. Asbestos: scientific developments and implications for public policy. *Science.* 1990;24:294–301.
4. Dunnigan, J. Linking chrysotile asbestos with mesothelioma. *Am J Ind Med.* 1988;14: 205–209.
5. Pooley FD, Clark NJ. Quantitative assessment of inorganic fibrous particulates in dust samples with an analytical transmission electron microscope. *Ann Occup Hyg.* 1979;22:253–271.
6. Addison J, Davies LST. Analysis of amphibole asbestos in chrysotile and other minerals. *Ann Occup Hyg.* 1990;34:159–175.
7. Pooley FD. An examination of the fibrous mineral content of asbestos lung tissue form the Canadian chrysotile mining industry. *Environ Res.* 1976;12:281–298.
8. Rowlands N, Gibbs GW, McDonald AD. Asbestos fibres in the lungs of chrysotile miners and millers—a preliminary report. *Ann Occup Hyg.* 1982;26:411–415.
9. Churg A, Warnock ML. Asbestos fibers in the general population. *Am Rev Respir Dis.* 1980;122:669–678.
10. Jones JSP, Roberts GS, Pooley FD, et al. The pathology and mineral content of lungs in cases of mesothelioma in the United Kingdom in 1976. In: Wagner JC, ed. *Biological Effects of Mineral Fibers.* Lyon, France: International Agency for Research on Cancer; 1980:188–199. Scientific Publication No. 30.
11. Wagner JC, Pooley FD, Berry G, et al. A pathological and mineralogical study of asbestos-related deaths in the United Kingdom in 1977. *Ann Occup Hyg.* 1982;26:423–431.
12. McDonald AD, McDonald JC, Pooley FD. Mineral fibre content of lung in mesothelial tumours in North America. *Ann Occup Hyg.* 1982;26:417–422.
13. Gaudichet A, Janson X, Monchaux G, et al. Assessment by analytical microscopy of the total lung fibre burden in mesothelioma patients matched with four other pathological series. *Ann Occup Hyg.* 1988; 32(suppl 1):213–223.
14. Wagner JC, Berry G, Pooley FD. Mesotheliomas and asbestos type in asbestos textile workers: a study of lung contents. *Br Med J.* 1982;285:603–606.
15. McDonald JC, Armstrong B, Case B, et al. Mesothelioma and asbestos fiber type: evidence from lung tissue analyses. *Cancer.* 1989;63:1544–1547.
16. Rogers AJ, Leigh J, Berry G, Ferguson DA, Mulder HB, Ackad M. Relationship between lung asbestos fiber type and concentration and relative risk of mesothelioma. *Cancer.* 1991;67:1912–1920.
17. Wagner JC, Berry G, Skidmore JW, Timbrell V. The effects of the inhalation of asbestos in rats. *Br J Cancer.* 1974;29:252–269.
18. Middleton AP, Beckett ST, Davis JMG. A study of the short-term retention and clearance of inhaled asbestos by rats, using U.I.C.C. standard reference samples. In: Walton WH, ed. *Inhaled Particles IV.* Edinburgh, Scotland: Institute of Occupational Medicine; 1975:247–258.
19. Churg A, Wright JL, Vedel S. Fiber burden and patterns of asbestos-related disease in chrysotile miners and millers. *Am Rev Respir Dis.* 1993;48:25–31.
20. Rendall RE. *Retention and Clearance of Glass Fibers and Different Varieties of Asbestos by the Lung.* Johannesburg, South Africa: University of Witwatersrand; 1988. Dissertation.
21. Selikoff IJ, Hammond EC, Seidman H. Mortality experience of insulation workers in the United States and Canada, 1943–1976. *Ann N Y Acad Sci.* 1979;330:91–116.
22. Sebastien P, Janson X, Gaudichet A, Hirsch A, Bigon J. Asbestos retention in human respiratory tissues: comparative measurements in lung parenchyma and in parietal pleura. In: Wagner JC, ed. *Biological Effects of Mineral Fibers.* Lyon, France: International Agency for Research on Cancer; 1980:237–246.
23. Churg A. Asbestos fiber content of the lungs in patients with and without asbestos airways disease. *Am Rev Respir Dis.* 1983; 127:470–473.
24. LeBouffant L, Martin JC, Duyif S, Daniel H. Structure and composition of pleural plaque. In: Bogovski P, Gilson JC, Timbrell V, Wagner JC, eds. *Biological Effects of Asbestos.* Lyon, France: International Agency for Research on Cancer; 1973:249–257. Scientific Publication No. 8.
25. Dodson RF, Williams MG, Corn CJ, Brollo A, Bianchi C. Asbestos content of lung tissue, lymph nodes, and pleural plaques from former shipyard workers. *Am Rev Respir Dis.* 1990;142:843–847.
26. Kohyama N, Suzuki Y. Analysis of asbestos fibers in lung parenchyma, pleural plaques, and mesothelioma tissues of North American insulation workers. *Ann N Y Acad Sci.* 1991;643:27–52.
27. Acheson ED, Gardner MJ, Pippard EC, Grime LP. Mortality of two groups of women who manufactured gas masks from chrysotile and crocidolite asbestos: a 40-year follow-up. *Br J Ind Med.* 1982;39:344–348.
28. Cheng W, Kong J. A retrospective mortality cohort study of chrysotile asbestos products workers in Tianjin 1972–1987. *Environ Res.* 1992;59:271–278.
29. Dement JM, Brown DP, Okun A. Mortality among chrysotile asbestos textile workers: Cohort mortality and case-control analyses. *Ann Occup Hyg.* 1994;38:525–532.
30. Finkelstein MM. Mortality among employees of an Ontario factory that manufactured construction materials using chrysotile asbestos and coal tar pitch. *Am J Ind Med.* 1989;16:281–287.
31. Finkelstein MM. Mortality rates among employees potentially exposed to chrysotile asbestos at two automotive parts factories. *Can Med Assoc J.* 1989;141:327–330.
32. Hughes JM, Weill H, Hammad YY. Mortality of workers employed in two asbestos cement manufacturing plants. *Br J Ind Med.* 1987;44:161–174.
33. Huilan Z, Zhiming W. Study of occupational lung cancer in asbestos factories in China. *Br J Ind Med.* 1993;50:1039–1042.
34. McDonald AD, Fry JS, Woolley AJ, McDonald JC. Dust exposure and mortality in an American chrysotile asbestos friction products plant. *Br J Ind Med.* 1984;41:151–157.
35. McDonald JC, Liddell FDK, Gibbs GW, Eyssen GE, McDonald AD. Dust exposure and mortality in chrysotile mining, 1910–1975. *Br J Ind Med.* 1980;37:11–24.
36. McDonald JC, Liddell FDK, Dufresne A, McDonald AD. The 1891–1920 birth cohort of Quebec chrysotile miners and millers: mortality 1976–88. *Br J Ind Med.* 1993;50:1073–1081.
37. Piolatto G, Negri E, LaVecchia C, Pira E, Decarli A, Peto J. An update of cancer mortality among chrysotile asbestos miners in Balangero, Northern Italy. *Br J Ind Med.* 1990;47:810–814.
38. Shiqu Z, Yongxian W, Fusheng M, Hongshuen M, Wenzhi S, Zhenhuan J. Retrospective mortality study of asbestos workers in Laiyuan. In: *Proceedings of the VII International Pneumoconioses Conference,* Part II; August 23–26, 1988; Pittsburgh, Pa. National Institute for Occupational Safety and Health; 1990:1242–1244. DHHS publication 90-109, part II.
39. Weiss W. Mortality of a cohort exposed to chrysotile asbestos. *J Occup Med.* 1977;19: 737–740.
40. Dement JM, Harris RL, Symons MJ, Shy CM. Exposures and mortality among chrysotile asbestos workers, part I: exposure estimates. *Am J Ind Med.* 1983;4:399–419.
41. Dement JM, Harris RL, Symons MJ, Shy CM. Exposures and mortality among chrysotile asbestos workers, part II: mortality. *Am J Ind Med.* 1983;4:421–433.
42. Peto J, Doll R, Hermon C, Binns W, Clayton R, Goffe T. Relationship of mortality to measures of environmental asbestos pollution in an asbestos textile factory. *Ann Occup Hyg.* 1985;29:305–355.
43. McDonald JC, McDonald AD, Armstrong B, Sebastien P. Cohort study of mortality of vermiculite miners exposed to tremolite. *Br J Ind Med.* 1986;43:436–444.
44. de Klerk NH, Armstrong BK, Musk AW, Hobbs MST. Cancer mortality in relation to measures of occupational exposure to crocidolite at Wittenoom Gorge in Western Australia. *Br J Ind Med.* 1989;46:529–536.
45. Henderson VL, Enterline PE. Asbestos exposure: Factors associated with excess cancer and respiratory disease mortality. *Ann N Y Acad Sci.* 1979;117–126.
46. Berry G, Newhouse ML. Mortality of workers manufacturing friction materials using asbestos. *Br J Ind Med.* 1983;40:1–7.
47. Sebastien P, McDonald JC, McDonald AD, Case B, Harley R. Respiratory cancer in chrysotile textile and mining industries: exposure inferences from lung analysis. *Br J Ind Med.* 1989;46:180–187.
48. Dement JM. Carcinogenicity of chrysotile

asbestos: a case control study of textile workers. *Cell Biol Toxicol.* 1991;7:59–65.
49. Tolbert P, Eisen E, Pothier LJ, Monson RR, Hallock MF, Smith TJ. Mortality studies of machining-fluid exposure in the automobile industry, II: risks associated with specific fluid types. *Scand J Work Environ Health.* 1992;18:351–360.
50. Dement JM and Wallingford KM. Comparison of phase contrast and electron microscopic methods for evaluation of occupational asbestos exposures. *Appl Occup Environ Hyg.* 1990;5:242–247.
51. *Vital Statistics of the United States, 1989,* Vol II—Mortality, Part B. Hyattsville, Md: National Center for Health Statistics; 1992. DHHS publication PHS 92-1102.
52. Cullen MR and Baloyi RS. Chrysotile asbestos and health in Zimbabwe, I: analysis of miners and millers compensated for asbestos-related diseases since independence (1980). *Am J Ind Med.* 1991;29:161–169.
53. Begin R, Gauthier J, Desmeules M, Ostiguy G. Work-related mesothelioma in Quebec, 1967–1990. *Am J Ind Med.* 1992;22:531–542.
54. McDonald JC, McDonald AD. Chrysotile, tremolite and mesothelioma. *Science.* February 10, 1995;267:775–776.
55. Peto J, Seidman H, Selikoff IJ. Mesothelioma mortality in asbestos workers: implications for models of carcinogenesis and risk assessment. *Br J Cancer.* 1982;45:124–135.
56. Hughes JM, Weill H. Asbestos Exposure-Quantitative Assessment of Risk. *Am Rev Respir Dis.* 1986;133:5–13.
57. Sluis-Cremer GK, Liddell FDK, Logan WPD, Bezuidenhout BN. The mortality of amphibole miners in South Africa, 1946–80. *Br J Ind Med.* 1992;49:566–575.
58. Davis JMG, Beckett ST, Bolton RE, Collins P, Middleton AP. Mass and number of fibres in the pathogenesis of asbestos-related lung disease in rats. *Br J Cancer.* 1978;37:673–688.
59. Davis JMG, Addison J, Bolton RE, Donaldson K, Jones AD, Smith T. The pathogenicity of long versus short fibre samples of amosite asbestos administered to rats by inhalation and intraperitoneal injection. *Br J Exp Pathol.* 1986;67:415–430.
60. Davis JMG, Jones AD. Comparisons of the pathogenicity of long and short fibres of chrysotile asbestos in rats. *Br J Exp Pathol.* 1988;69:717–737.
61. Davis JMG, Addison J, Bolton RE, Donaldson K, Jones AD, Miller BG. Inhalation studies on the effects of tremolite and brucite dust in rats. *Carcinogenesis.* 1985;6:667–674.
62. Coffin DL, Cook PM, Creason JP. Relative mesothelioma induction in rats by mineral fibers: comparison with residual pulmonary mineral fiber number and epidemiology. *Inhal Toxicol.* 1992;4:273–300.
63. Timbrell V, Gibson JC, Webster I. UICC standard reference samples of asbestos. *Int J Cancer.* 1968;3:406–408.
64. Wagner JC, Berry G, Timbrell V. Mesotheliomata in rats after inoculation with asbestos and other materials. *Br J Cancer.* 1973;28:173–185.
65. Stanton MF, Layard M, Tegeris A, et al. Relation of particle dimension to carcinogenicity in amphibole asbestoses and other fibrous minerals. *J Natl Cancer Inst.* 1981;67:965–975.
66. Mossman BT. Mechanisms of asbestos carcinogenesis and toxicity: the amphibole hypothesis revisited. *Br J Ind Med.* 1993;50:673–676. Letter.
67. Weinberg ED. Association of iron with respiratory tract neoplasia. *J Trace Elem Exp Med.* 1993;6:117–123.
68. *Kirk-Othmer Encyclopedia of Chemical Technology.* 3rd ed. Vol 15. New York, NY: John Wiley & Sons; 1981:639.
69. Davis JMG. Mineral fibre carcinogenesis: experimental data relating to the importance of fibre type, size, deposition, dissolution, and migration. In: Bignon J, Peto J, Saracci R, eds. *Non-occupational Exposure to Mineral Fibers.* Lyon, France: International Agency for Research on Cancer; 1989:33–45.
70. Baloyi R. *Exposure to Asbestos among Chrysotile Miners, Millers and Mine Residents and Asbestosis in Zimbabwe.* Helsinki, Finland: University of Kuopio; 1989. Dissertation.
71. Peto J. Fibre carcinogenesis and environmental hazards. In: Bignon J, Peto J, Saracci R, eds. *Non-occupational Exposure to Mineral Fibres.* Lyon, France: International Agency for Research on Cancer; 1989:457–470.
72. *Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices.* Cincinnati, Ohio: American Conference of Governmental Industrial Hygienists; 1994–1995.

WORK-RELATED LUNG DISEASE SURVEILLANCE REPORT 1996

Division of Respiratory Disease Studies
National Institute for Occupational Safety and Health

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

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Preface

This 1996 *Work-Related Lung Disease (WoRLD) Surveillance Report* is the fourth in a series of occupational respiratory disease surveillance reports produced by the National Institute for Occupational Safety and Health (NIOSH). The purpose of this 1996 report is to provide national and state-specific summaries of occupational respiratory disease surveillance data focusing on pneumoconiosis mortality. Selected occupational respiratory hazard sampling data relevant to pneumoconiosis are also presented.

The 1996 *WoRLD Surveillance Report* has three sections: 1) a highlights and limitations section that provides data highlights and data usage limitations; 2) a United States section that serves to update and expand overall national data provided in the 1994 *WoRLD Surveillance Report;* and 3) a state section that provides detailed profiles of pneumoconiosis data for each state in the U.S..

The United States section updates pneumoconiosis mortality surveillance data published previously in the 1994 *WoRLD Surveillance Report*, by including data available for 1991 and 1992. For each condition, this section presents national data such as counts, crude and age-adjusted mortality rates, and years of potential life lost to age 65 to and life expectancy. Proportionate mortality ratios by industry and occupation, are based on data from a subset of states (see state list, Appendix C) for which usual industry and occupation have been coded for decedents. Also presented are U.S. county level maps showing the geographic distribution of mortality for each pneumoconiosis condition. In addition, this section presents selected occupational exposure sampling data for asbestos, coal and coal mine dust, silica dust, cotton dust, etc. (see agent categories, Appendix D).

The State section provides more detailed pneumoconiosis mortality surveillance data for each state and for the District of Columbia. The State section is organized so that tables and graphs of data for each state are grouped together. Selected graphs, tables, and maps present pneumoconiosis mortality from 1968 to 1992 for each state, as well as for counties within each state. Surveillance data include counts, crude and age-adjusted mortality rates, and years of potential life lost to life expectancy.

Pneumoconiosis conditions highlighted in the report include asbestosis, coal workers' pneumoconiosis, silicosis, byssinosis, and pneumoconioses classified as either "unspecified" or "other," as well as all pneumoconioses aggregated. Although some experts do not consider byssinosis a typical pneumoconiosis, it is included because the International Classification of Disease (ICD) system places byssinosis (code 504) within the series of codes for the pneumoconioses (500-505) and because byssinosis is included with other pneumoconioses in a new occupational safety and health objective for the nation (#10.17 in *Healthy People 2000: Midcourse Review and 1995 Revisions*).

Data contained in the report originate from publications, reports, and data provided by the National Center for Health Statistics (NCHS), the Occupational Safety and Health Administration (OSHA), the Mine Safety and Health Administration (MSHA), and the Bureau of Mines (BoM). Details on the major data sources and on the methods used to compute specific statistics can be found in Appendices A and B, respectively. Interpreted with appropriate care, information contained in this report can help to establish priorities for investigation and intervention, as well as to track progress toward the elimination of an important subset of preventable occupational respiratory diseases.

A description of previous editions of the *WoRLD Surveillance Report*, along with revisions and errata can be found in Appendix E. Comments and suggestions from users of earlier editions have influenced the content and format of this 1996 edition. To increase the utility of future editions, comments on the current report and descriptions of how the information is used are invited.

Send comments, suggestions, tear-out reader response card and other correspondence to:

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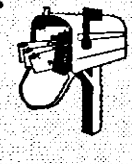

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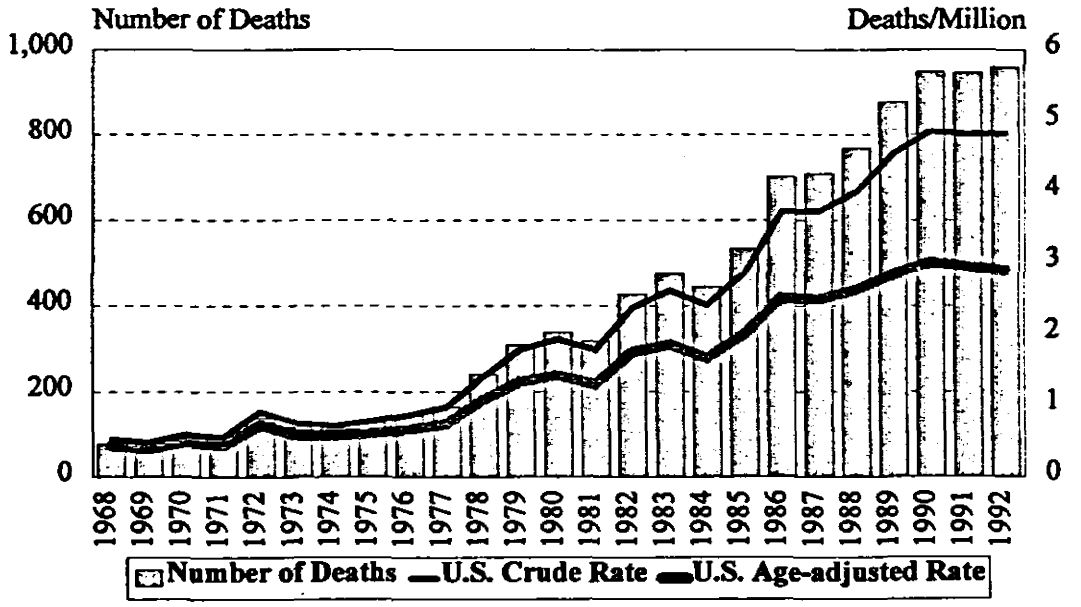
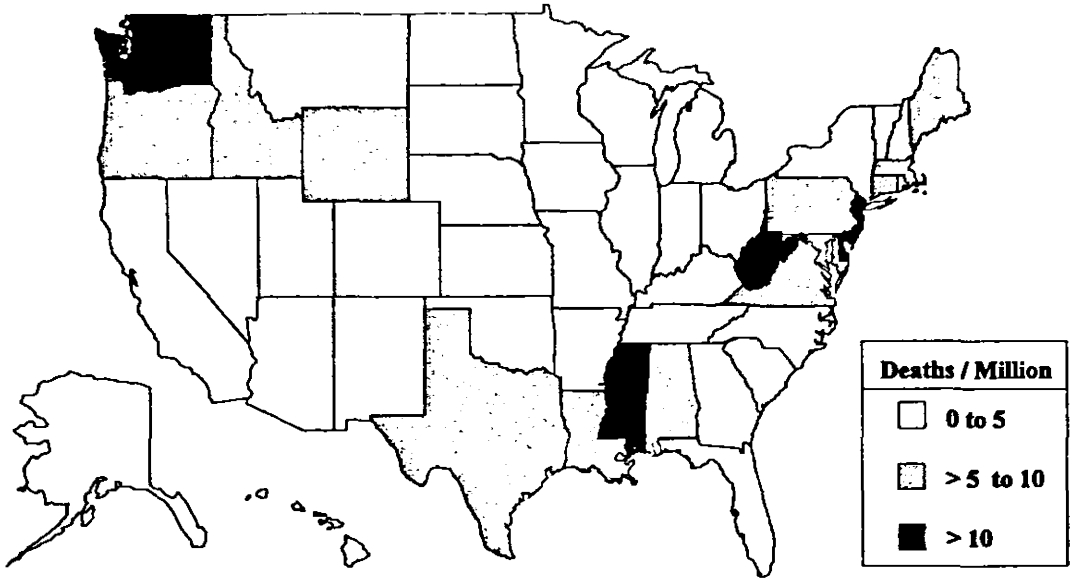
Figure 1-1. Asbestosis: Number of deaths, crude and age-adjusted mortality rates, U.S. residents age 15 and over, 1968-1992



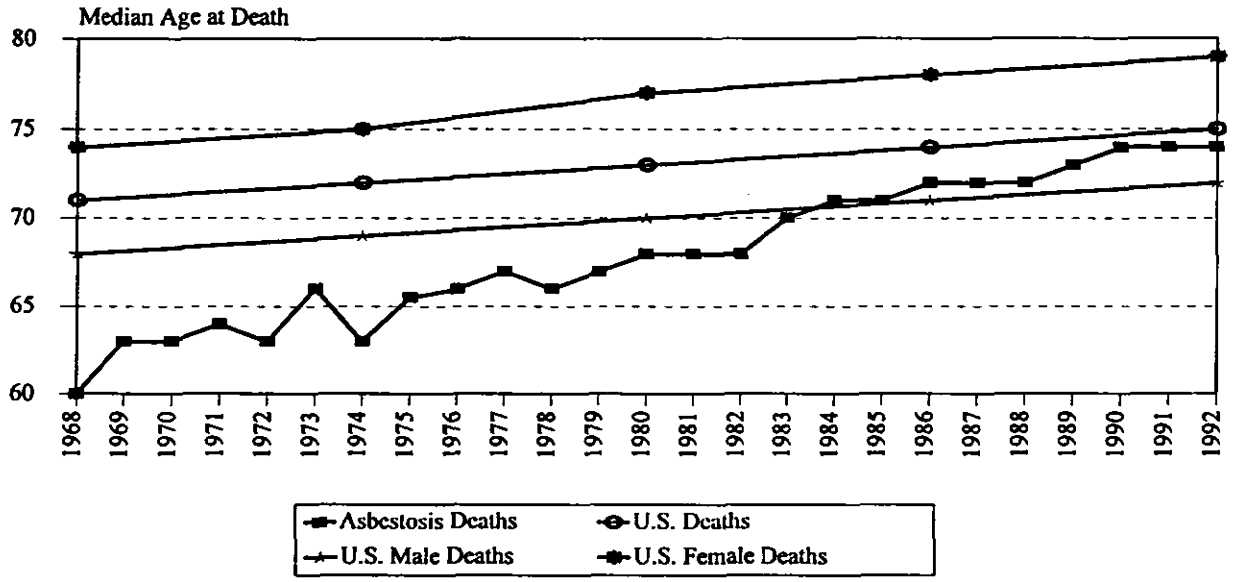
NOTE: See Appendix A for source description and Appendix B for methods and ICD-8 and ICD-9 codes.
SOURCE: National Center for Health Statistics multiple cause of death data. Population estimates from U.S. Bureau of the Census.

Figure 1-2. Asbestosis: Crude mortality rates by state, U.S. residents age 15 and over, 1991-1992



NOTE: See Appendix A for source description and Appendix B for methods and ICD-8 and ICD-9 codes.
SOURCE: National Center for Health Statistics multiple cause of death data. Population estimates from U.S. Bureau of the Census.

Figure 1-3. Asbestosis: Median age at death, U.S. residents age 15 and over, 1968-1992



NOTE: See Appendix A for source description and Appendix B for methods and ICD-8 and ICD-9 codes.
SOURCE: National Center for Health Statistics multiple cause of death data.

Table 1-1. Asbestosis: Number of deaths by sex, race, and age, U.S. residents age 15 and over, 1991-1992

		1991		1992	
		Number	Percent	Number	Percent
Total deaths		946	100.0	959	100.0
Sex	Male	908	96.0	923	96.2
	Female	38	4.0	36	3.8
Race	White	877	92.7	898	93.6
	Black	63	6.7	57	5.9
	Other	6	0.6	4	0.4
Age	15-24	1	0.1	0	0.0
	25-34	0	0.0	0	0.0
	35-44	1	0.1	3	0.3
	45-54	25	2.6	13	1.4
	55-64	114	12.1	124	12.9
	65-74	370	39.1	371	38.7
	75-84	358	37.8	355	37.0
	85 and over	77	8.1	93	9.7
	Mean age	73.1		73.5	
	Range for age	19-96		38-100	

NOTE: Percentages may not total to 100% due to rounding. See Appendix A for source description and Appendix B for methods and ICD-8 and ICD-9 codes. Data for 1968-1990 can be found in the Work-Related Lung Disease Surveillance Report, 1994, see Appendix E.
SOURCE: National Center for Health Statistics multiple cause of death data.

Table 1-2. Asbestosis: Number of deaths by state, U.S. residents age 15 and over, 1968-1992

State	1968-1978	1979-1990	1991	1992	TOTAL
Alabama	6	92	23	18	139
Alaska	3	4	1	1	9
Arizona	20	64	6	8	98
Arkansas	1	35	6	5	47
California	157	954	94	95	1,300
Colorado	5	38	5	4	52
Connecticut	22	94	14	17	147
Delaware	4	37	14	8	63
District of Columbia	2	6	1	-	9
Florida	37	348	54	52	491
Georgia	16	78	10	18	122
Hawaii	1	31	4	4	40
Idaho	3	33	6	3	45
Illinois	40	147	20	21	228
Indiana	9	49	4	4	66
Iowa	5	25	3	7	40
Kansas	1	23	3	7	34
Kentucky	2	31	5	5	43
Louisiana	13	113	20	14	160
Maine	9	98	8	8	123
Maryland	19	172	27	33	251
Massachusetts	104	340	27	48	519
Michigan	19	85	15	16	135
Minnesota	11	68	6	17	102
Mississippi	2	97	25	25	149
Missouri	23	84	11	14	132
Montana	3	30	2	4	39
Nebraska	2	20	3	2	27
Nevada	3	19	3	1	26
New Hampshire	5	48	1	4	58
New Jersey	232	800	93	80	1,205
New Mexico	4	19	3	1	27
New York	91	272	37	30	430
North Carolina	24	140	21	25	210
North Dakota	-	6	3	-	9
Ohio	32	167	24	32	255
Oklahoma	3	39	6	5	53
Oregon	25	144	22	22	213
Pennsylvania	165	640	83	100	988
Rhode Island	5	48	1	5	59
South Carolina	37	100	8	13	158
South Dakota	1	2	-	-	3
Tennessee	12	73	8	4	97
Texas	34	354	95	54	537
Utah	1	16	-	4	21
Vermont	2	11	-	3	16
Virginia	39	271	36	43	389
Washington	81	342	50	40	513
West Virginia	6	88	20	28	142
Wisconsin	16	56	11	6	89
Wyoming	2	5	4	1	12
TOTAL	1,359	6,856	946	959	10,120

- indicates no deaths listed.
NOTE: See Appendix A for source description and Appendix B for methods and ICD-8 and ICD-9 codes.
SOURCE: National Center for Health Statistics multiple cause of death data.

Table 1-3. Asbestosis: Mortality rates (per 1,000,000 population) by race and sex, U.S. residents age 15 and over, 1991-1992

Year	Overall rate	White		Black	
		Males	Females	Males	Females
	Crude mortality rate				
1991	4.80	10.42	0.42	5.86	0.16
1992	4.82	10.63	0.37	5.00	0.32
	Age-adjusted mortality rate				
1991	2.94	6.91	0.20	5.79	0.14
1992	2.90	6.93	0.17	4.99	0.28

NOTE: See Appendix A for source description and Appendix B for methods and ICD-8 and ICD-9 codes. Data for 1968-1990 can be found in the Work-Related Lung Disease Surveillance Report, 1994, see Appendix E.
See Appendix E for revised rates for 1968-1990.
SOURCE: National Center for Health Statistics multiple cause of death data. Population estimates from U.S. Bureau of the Census.

Table 1-4. Asbestosis: Years of potential life lost by race and sex, U.S. residents age 15 and over, 1991-1992

Year	Overall	White		Black	
		Males	Females	Males	Females
	Years of potential life lost to age 65				
1991	1,015	845	30	130	0
1992	890	780	15	50	30
	Years of potential life lost to life expectancy				
1991	11,883	9,294	466	664	28
1992	11,850	9,441	389	540	80

NOTE: See Appendix A for source description and Appendix B for methods and ICD-8 and ICD-9 codes. Data for 1968-1990 can be found in the Work-Related Lung Disease Surveillance Report, 1994, see Appendix E.
SOURCE: National Center for Health Statistics multiple cause of death data.

Table 1-5. Asbestosis: Total number of deaths, crude and age-adjusted mortality rates (per 1,000,000 population), and total years of potential life lost (YPLL) to life expectancy, by state, U.S. residents age 15 and over, 1988-1992

State	Total deaths	Rank	Crude mortality Rate	Crude mortality Rank	Age-adjusted mortality Rate	Age-adjusted mortality Rank	YPLL to life expectancy Years	YPLL to life expectancy Rank	YPLL to life expectancy Years/death	YPLL to life expectancy Rank
Alabama	94	15	5.94	14	3.79	15	1,254	14	13.3	17
Alaska	5	47	2.49	29	3.96	13	84	48	16.8	2
Arizona	30	29	2.11	37	1.39	35	414	29	13.8	13
Arkansas	20	36	2.18	34	1.34	37	283	35	14.2	8
California	459	2	3.96	23	2.75	23	5,503	2	12.0	40
Colorado	22	33	1.71	43	1.27	40	309	33	14.0	9
Connecticut	66	19	4.98	19	2.87	22	797	21	12.1	39
Delaware	49	25	18.55	1	13.08	1	727	23	14.8	4
District of Columbia	1	50	0.39	50	0.29	50	14	50	14.0	12
Florida	264	5	5.00	18	2.35	26	3,287	5	12.5	32
Georgia	55	22	2.18	34	1.79	28	806	20	14.7	5
Hawaii	20	36	4.58	22	2.91	21	227	39	11.4	43
Idaho	22	33	5.88	15	3.72	17	279	36	12.7	29
Illinois	96	14	2.15	36	1.40	34	1,288	13	13.4	16
Indiana	26	32	1.20	49	0.73	49	316	32	12.2	37
Iowa	18	40	1.66	45	0.91	47	232	38	12.9	20
Kansas	20	36	2.09	38	1.05	45	226	40	11.3	44
Kentucky	19	39	1.31	48	0.87	48	260	37	13.7	14
Louisiana	86	17	5.41	16	3.59	18	1,102	16	12.8	23
Maine	53	23	10.94	4	5.96	6	610	25	11.5	42
Maryland	136	10	7.16	12	5.30	8	1,825	10	13.4	15
Massachusetts	184	8	7.56	10	4.05	12	2,054	9	11.2	47
Michigan	66	19	1.83	41	1.32	38	987	19	15.0	3
Minnesota	47	26	2.78	26	1.78	29	585	26	12.4	33
Mississippi	98	13	10.05	6	6.22	4	1,247	15	12.7	27
Missouri	52	24	2.59	28	1.44	31	635	24	12.2	36
Montana	22	33	7.20	11	4.57	10	309	33	14.0	9
Nebraska	12	43	1.98	39	0.94	46	127	43	10.6	49
Nevada	7	46	1.46	47	1.06	44	93	45	13.3	18
New Hampshire	17	41	3.90	24	2.74	24	214	41	12.6	30
New Jersey	473	1	15.21	2	8.88	2	5,821	1	12.3	34
New Mexico	13	42	2.29	32	1.57	30	167	42	12.8	21
New York	172	9	2.39	31	1.42	33	2,196	7	12.8	25
North Carolina	100	12	3.77	25	2.51	25	1,320	12	13.2	19
North Dakota	4	49	1.63	46	1.36	36	87	47	21.8	1
Ohio	118	11	2.78	26	1.88	27	1,715	11	14.5	6
Oklahoma	30	29	2.46	30	1.43	32	376	30	12.5	31
Oregon	94	15	8.41	8	4.32	11	1,060	18	11.3	45
Pennsylvania	430	3	9.02	7	4.85	9	5,493	3	12.8	24
Rhode Island	29	31	7.14	13	3.86	14	351	31	12.1	38
South Carolina	64	21	4.70	21	3.11	19	784	22	12.3	35
South Dakota	-	-	-	-	-	-	-	-	-	-
Tennessee	35	28	1.81	42	1.26	41	500	28	14.3	7
Texas	335	4	5.18	17	3.79	15	4,269	4	12.7	26
Utah	10	44	1.68	44	1.26	41	119	44	11.9	41
Vermont	5	47	2.26	33	1.22	43	50	49	10.0	50
Virginia	196	6	7.95	9	5.79	7	2,516	6	12.8	22
Washington	195	7	10.25	5	6.10	5	2,191	8	11.2	46
West Virginia	86	17	12.03	3	6.53	3	1,091	17	12.7	28
Wisconsin	37	27	1.95	40	1.28	39	519	27	14.0	11
Wyoming	8	45	4.72	20	2.94	20	88	46	11.0	48

- indicates no deaths listed.

NOTE: Ranks are based on unrounded values. See Appendix A for source description and Appendix B for methods and ICD-8 and ICD-9 codes.

SOURCE: National Center for Health Statistics multiple cause of death data. Population estimates from U.S. Bureau of the Census.

Table 1-6. Asbestosis: Most frequently recorded occupations on death certificate, selected states, U.S. residents age 15 and over, 1991-1992

COC	Occupation	Number	Percent
585	Plumbers, pipefitters, and steamfitters	46	8.0
575	Electricians	31	5.4
593	Insulation workers	29	5.0
567	Carpenters	25	4.3
889	Laborers, except construction	24	4.2
633	Supervisors, precision production occupations	18	3.1
643	Boilermakers	18	3.1
019	Managers and administrators, n.e.c.	17	2.9
783	Welders and cutters	17	2.9
453	Janitors and cleaners	15	2.6
	All other occupations	322	55.7
	Occupation not reported	16	2.8
	TOTAL	578	100.0

COC - 1980 Census Occupation Code n.e.c. - not elsewhere classified
NOTE: See Appendix A for source description, Appendix B for methods and ICD-8 and ICD-9 codes, and Appendix C for list of 25 states reporting usual occupation and years reporting. Data for 1985-1990 can be found in the Work-Related Lung Disease Surveillance Report, 1994, see Appendix E.
SOURCE: National Center for Health Statistics multiple cause of death data.

Table 1-7. Asbestosis: Most frequently recorded industries on death certificate, selected states, U.S. residents age 15 and over, 1991-1992

CIC	Industry	Number	Percent
060	Construction	149	25.8
360	Ship and boat building and repairing	50	8.7
192	Industrial and miscellaneous chemicals	23	4.0
262	Miscellaneous nonmetallic mineral stone products	23	4.0
400	Railroads	17	2.9
901	General government, n.e.c.	17	2.9
142	Yarn, thread, and fabric mills	11	1.9
211	Other rubber products, and plastic footwear and belting	9	1.6
392	Not specified manufacturing industries	9	1.6
410	Trucking service	9	1.6
	All other industries	244	42.2
	Industry not reported	17	2.9
	TOTAL	578	100.0

CIC - 1980 Census Industry Code n.e.c. - not elsewhere classified
NOTE: See Appendix A for source description, Appendix B for methods and ICD-8 and ICD-9 codes, and Appendix C for list of 25 states reporting usual industry and years reporting. Data for 1985-1990 can be found in the Work-Related Lung Disease Surveillance Report, 1994, see Appendix E.
SOURCE: National Center for Health Statistics multiple cause of death data.

Table 1-8. Asbestosis: Proportionate mortality ratio (PMR) by usual occupation, selected states and years, U.S. residents age 15 and over, 1985-1992

COC	Occupation	Number of deaths	PMR	95% confidence interval	
				LCL	UCL
593	Insulation workers	110	261.01	212.20	317.53
643	Boilermakers	48	50.47	36.84	67.56
646	Lay-out workers	9	30.60	14.04	58.06
585	Plumbers, pipefitters, and steamfitters	153	19.06	16.03	22.50
653	Sheet metal workers	39	14.03	9.74	19.51
534	Heating, air conditioning, and refrigeration mechanics	11	11.20	5.60	20.04
584	Plasterers	6	10.61	3.89	23.12
575	Electricians	74	8.14	6.36	10.29
759	Painting, paint spray machine operators	11	7.06	3.53	12.63
829	Sailors and deckhands	6	6.41	2.35	13.97
757	Separate, filter, clarify machine operators	8	6.01	2.59	11.83
547	Specified mechanics and repairers, n.e.c.	13	5.29	2.81	9.04
783	Welders and cutters	40	4.91	3.51	6.69
363	Production coordinators	5	4.68	1.51	10.93
518	Industrial machinery repairers	19	4.41	2.66	6.89
563	Brickmasons and stonemasons	19	4.36	2.63	6.81
544	Millwrights	11	4.21	2.10	7.53
516	Heavy equipment mechanics	9	4.20	1.93	7.97
056	Industrial engineer	6	3.89	1.42	8.47
756	Mixing, blending machine operators	5	3.64	1.18	8.50
558	Supervisors, construction, n.e.c.	26	3.30	2.16	4.84
696	Stationary engineers	14	3.13	1.71	5.25
549	Not specified mechanics and repairers	12	2.99	1.54	5.22
579	Painters, construction and maintenance	22	2.85	1.78	4.32
856	Industrial truck, tractor equipment operators	8	2.76	1.19	5.43
637	Machinists	47	2.65	1.93	3.55
777	Miscellaneous machine operators, n.e.c.	27	2.61	1.72	3.80
779	Machine operators, not specified	37	2.47	1.72	3.44
633	Supervisors, production occupations	47	2.35	1.72	3.15
567	Carpenters	51	2.33	1.73	3.07
869	Construction laborers	32	1.79	1.21	2.56
453	Janitors and cleaners	43	1.46	1.04	1.99

COC - 1980 Census Occupation Code n.e.c. - not elsewhere classified LCL - lower confidence limit UCL - upper confidence limit

NOTE: See Appendix A for source description, Appendix B for methods and ICD-8 and ICD-9 codes, and Appendix C for list of 25 states reporting usual occupation and years reporting.

SOURCE: National Center for Health Statistics multiple cause of death data.

Table 1-9. Asbestosis: Proportionate mortality ratio (PMR) by usual industry, selected states and years, U.S. residents age 15 and over, 1985-1992

CIC	Industry	Number of deaths	PMR	95% confidence interval LCL	UCL
360	Ship and boat building and repairing	164	43.01	36.60	50.19
262	Miscellaneous nonmetallic mineral and stone products	54	29.17	21.61	38.48
192	Industrial and miscellaneous chemicals	54	7.02	5.20	9.26
502	Lumber and construction materials	7	6.64	2.67	13.69
282	Fabricated structural metal products	29	6.22	4.17	8.94
462	Electric and gas, and other combinations	9	6.13	2.81	11.63
200	Petroleum refining	19	5.82	3.51	9.09
521	Hardware, plumbing and heating supplies	8	4.74	2.04	9.33
420	Water transportation	16	4.61	2.63	7.48
211	Other rubber products, and plastics footwear and belting	19	4.49	2.70	7.02
060	Construction	435	4.42	4.00	4.87
181	Drugs	8	4.38	1.89	8.62
881	Membership organizations	12	3.96	2.04	6.91
180	Plastics, synthetics, and resins	6	3.31	1.21	7.21
460	Electric light and power	25	3.29	2.12	4.85
210	Tires and inner tubes	10	2.95	1.42	5.42
272	Primary aluminum industries	7	2.88	1.16	5.94
400	Railroads	46	2.15	1.57	2.88
160	Pulp, paper, and paperboard mills	15	2.02	1.13	3.33
392	Not specified manufacturing industries	43	1.66	1.19	2.26

CIC - 1980 Census Industry Code LCL - lower confidence limit UCL - upper confidence limit
NOTE: See Appendix A for source description, Appendix B for methods and ICD-8 and ICD-9 codes, and Appendix C for list of 25 states reporting usual industry and years reporting.
SOURCE: National Center for Health Statistics multiple cause of death data.

Table 1-10. Asbestos: Number of MSHA and OSHA inspector samples, percent exceeding the permissible exposure limit (PEL) and average severity level, by industry, 1993-1994

CIC	Industries most frequently recorded on 1991-1992 death certificates with asbestosis	Number of samples	% > PEL	Average severity
060	Construction	221	4.1	0.17
360	Ship and boat building and repairing	3	0.0	0.00
192	Industrial and miscellaneous chemicals	2	0.0	0.00
262	Miscellaneous nonmetallic mineral and stone products	65	10.8	0.43
400	Railroads	0	-	-
901	General government, n.e.c.	20	0.0	0.05
142	Yarn, thread, and fabric mills	1	0.0	0.00
211	Other rubber products, and plastics footwear and belts	6	0.0	0.03
392	Not specified manufacturing industries	0	-	-
410	Trucking service	1	0.0	0.00
	All other industries	483	0.8	0.08
	Industry not reported	3	0.0	0.00
	TOTAL	805	2.5	0.13

CIC - 1980 Census Industry Code n.e.c. - not elsewhere classified - indicates incalculable field
NOTE: See Appendix A for source description, Appendix B for methods, Appendix C for list of 25 states reporting usual industry and years reporting, and Appendix D for agents.
SOURCE: Bureau of Mines: Mine Inspection Data Analysis System. Occupational Safety and Health Administration: Integrated Management Information System. National Center for Health Statistics: multiple cause of death data.

Table 1-11. Asbestos: Number of MSHA and OSHA inspector samples, percent exceeding the permissible exposure limit (PEL) and average severity level, by industry, 1993-1994

CIC	Industries most frequently sampled in 1993-1994	Number of samples	% > PEL	Average severity
060	Construction	221	4.1	0.17
262	Miscellaneous nonmetallic mineral and stone products	65	10.8	0.43
910	Justice, public order, and safety	43	0.0	0.02
831	Hospitals	22	0.0	0.00
050	Nonmetallic mining	21	0.0	0.04
901	General government, n.e.c.	20	0.0	0.05
842	Elementary and secondary schools	19	0.0	0.06
751	Automotive repair shops	18	0.0	0.03
591	Department stores	16	0.0	0.00
351	Motor vehicles and motor vehicle equipment	15	0.0	0.04
	All other industries	342	1.2	0.11
	Industry not reported	3	0.0	0.00
	TOTAL	805	2.5	0.13

CIC - 1980 Census Industry Code n.e.c. - not elsewhere classified
NOTE: See Appendix A for source description, Appendix B for methods, and Appendix D for agents.
SOURCE: Bureau of Mines: Mine Inspection Data Analysis System. Occupational Safety and Health Administration: Integrated Management Information System.

Table 1-12 (page 1 of 2). Asbestos: Number of MSHA inspector samples, percent exceeding the permissible exposure limit (PEL) and average severity levels (Avg. Sev.), by state, 1974-1994

State	1974-1984				1985-1994				1993-1994			
	Total samples		Samples > PEL		Total samples		Samples > PEL		Total samples		Samples > PEL	
	Number	Avg. Sev.	%	Avg. Sev.	Number	Avg. Sev.	%	Avg. Sev.	Number	Avg. Sev.	%	Avg. Sev.
Alabama	0	-	-	-	0	-	-	-	0	-	-	-
Alaska	0	-	-	-	0	-	-	-	0	-	-	-
Arizona	86	0.94	14.0	4.78	0	-	-	-	0	-	-	-
Arkansas	0	-	-	-	0	-	-	-	0	-	-	-
California	156	0.73	21.8	2.33	64	0.20	0.0	-	9	0.09	0.0	-
Colorado	25	0.19	0.0	-	20	0.03	0.0	-	0	-	-	-
Connecticut	0	-	-	-	0	-	-	-	0	-	-	-
Delaware	0	-	-	-	0	-	-	-	0	-	-	-
District of Columbia	0	-	-	-	0	-	-	-	0	-	-	-
Florida	4	0.03	0.0	-	0	-	-	-	0	-	-	-
Georgia	16	1.79	25.0	5.45	4	0.00	0.0	-	0	-	-	-
Hawaii	0	-	-	-	1	0.00	0.0	-	1	0.00	0.0	-
Idaho	0	-	-	-	5	0.01	0.0	-	0	-	-	-
Illinois	55	0.05	0.0	-	2	0.00	0.0	-	1	0.00	0.0	-
Indiana	6	0.07	0.0	-	1	0.01	0.0	-	0	-	-	-
Iowa	0	-	-	-	0	-	-	-	0	-	-	-
Kansas	0	-	-	-	0	-	-	-	0	-	-	-
Kentucky	0	-	-	-	0	-	-	-	0	-	-	-
Louisiana	4	0.00	0.0	-	32	0.00	0.0	-	5	0.00	0.0	-
Maine	0	-	-	-	0	-	-	-	0	-	-	-
Maryland	91	0.27	3.3	1.58	12	0.00	0.0	-	0	-	-	-
Massachusetts	0	-	-	-	1	0.00	0.0	-	0	-	-	-
Michigan	2	0.00	0.0	-	5	0.01	0.0	-	5	0.01	0.0	-
Minnesota	208	0.10	0.5	1.20	31	0.02	0.0	-	2	0.05	0.0	-
Mississippi	0	-	-	-	0	-	-	-	0	-	-	-
Missouri	0	-	-	-	4	0.00	0.0	-	0	-	-	-
Montana	180	0.28	2.8	1.42	17	0.17	0.0	-	0	-	-	-

See footnotes at end of table.

Table 1-12 (page 2 of 2). Asbestos: Number of MSHA inspector samples, percent exceeding the permissible exposure limit (PEL) and average severity levels (Avg. Sev.), by state, 1974-1994

State	1974-1984				1985-1994				1993-1994			
	Total samples		Samples > PEL		Total samples		Samples > PEL		Total samples		Samples > PEL	
	Number	Avg. Sev.	%	Avg. Sev.	Number	Avg. Sev.	%	Avg. Sev.	Number	Avg. Sev.	%	Avg. Sev.
Nebraska	0	-	-	-	0	-	-	-	0	-	-	-
Nevada	0	-	-	-	4	0.00	0.0	-	0	-	-	-
New Hampshire	0	-	-	-	0	-	-	-	0	-	-	-
New Jersey	14	0.13	0.0	-	0	-	-	-	0	-	-	-
New Mexico	96	0.18	2.1	3.30	2	0.44	0.0	-	0	-	-	-
New York	63	0.35	1.6	2.60	2	0.00	0.0	-	2	0.00	0.0	-
North Carolina	13	0.39	0.0	-	3	0.00	0.0	-	0	-	-	-
North Dakota	0	-	-	-	0	-	-	-	0	-	-	-
Ohio	1	0.01	0.0	-	0	-	-	-	0	-	-	-
Oklahoma	21	0.17	0.0	-	0	-	-	-	0	-	-	-
Oregon	0	-	-	-	0	-	-	-	0	-	-	-
Pennsylvania	12	0.10	0.0	-	11	0.00	0.0	-	1	0.00	0.0	-
Rhode Island	0	-	-	-	0	-	-	-	0	-	-	-
South Carolina	62	0.06	0.0	-	11	0.00	0.0	-	0	-	-	-
South Dakota	27	0.27	0.0	-	43	0.09	0.0	-	0	-	-	-
Tennessee	0	-	-	-	0	-	-	-	0	-	-	-
Texas	72	1.12	1.4	75.63	5	0.00	0.0	-	4	0.00	0.0	-
Utah	0	-	-	-	2	0.00	0.0	-	0	-	-	-
Vermont	221	0.62	14.9	2.07	34	0.55	17.6	1.96	0	-	-	-
Virginia	12	0.16	0.0	-	8	0.00	0.0	-	2	0.00	0.0	-
Washington	1	0.02	0.0	-	10	0.00	0.0	-	0	-	-	-
West Virginia	0	-	-	-	2	0.00	0.0	-	2	0.00	0.0	-
Wisconsin	35	0.13	0.0	-	1	0.00	0.0	-	0	-	-	-
Wyoming	0	-	-	-	7	0.00	0.0	-	0	-	-	-
TOTAL	1,483	0.41	6.5	3.38	344	0.12	1.7	1.96	34	0.03	0.0	-

- Indicates incalculable field.

NOTE: See Appendix A for source description, Appendix B for methods, and Appendix D for agents.

SOURCE: Bureau of Mines: Mine Inspection Data Analysis System.

Table 1-13 (page 1 of 2). Asbestos: Number of OSHA inspector samples, percent exceeding the permissible exposure limit (PEL) and average severity levels (Avg. Sev.), by state, 1979-1994

State	1979-1984				1985-1994				1993-1994			
	Total samples		Samples > PEL		Total samples		Samples > PEL		Total samples		Samples > PEL	
	Number	Avg. Sev.	%	Avg. Sev.	Number	Avg. Sev.	%	Avg. Sev.	Number	Avg. Sev.	%	Avg. Sev.
Alabama	91	0.73	9.9	6.53	82	0.06	2.4	1.71	1	0.00	0.0	-
Alaska	76	0.01	0.0	-	110	0.14	2.7	4.52	4	0.00	0.0	-
Arizona	24	0.03	0.0	-	47	0.10	2.1	1.90	0	-	-	-
Arkansas	44	0.07	0.0	-	212	0.66	7.5	7.62	67	0.40	10.4	1.90
California	23	0.14	4.3	1.27	153	0.55	6.5	7.13	6	0.01	0.0	-
Colorado	212	0.08	0.9	3.57	123	0.14	3.3	3.23	6	1.41	33.3	3.98
Connecticut	164	0.39	4.9	6.12	223	0.04	0.0	-	26	0.05	0.0	-
Delaware	20	0.13	0.0	-	4	0.01	0.0	-	0	-	-	-
District of Columbia	50	0.10	2.0	2.04	10	0.06	0.0	-	2	0.00	0.0	-
Florida	82	1.74	7.3	23.09	177	0.13	2.3	4.73	30	0.25	10.0	1.67
Georgia	197	0.45	12.2	3.01	147	0.09	2.7	1.91	5	0.00	0.0	-
Hawaii	9	0.01	0.0	-	14	0.00	0.0	-	3	0.00	0.0	-
Idaho	14	0.05	0.0	-	81	0.02	0.0	-	3	0.00	0.0	-
Illinois	222	0.06	1.8	1.73	434	0.03	0.2	1.20	65	0.02	0.0	-
Indiana	170	0.28	6.5	1.44	216	0.04	0.0	-	17	0.00	0.0	-
Iowa	87	0.24	6.9	2.72	209	0.03	0.5	1.15	3	0.00	0.0	-
Kansas	44	0.21	9.1	1.74	33	0.04	0.0	-	3	0.00	0.0	-
Kentucky	79	0.05	1.3	1.94	132	0.08	0.0	-	13	0.03	0.0	-
Louisiana	57	0.07	1.8	1.58	82	0.07	2.4	1.63	0	-	-	-
Maine	63	0.06	0.0	-	17	0.05	0.0	-	0	-	-	-
Maryland	20	0.03	0.0	-	51	0.31	9.8	2.64	4	0.08	0.0	-
Massachusetts	291	0.40	15.5	1.59	241	0.39	8.7	3.71	20	0.55	20.0	2.43
Michigan	0	-	-	-	342	0.10	2.0	3.36	61	0.04	0.0	-
Minnesota	8	0.31	25.0	1.15	23	0.00	0.0	-	0	-	-	-
Mississippi	17	0.08	0.0	-	105	0.06	1.0	1.05	16	0.09	0.0	-
Missouri	372	0.18	2.4	6.86	155	0.04	1.3	1.66	11	0.00	0.0	-
Montana	120	0.09	0.8	1.95	63	0.12	3.2	1.13	0	-	-	-

See footnotes at end of table.

Table 1-13 (page 2 of 2). Asbestos: Number of OSHA inspector samples, percent exceeding the permissible exposure limit (PEL) and average severity levels (Avg. Sev.), by state, 1979-1994

State	1979-1984				1985-1994				1993-1994			
	Total samples		Samples > PEL		Total samples		Samples > PEL		Total samples		Samples > PEL	
	Number	Avg. Sev.	%	Avg. Sev.	Number	Avg. Sev.	%	Avg. Sev.	Number	Avg. Sev.	%	Avg. Sev.
Nebraska	36	0.04	0.0	-	109	0.01	0.0	-	3	0.14	0.0	-
Nevada	16	0.03	0.0	-	50	0.28	6.0	3.90	6	0.12	0.0	-
New Hampshire	100	0.82	12.0	5.84	70	0.05	1.4	1.50	5	0.00	0.0	-
New Jersey	268	0.23	3.4	3.39	307	0.81	14.7	5.13	13	0.02	0.0	-
New Mexico	2	0.00	0.0	-	22	0.02	0.0	-	6	0.02	0.0	-
New York	613	0.08	1.8	1.25	952	0.22	1.7	10.74	126	0.27	2.4	10.10
North Carolina	87	0.26	4.6	2.09	231	0.20	4.3	3.34	46	0.08	2.2	1.15
North Dakota	7	0.00	0.0	-	1	0.00	0.0	-	0	-	-	-
Ohio	282	0.46	4.3	9.27	545	0.87	5.3	15.45	42	0.00	0.0	-
Oklahoma	91	0.12	2.2	4.35	78	0.14	6.4	1.83	3	0.00	0.0	-
Oregon	78	0.13	5.1	1.37	76	0.09	0.0	-	15	0.08	0.0	-
Pennsylvania	365	0.30	4.7	4.14	393	0.62	6.6	8.33	11	0.00	0.0	-
Rhode Island	46	0.38	13.0	2.43	56	1.01	16.1	6.17	0	-	-	-
South Carolina	24	0.08	0.0	-	44	0.28	9.1	2.70	8	0.00	0.0	-
South Dakota	4	0.00	0.0	-	29	0.00	0.0	-	0	-	-	-
Tennessee	12	0.00	0.0	-	111	4.59	0.9	500.00	29	0.07	0.0	-
Texas	317	0.05	0.6	1.30	434	0.30	4.6	5.42	41	0.01	0.0	-
Utah	2	0.02	0.0	-	2	0.00	0.0	-	0	-	-	-
Vermont	0	-	-	-	1	0.00	0.0	-	0	-	-	-
Virginia	106	0.38	11.3	2.41	125	0.57	16.8	2.43	0	-	-	-
Washington	5	1.42	80.0	1.68	67	0.05	0.0	-	39	0.01	0.0	-
West Virginia	73	0.04	0.0	-	29	1.76	27.6	5.81	0	-	-	-
Wisconsin	161	0.16	5.6	1.87	160	0.73	6.3	10.65	12	0.03	0.0	-
Wyoming	15	0.00	0.0	-	0	-	-	-	0	-	-	-
TOTAL	5,266	0.24	4.5	3.78	7,378	0.37	4.0	8.10	771	0.14	2.6	3.37

- indicates incalculable field.
NOTE: See Appendix A for source description, Appendix B for methods, and Appendix D for agents.
SOURCE: Occupational Safety and Health Administration: Integrated Management Information System.

NIOSH MANUAL OF ANALYTICAL METHODS

4th EDITION

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DHHS (NIOSH) Publication No. 94-113

Various MW: Various CAS: Various RTECS: Various

METHOD: 7400, Issue 2 EVALUATION: FULL Issue 1: Rev. 3 on 15 May 1989
Issue 2: 15 August 1994

OSHA: 0.1 asbestos fiber (> 5 μm long)/cc; 1 f/cc/30 min excursion; carcinogen
MSHA: 2 asbestos fibers/cc
NIOSH: 0.1 f/cc (fibers > 5 μm long)/400 L; carcinogen
ACGIH: 0.2 crocidolite; 0.5 amosite; 2 chrysotile and other asbestos, fibers/cc; carcinogen

PROPERTIES: solid, fibrous, crystalline, anisotropic

SYNONYMS [CAS #]: actinolite [77536-66-4] or ferroactinolite [15669-07-5]; amosite [12172-73-5]; anthophyllite [77536-67-5]; chrysotile [12001-29-5]; serpentine [18786-24-8]; crocidolite [12001-28-4]; tremolite [77536-68-6]; amphibole asbestos [1332-21-4]; refractory ceramic fibers [142844-00-6]; fibrous glass.

SAMPLING		MEASUREMENT	
SAMPLER:	FILTER (0.45- to 1.2-μm cellulose ester membrane, 25-mm; conductive cowl on cassette)	**TECHNIQUE:**	LIGHT MICROSCOPY, PHASE CONTRAST
FLOW RATE*:	0.5 to 16 L/min	**ANALYTE:**	fibers (manual count)
VOL-MIN*: -MAX*:	400 L @ 0.1 fiber/cc (step 4, sampling) *Adjust to give 100 to 1300 fiber/mm²	**SAMPLE PREPARATION:**	acetone - collapse/triacetin - immersion method [2]
SHIPMENT:	routine (pack to reduce shock)	**COUNTING RULES:**	described in previous version of this method as "A" rules [1,3]
SAMPLE STABILITY:	stable	**EQUIPMENT:**	1. positive phase-contrast microscope 2. Walton-Beckett graticule (100-μm field of view) Type G-22 3. phase-shift test slide (HSE/NPL)
BLANKS:	2 to 10 field blanks per set	**CALIBRATION:**	HSE/NPL test slide
ACCURACY		**RANGE:**	100 to 1300 fibers/mm² filter area
RANGE STUDIED:	80 to 100 fibers counted	**ESTIMATED LOD:**	7 fibers/mm² filter area
BIAS:	see EVALUATION OF METHOD	**PRECISION ($\hat{S}_r$):**	0.10 to 0.12 [1]; see EVALUATION OF METHOD
OVERALL PRECISION ($\hat{S}_{rT}$):	0.115 to 0.13 [1]		
ACCURACY:	see 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. 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. Fibers < ca. 0.25 μm diameter will not be detected by this method [4]. This method may be used for other materials such as fibrous glass by using alternate counting rules (see Appendix C).

INTERFERENCES: If the method is used to detect a specific type of fiber, 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 revision replaces Method 7400, Revision #3 (dated 5/15/89).

REAGENTS:

1. Acetone,* reagent grade.
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-μm pore size, and backup pad.
   NOTE 1: Analyze representative filters for fiber background before use to check for clarity and background. 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.
   NOTE 3: Use 0.8-μm pore size filters for personal sampling. The 0.45-μm 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.
   NOTE 4: Other cassettes have been proposed that exhibit improved uniformity of fiber deposit on the filter surface, e.g., bellmouthed sampler (Envirometrics, Charleston, SC). These may be used if shown to give measured concentrations equivalent to sampler indicated above for the application.
2. Personal sampling pump, battery or line-powered vacuum, of sufficient capacity to meet flow-rate requirements (see step 4 for flow rate), with flexible connecting tubing.
3. Wire, multi-stranded, 22-gauge; 1", hose clamp to attach wire to cassette.
4. Tape, shrink- or adhesive-.
5. Slides, glass, frosted-end, pre-cleaned, 25 x 75-mm.
6. Cover slips, 22- x 22-mm, No. 1-1/2, unless otherwise specified by microscope manufacturer.
7. Lacquer or nail polish.
8. Knife, #10 surgical steel, curved blade.
9. Tweezers.

EQUIPMENT:

10. Acetone flash vaporization system for clearing filters on glass slides (see ref. [5] for specifications or see manufacturer's instructions for equivalent devices).
11. Micropipets or syringes, 5-μL and 100- to 500-μL.
12. Microscope, positive phase (dark) contrast, with green or blue filter, adjustable field iris, 8 to 10X eyepiece, and 40 to 45X phase objective (total magnification ca. 400X); numerical aperture = 0.65 to 0.75.
13. Graticule, Walton-Beckett type with 100-μm diameter circular field (area = 0.00785 mm^2) at the specimen plane (Type G-22). Available from Optometrics USA, P.O. Box 699, Ayer, MA 01432 [phone (508)-772-1700], 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).
14. HSE/NPL phase contrast test slide, Mark II. Available from Optometrics USA (address above).
15. Telescope, ocular phase-ring centering.
16. Stage micrometer (0.01-mm divisions).

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.

SAMPLING:

1. Calibrate each personal sampling pump with a representative sampler in line.
2. To reduce contamination and to hold the cassette tightly together, seal the crease between the cassette base and the cowl with a shrink band or light colored adhesive tape. For personal sampling, fasten the (uncapped) open-face cassette to the worker's lapel. The open face should be oriented downward.
    NOTE: The cowl should be electrically grounded during area sampling, especially under conditions of low relative humidity. Use a hose clamp to secure one end of the wire (Equipment, Item 3) to the monitor's cowl. Connect the other end to an earth ground (i.e., 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 a manner representative of actual handling of associated samples in the set. Open field blank cassettes at the same time as other cassettes just prior to sampling. Store top covers and cassettes in a clean area (e.g., a closed bag or box) with the top covers from the sampling cassettes during the sampling period.
4. Sample at 0.5 L/min or greater [6]. Adjust sampling flow rate, Q (L/min), and time, t (min), to produce a fiber density, E, of 100 to 1300 fibers/mm^2 ($3.85 \cdot 10^4$ to $5 \cdot 10^5$ fibers per 25-mm filter with effective collection area A_c = 385 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. The collection efficiency does not appear to be a function of flow rate in the range of 0.5 to 16 L/min for asbestos fibers [7]. Relatively large diameter fibers (>3 μm) may exhibit significant aspiration loss and inlet deposition. A sampling rate of 1 to 4 L/min for 8 h 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 minimum sampling volume of 48 L for an excursion measurement, and a maximum sampling rate of 2.5 L/min [3].

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 $\leq$1.46. This method collapses the filter for easier focusing and produces permanent (1 - 10 years) 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 [2]. 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 [3,7,8,9]). Unless the effective filtration area is known, determine the area and record the information referenced against the sample ID number [1,9,10,11].

NOTE 2: Excessive water in the acetone may slow the clearing of the filter, 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 [2].
   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 surgical steel 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 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 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. Mark the outline of the filter segment with a glass marking pen to aid in microscopic evaluation.

e. Glue the edges of the cover slip to the slide using lacquer or nail polish [12]. 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 manufacturers 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 microscope cleanings and major servicing.

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 contract 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 and 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 [13]. 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 reference samples (e.g., PAT, AAR, commercial 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 precision. Obtain separate values of relative standard deviation (S_r) 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, and >50 to 100 fibers in 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 [9]

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^2) exceeds 2.77 $(X)S_r'$, where X = average of the square roots of the two fiber counts (in fiber/mm^2) and $S_r' = \frac{S_r}{2}$, where S_r is the intracounter relative standard deviation for the appropriate count range (in fibers) determined in step 11. For more complete discussions see reference [13].

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 [13].

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 [14]. 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 for asbestos and routinely exchange field samples with other laboratories to compare performance of counters.

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 μm) [4].

18. Counting rules: (same as P&CAM 239 rules [1,10,11]: see examples in APPENDIX B).

a. Count any fiber longer than 5 μm which lies entirely within the graticule area.
   (1) Count only fibers longer than 5 μm. Measure length of curved fibers along the curve.
   (2) Count only fibers with a length-to-width ratio equal to or greater than 3:1.

b. For fibers which cross the boundary of the graticule field:

(1) Count as 1/2 fiber any fiber with only one end lying within the graticule area, provided that the fiber meets the criteria of rule a above.
(2) Do not count any fiber which crosses the graticule boundary more than once.
(3) Reject and do not count all other fibers.

c. Count bundles of fibers as one fiber unless individual fibers can be identified by observing both ends of a fiber.
d. 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 or bubble 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 <u>total</u> 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 μm), polarized light microscopy (as in NIOSH Method 7403) may be used to identify and eliminate interfering non-crystalline fibers [15].

NOTE 3: Do not count at edges where filter was cut. Move in at least 1 mm from the edge.

NOTE 4: 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 [5].

NOTE 5: Counts are to be recorded on a data sheet that provides, as a minimum, spaces on which to record the counts for each field, filter identification number, analyst's name, date, total fibers counted, total fields counted, average count, fiber density, and commentary. Average count is calculated by dividing the total fiber count by the number of fields observed. Fiber density (fibers/mm^2) is defined as the average count (fibers/field) divided by the field (graticule) area (mm^2/field).

CALCULATIONS AND REPORTING OF RESULTS

20. Calculate and report fiber density on the filter, E (fibers/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 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^2 and fiber counts from samples with >50% of filter area covered with particulate should be reported as "uncountable" or "probably biased." Other fiber counts outside the 100-1300 fiber/mm^2 range should be reported as having "greater than optimal variability" and as being "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_c (approx. 385 mm^2 for a 25-mm filter):

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

NOTE: Periodically check and adjust the value of A_c, if necessary.

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

NOTE: Precision depends on the total number of fibers counted [1,16]. Relative standard deviation is documented in references [1,15-17] 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 [10]. 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^3 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 [10].
   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 [2,4,10]. The aluminum "hot block" technique minimizes the amount of acetone needed to prepare each sample.
   3. Measurement:
      a. The Walton-Beckett graticule standardizes the area observed [14,18,19].
      b. The HSE/NPL test slide standardizes microscope optics for sensitivity to fiber diameter [4,14].
      c. Because of past inaccuracies associated with low fiber counts, the minimum recommended loading has been increased to 100 fibers/mm^2 filter area (a total of 78.5 fibers counted in 100 fields, each with field area = .00785 mm^2.) Lower levels generally result in an overestimate of the fiber count when compared to results in the recommended analytical range [20]. The recommended loadings should yield intracounter S_r in the range of 0.10 to 0.17 [21,22,23].

B. Interlaboratory comparability:
   An international collaborative study involved 16 laboratories using prepared slides from the asbestos cement, milling, mining, textile, and friction material industries [9]. The relative standard deviations (S_r) varied with sample type and laboratory. The ranges were:

	Intralaboratory S_r	Interlaboratory S_r	Overall S_r
AIA (NIOSH A Rules)*	0.12 to 0.40	0.27 to 0.85	0.46
Modified CRS (NIOSH B Rules)**	0.11 to 0.29	0.20 to 0.35	0.25

* 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 A Rules are otherwise similar to the AIA rules.
** See Appendix C.

A NIOSH study conducted using field samples of asbestos gave intralaboratory S_r in the range 0.17 to 0.25 and an interlaboratory S_r of 0.45 [21]. This agrees well with other recent studies [9,14,16].

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 indicated that lightly loaded samples are as accurate as properly loaded ones).

Theoretically, the process of counting randomly (Poisson) distributed 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 [17,19,20,21].

An additional component of variability comes primarily from subjective interlaboratory differences. In a study of ten counters in a continuing sample exchange program, Ogden [15] 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 [14]. 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 [21]. 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 PAT program for laboratory-generated samples [17].

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 chosen as 0.45. It is hoped that the 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 [16].

For example, if a sample yields a count of 24 fibers, Figure 1 indicates that the mean interlaboratory 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^2 counting field 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 [9].

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 x 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.

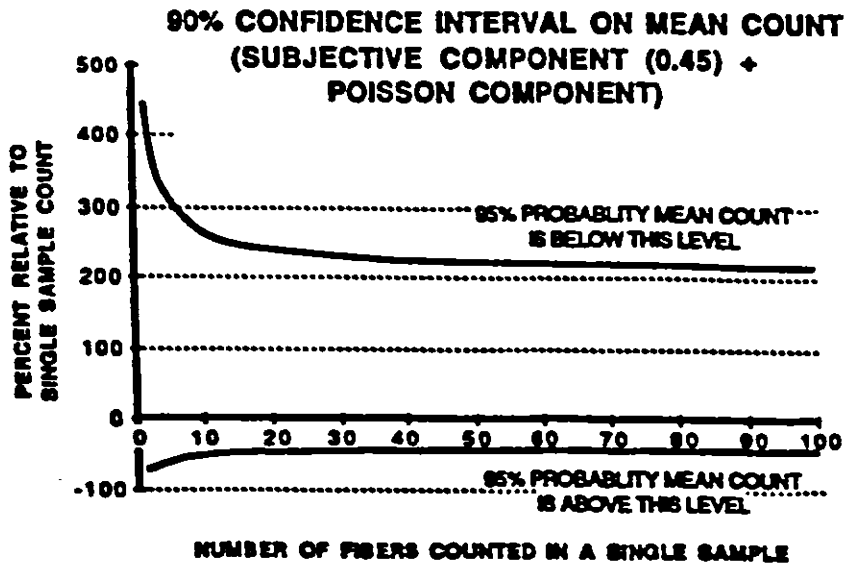

Figure 1. Interlaboratory Precision of Fiber Counts

The curves in Figures 1 are defined by the following equations:

$$UCL = \frac{2X + 2.25 + [(2.25 + 2X)^2 - 4(1 - 2.25 S_r^2) X^2]^{1/2}}{2(1 - 2.25 S_r^2)} \quad (3)$$

$$LCL = \frac{2X + 4 - [(4 + 2X)^2 - 4(1 - 4 S_r^2) X^2]^{1/2}}{2(1 - 4 S_r^2)} \quad (4)$$

where S_r = subjective interlaboratory relative standard deviation, which is close to the total interlaboratory 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.

REFERENCES:

[1] Leidel, N. A., S. G. Bayer, R. D. Zumwalde, and K. A. Busch. USPHS/NIOSH Membrane Filter Method for Evaluating Airborne Asbestos Fibers, U.S. Department of Health, Education, and Welfare, Publ. (NIOSH) 79-127 (1979).

[2] Baron, P. A. and G. C. Pickford. "An Asbestos Sample Filter Clearing Procedure," Appl. Ind. Hyg., 1:169-171, 199 (1986).

[3] Occupational Safety and Health Administration, U.S. Department of Labor, Occupational Exposure to Asbestos, Tremolite, Anthophyllite, and Actinolite Asbestos; Final Rules, 29 CFR Part 1910.1001 Amended June 20, 1986.

[4] Rooker, S. J., N. P. Vaughn, and J. M. LeGuen. "On the Visibility of Fibers by Phase Contrast Microscopy," Amer. Ind. Hyg. Assoc. J., 43, 505-515 (1982).

[5] Baron, P. and G. Deye, "Electrostatic Effects in Asbestos Sampling," Parts I and II Amer. Ind. Hyg. Assoc. J., 51, 51-69 (1990).

[6] Johnston, A. M., A. D. Jones, and J. H. Vincent. "The Influence of External Aerodynamic Factors on the Measurement of the Airborne Concentration of Asbestos Fibers by the Membrane Filter Method," Ann. Occup. Hyg., 25, 309-316 (1982).

[7] Beckett, S.T., "The Effects of Sampling Practice on the Measured Concentration of Airborne Asbestos," Ann. Occup. Hyg., 21, 259-272 (1980).

[8] Jankovic, J. T., W. Jones, and J. Clere. "Field Techniques for Clearing Cellulose Ester Filters Used in Asbestos Sampling," Appl. Ind. Hyg., 1, 145-147 (1986).

[9] Crawford, N. P., H. L. Thorpe, and W. Alexander. "A Comparison of the Effects of Different Counting Rules and Aspect Ratios on the Level and Reproducibility of Asbestos Fiber Counts," Part I: Effects on Level (Report No. TM/82/23), Part II: Effects on Reproducibility (Report No. TM/82/24), Institute of Occupational Medicine, Edinburgh, Scotland (December, 1982).

[10] NIOSH Manual of Analytical Methods, 2nd ed., Vol. 1., P&CAM 239, U.S. Department of Health, Education, and Welfare, Publ. (NIOSH) 77-157-A (1977).

[11] Revised Recommended Asbestos Standard, U.S. Department of Health, Education, and Welfare, Publ. (NIOSH) 77-169 (1976); as amended in NIOSH statement at OSHA Public Hearing, June 21, 1984.

[12] Asbestos International Association, AIA Health and Safety Recommended Technical Method #1 (RTMI). "Airborne Asbestos Fiber Concentrations at Workplaces by Light Microscopy" (Membrane Filter Method), London (1979).

[13] Abell, M., S. Shulman and P. Baron. The Quality of Fiber Count Data, Appl. Ind. Hyg., 4, 273-285 (1989).

[14] "A Study of the Empirical Precision of Airborne Asbestos Concentration Measurements in the Workplace by the Membrane Filter Method," Asbestos Information Association, Air Monitoring Committee Report, Arlington, VA (June, 1983).
[15] McCrone, W., L. McCrone and J. Delly, "Polarized Light Microscopy," Ann Arbor Science (1978).
[16] Ogden, T. L. "The Reproducibility of Fiber Counts," Health and Safety Executive Research Paper 18 (1982).
[17] Schlecht, P. C. and S. A. Schulman. "Performance of Asbestos Fiber Counting Laboratories in the NIOSH Proficiency Analytical Testing (PAT) Program," Am. Ind. Hyg. Assoc. J., 47, 259-266 (1986).
[18] Chatfield, E. J. Measurement of Asbestos Fiber Concentrations in Workplace Atmospheres, Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario, Study No. 9, 180 Dundas Street West, 22nd Floor, Toronto, Ontario, CANADA M5G 1Z8.
[19] Walton, W. H. "The Nature, Hazards, and Assessment of Occupational Exposure to Airborne Asbestos Dust: A Review," Ann. Occup. Hyg., 25, 115-247 (1982).
[20] Cherrie, J., A.D. Jones, and A.M. Johnston. "The Influence of Fiber Density on the Assessment of Fiber Concentration Using the membrane filter Method." Am. Ind. Hyg. Assoc. J., 47(8), 465-74 (1986).
[21] Baron, P. A. and S. Shulman. "Evaluation of the Magiscan Image Analyzer for Asbestos Fiber Counting." Am. Ind. Hyg. Assoc. J., (in press).
[22] Taylor, D. G., P. A. Baron, S. A. Shulman and J. W. Carter. "Identification and Counting of Asbestos Fibers," Am. Ind. Hyg. Assoc. J. 45(2), 84-88 (1984).
[23] "Potential Health Hazards of Video Display Terminals," NIOSH Research Report, June 1981.
[24] "Reference Methods for Measuring Airborne Man-Made Mineral Fibers (MMMF)," WHO/EURO Technical Committee for Monitoring an Evaluating Airborne MMMF, World Health Organization, Copenhagen (1985).
[25] Criteria for a Recommended Standard...Occupational Exposure to Fibrous Glass, U.S. Department of Health, Education, and Welfare, Publ. (NIOSH) 77-152 (1977).

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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 μm 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 (μm), 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:

$$d_c = \frac{L_a}{L_o} \times D. \qquad (5)$$

Example: If L_o = 112 μm, L_a = 4.5 mm and D = 100 μm, then d_c = 4.02 mm.

8. Check the field diameter, D (acceptable range 100 μm ± 2 μm) with a stage micrometer upon receipt of the graticule from the manufacturer. Determine field area (acceptable range 0.00754 mm^2 to 0.00817 mm^2).

APPENDIX B: COMPARISON 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.

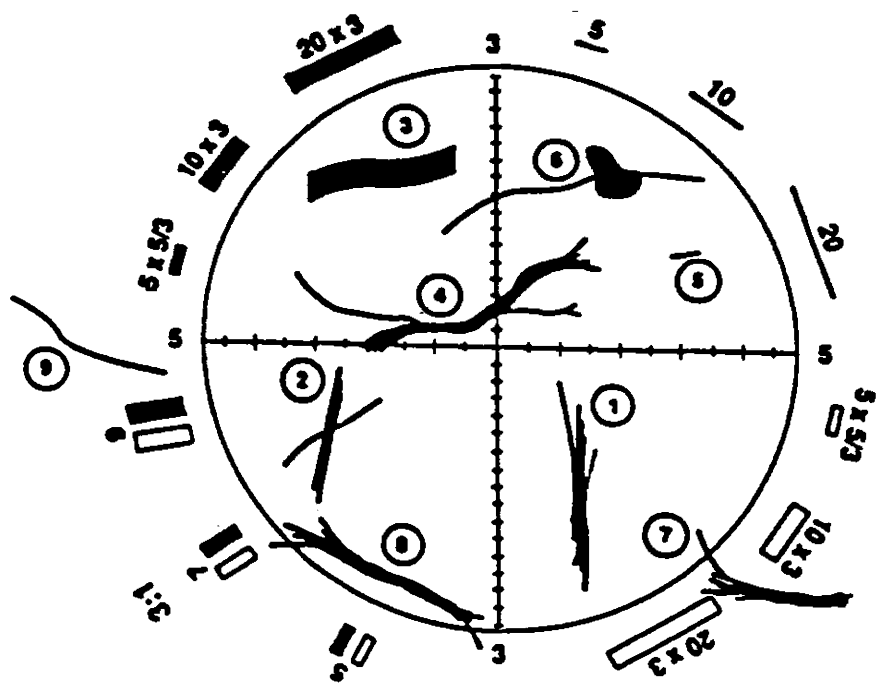

Figure 2. Walton-Beckett graticule with fibers.

These rules are sometimes referred to as the "A" rules.

FIBER COUNT

Object	Count	DISCUSSION
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 μm 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 μm), 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 μm 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 graticulate boundary more than once. count
9	Do not count	Ignore fibers that lie outside the graticule boundary.

APPENDIX C. ALTERNATE COUNTING RULES FOR NON-ASBESTOS FIBERS

Other counting rules may be more appropriate for measurement of specific non-asbestos fiber types, such as fibrous glass. These include the "B" rules given below (from NIOSH Method 7400, Revision #2, dated 8/15/87), the World Health Organization reference method for man-made mineral fiber [24], and the NIOSH fibrous glass criteria document method [25]. The upper diameter limit in these methods prevents measurements of non-thoracic 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.

"B" COUNTING RULES:

1. Count only ends of fibers. Each fiber must be longer than 5 μm and less than 3 μm 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 1 and 2 above. 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 μm 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.

APPENDIX D. EQUIVALENT LIMITS OF DETECTION AND QUANTITATION

fiber density on filter*		fiber concentration in air, f/cc	
fibers per 100 fields	fibers/mm^2	400-L air sample	1000-L air sample
200	255	0.25	0.10
100	127	0.125	0.05
LOQ.....80.....	.....102.....	.....0.10.....	.....0.04
50	64	0.0625	0.025
25	32	0.03	0.0125
20	25	0.025	0.010
10	12.7	0.0125	0.005
8	10.2	0.010	0.004
LOD.....5.5.....	.....7.....	.....0.00675.....	.....0.0027

* Assumes 385 mm^2 effective filter collection area, and field area = 0.00785 mm^2, for relatively "clean" (little particulate aside from fibers) filters.

ASBESTOS by TEM

FORMULA: Various MW: Various CAS: Various RTECS: Various

METHOD: 7402 EVALUATION: PARTIAL Issue 1: 15 May 1989
Issue 2: 15 August 1994

OSHA: 0.1 asbestos fibers (>5 μm long)/cc; 1 f/cc/30 min excursion; carcinogen
MSHA: 2 asbestos fibers/cc
NIOSH: 0.1 f/cc (fibers > 5 μm long)/400 L; carcinogen
ACGIH: 0.2 crocidolite; 0.5 amosite; 2 chrysotile and other asbestos, fibers/cc; carcinogen

PROPERTIES: solid, fibrous, crystalline, anistropic

SYNONYMS [CAS#]: actinolite [77536-66-4] or ferroactinolite [15669-07-5]; amosite [12172-73-5]; anthophyllite [77536-67-5]; chrysotile [12001-29-5]; serpentine [18786-24-8]; crocidolite [12001-28-4]; tremolite [77536-68-6]; amphibole asbestos [1332-21-4].

SAMPLING

SAMPLER: FILTER (0.45- to 1.2-μm cellulose ester membrane, 25-mm diameter; conductive cassette)

FLOW RATE: 0.5 to 16 L/min

VOL-MIN*: 400 L @ 0.1 fiber/cc
-MAX*: (step 4, sampling)
*Adjust for 100 to 1300 fibers/mm²

SHIPMENT: routine (pack to reduce shock)

SAMPLE STABILITY: stable

BLANKS: 2 to 10 field blanks per set

ACCURACY

RANGE STUDIED: 80 to 100 fibers counted

BIAS: not determined

OVERALL PRECISION ($\hat{S}_{rT}$): see EVALUATION OF METHOD

ACCURACY: not determined

MEASUREMENT

TECHNIQUE: MICROSCOPY, TRANSMISSION ELECTRON (TEM)

ANALYTE: asbestos fibers

SAMPLE PREPARATION: modified Jaffe wick

EQUIPMENT: transmission electron microscope; energy dispersive X-ray system (EDX) analyzer

CALIBRATION: qualitative electron diffraction; calibration of TEM magnification and EDX system

RANGE: 100 to 1300 fibers/mm² filter area [1]

ESTIMATED LOD: 1 confirmed asbestos fiber above 95% of expected mean blank value

PRECISION ($\bar{S}_r$): 0.28 when 65% of fibers are asbestos; 0.20 when adjusted fiber count is applied to PCM count [2].

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. Some non-asbestos amphibole minerals may give electron diffraction patterns similar to asbestos amphiboles.

OTHER METHODS: This method is designed for use with Method 7400 (phase contrast microscopy).

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-μm 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-μm pore size filters are recommended for personal sampling. 0.45-μm 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, 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, (about 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-.

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. Asbestos is a confirmed human carcinogen. Handle only in a well-ventilated fume hood.

SAMPLING:

1. Calibrate each personal sampling pump with a representative sampler in line.
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 [3]. Adjust sampling rate, Q (L/min), and time, t (min), to produce fiber density, E, of 100 to 1300 fibers/mm^2 [3.85 · 10^4 to 5 · 10^5 fibers per 25-mm filter with effective collection area (A_c= 385 mm^2)] 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 h (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 (≤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 [3].
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 circular sections from any of three quadrants of each sample and blank filter using a cork borer [4]. 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 [5] of Method 7400 or the DMF clearing technique [6] 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 [4].
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 [7].

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 grid 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) [7].
    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 μm 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 [7]. 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. 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 μm of each asbestos variety obtained from standard reference materials [7].

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

a. Prepare TEM grids of all asbestos varieties.
b. 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.
c. Estimate the elemental peak heights visually as follows:
   (1) Normalize all peaks to silicon (assigned an arbitrary value of 10).
   (2) Visually interpret all other peaks present and assign values relative to the silicon peak.
   (3) Determine an elemental profile for the fiber using the elements Na, Mg, Si, Ca, and Fe. Example: 0-4-10-3-<1 [7].
      NOTE: In fibers other than asbestos, determination of Al, K, Ti, S, P, and F may also be required for fiber characterization.
   (4) 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:
   a. chrysotile;
   b. amphibole;
   c. ambiguous;
   d. 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:
   a. 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."
   b. 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.
   c. 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:
    a. 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 [7,8]:
        (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 μm that meet the definition of a fiber (aspect ratio $\geq$3:1, longer than 5 μm). 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 μm 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 μm and later converted to μm 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.

EVALUATION OF METHOD:

The TEM method, using the direct count of asbestos fibers, has been shown to have a precision of 0.275 (s_r) in an evaluation of mixed amosite and wollastonite fibers. The estimate of the asbestos fraction, however, had a precision of 0.11 (s_r). When this fraction was applied to the PCM count, the overall precision of the combined analysis was 0.20 [2].

REFERENCES:

[1] Walton, W. H. "The Nature, Hazards, and Assessment of Occupational Exposure to Airborne Asbestos Dust: A Review," Ann. Occup. Hyg., 25, 115-247 (1982).
[2] Taylor, D. G., P. A. Baron, S. A. Shulman and J. W. Carter. "Identification and Counting of Asbestos Fibers," Am. Ind. Hyg. Assoc. J. 45(2), 84-88 (1984).
[3] Johnston, A. M., A. D. Jones, and J. H. Vincent. "The Influence of External Aerodynamic Factors on the Measurement of the Airborne Concentration of Asbestos Fibers by the Membrane Filter Method," Ann. Occup. Hyg., 25, 309-316 (1982).
[4] Zumwalde, R. D. and J. M. Dement. Review and Evaluation of Analytical Methods for Environmental Studies of Fibrous Particulate Exposures, NIOSH Technical Information Bulletin #77-204 (1977).
[5] Baron, P. A. and G. C. Pickford. "An Asbestos Sample Filter Clearing Procedure," Appl. Ind. Hyg., 1:169-171,199 (1986).
[6] LeGuen, J. M. and S. Galvin "Clearing and Mounting Techniques for the Evaluation of Asbestos Fibers by the Membrane Filter Method" Ann. Occup. Hyg. 24, 273-280 (1981).
[7] Yamate, G., S. A. Agarwal, and R. D. Gibbons. "Methodology for the Measurement of Airborne Asbestos by Electron Microscopy," EPA Contract No. 68-02-3266 (in press).
[8] Steel, E. B. and J. A. Small. "Accuracy of Transmission Electron Microcopy for the Analysis of Asbestos in Ambient Environments," Anal. Chem., 57, 209-213 (1985).

METHOD REVISED BY:

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ASBESTOS, CHRYSOTILE by XRD 9000

$Mg_3Si_2O_5(OH)_4$ MW: ca. 283 CAS: 12001-29-5 RTECS: CI6478500

METHOD: 9000, Issue 2 **EVALUATION:** FULL **Issue 1:** 15 May 1989 **Issue 2:** 15 August 1994

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

SAMPLING

BULK SAMPLE: 1 to 10 grams

SHIPMENT: seal securely to prevent escape of asbestos

SAMPLE STABILITY: indefinitely

BLANKS: none required

ACCURACY

RANGE STUDIED: 1 to 100% in talc [1]

BIAS: negligible if standards and samples are matched in particle size [1]

OVERALL PRECISION ($\hat{S}_{rT}$): unknown; depends on matrix and concentration

ACCURACY: ± 14% to ± 25%

MEASUREMENT

TECHNIQUE: X-RAY POWDER DIFFRACTION

ANALYTE: chrysotile

PREPARATION: grind under liquid N_2; wet-sieve through 10-μm sieve

DEPOSIT: 5 mg dust on 0.45-μm Ag membrane filter

XRD: Cu target X-ray tube; Optimize for intensity; 1° slit; integrated intensity with background subtraction

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 Fe_2O_3

PRECISION ($\bar{S}_r$): 0.07 (5 to 100%); 0.10 (@ 3%); 0.125 (@ 1%)

APPLICABILITY: Analysis of percent chrysotile asbestos in bulk samples.

INTERFERENCES: Antigorite (massive serpentine), Chlorite, Kaolinite, Bementite, and Brushite interfere. 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 [3]. Method 7400 is an optical counting procedure for airborne fibers in personal samples. Methods 7402 (Asbestos by Transmission Electron Microscopy) and 9002 (Asbestos by Polarized Light Microscopy) are also useful for positive identification of asbestos.

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_2-cooled, (Spex Model 6700 or equivalent), grinding vials (Spex 6701), extractor (Spex 6704).
3. Ultrasonic bath.
4. Sieve, 10-μm, for wet-sieving.
5. Filters, polycarbonate, 1.0-μm, 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-μm pore size (Millipore Corp., Poretics Corp., 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.

SPECIAL PRECAUTIONS: Asbestos, a human carcinogen, should be handled in a hood [4].

2-Propanol is flammable.

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-μm 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 h or more.

9 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 h or more. Store in a desiccator.

   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 μg.

   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° and are then normalized (12.e.) to obtain $\hat{I}_x^\circ$. The intensities for standards greater than 200 mg should be corrected for matrix absorption (12.f. and 13).

   g. Prepare a calibration graph by plotting $\hat{I}_x^\circ$, as a function of μg of each standard.

      NOTE: Poor repeatability (greater than 10% above 0.04 mg chrysotile) 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 $1/\sigma^2$ weighing, where σ^2 is the variance of the data at a given loading.

   h. Determine the slope, m, of the calibration curve in counts/μg. The intercept on the abscissa should be within ± 5 μg 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 (for determination of sample self-absorption, step 13) 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, $\hat{I}^{\circ}_{Ag}$, 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:

	Peak (2-Theta Degrees)	
Mineral	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:
    a. 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.
    b. Measure the diffraction peak area of a chrysotile peak that is free of interference. Scan times should be long, e.g., 15 min.
    c. 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.
    d. Calculate the net intensity, I_x (the difference between the peak integrated count and the total background count).
    e. Calculate and record the normalized intensity, $\hat{I}_x$, for the sample peak on each sample and standard:

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

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.
    g. Scan each field blank over the same 2-theta range used for the analyte and silver peaks. These analyses serve only to verify that contamination of the filters has not occurred. The analyte peak should be absent. The normalized intensity of the silver peak should match that of the media blanks.

CALCULATIONS:

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

$$C = \frac{(\hat{I}_x \cdot f(T) - b) \cdot 100}{m \cdot W}, \%$$

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

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

m = slope of calibration graph (counts/μg)

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

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

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

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

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

W = mass of deposited sample in μg.

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 [1,2]. 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	$\bar{S}_r$ (%)
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 negligible.

REFERENCES:

[1] Lange, B. A. Determination of Microgram Quantities of Asbestos by X-Ray Diffraction: Chrysotile in Thin Dust Layers of Matrix Material, Anal. Chem., 51:520(1979).

[2] NIOSH Manual of Analytical Methods, 2nd ed., V. 5, P&CAM 309, U.S. Department of Health, Education, and Welfare, Publ. (NIOSH) 79-141 (1979).

[3] Perkins, R.L. and B.W. Harvey. U.S. Environmental Protection Agency Test Method for the Determination of Asbestos in Bulk Building Materials, EPA/600/R-93/116 (June, 1993).

[4] Criteria for a Recommended Standard...Occupational Exposure to Asbestos (Revised), U.S. Department of Health, Education, and Welfare, Publ. (NIOSH) 77-169 (1976).

[5] Leroux, J. and C. Powers. Direct X-Ray Diffraction Quantitative Analysis of Quartz in Industrial Dust Films Deposited on Silver Membrane Filters, Occup. Health Rev., 21:26 (1970).

[6] Williams, D. D. Direct Quantitative Diffractometric Analysis, Anal. Chem., 31:1841 (1959).

[7] Abell, M. T., D. D. Dollberg, B. A. Lange, R. W. Hornung and J. C. Haartz. Absorption Corrections in X-ray Diffraction Dust Analyses: Procedures Employing Silver Filters, Electron Microscopy and X-ray Applications, V. 2, 115, Ann Arbor Science Publishers, Inc. (1981).

[8] Dollberg, D. D., M. T. Abell, and B. A. Lange. Occupational Health Analytical Chemistry: Quantitation Using X-Ray Powder Diffraction, ACS Symposium Series, No. 120, 43 (1980).

METHOD REVISED BY:

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TABLE 1. ABSORPTION CORRECTION FACTOR AS A FUNCTION OF TRANSMITTANCE FOR SOME CHRYSOTILE-SILVER PEAK COMBINATIONS.

Transmittance	Chrysotile / Silver	f(T) 12.08 / 38.12	f(T) 24.38 / 38.12	Transmittance	f(T) 12.08 / 38.12	f(T) 24.38 / 38.12
T				*T*		
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			

various MW: various CAS: 1332-21-4 RTECS: CI6475000

METHOD: 9002, Issue 2 **EVALUATION: PARTIAL** **Issue 1: 15 May 1989**
Issue 2: 15 August 1994

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

SYNONYMS [CAS #]: actinolite [77536-66-4], or ferroactinolite [15669-07-5]; amosite [12172-73-5]; anthophyllite [77536-67-5]; chrysotile [12001-29-5]; serpentine [18786-24-8]; crocidolite [12001-28-4]; tremolite [77536-68-6]; amphibole asbestos [1332-21-4].

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, crocidolite, tremolite asbestos
SAMPLE STABILITY:	stable	**EQUIPMENT:**	microscope, polarized light; 100-400X dispersion staining objective, stereo microscope: 10-45X
BLANKS:	none required	**RANGE:**	1% to 100% asbestos
		ESTIMATED LOD:	<1% asbestos [1]
		PRECISION:	not determined

ACCURACY	
RANGE STUDIED:	<1% to 100% asbestos
BIAS:	not determined
PRECISION:	not determined
ACCURACY:	not determined

APPLICABILITY: this method is useful for the qualitative identification of asbestos and the semi-quantitative determination of asbestos content of bulk samples. 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 μm) 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].

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.

* 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 ± 20 nm, retardation: "first order red" compensator.
3. Microscope slides: 75 mm x 25 mm.
4. Cover slips.
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).

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].

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 (step 14) and quantify (step 15).

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 of 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, and may require transmission electron microscopy for detection of the short asbestos fibers which are characteristic of floor tiles.
   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 washing and sodium metaphosphate treatment and ashing may be necessary, especially to detect low concentrations of asbestos. 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 or 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 each day of operation. Wipe microscope slides and cover slips with lens paper before using. Check refractive index liquids. 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 complete 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. Owing 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 (Table 1). 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. Observation of 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 color 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. Observed 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 were 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 clip 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 determine 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 since 1982 [1,5]. However, no formal evaluation of this method, as written, has been performed.

REFERENCES:

[1] Perkins, R.L. and B.W. Harvey, U.S. Environmental Protection Agency. Test Method for the Determination of Asbestos in Bulk Building Materials. EPA/600/R-93/116 (June. 1993).
[2] Criteria for a Recommended Standard...Occupational Exposure to Asbestos (Revised), U.S. Department of Health, Education, and Welfare, Publ. (NIOSH) 77-169 (1976), AS AMENDED IN NIOSH Statement at OSHA Public Hearing, (June 21, 1984).
[3] Jankovic, J.T. Asbestos Bulk Sampling Procedure, Amer. Ind. Hyg. Assoc. J., B-8 to B-10, (February, 1985).
[4] U.S. Environmental Protection Agency, "Asbestos Waste Management Guidance" EPA/530-SW-85-007, (May, 1985).
[5] National Voluntary Laboratory Accreditation Program, National Institute of Standards and Technology, Bldg 101, Room A-807 Gaithersburg, MD. 20899.
[6] Jankovic, J.T., J.L. Clere, W. Sanderson, and L. Piacitelli. Estimating Quantities of Asbestos in Building Materials. National Asbestos Council Journal, (Fall, 1988).
[7] Title 40, Code of Federal Regulations, Part 763. Appendix A to Subpart F. Interim Method of the Determination of Asbestos in Bulk Insulation Samples, (April 15, 1988).
[8] Bloss, F. Donald, Introduction to the Methods of Optical Crystallography, Holt, Rinehart, & Winston, (1961).
[9] Kerr, Paul F., Optical Mineralogy, 4th Ed., New York, McGraw-Hill, (1977).
[10] Shelley, David, Optical Mineralogy, 2nd Ed., New York, Elsevier, (1985).
[11] Phillips, W.R. and D.T. Griffen, Optical Mineralogy, W. H. Freeman and Co., (1981).
[12] McCrone, Walter, The Asbestos Particle Atlas, Ann Arbor Science, Michigan, (1980).
[13] "Selected Silicate Minerals and their Asbestiform Varieties," Bureau of Mines Information Circular IC 8751, (1977).

METHOD WRITTEN BY:

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Figure 1. Percent estimate comparator.

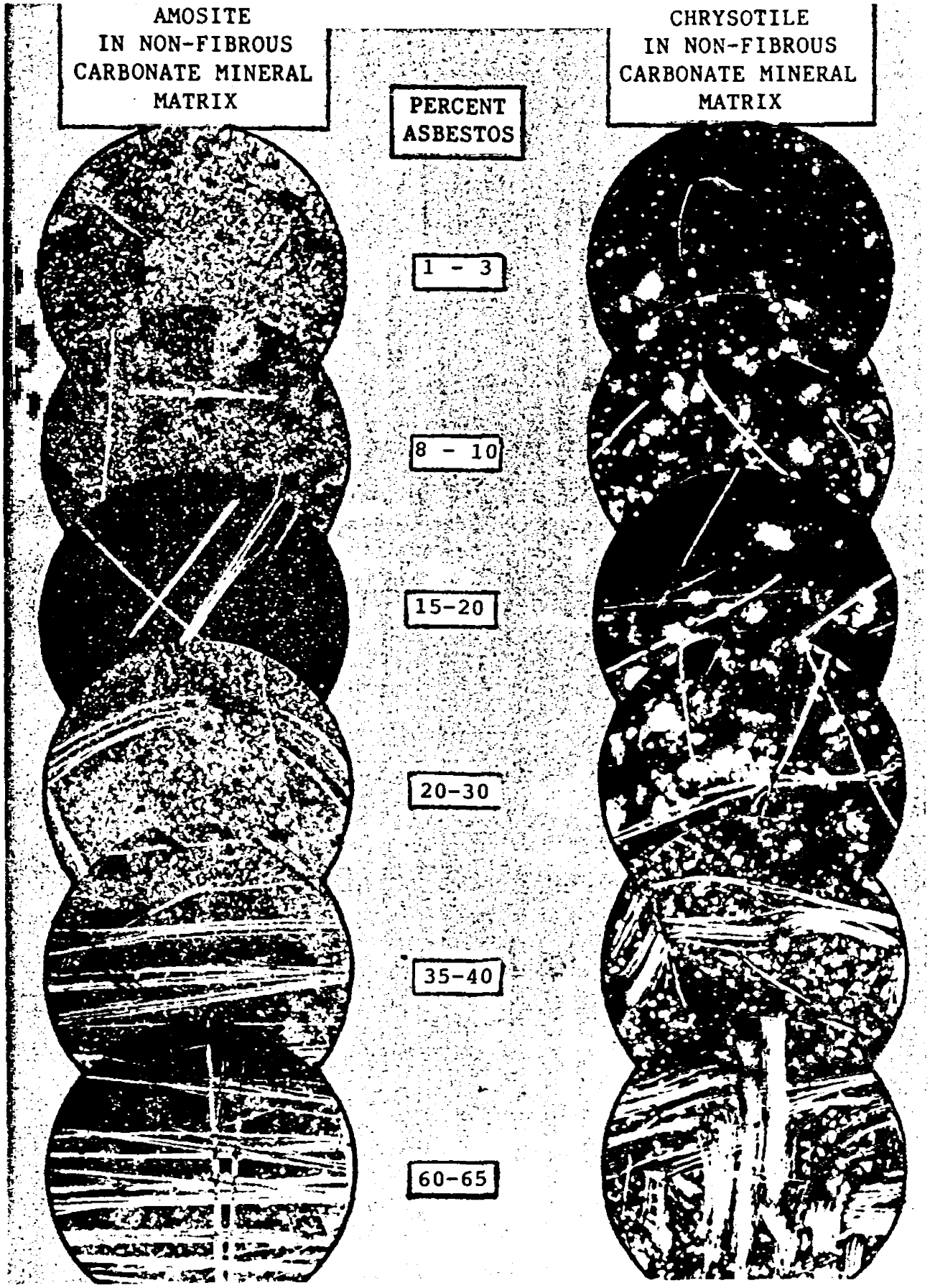

Table 1. Optical Properties of Asbestos Fibers

		Refractive Index (Approximate Values)		
Mineral	Morphology and Color	⊥ to Elongation	∥ to Elongation	Birefringence
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	0.019 - 0.024
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 - 1.67 (actinolite)	1.62 - 1.64 (tremolite) 1.64 - 1.68 (actinolite)	0.02 - 0.03

Table 1. Optical Properties of Asbestos Fibers (Continued)

			Central Stop Dispersion Staining Colors		
Mineral	Extinction	Sign of Elongation	RI Liquid	⊥ to Vibration	∥ to Vibration
Chrysotile	Parallel to fiber length	+ (length slow)	1.550 HD	Blue	Blue-magenta
Cummingtonite-Grunerite (Amosite)	Parallel to fiber length	+ (length slow)	1.670 Fibers subjected to high temperatures will not dispersion-stain.	Red magenta to blue	Yellow
Cummingtonite Grunerite			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 HD 1.620 HD	Blue Blue-green	Gold to gold-magenta Golden-yellow
Tremolite-Actinolite	Oblique - 10 to 20° for fragments. Some composite fibers show ∥ extinction.	+ (length slow)	1.605 HD	Pale blue (tremolite) Yellow (actinolite)	Yellow (tremolite) Pale yellow (actinolite)

HD = high-dispersion RI liquid series.

Building Air Quality

A Guide for Building Owners and Facility Managers

U.S. Environmental Protection Agency
Office of Air and Radiation
Office of Atmospheric and Indoor Air Programs
Indoor Air Division

U.S. Department of Health and Human Services
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health

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DISCLAIMER

This document has been reviewed in accordance with policies at the U.S. Environmental Protection Agency and the National Institute for Occupational Safety and Health. Information provided is based upon current scientific and technical understanding of the issues presented. Following the advice given will not necessarily provide complete protection in all situations or against all health hazards that may be caused by indoor air pollution. Mention of any trade names or commercial products does not constitute endorsement or recommendation for use.

Appendix D: Asbestos

"Asbestos" describes six naturally occurring fibrous minerals found in certain types of rock formations. When mined and processed, asbestos is typically separated into very thin fibers that are normally invisible to the naked eye. They may remain in the air for many hours if released from asbestos-containing material (ACM) and may be inhaled during this time. Three specific diseases — asbestosis (a fibrous scarring of the lungs), lung cancer, and mesothelioma (a cancer of the lining of the chest or abdominal cavity) — have been linked to asbestos exposure. It may be 20 years or more after exposure before symptoms of these diseases appear; however, high levels of exposure can result in respiratory diseases within a shorter period of time.

Most of the health problems resulting from asbestos exposure have been experienced by workers whose jobs exposed them to asbestos in the air over a prolonged period without the worker protection that is now required. Asbestos fibers can be found nearly everywhere in our environment, usually at very low levels. While the risk to occupants is likely to be small, health concerns remain, particularly for the custodial and maintenance workers in a building. Their jobs are likely to bring them into proximity to ACM and may sometimes require them to disturb the ACM in the performance of maintenance activities.

EPA estimates that "friable" (easily crumbled) ACM can be found in an estimated 700,000 public and commercial buildings. About 500,000 of those buildings are believed to contain at least some damaged asbestos. Significantly damaged ACM is found primarily in building areas not generally accessible to the public, such as boiler and mechanical rooms, where asbestos exposures generally would be limited to service and maintenance workers. However, if friable ACM is present in air plenums, it can be distributed throughout the building, thereby possibly exposing building occupants.

When is asbestos a problem? **Intact and undisturbed asbestos materials do not pose a health risk.** The mere presence of asbestos in a building does not mean that the health of building occupants is endangered. ACM which is in good condition, and is not damaged or disturbed, is not likely to release asbestos fibers into the air. When ACM is properly managed, release of asbestos fibers into the air is reduced, and the risk of asbestos-related disease is thereby correspondingly reduced.

There are a number of guidelines and regulations that govern asbestos exposure. Occupational standards for preventing asbestos-related diseases are recommended by NIOSH and promulgated by OSHA. NIOSH guidance contains Recommended Exposure Limits (RELs) and OSHA standards set Permissible Exposure Limits (PELs). The standards also contain many other measures, such as surveillance, medical screening, analytical methods, and methods of control. OSHA regulations and the EPA Worker Protection Rule also provide guidance on day-to-day activities that may bring workers in contact with ACM. EPA National Emission Standards for Hazardous Air Pollutants (NESHAP) define acceptable practices for renovation and demolition activities that involve asbestos-containing materials. In addition, many States have set exposure standards and other regulations concerning asbestos.

EPA and NIOSH recommend a practical approach that protects public health by emphasizing that ACM in buildings should be identified and appropriately managed, and that those workers who might disturb it should be properly trained and protected.

EPA AND NIOSH POSITIONS ON ASBESTOS

In an effort to calm unwarranted fears that a number of people seem to have about the mere presence of asbestos in their buildings and to discourage the decisions by some building owners to remove all ACM regardless of its condition, the EPA Administrator issued an *Advisory to the Public on Asbestos in Buildings* in 1991. This advisory summarized EPA's policies for asbestos control in the presentation of the following "five facts":

- Although asbestos is hazardous, the risk of asbestos-related disease depends upon exposure to airborne asbestos fibers.
- Based upon available data, the average airborne asbestos levels in buildings seem to be very low. Accordingly, the health risk to most building occupants also appears to be very low.
- Removal is often not a building owner's best course of action to reduce asbestos exposure. In fact, an improper removal can create a dangerous situation where none previously existed.
- EPA only requires asbestos removal in order to prevent significant public exposure to airborne asbestos fibers during building demolition or renovation activities.
- EPA does recommend a pro-active, in-place management program whenever asbestos-containing material is discovered.

NIOSH's position on asbestos exposure has been expressed in NIOSH policy statements and internal reports and at OSHA public hearings:

- NIOSH recommends the goal of eliminating asbestos exposure in the workplace. Where exposures cannot be eliminated, exposures should be limited to the lowest concentration possible.
- NIOSH contends that there is no safe airborne fiber concentration for asbestos. NIOSH therefore believes that any detectable concentration of asbestos in the workplace warrants further evaluation and, if necessary, the implementation of measures to reduce exposures.
- NIOSH contends that there is no scientific basis to support differentiating health risks between types of asbestos fibers for regulatory purposes.

Copies of the EPA and NIOSH policy statements and public advisories are available, respectively, from those agencies. See the last section in this appendix and *Appendix G* for information on how to obtain them.

OSHA requires that signs be posted around areas where work is being done that involves damaged asbestos-containing materials. These signs must communicate specific types of information.

Asbestos
Cancer and Lung Disease Hazard
Authorized Personnel Only
Respirators and Protective Clothing Are Required in This Area

PROGRAMS FOR MANAGING ASBESTOS IN-PLACE

In some cases, an asbestos operations and maintenance program is more appropriate than other asbestos control strategies, including removal. Proper asbestos management is neither to rip it all out in a panic nor to ignore the problem under the false presumption that asbestos is "risk free." Health concerns remain, particularly for custodial and maintenance workers.

In-place management does not mean "do nothing." It means having a program to ensure that the day-to-day management of the building is carried out in a manner that minimizes release of asbestos fibers into the air, and that ensures that when asbestos fibers are released, either accidentally or intentionally, proper control and clean-up procedures are implemented. Such a program may be all that is necessary to control the release of asbestos fibers until the asbestos-containing material in a building is scheduled to be disturbed by renovation or demolition activities.

The first responsibility of a building owner or manager is to identify asbestos-containing materials, through a building-wide inventory or on a case-by-case basis, before suspect materials are disturbed by renovations or other actions. The Asbestos Hazard Emergency Response Act (AHERA) program requires that in schools an inventory of asbestos materials be done by properly accredited individuals. Starting in late 1991 or 1992, there will also be a requirement that if an inventory of asbestos materials is done in public and commercial buildings, the inventory must be done by properly accredited individuals. In public and commercial buildings facing major renovations or demolition, inspections for the presence of ACM are required, according to the 1990 revision of the EPA Asbestos NESHAP. A carefully designed air monitoring program can be used as an adjunct to visual and physical evaluations of the asbestos-containing materials.

After the material is identified, the building management and staff can then institute controls to ensure that the day-to-day management of the building is carried out in a manner that prevents or minimizes the release of asbestos fibers into the air. These controls will ensure that when asbestos fibers are released, either accidentally or intentionally, proper management and clean-up procedures are implemented.

Another concern of EPA, NIOSH, and other Federal, State, and local agencies that are concerned with asbestos and public health is to ensure proper worker training and protection. In the course of their daily activities, maintenance and service workers in buildings may disturb materials and thereby elevate asbestos fiber levels and asbestos exposure, especially for themselves, if they are not properly trained and protected. For these persons, risk may be significantly higher than for other building occupants. Proper worker training and protection, as part of an active in-place management program, can reduce any unnecessary asbestos exposure for these workers and others. AHERA requires this training for school employees whose job activities may result in asbestos disturbances.

In addition to the steps outlined above, an in-place management program will usually include notification to workers and occupants of the existence of asbestos in their building, periodic surveillance of the material, and proper recordkeeping. EPA requires all of these activities for schools and strongly recommends that other building owners also establish comprehensive asbestos management programs. Without such programs, asbestos materials could be damaged or could deteriorate, which might result in elevated levels of airborne asbestos fibers. While the management costs of all the above activities will depend upon the amount, condition, and location of the materials, such a program need not be expensive.

WHERE TO GO FOR ADDITIONAL INFORMATION

For guidance on asbestos, building owners and managers are urged to become familiar with two EPA documents: *Managing Asbestos in Place* (published in 1990 and also known as the "Green Book") and *Guidance for Controlling Asbestos-Containing Materials in Buildings* (published in 1985 and also known as the "Purple Book").

To obtain copies of the guidance publications and other materials mentioned above, or to get additional information on technical issues, call or write:

Environmental Assistance Division
Office of Toxic Substances
U.S. EPA (TS-799)
401 M Street SW
Washington, DC 20460
Telephone (TSCA Information Hotline):
202-554-1404

National Institute for Occupational Safety and Health
Technical Information Branch
4676 Columbia Parkway
Cincinnati, OH 45226
Telephone: 1-800-35-NIOSH or
1-800-356-4674

Contact State air pollution control or health agencies for information on pertinent State activities and regulations. To find an asbestos contact in State agencies, consult the EPA Directory of State Indoor Air Contacts. For a more complete listing of publications concerning asbestos, refer to *Appendix G.*

DANGER asbestos

Working on brakes? Think about this:

- **Asbestos can cause fatal diseases years after exposure.**
- **Asbestos is used in clutches and brakes.**
- **Anyone working on brakes needs protection from asbestos.**

We can't see it, smell it, taste it, or feel it, but we know that asbestos can cause debilitating and often fatal diseases. We also know that these diseases take as long as 20 years to develop. What we *don't* know is how much–*or how little*–exposure to asbestos can cause them.

There is *no known safe level of exposure.* Anyone who works with any quantity of asbestos for any length of time risks developing serious disease later in life. And that person puts *others* at risk. Invisible asbestos fibers cling to clothes, hair, and skin. When they become airborne, as they do in natural movement, anyone near that person can inhale them.

Since asbestos is used in clutches and brakes, you may be exposing yourself and your loved ones to that risk. Yet protection from asbestos exposure can be simple and inexpensive. This bulletin will give you the facts about asbestos and how to take care of yourself when working with it.

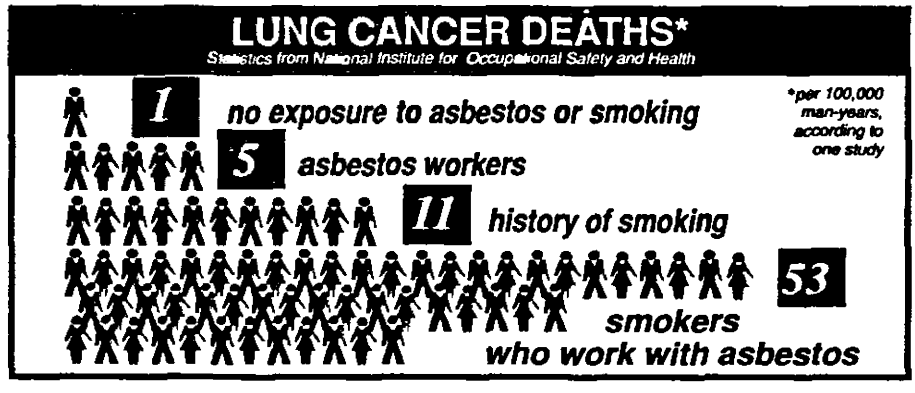
What is asbestos?

Asbestos is a mined mineral. It is a needle-like fiber that is very resistant to pressure and heat, which is why it is used in brakes and clutches.

It is very brittle, and it fractures lengthwise, creating smaller and smaller fibers with sharp, needle-like edges.

You can't see these fibers in the air—some asbestos particles are so small that nearly 200 of them would have to be bundled together to equal the diameter of one human hair.

These invisible particles are in brake dust and you can inhale them right through unapproved protective dust masks. They can then travel through the respiratory system and lodge in the lungs, where they become permanently embedded in lung tissue.



How dangerous is asbestos exposure?

Asbestos fibers are often found at tumor sites in the lung, and exposure to asbestos causes deadly lung diseases. Among these are:

•Lung cancer: About 95% of lung cancer victims die quickly — by the time the disease is detectable by x-ray, it is often widespread. Lung cancer accounts for the largest number of deaths attributed to asbestos exposure, and asbestos is almost as deadly as smoking as a cause of this disease. In fact, a smoker who works with asbestos is 10 times more likely to contract and die from lung cancer than than a non-smoker who works with asbestos.

•Mesothelioma: This deadly cancer is *100% fatal,* usually within one year of diagnosis — and asbestos is the major cause of this disease. No one knows how little it takes. Even *indirect* exposure is deadly. Wives, children, and pets of people who work with asbestos have died just from exposure to the clothes of the worker. This disease damages the lining of the chest and abdominal cavities.

Dangers continued on side 2

Doing a Brake Job? It's Easy to Do It Right!

If you can recognize hazards and know how to control them, you are better equipped to protect yourself. Therefore, the National Institute for Occupational Safety and Health (NIOSH) recommends a few basic rules for doing brake jobs. They include:

•Don't Blow It! Asbestos dust that doesn't get into the air will never get into your lungs. So *never* use an air hose, dry brush, rag, or ordinary shop-vac. They all stir up deadly dust. And for the same reason, don't grind brake linings. Slow lathe-turning will reduce dust significantly.

•Keep It Clean! Clean spills by vacuuming with HEPA (High Efficiency Particulate Air) systems or wet mopping immediately. (Dry sweeping and air hoses will blow dust into the air, and into your lungs.) Dispose of waste in sealed, labelled containers. And if you use a HEPA vacuum system, vacuum each component as you remove it.

Remember:

- ***Keep brake dust out of the air!***
- ***Keep work areas free of food and drink.***
- ***Dispose of all spills, contaminated solutions, and rags in accordance with state and local regulations.***
- ***Keep work clothing separate from street clothing—and launder work clothing after each shift.***
- ***Wash hands after working, but never with the same rags used to clean brakes.***

- ***Masks do not filter asbestos fibers. Respirators equipped with HEPA filters are the only approved protection.***
- ***New substitute friction materials have not been tested for safety over time. Use these safety precautions even when you believe the brake shoes do not contain asbestos.***

•Wet It Down! When using a wet brush, thoroughly wet the wheel hub and back of the assembly first. After removing the drum, wash all components with the brush. If using aerosol spray, remember to keep the nozzle far enough away from the surface to keep fibers from splashing back at you in the liquid. Also, if you must hammer drums, place a pan with water beneath the wheel to catch the dust.

•Seal It Tight! Enclosure systems should fit completely around the brake drum backing plate and should provide a tight seal around the axle. Turn on the HEPA vacuum before positioning enclosure over wheel, and leave it on while removing the enclosure.

•Check It Out! Make sure you have enough light to see and space to work. Respect the hazards and know how to control them. And if you have questions about safety practices, call:

The Ohio Industrial Commission
1-800-282-3045 or
The National Institute for Occupational Safety and Health
1-800-35-NIOSH

Dangers, continued

•Other Cancers: Cancer of the voice box has been linked to asbestos exposure. Asbestos also appears to cause cancers of the stomach and large intestine.

•Asbestosis: When microscopic asbestos fibers get caught in lung tissue, they cause scars. When this scarring spreads, the lungs can't expand and contract as easily as they should — and the victim finds it harder and harder to breathe. The condition is permanent, and studies suggest that if you work with asbestos for many years without protecting yourself, you stand about a 50-50 chance of developing this debilitating disease.

How do you know you have asbestos-related disease?

Unfortunately, you probably won't. Asbestos, like radiation, *seems* harmless — and no one has ever itched, sneezed, wheezed, or scratched because of it, even after asbestos particles have penetrated the lungs.

The victim often feels fine for years and may no longer even be working with asbestos when symptoms begin.

Usually symptoms take between *15 and 30 years* to become troublesome. By that time, as we have seen, disease can suddenly make it impossible to work. And in the case of lung cancer and mesothelioma, which are not curable, actually threaten life. The danger is deadly serious.

This bulletin *was produced at the Ohio State University office of the Ohio Technology Transfer Organization (OSU/OTTO) under a cooperative agreement with the National Institute for Occupational Safety and Health, Centers for Disease Control.*

Sources *for some of the information include:*
- *National Institute for Occupational Safety and Health*
- *United States Environmental Protection Agency*
- *Ohio Industrial Commission*
- *Ohio Department of Education*
- *Ohio Automotive Wholesalers Association*
- *Ohio Automotive Service Association*
- *Clayton Associates*
- *The Ohio State University*

*For more information about the Ohio Technology Transfer Organization, call **614-466-4286***

PART II

NIOSH BIBLIOGRAPHY ON ASBESTOS

A. NIOSH-AUTHORED DOCUMENTS

1. NUMBERED PUBLICATIONS

NIOSH 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. The following publications on asbestos are listed alphabetically by title.

Building Air Quality. A Guide for Building Owners and Facility Managers, *1991*. (Joint NIOSH-EPA Publication)
NIOSH PUB NO: 91-114. 253 pp.
GPO NO: 055-000-00390-4 $25.00
(A copy of the section on asbestos is contained in Part I of this Bibliography.)

Control of Asbestos Exposure During Brake Drum Service, *1989*.
NIOSH PUB NO: 89-121. 79 pp.
NTIS NO: PB90-168501 $31.50

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 $39.00

Criteria for a Recommended Standard: Occupational Exposure to Asbestos (Revised), *1976*.
NIOSH PUB NO: 77-169. 100 pp.
NTIS NO: PB-273965 $31.50
(An abstract of this recommended standard is contained in Part I one of this Bibliography.)

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.)
NTIS NO: PB83-105080 $35.00

Estimates of Pulmonary and Gastrointestinal Deposition for Occupational Fiber Exposures, *1979*.
NIOSH PUB NO: 79-135. 84 pp.
NTIS NO: PB80-149644 $31.50

An Evaluation of Glove Bag Containment in Asbestos Removal, *1990*.
NIOSH PUB NO: 90-119. 131 pp.
Available from NIOSH No Charge
NTIS NO: PB91-188995 $39.00

An Evaluation of Vacuum Equipment for Collection of Asbestos Waste, *1980*.
NIOSH PUB NO: 80-137. 77 pp.
NTIS NO: PB82-150236 $31.50

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 NO: PB87-157574 $44.00

Good Practices Manual for Insulation Installers, *1977*.
NIOSH PUB NO: 77-188. 40 pp.
NTIS NO: PB83-178822 $27.00

Laboratory Evaluations and Performance Reports for the Proficiency Analytical Testing (PAT) and Environmental Lead Proficiency Analytical Testing (ELPAT) Programs, *1994*.
NIOSH PUB NO: 95-104. 34 pp.
Available from NIOSH No Charge
NTIS NO: PB95-219515 $27.00

Laboratory Reports and Rating Criteria for the Proficiency Analytical Testing Program (PAT), *1990*.
NIOSH PUB NO: 91-102. 31 pp.
NTIS NO: PB91-227553 $27.00

NIOSH Guide to Industrial Respiratory Protection, *1987*.
NIOSH PUB NO: 87-116
Available from NIOSH No Charge
NTIS NO: PB88-188347 $61.50

NIOSH Manual of Analytical Methods - Fourth Edition, *1994*, and Supplement, *1996*.
NIOSH PUB NO: 94-113. 1373 pp.
NIOSH PUB NO: 96-135. 362 pp. (Supplement)
Available from NIOSH No Charge
GPO NO: 917-011-00000-1
(Subscription) $56.00
NTIS NO: PB95-154191 (Fourth Ed.) $126.00
NTIS NO: PB97-146138 (Supplement) $71.50
(Copies of the current NIOSH analytical methods for asbestos are contained in Part I of this Bibliography.)

Occupational Diseases: A Guide to Their Recognition (Revised), *1977.*
NIOSH PUB NO: 77-181. 619 pp.
NTIS NO: PB83-129528 $106.00

Occupational Exposure to Talc Containing Asbestos, *1980.*
NIOSH PUB NO: 80-115. 117 pp.
NTIS NO: PB80-193352 $35.00

Occupational Respiratory Diseases, *1986.*
NIOSH PUB NO: 86-102. 825 pp.
NTIS NO: PB87-205662 $125.00
(A copy of the sections on asbestos-related disease is contained in Part I of this Bibliography.)

Occupational Safety and Health Guidelines for Chemical Hazards, *1981*, and Supplement I, *1988.*
NIOSH PUB NO: 81-123. 1565 pp.
NIOSH PUB NO: 88-118. 214 pp. (Supplement I)
NTIS NO: PB83-154609 $193.00
NTIS NO: PB89-203129 (Suppl. I) $47.50
(A copy of the revised Guideline for asbestos is contained in Part I of this Bibliography.)

Proceedings of a Roundtable Discussion on Thermal Analysis Techniques, *1976.*
NIOSH PUB NO: 76-173. 260 pp.
NTIS NO: PB-266511 $59.00

Proceedings of the VIIth International Pneumoconioses Conference, Part I and Part II, Pittsburgh, PA, *August 23-26, 1988.*
NIOSH PUB NO: 90-108. 1608 pp.
NTIS NO: PB91-188821 (Part I) $125.00
NTIS NO: PB91-188839 (Part II) $115.00
(This Proceedings includes 75 papers on asbestos.)

Proceedings of the 9th International Symposium on Epidemiology in Occupational Health, *September 1994.*
NIOSH PUB NO: 94-112. 718 pp.
NTIS NO: PB95-173001 $92.00
(This Proceedings includes 9 papers on asbestos.)

Protect Your Family. Reduce Contamination at Home, *1997.*
NIOSH PUB NO: 97-125. 16 pp.
Available from NIOSH No Charge

Recommended Industrial Ventilation Guidelines, *1976.*
NIOSH PUB NO: 76-162. 333 pp.
NTIS NO: PB-266227 $61.50

Report to Congress on Workers' Home Contamination Study Conducted Under the Workers' Family Protection Act (29 U.S.C. 671a), *1995.*
NIOSH PUB NO: 95-123. 304 pp.
Available from NIOSH No Charge
NTIS NO: PB96-192000 $61.50

Results from the National Occupational Health Survey of Mining (NOHSM), *1996.*
NIOSH PUB NO: 96-136. 223 pp.
Available from NIOSH No Charge
NTIS NO: PB97-124473 $44.00

Review and Evaluation of Analytical Methods for Environmental Studies of Fibrous Particulate Exposures, *1977.*
NIOSH PUB NO: 77-204. 73 pp.
NTIS NO: PB-274750 $27.00

To B or not to B . . . a NIOSH B Reader, *1997.*
NIOSH PUB NO: 97-104. 2 pp.
Available from NIOSH No Charge

The Use of Light Scattering for the Detection of Filter Samples of Fibrous Aerosols, *1978.*
NIOSH PUB NO: 78-105. 59 pp.
NTIS NO: PB80-176977 $27.00

USPHS/NIOSH Membrane Filter Method for Evaluating Airborne Asbestos Fibers, *1979.*
NIOSH PUB NO: 79-127. 89 pp.
NTIS NO: PB-297731 $31.50

Work-Related Lung Disease Surveillance Report, *1991.*
NIOSH PUB NO: 91-113. 84 pp.
NTIS NO: PB92-136266 $31.50

Work-Related Lung Disease Surveillance Report, Supplement, *1992.*
NIOSH PUB NO: 91-113s. 43 pp.
NTIS NO: PB93-145969 $27.00

Work-Related Lung Disease Surveillance Report, *1994.*
NIOSH PUB NO: 94-120. 162 pp.
NTIS NO: PB95-181988 $44.00

Work-Related Lung Disease Surveillance Report, *1996.*
NIOSH PUB NO: 96-134. 484 pp.
Available from NIOSH No Charge
NTIS NO: PB97-128607 $57.00
(Excerpts from this report are contained in Part I of this Bibliography.)

Workplace Exposure to Asbestos, Review and Recommendations, *1980.*
NIOSH PUB NO: 81-103. 39 pp.
NTIS NO: PB83-176677 $27.00
(Excerpts from this publication are contained in Part I of this Bibliography.)

2. TESTIMONY

NIOSH testimony consists of both written comments and oral testimony presented before Congressional committees and at hearings convened by regulatory agencies. The following list of NIOSH testimony on asbestos is arranged in reverse chronological order.

NIOSH [1993]. Comments by R.W. Niemeier to the Coast Guard on their advanced notice of proposed rulemaking on controlling the marine asbestos hazard. 9 pp.
NTIS NO: PB93-215127 $10.00

NIOSH [1993]. Supplemental comments to the Department of Labor on the Occupational Safety and Health Administration proposed rule on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 7 pp.
NTIS NO: PB93-215119 $10.00

NIOSH [1991]. Post-hearing comments to the Department of Labor on the Occupational Safety and Health Administration proposed rule on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 6 pp.
NTIS NO: PB92-136043 $10.00

NIOSH [1991]. Testimony by R.A. Lemen to the Department of Labor on the Occupational Safety and Health Administration proposed rule on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 16 pp.
NTIS NO: PB92-139088 $19.50

NIOSH [1990]. Post-hearing comments to the Department of Labor on the Occupational Safety and Health Administration proposed rule on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 22 pp.
NTIS NO: PB91-212514 $19.50

NIOSH [1990]. Testimony by R.A. Lemen to the Department of Labor on the Occupational Safety and Health Administration notice of proposed rulemaking on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 17 pp.
NTIS NO: PB91-152439 $19.50

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: PB91-132852 $10.00
(A copy of this statement is contained in Part I of this Bibliography.)

NIOSH [1990]. Comments by R.A. Lemen to the Department of Labor on the Occupational Safety and Health Administration notice of proposed rulemaking on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 15 pp.
NTIS NO: PB91-152413 $19.50

NIOSH [1989]. Comments by R.W. Niemeier to the Environmental Protection Agency on their notice of proposed rule revision, including disposal of asbestos containing materials removed from schools. 6 pp.
NTIS NO: PB90-129404 $10.00

NIOSH [1987]. Comments by J.D. Millar to the Department of Labor on the Occupational Safety and Health Administration final rule on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 6 pp.
NTIS NO: PB90-129370 $10.00

NIOSH [1987]. Comments by R.A. Lemen to the Environmental Protection Agency on their proposed rule on asbestos-containing materials in schools. 6 pp.
NTIS NO: PB91-169003 $10.00

NIOSH [1986]. Comments by J.D. Millar to the Department of Labor on the Occupational Safety and Health Administration final rule on occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. 2 pp.
NTIS NO: PB90-130048 $6.50

NIOSH [1985]. Comments by J.D. Millar to the Environmental Protection Agency on their proposed rule: asbestos abatement projects. 15 pp.
NTIS NO: PB91-152777 $19.50

NIOSH [1984]. Testimony by R.A. Lemen before the Subcommittee on Public Buildings and Grounds, House Committee on Public Works and Transportation. 11 pp.
NTIS NO: PB90-179128 $19.50

NIOSH [1984]. Testimony by R.A. Lemen before the Department of Labor, public hearing on occupational exposure to asbestos. 16 pp.
NTIS NO: PB87-222642 $19.50

NIOSH [1980]. Remarks by A. Robbins on the need for a new asbestos standard. 6 pp.
NTIS NO: PB90-180753 $10.00

NIOSH [1980]. Statement by R.A. Lemen before the Subcommittee on Coast Guard and Navigation, House Committee on Merchant Marine and Fisheries. 11 pp.
NTIS NO: PB90-192964 $19.50

NIOSH [1979]. Statement by A. Robbins before the Subcommittee on Labor Standards, House Committee on Education and Labor. 16 pp.
NTIS NO: PB90-130063 $19.50

NIOSH [1979]. Statement by P. Landrigan on the jewelry industry before the Subcommittee on Labor Standards, House Committee on Education and Labor. 8 pp.
NTIS NO: PB90-192972 $10.00

3. JOURNAL ARTICLES, BOOK CHAPTERS, and PROCEEDINGS

Journal articles, book chapters, and proceedings by NIOSH authors may appear in either U.S. or foreign journals or symposia. The following list, which is in alphabetical order by author, includes the bibliographic information to permit retrieval of the references from public or university libraries.

Abell M [1988]. Quality audit samples and the asbestos analyst registry. Applied Industrial Hygiene *3*(6):R4-R5.

Abell M, Doemeny L [1991]. Monitoring the performance of occupational health laboratories. American Industrial Hygiene Association Journal *52*(8):336-339.

Abell M, Schulman S, Baron P [1989]. The quality of fiber count data. Applied Industrial Hygiene *4*(11):273-285.

Althouse R, Castellan R, Wagner G [1992]. Pneumoconioses in the United States: highlights of surveillance data from NIOSH and other federal sources. Occupational Medicine: State of the Art Reviews *7*(2):197-208.

Amandus H, Wheeler R, Jankovic J, Tucker J [1987]. The morbidity and mortality of vermiculite miners and millers exposed to tremolite-actinolite: part I. Exposure estimates. American Journal of Industrial Medicine *11*(1):1-14.

Amandus H, Wheeler R [1987]. The morbidity and mortality of vermiculite miners and millers exposed to tremolite-actinolite: part II. Mortality. American Journal of Industrial Medicine *11*(1):15-26.

Amandus H, Althouse R, Morgan W, Sargent E, Jones R [1987]. The morbidity and mortality of vermiculite miners and millers exposed to tremolite-actinolite: part III. Radiographic findings. American Journal of Industrial Medicine *11*(1):27-37.

Bang K [1996]. Epidemiology of occupational cancer. Occupational Medicine: State of the Art Reviews *11*(3):467-485.

Baron P [1987]. Asbestos analysis—NIOSH method 7400. Applied Industrial Hygiene *2*(6):R8-R10.

Baron P [1989]. Asbestos measurement and quality control. In: Proceedings of the Asbestos International Association (AIA) 6th International Colloquium on Dust Measurement Technique and Strategy, Jersey, Channel Islands, November 28-30, pp. 142-151.

Baron P [1993]. Exposure to airborne asbestos associated with simulated cable installation above a suspended ceiling [letter to the editor]. American Industrial Hygiene Association Journal *54*(3):A87-A89.

Baron P [1987]. Overloading and electrostatic effects in asbestos fiber sampling. American Industrial Hygiene Association Journal *48*(11):A709-A710.

Baron P [1991]. Phase contrast microscope asbestos fiber counting. Applied Occupational and Environmental Hygiene *6*(3):182.

Baron P, Chen CC, Hemenway D, O'Shaughnessy P [1994]. Nonuniform air flow in inlets: the effect on filter deposits in the fiber sampling cassette. American Industrial Hygiene Association Journal *55*(8):722-732.

Baron P, Deye G [1990]. Electrostatic effects in asbestos sampling I: experimental measurements. American Industrial Hygiene Association Journal *51*(2):51-62.

Baron P, Deye G [1990]. Electrostatic effects in asbestos sampling II: comparison of theory and experiment. American Industrial Hygiene Association Journal *51*(2):63-69.

Baron P, Deye G [1987]. Generation of replicate asbestos aerosol samples for quality assurance. Applied Industrial Hygiene *2*(3):114-118.

Baron P, Deye G, Fernback J [1994]. Length separation of fibers. Aerosol Science and Technology *21*(2):179-192.

Baron P, Pickford G [1986]. An asbestos sample filter clearing procedure. Applied Industrial Hygiene *1*(4):169-171.

Baron P, Platek S [1990]. NIOSH method 7402–asbestos fibers (revision #1)—low-temperature ashing of filter samples. American Industrial Hygiene Association Journal *51*(11):A730-A731.

Baron P, Shulman S [1987]. Evaluation of the Magiscan image analyzer for asbestos fiber counting. American Industrial Hygiene Association Journal *48*(1):39-46.

Bernstein R, Kuempel E, Lemen R [1988]. Environmental exposures to asbestos: a guide for physicians, toxicologists, and public health officials. In: Proceedings Asia-Pacific Symposium on Environmental and Occupational Toxicology, Singapore, October 4-7, 1987, Environmental and Occupational Chemical Hazards, No. 8, pp. 63-78.

Brodkin C, Barnhart S, Checkoway H, Balmes J, Omenn G, Rosenstock L [1996]. Longitudinal pattern of reported respiratory symptoms and accelerated ventilatory loss in asbestos-exposed workers. Chest *109*(1):120-126.

Brown D, Dement J, Okun A [1994]. Mortality patterns among female and male chrysotile asbestos textile workers. Journal of Occupational Medicine *36*(8):882-888.

Brown D, Dement J, Wagoner J [1979]. Mortality patterns among miners and millers occupationally exposed to asbestiform talc. In: Lemen R, Dement J, eds. Dusts and Disease, Proceedings of the Conference on Occupational Exposures to Fibrous and Particulate Dust and Their Extension into the Environment. Park Forest South, IL: Pathotox Publisher, Inc., pp. 317-324.

Burkhart G, Schulte P, Robinson C, Sieber W, Vossenas P, Ringen K [1993]. Job tasks, potential exposures, and health risks of laborers employed in the construction industry. American Journal of Industrial Medicine *24*(4):413-425.

Busch K, Hornung R, Smith R, Leidel N [1979]. Unbiased estimates of coefficients of variation for asbestos counting determined from Johns-Manville data. In: Lemen R, Dement J, eds. Dusts and Disease, Proceedings of the Conference on Occupational Exposures to Fibrous and Particulate Dust and Their Extension into the Environment. Park Forest South, IL: Pathotox Publisher, Inc., pp. 185-197.

Castranova V, Pailes W, Judy D, Schwegler-Berry D, Jones W [1994]. Comparative cytotoxic effects of crocidolite and its non-asbestiform polymorph on rat alveolar macrophages. Inhaled Particles VII, The Annals of Occupational Hygiene *38*(Suppl. 1):665-673.

Costello J, Castellan R, Swecker G, Kullman G [1995]. Mortality of a cohort of U.S. workers employed in the crushed stone industry, 1940-1980. American Journal of Industrial Medicine *27*(5):625-640.

Craighead J, Abraham J, Churg A, Green F, Kleinerman J, Pratt P, Seemayer T, Vallyathan V, Weill H [1982]. Asbestos-associated diseases, special issue. The pathology of asbestos-associated diseases of the lungs and pleural cavities: diagnostic criteria and proposed grading schema. Archives of Pathology and Laboratory Medicine *106*(11):541-596.

Cralley L [1971]. Electromotive phenomenon in metal and mineral particulate exposures: relevance to exposure to asbestos and occurrence of cancer. American Industrial Hygiene Association Journal *32*(10):653-661.

Cralley L, Lainhart W [1973]. Are trace metals associated with asbestos fibers responsible for the biologic effects attributed to asbestos? Journal of Occupational Medicine *15*(3):262-266.

Curtis R, Bierbaum P [1975]. Technological feasibility of the 2 fibers/cc asbestos standard in asbestos textile facilities. American Industrial Hygiene Association Journal *36*(2):115-125.

Dement J, Brown D, Okun A [1994]. Follow-up study of chrysotile asbestos textile workers: cohort mortality and case-control analyses. American Journal of Industrial Medicine *26*(4):431-447.

Dement J, Harris R, Symons M, Shy C [1982]. Estimates of dose-response for respiratory cancer among chrysotile asbestos textile workers. Annals of Occupational Hygiene *26*(1-4):869-887.

Dement J, Wallingford K [1990]. Comparison of phase contrast and electron microscopic methods for evaluation of occupational asbestos exposures. Applied Occupational and Environmental Hygiene *5*(4):242-247.

Dement J, Zumwalde R [1979]. Occupational exposures to talcs containing asbestiform minerals. In: Lemen R, Dement J, eds. Dusts and Disease, Proceedings of the Conference on Occupational Exposures to Fibrous and Particulate Dust and Their Extension into the Environment. Park Forest South, IL: Pathotox Publisher, Inc., pp. 287-305.

Dement J, Zumwalde R, Wallingford K [1976]. Discussion paper: asbestos fiber exposures in a hard rock gold mine. Annals of the New York Academy of Sciences *271*:345-352.

Esche C, Groff J [1997]. Proficiency analytical testing (PAT) program: background and current status. Applied Occupational and Environmental Hygiene *12*(7):456-457.

Fleeger A [1989]. Methylene chloride—minimizing the danger. Asbestos Issues '89, March, pp. 36-37,125-126.

Fleeger A, Lee J [1988]. Characterization of worker exposures to methylene chloride resulting from application of aerosol glue in the asbestos abatement industry. Applied Industrial Hygiene *3*(9):245-250.

Froehlich P [1992]. Analysis of PCM asbestos air monitoring results for a major abatement project [letter to the editor]. Applied Occupational and Environmental Hygiene *7*(5):288-289.

Froehlich P [1993]. Evaluation of two negative-air glove bag techniques for asbestos removal. Applied Occupational and Environmental Hygiene *8*(10):871-882.

Froehlich P, Hollett B [1993]. Evaluation of glove bag containment and asbestos abatement clearance: methodologies for asbestos removal. Applied Occupational and Environmental Hygiene *8*(11):937-944.

Gillam J, Dement J, Lemen R, Wagoner J, Archer V, Blejer H [1976]. Mortality patterns among hard rock gold miners exposed to an asbestiform mineral. Annals of the New York Academy of Sciences *271*:336-344.

Gorski C, Stettler L [1974]. The surface energetics of asbestos minerals. American Industrial Hygiene Association Journal *35*(6):345-353.

Gorski C, Stettler L, Lempert B [1976]. The porosity of amosite and chrysotile asbestos. American Industrial Hygiene Association Journal *37*(2):117-122.

Green F, Attfield M [1983]. Pathology standards for asbestosis. Scandinavian Journal of Work, Environment and Health *9*(2):162-168.

Greenland S, Salvan A, Wegman D, Hallock M, Smith T [1994]. A case-control study of cancer mortality at a transformer-assembly facility. International Archives of Occupational and Environmental Health *66*(1):49-54.

Haartz J, Lange B, Draftz R, Scholl R [1978]. Selection and characterization of fibrous and nonfibrous amphiboles for analytical methods development. In: Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods, National Bureau of Standards (NBS), Gaithersburg, MD, July 18-20, 1977, pp. 295-312.

Hahon N, Booth J [1986]. Coinhibition of viral interferon induction by benzo[a]pyrene and chrysotile asbestos. Environmental Research *40*(1):103-109.

Hahon N, Booth J, Eckert H [1977]. Antagonistic activity of poly(4-vinylpyridine-N-oxide) to the inhibition of viral interferon induction by asbestos fibres. British Journal of Industrial Medicine *34*(2):119-125.

Hahon N, Booth J, Flowers L [1990]. Coinhibition of viral interferon induction by benzo[a]pyrene in association with occupation-related particles. Environmental Research *52*(1):83-98.

Henderson D, Rantanen J, Barnhart S, Dement J, De Vuyst P, Hillerdal G, Huuskonen M, Kivisaari L, Kusaka Y, Lahdensuo A, Langard S, Mowe G, Okubo T, Parker J, Roggli V [1997]. Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. Scandinavian Journal of Work, Environment and Health *23*(4):311-316.

Hodous T [1990]. ILO "A" and "B" reader classification [letter to the editor]. Journal of Occupational Medicine *32*(3):267.

Ing R, Falk H, Lemen R, Carson G, Kelter A, Sarn J, Gray M [1981]. Community asbestos exposure in Globe, Arizona. Journal of Pediatrics *99*(3):409-411.

Jankovic J [1985]. Industrial hygiene forum: asbestos bulk sampling procedure. American Industrial Hygiene Association Journal *46*(2):B8-B10.

Jankovic J, Jones W, Clere J [1986]. Field technique for clearing cellulose esters filters used in asbestos sampling. Applied Industrial Hygiene *1*(3):145-147.

Johnson W, Lemen R, Hurst G, Spiegel R, Lui F [1982]. Respiratory morbidity among workers in an amosite asbestos insulation plant. Journal of Occupational Medicine *24*(12):994-999.

Kang S, Burnett C, Freund E, Walker J, Lalich N, Sestito J [1996]. Gastrointestinal cancer mortality of workers in occupations with high asbestos exposure. American Journal of Industrial Medicine *31*(6):713-718.

Kelley J [1993]. Mastic removal—an educational experience. Applied Occupational and Environmental Hygiene *8*(7):607-608.

Kullman G, Greife A, Costello J, Hearl F [1995]. Occupational exposures to fibers and quartz at 19 crushed stone mining and milling operations. American Journal of Industrial Medicine *27*(5):641-660.

Lange B, Haartz J [1979]. Determination of microgram quantities of asbestos by x-ray diffraction: chrysotile in thin dust layers of matrix material. Analytical Chemistry *51*(4):520-525.

Lemen R, Bingham E [1994]. A case study in avoiding a deadly legacy in developing countries. Toxicology and Industrial Health *10*(1/2):59-87.

Lemen R, Dement J, Wagoner J [1980]. Epidemiology of asbestos-related diseases. Environmental Health Perspectives *34*:1-11.

Lemen R, Meinhardt T, Becking G, Cantor K, Cherner J, Cordle F, Groth D, Keller C, Lybarger J, McConnell E, Millette J, Patel Y, Sonich-Mullin C, Tollefson L [1987]. Report on cancer risks associated with the ingestion of asbestos. Environmental Health Perspectives *72*:253-265.

Lu J, Keane M, Ong T, Wallace W [1994]. In vitro genotoxicity studies of chrysotile asbestos fibers dispersed in simulated pulmonary surfactant. Mutation Research *320*(4):253-259.

Lynch J [1971]. Environmental sampling and analysis of particulate matter: asbestos. In: Transactions of the 35th Annual Meeting of Industrial Health Foundation, Inc., October 13-14, 1970, Chatham Center, Pittsburgh, Pennsylvania, Transactions Bulletin No. 44, Industrial Hygiene Foundation of America, Inc., Pittsburgh, Pennsylvania, pp. 81-86.

McCammon C, Lee S [1991]. Asbestos exposure during low-income housing weatherization. Applied Occupational and Environmental Hygiene *6*(5):347-348.

Morgan A, Cralley L [1973]. Chemical characteristics of asbestos and associated trace elements. In: Biological Effects of Asbestos, IARC Scientific Publication No. 8. Geneva: World Health Organization, International Agency for Research on Cancer, pp. 113-118.

Nevitt C, Daniell W, Rosenstock L [1994]. Workers' compensation for nonmalignant asbestos-related lung disease. American Journal of Industrial Medicine *26*(6):820-830.

Pailes W, Judy D, Resnick H, Castranova V [1984]. Relative effects of asbestos and wollastonite on alveolar macrophages. Journal of Toxicology and Environmental Health *14*(4):497-510.

Petsonk E [1996]. Electric power generation. In: Harber P, Schenker M, Balmes J, eds. Occupational and Environmental Respiratory Disease. St. Louis: Mosby-Year Book Inc., pp. 697-703.

Platek S, Groth D, Ulrich C [1985]. Chronic inhalation of short asbestos fibers. Fundamental and Applied Toxicology *5*(2):327-340.

Platek S, Riley R, Simon S [1992]. The classification of asbestos fibres by scanning electron microscopy and computer-digitizing tablet. The Annals of Occupational Hygiene *36*(2):155-171.

Reger R, Ames R, Merchant J, Polakoff P, Sargent E, Silbiger M, Whittlesey P [1982]. The detection of thoracic abnormalities using posterior-anterior (pa) vs pa and oblique roentgenograms. Chest *81*(3):290-295.

Rinsky R, Melius J, Murray W, Hornung R, Zumwalde R, Waxweiler R, Landrigan P, Bierbaum P [1988]. Case-control study of lung cancer in civilian employees at the Portsmouth Naval Shipyard, Kittery, Maine. American Journal of Epidemiology *127*(1):55-64.

Robinson C, Burnett C [1994]. Mortality patterns of US female construction workers by race, 1979-1990. Journal of Occupational Medicine *36*(11):1228-1233.

Robinson C, Lemen R, Wagoner J [1979]. Mortality patterns, 1940-1975 among workers employed in an asbestos textile friction and packing products manufacturing facility. In: Lemen R, Dement J, eds. Dusts and Disease, Proceedings of the Conference on Occupational Exposures to Fibrous and Particulate Dust and Their Extension in the Environment. Park Forest South, IL: Pathotox Publisher, Inc., pp. 131-143.

Robinson C, Stern F, Halperin W, Venable H, Petersen M, Frazier T, Burnett C, Lalich N, Salg J, Sestito J, Fingerhut M [1995]. Assessment of mortality in the construction industry in the United States, 1984-1986. American Journal of Industrial Medicine *28*(1):49-70.

Ryan G, Buchan R, Keefe T, McCammon C [1996]. A longitudinal study of an American public building following asbestos removal. Applied Occupational and Environmental Hygiene *11*(12):1417-1423.

Schlecht P, Shulman S [1986]. Performance of asbestos fiber counting laboratories in the NIOSH Proficiency Analytical Testing (PAT) program. American Industrial Hygiene Association Journal *47*(5):259-269.

Schlecht P, Shulman S [1995]. Phase contrast microscopy asbestos fiber counting performance in the proficiency analytical testing program. American Industrial Hygiene Association Journal *56*(5):480-489.

Schulte P, Halperin W, Herrick M, Connally B [1987]. The current focus of biological monitoring. In: Foa V, Emmett E, Maroni M, Colombi A, eds. Occupational and Environmental Chemical Hazards: Cellular and Biochemical Indices for Monitoring Toxicity. Chichester, UK: Ellis Horwood Limited, pp. 50-60.

Seitz T, Kinnes G [1991]. Workplace exposures at a waste-to-energy facility. Applied Occupational and Environmental Hygiene *6*(7):573-574.

Selikoff I, Stokinger H [1973]. Cancer risk to allergy patients from syringes with asbestos-wound plunger. Journal of the American Medical Association *225*(4):423.

Sepulveda M, Merchant J [1983]. Roentgenographic evidence of asbestos exposure in a select population of railroad workers. American Journal of Industrial Medicine *4*(5):631-639.

Sheehy J, Cooper T, O'Brien D [1989]. Control of asbestos exposure during brake drum service. Applied Industrial Hygiene *4*(12):313-319.

Short S, Petsonk E [1993]. Respiratory health risks among nonmetal miners. Occupational Medicine: State of the Art Reviews *8*(1):57-70.

Simeonova P, Luster M [1996]. Asbestos induction of nuclear transcription factors and interleukin 8 gene regulation. American Journal of Respiratory Cell and Molecular Biology *15*:787-795.

Sinks T, Goodman M, Kolonel K, Anderson B [1994]. A case-control study of mesothelioma and employment in the Hawaii sugarcane industry. Epidemiology *5*(4):466-468.

Sinks T, Hartle R, Boeniger M, Mannino D, Boyd J, Fernback J, Hawkins M, Grimes G, Watkins K, Dill P, Anderson B [1994]. Exposure to biogenic silica fibers and respiratory health in Hawaii sugarcane workers. Journal of Occupational Medicine *36*(12):1329-1334.

Stayner L, Dankovic D, Lemen R [1996]. Occupational exposure to chrysotile asbestos and cancer risk: a review of the amphibole hypothesis. American Journal of Public Health *86*(2):179-186.
(A copy of this article is included in Part I of this Bibliography.)

Stayner L, Dankovic D, Lemen R [1997]. Asbestos-related cancer and the amphibole hypothesis [reply to letters to the editor]. American Journal of Public Health *87*(4):687-691.

Stayner L, Kuempel E, Rice F, Prince M, Althouse R [1996]. Approaches for assessing the efficacy of occupational health and safety standards. American Journal of Industrial Medicine *29*(4):353-357.

Stayner L, Smith R, Bailer J, Gilbert S, Steenland K, Dement J, Brown D, Lemen R [1997]. Exposure-response analysis of risk of respiratory disease associated with occupational exposure to chrysotile asbestos. Occupational and Environmental Medicine *54*:646-652.

Steenland K, Loomis D, Shy C, Simonsen N [1996]. Occupational causes of lung cancer. In: Harbor P, Schenker M, Balmes J, eds. Occupational and Environmental Respiratory Disease. St. Louis: Mosby-Year Book, Inc., pp. 589-607.

Steenland K, Loomis D, Shy C, Simonsen N [1996]. Review of occupational lung carcinogens. American Journal of Industrial Medicine *29*(5):474-490.

Stern F, Schulte P, Haring Sweeney M, Fingerhut M, Vossenas P, Burkhardt G, Kornak M [1995]. Proportionate mortality among construction laborers. American Journal of Industrial Medicine *27*(4):485-509.

Sullivan P, Bang K, Hearl F, Wagner G [1995]. Respiratory disease risks in the construction industry. Occupational Medicine: State of the Art Reviews *10*(2):313-334.

Taylor D, Baron P, Shulman S, Carter J [1984]. Identification and counting asbestos fibers. American Industrial Hygiene Association Journal *45*(2):84-88.

Tillett S, Ringen K, Schulte P, McDougall V, Miller K, Samuels S [1986]. Interventions in high-risk occupational cohorts: a cross-sectional demonstration project. Journal of Occupational Medicine *28*(8):719-727.

Vallyathan V [1994]. Oxygen radical generation by asbestos and its correlation to cytotoxicity. In: Davis J and Jaurand M, eds. Cellular and Molecular Effects of Mineral and Synthetic Dusts and Fibres. Berlin: Springer-Verlag, NATO ASI Series, Volume H 85, pp. 9-21.

Vallyathan V, Green F [1985]. The role of analytical techniques in the diagnosis of asbestos-associated disease. CRC Critical Reviews in Clinical Laboratory Sciences *22*(1):1-42.

Vallyathan V, Hahon N, Booth J, Schwegler D, Sepulveda M [1985]. Cytotoxicity of native- and surface-modified asbestos. In: Beck E, Bignon J, eds. In Vitro Effects of Mineral Dusts. Third International Workshop. Berlin: Springer-Verlag, pp. 159-165.

Vallyathan V, Mega J, Shi X, Dalal N [1992]. Enhanced generation of free radicals from phagocytes induced by mineral dusts. American Journal of Respiratory Cell and Molecular Biology *6*(4):404-413.

Wagner G [1997]. Asbestosis and silicosis. The Lancet *349*(9061):1311-1315.

Wagner G [1994]. Mineral dusts. In: Rosenstock L, Cullen M, eds. Textbook of Clinical Occupational and Environmental Medicine. Philadelphia: W.B. Saunders Company, pp. 825-837.

Wagner G, Attfield M, Parker J [1993]. Chest radiography in dust-exposed miners: promise and problems, potential and imperfections. Occupational Medicine: State of the Art Reviews *8*(1):127-141.

Waxweiler R, Robinson C [1983]. Asbestos and non-Hodgkin's lymphoma. The Lancet *1*(8317):189-190.

Weidner R, Ayer H [1972]. Dust exposure in asbestos processing. In: Transactions of the 34th Annual Meeting of the American Conference of Governmental Industrial Hygienists, San Francisco, CA, May 14-19, 1972, pp. 103-124.

White M, Hodous T, Hudnall J [1989]. Physiological and subjective responses to working in disposable protective coveralls and respirators commonly used by the asbestos abatement industry. American Industrial Hygiene Association Journal *50*(6):313-319.

4. MISCELLANEOUS REPORTS

Miscellaneous reports include all other NIOSH-authored documents not included in the previous three sections. In addition to the reports listed below, NIOSH also has produced Health Hazard Evaluation (HHE) reports, Industrywide Study (IWS) reports, and Control Technology (CT) reports related to asbestos that are not listed in this bibliography. These three types of reports examine conditions at specific worksites. HHE reports are the results of requests from employees, employee representatives, or employers to NIOSH to determine if a hazard exists in the workplace. IWS reports document information obtained during brief 1- to 2-day walk-through surveys at plant sites and represent the results of industrial hygiene field studies that assess whether specific occupational exposures of particular workers are associated with adverse health effects. CT reports represent the results of field study surveys that evaluate health hazard control systems at individual worksites. A list of HHE, IWS, and CT reports on asbestos, along with ordering information, is available from NIOSH at the address shown on page ii.

Anonymous [1973]. Caution: asbestos dust . . . is hazardous to your health. 16 pp.
NTIS NO: PB88-204649 $19.50

Anonymous [1979]. Mining surveillance: potentially toxic occupational exposures. 24 pp.
NTIS NO: PB298973 $19.50

Anonymous [1988]. National occupational health survey of mining, asbestos report. 82 pp.
NTIS NO: PB90-163676 $25.00

Anonymous [1984]. A review of the scientific basis for EPA's school asbestos hazard program with recommendations to state health officials. 24 pp.
NTIS NO: PB85-221448 $19.50

Anonymous [1979]. Sampling and evaluating airborne asbestos dust - NIOSH Course 582. 66 pp.
NTIS NO: PB85-246023 $21.50

Ayer H, Zumwalde R [1979]. Physical and chemical characteristics of airborne fibers. 41 pp.
NTIS NO: PB87-234662 $21.50

Baron P, Crouch K [1982]. Report on testing of the automated fiber counter. 16 pp.
NTIS NO: PB83-192997 $19.50

Conroy C, Smith D, Bender T [1988]. Fatal accident circumstances and epidemiology (FACE) report 88-28, asbestos worker electrocuted. 7 pp.
NTIS NO: PB89-187827 $10.00

Dement J [1981]. Epidemiology of asbestos related diseases. 49 pp.
NTIS NO: PB90-155904 $21.50

Dye G, Baron P [1990]. Application of neural network technology to fiber image analysis, final report: analytical methods for asbestos fibers. 46 pp.
NTIS NO: PB91-169367 $21.50

Geraci C, Baron P, Carter J, Smith D [1979]. Testing of hair dryers for asbestos emissions, NIOSH/CPSC Interagency Agreement (IA) 79-29. 61 pp.
NTIS NO: PB89-165237 $21.50

Hankinson J, Stettler L, Baron P, Lewis D, Zumwalde R, Brown D, Noonan G, Haag W, Okun A, Wagner G, Lemen R [1993]. NIOSH occupational fiber exposures and lung disease research strategy (version 7.4). 23 pp.
NTIS NO: PB94-173739 $19.50

Leidel N, Busch K [1974]. An evaluation of phase contrast microscopes for asbestos counting. 12 pp.
NTIS NO: PB90-130022 $19.50

Lemen R, Selikoff I, Hurst G, Wagoner J [1971]. Asbestos related disease - a community epidemic in the making. 7 pp.
NTIS NO: PB90-155896 $10.00

Pailes W, Judy D, Resnick H, Castranova V [1983]. Cellular toxicity of mineral dusts. 23 pp.
NTIS NO: PB83-232264 $19.50

Wallingford K [1978]. Chrysotile asbestos in industry. 22 pp.
NTIS NO: PB90-155953 $19.50

B. NIOSH-FUNDED DOCUMENTS

1. GRANT and COOPERATIVE AGREEMENT REPORTS

Grant and cooperative agreement (CA) reports are generated primarily from an agreement between NIOSH and a non-governmental organization. They typically describe scientific research conducted by that organization and funded by NIOSH. Grant and CA reports, listed below in alphabetical order by author, may be published either as final reports available from NTIS or as journal articles. For the former, NTIS ordering information is shown; for the latter, bibliographic information is provided to permit retrieval from public or university libraries.

Allen M [1971]. Dissolution and cation exchange properties of some asbestos minerals in aqueous media, minerals research progress report.
GRANT NO: R01-OH-00332. 96 pp.
NTIS NO: PB88-247838 $25.00

Allen M [1973]. Dissolution of asbestos minerals in neutral salt solutions, minerals research progress report no. 2.
GRANT NO: R01-OH-00332. 52 pp.
NTIS NO: PB88-237748 $21.50

Anderson H, Hanrahan L, Higgins D, Sarow P [1992]. A radiographic survey of public school building maintenance and custodial employees. Environmental Research *59*(1):159-166.
CA NO: U50/CCU-502661

Anderson H, Hanrahan L, Schirmer J, Higgins D, Sarow P [1991]. Mesothelioma among employees with likely contact with in-place asbestos-containing building materials. Annals of the New York Academy of Sciences *643*:550-572.
CA NO: U53/CCU-500801 and U60/CCU-502984

Ayer H, Burg J [1975]. Cumulative asbestos exposure and forced vital capacity.
GRANT NO: T01-OH-00028. 21 pp.
NTIS NO: PB90-153552 $19.50

Baldwin C, Beaulieu H, Buchan R, Johnson H [1982]. Asbestos in Colorado schools. Public Health Reports *97*(4):325-331.
GRANT NO: R18-OH-01053

Barker R [1981]. Asbestos substitute fabrics for safety clothing, final progress report.
GRANT NO: R01-OH-00910. 10 pp.
NTIS NO: PB84-238385 $10.00

Brandt-Rauf P, Smith S, Hemminki K, Koskinen H, Vainio H, Niman H, Ford J [1992]. Serum oncoproteins and growth factors in asbestosis and silicosis patients. International Journal of Cancer *50*(6):881-885.
GRANT NO: K01-OH-00076

Broderick A, Fuortes L, Merchant J, Galvin J, Schwartz D [1992]. Pleural determinants of restrictive lung function and respiratory symptoms in an asbestos-exposed population. Chest *101*(3):684-691.
GRANT NO: K01-OH-00093

Brosseau L, Ellenbecker M, Evans J [1990]. Collection of silica and asbestos aerosols by respirators at steady and cyclic flow. American Industrial Hygiene Association Journal *51*(8):420-426
GRANT NO: R01-OH-02154 and T15-OH-07096

Brousseau L, Evans J, Ellenbecker M [1993]. An empirical model for estimating the collection efficiency of dust-mist respirators. Annals of Occupational Hygiene *37*(2):135-150.
GRANT NO: R01-OH-02154 and T15-OH-07096

Buchan R [1982]. The safe asbestos treatment program, project report.
GRANT NO: R18-OH-01053. 148 pp.
NTIS NO: PB84-238351 $31.00

Buchan R, Richardson D, Beaulieu H, Keefe T [1984]. Development of a mathematical model for predicting concentrations of small asbestos fibers. Environmental Research *33*(2):296-299.
GRANT NO: T01-OH-07039

Bunn W, Hunninghake G, Broderick A, Wilson J, Galvin J, Merchant J, Watt J, Dayton C, Schwartz D [forthcoming]. The clinical relevance of dsypnea in workers exposed to asbestos.
GRANT NO: K01-OH-00093

Burns P, Swanson G [1991]. The occupational cancer incidence surveillance study (OCISS): risk of lung cancer by usual occupation and industry in the Detroit metropolitan area. American Journal of Industrial Medicine *19*(5):655-671.
GRANT NO: R01-OH-02067

Burrell R [1974]. Immunological reflections on asbestos. Environmental Health Perspectives *9*:297-298.
GRANT NO: R01-OH-00360

Cheng Y, Fan B, Holmes T, Yeh H [1996]. Evaluation of respirator filters for asbestos fibers, final performance report.
GRANT NO: R01-OH-02922. 56 pp.
NTIS NO: PB97-105209 $21.50

Choi I, Smith R [1972]. Kinetic study of dissolution of asbestos fibers in water. Journal of Colloid Interface Science *40*(2):253-262.
GRANT NO: R01-OH-00332

Cicioni C, London S, Garabrant D, Bernstein L, Phillips K, Peters J [1991]. Occupational asbestos exposure and mesothelioma risk in Los Angeles County: application of an occupational hazard survey job-exposure matrix. American Journal of Industrial Medicine *20*(3):371-379.
GRANT NO: T15-OH-07214

Craighead J, Mossman B, Bradley B [*1980*]. Comparative studies on the cytotoxicity of amphibole and serpentine asbestos. Environmental Health Perspectives *34*:37-46.
GRANT NO: R01-OH-00653

Cullen M, Merrill W, Marenberg M [1994]. A model for staging asbestos-related lung effects after cessation of exposure based on clinical demographic and bronchoscopic data. In: Mehlman M, Upton A, eds. The Identification and Control of Environmental and Occupational Diseases: Asbestos and Cancers, Advances in Modern Environmental Toxicology, Volume XXII. Princeton: Princeton Scientific Publishing Company, Inc., pp. 287-303.
GRANT NO: R01-OH-02114

Davis H, Reeves A [1971]. Collagen biosynthesis in rat lungs during exposure to asbestos. American Industrial Hygiene Association Journal *32*(9):599-602.
GRANT NO: R01-OH-00323.

Demers R, Burns P, Swanson G [1994]. Construction occupations, asbestos exposure, and cancer of the colon and rectum. Journal of Occupational Medicine *36*(9):1027-1031.
GRANT NO: R01-OH-02067

Evans J, Brosseau L, Ellenbecker M [1989]. Asbestos fiber collection by NIOSH-approved respirators, final performance report.
GRANT NO: R01-OH-02154. 7 pp.
NTIS NO: PB90-153545 $10.00

Ferin J, Leach L [1976]. The effect of amosite and chrysotile asbestos on the clearance of TiO2 particles from the lung. Environmental Research *12*(2):250-254.
GRANT NO: R01-OH-00334

Ghio A, Crumbliss A [1991]. Surface complexation of Fe3+ by silica and silicate dusts increases in vitro oxidant generation but diminishes in vitro cytotoxicity.
GRANT NO: R01-OH-02264. 24 pp.
NTIS NO: PB92-136357 $19.50

Gross P [1972]. Dose-effect relationship of asbestos dust, final technical report.
GRANT NO: R01-OH-00354. 17 pp.
NTIS NO: PB90-129859 $19.50

Gross P, Harley R [1972]. Asbestos dust: a study on the pathogenetic mechanism.
GRANT NO: R01-OH-00326. 22 pp.
NTIS NO: PB90-129396 $19.50

Gross P, Harley R [1972]. Asbestos-induced intrathoracic tissue reactions.
GRANT NO: R01-OH-00326. 20 pp.
NTIS NO: PB88-248380 $19.50

Hammond E, Selikoff I [1972]. Relation of cigarette smoking to risk of death of asbestos-associated disease among insulation workers in the United States.
GRANT NO: R01-OH-00305. 13 pp.
NTIS NO: PB90-103433 $19.50

Hammond C, Selikoff I, Churg J [1972]. Carcinogenicity of amosite asbestos. Archives of Environmental Health *25*:183-186.
GRANT NO: R01-OH-00305

Harkin T, McGuinness G, Goldring R, Cohen H, Parker J, Crane M, Naidich D, Rom W [1996]. Differentiation of the ILO boundary chest roentgenograph (0/1 to 1/0) in asbestosis by high-resolution computed tomography scan, alveolitis, and respiratory impairment. Journal of Occupational and Environmental Medicine *38*(1):46-52.
CA NO: U60/CCU-206153

Hartley P, Galvin J, Hunninghake G, Merchant J, Yagla S, Speakman S, Schwartz D [1994]. High-resolution CT-derived measures of lung density are a valid index of interstitial lung disease. Journal of Applied Physiology *76*(1):271-277.
GRANT NO: K01-OH-00093

Hawaii Department of Health [1981]. School asbestos fund for removal activities, terminal progress report.
GRANT NO: R18-OH-01062. 105 pp.
NTIS NO: PB84-239474 $28.00

Homa D, Garabrant D, Gillespie B [1994]. A meta-analysis of colorectal cancer and asbestos exposure. American Journal of Epidemiology *139*(12):1210-1222.
GRANT NO: T15-OH-07207

Kannerstein M, Churg J [1972]. Pathology of carcinoma of the lung associated with asbestos exposure. Cancer *30*(1):14-21.
GRANT NO: R01-OH-00305

Kaselaan & D'Angelo Associates [1982]. Evaluation of a commercial vacuum system for asbestos removal, a safety and cost evaluation.
GRANT NO: R18-OH-01072. 175 pp.
NTIS NO: PB85-222982 $35.00

Kline J, Schwartz D, Monick M, Floerchinger C, Hunninghake G [1993]. Relative release of interleukin-1 beta and interleukin-1 receptor antagonist by alveolar macrophages. A study in asbestos-induced lung disease, sarcoidosis, and idiopathic pulmonary fibrosis. Chest *104*(1):47-53.
GRANT NO: K01-OH-00093

Langer A, Hammond E, Selikoff I [1971]. Inorganic fibers, including chrysotile, in lungs at autopsy: preliminary report. Inhaled Particles III, Proceedings of an International Symposium. Surrey, UK: Unwin Brothers Ltd., Gresham Press, pp. 683-694.
GRANT NO: R01-OH-00320

Langer A, Rubin I, Selikoff I, Pooley F [1972]. Chemical characterization of uncoated asbestos fibers from the lungs of asbestos workers by electron microprobe analysis. Journal of Histochemistry and Cytochemistry *20*(9):735-740.
GRANT NO: R01-OH-00305

Langer A, Selikoff I, Rosenberg C [1979]. Asbestos in brake worker's lungs: an exposure index. Defining new asbestos high risk groups (abstract). In: Proceedings of International Conference on Critical Current Issues in Environmental Health Hazards, Tel Aviv, Israel, March 4-7, p. 11.
GRANT NO: OH-00734-01

Langer A, Selikoff I, Sastre A [1971]. Chrysotile asbestos in the lungs of persons in New York City. Archives of Environmental Health *22*:348-361.
GRANT NO: R01-OH-00320

Levin S [1994]. Abnormalities consistent with asbestos-related disease among long-term demolition workers.
CA NO: U60/CCU-306169. 16 pp.
NTIS NO: PB97-151757 $19.50

Luo J, Zehab R, Antilla S, Ridanpaa M, Husgafvel-Pursiainen K, Vainio H, Carney W, DeVivo I, Milling C, Brandt-Rauf P [1994]. Detection of serum p53 protein in lung cancer patients. Journal of Occupational Medicine *36*(2):155-160.
GRANT NO: K01-OH-00076

Martin B [1981]. Asbestos removal or treatment in Oklahoma schools, terminal progress report.
GRANT NO: R18-OH-01049. 6 pp.
NTIS NO: PB88-248422 $10.00

Martin B, Hodges J, Hallett P, Myers R [1981]. Bidding documents for asbestos abatement in Oklahoma public buildings.
GRANT NO: R18-OH-01049. 59 pp.
NTIS NO: PB88-248430 $21.50

Merrill W, Cullen M, Carter D, Horwitz R, Matthay R, Gee J [1991]. Epithelial surface proteins: markers of cancer risk.
GRANT NO: R01-OH-02114. 49 pp.
NTIS NO: PB92-115674 $21.50

Miller A, Langer A, Teirstein A, Selikoff I [1975]. "Nonspecific" interstitial pulmonary fibrosis - association with asbestos fiber detected by electron microscopy. New England Journal of Medicine *292*(2):91-93.
GRANT NO: R01-OH-00320

Mossman B, Craighead J [1982]. Comparative cocarcinogenic effects of crocidolite asbestos, hematite, kaolin and carbon in implanted tracheal organ cultures. Inhaled Particles V, The Annals of Occupational Hygiene *26*(1-4):553-567.
GRANT NO: R01-OH-00888

Mossman B, Craighead J [1983]. Mechanisms of asbestos and nonasbestiform particles and fibers in bronchogenic carcinoma. In: Wagner W, Rom W, Merchant J, eds. Health Issues Related to Metal and Nonmetallic Mining. Boston: Butterworth Publishers, pp. 123-134.
GRANT NO: R01-OH-00888

Mossman B, Craighead J, MacPherson B [1980]. Asbestos-induced epithelial changes in organ cultures of hamster trachea: inhibition by retinyl methyl ether. Science *207*(4428):311-313.
GRANT NO: R01-OH-00653

Mossman B, Marsh J, Gilbert R, Shatos M, Doherty J, Cutroneo K [1986]. Cellular and molecular mechanisms of asbestosis. Chest *89*(3):160S-161S.
GRANT NO: R01-OH-00007

Mossman B, Marsh J, Hardwick D, Gilbert R, Hill S, Sesko A, Shatos M, Doherty J, Weller A, Bergeron M [1986]. Approaches to prevention of asbestos-induced lung disease using polyethylene glycol (peg)-conjugated catalase. Journal of Free Radicals in Biology and Medicine 2:335-338.
GRANT NO: R01-OH-00007

Mossman B, Marsh J, Shatos M [1986]. Alteration of superoxide dismutase activity in tracheal epithelial cells by asbestos and inhibition of cytotoxicity by antioxidants. Laboratory Investigation *54*(2):204-212.
GRANT NO: R01-OH-00007

Murphy R, Ferris B, Burgess W, Worcester J, Gaensler E [1971]. Effects of low concentrations of asbestos - clinical, environmental, radiologic and epidemiologic observations in shipyard pipe coverers and controls. New England Journal of Medicine *285*(23):1271-1278.
GRANT NO: R01-OH-00310

Nicholson W, Maggiore C, Selikoff I [1972]. Asbestos contamination of parenteral drugs. Science *177*(4044): 171-173.
GRANT NO: R01-OH-00305

Ohio Technology Transfer Organization [1990]. Danger asbestos. Working on brakes? Think about this.
(A copy of this bulletin, which was produced under a cooperative agreement with NIOSH, is included in Part I of this Bibliography.)

Partanen R, Hemminki K, Koskinen H, Luo J, Carney W, Brandt-Rauf P [1994]. The detection of increased amounts of the extracellular domain of the epidermal growth factor receptor in serum during carcinogenesis in asbestosis patients. Journal of Occupational Medicine *36*(12):1324-1328.
GRANT NO: K01-OH-00076

Reeves A, Puro H, Smith R, Vorwald A [1971]. Experimental asbestos carcinogenesis. Environmental Research *4*(6):496-511.
GRANT NO: R01-OH-00323

Reitze W, Nicholson W, Holaday D, Selikoff I [1972]. Application of sprayed inorganic fiber containing asbestos: occupational health hazards. American Industrial Hygiene Association Journal *33*(1):178-191.
GRANT NO: R01-OH-00305

Schwartz D [1991]. The clinical relevance of asbestos-induced pleural fibrosis. Annals of the New York Academy of Sciences *643*:169-177.
GRANT NO: K01-OH-00093

Schwartz D [1991]. New developments in asbestos-induced pleural disease. Chest *99*(1):191-198.
GRANT NO: K01-OH-00093

Schwartz D, Davis C, Merchant J, Bunn W, Galvin J, Van Fossen D, Dayton C, Hunninghake G [1994]. Longitudinal changes in lung function among asbestos-exposed workers. American Journal of Respiratory and Critical Care Medicine *150*(5):1243-1249.
GRANT NO: K01-OH-00093

Schwartz D, Fuortes L, Galvin J, Burmeister L, Schmidt L, Leistikow B, Lamarte F, Merchant J [1990]. Asbestos-induced pleural fibrosis and impaired lung function. American Review of Respiratory Disease *141*(2):321-326.
GRANT NO: K01-OH-00093

Schwartz D, Galvin J, Burmeister L, Merchant R, Dayton C, Merchant J, Hunninghake G [1991]. The clinical utility and reliability of asbestos bodies in bronchoalveolar fluid. American Review of Respiratory Disease *44*(3):684-688.
GRANT NO: K01-OH-00093

Schwartz D, Galvin J, Dayton C, Stanford W, Merchant J, Hunninghake G [1990]. Determinants of restrictive lung function in asbestos-induced pleural fibrosis. Journal of Applied Physiology *68*(5):1932-1937.
GRANT NO: K01-OH-00093

Schwartz D, Galvin J, Frees K, Dayton C, Burmeister L, Merchant J, Hunninghake G [1993]. Clinical relevance of cellular mediators of inflammation in workers exposed to asbestos. American Review of Respiratory Disease *148*(1):68-74.
GRANT NO: K01-OH-00093

Schwartz D, Galvin J, Merchant R, Dayton C, Burmeister L, Merchant J, Hunninghake G [1992]. Influence of cigarette smoking on bronchoalveolar lavage cellularity in asbestos-induced lung disease. American Review of Respiratory Disease *145*(2):400-405.
GRANT NO: K01-OH-00093

Schwartz D, Galvin J, Yagla S, Speakman S, Merchant J, Hunninghake G [1993]. Restrictive lung function and asbestos-induced pleural fibrosis. A quantitative approach. Journal of Clinical Investigation *91*(6):2685-2692.
GRANT NO: K01-OH-00093

Selikoff I [1974]. Asbestos criteria document highlights. American Society of Safety Engineers Journal *19*(3):26-33.
GRANT NO: R01-OH-00320

Selikoff I, Hammond E, Seidman H [1972]. Cancer risk of insulation workers in the United States.
GRANT NO: R01-OH-00305. 25 pp.
NTIS NO: PB90-103813 $19.50

Selikoff I, Hammond E, Seidman H [1979]. Mortality experience of insulation workers in the United States and Canada, 1943-1976. Annals of the New York Academy of Sciences *330*:91-116.
GRANT NO: R01-OH-00320

Selikoff I, Nicholson W, Langer A [1972]. Asbestos air pollution. Archives of Environmental Health 25:1-13.
GRANT NO: R01-OH-00305

Shatos M, Doherty J, Marsh J, Mossman B [1987]. Prevention of asbestos-induced cell death in rat lung fibroblasts and alveolar macrophages by scavengers of active oxygen species. Environmental Research *44*(1):103-116.
GRANT NO: K01-OH-00007

Shih J, Hunninghake G, Goeken N, Galvin, J, Merchant J, Schwartz D [1993]. The relationship between HLA-A, B, DQ, and DR antigens and asbestos-induced lung disease. Chest *104*(1):26-31.
GRANT NO: K01-OH-00093

Shih J, Wilson J, Broderick A, Watt J, Galvin J, Merchant J, Schwartz D [1994]. Asbestos-induced pleural fibrosis and impaired exercise physiology. Chest *105*(5):1370-1376.
GRANT NO: K01-OH-00093

Smith R [1973]. Aqueous surface chemistry of asbestos minerals, final progress report.
GRANT NO: R01-OH-00332. 21 pp.
NTIS NO: PB90-100942 $19.50

Smith R, Choi I [1984]. The behavior in aqueous solutions of transition metals associated with asbestos minerals and its implication in tumor initiation in the lung. Speculations in Science and Technology *7*(1):27-36.
GRANT NO: R01-OH-00332

Smith C, Kelsey K, Wiencke J, Leyden K, Levin S, Christiani D [1994]. Inherited glutathione-s-transferase deficiency is a risk factor for pulmonary asbestosis. Cancer Epidemiology, Biomarkers and Prevention *3*(6):471-477.
GRANT NO: K01-OH-00110

Stolwijk J [1980]. Continuous optical monitoring of asbestos in air.
GRANT NO: R01-OH-00647. 8 pp.
NTIS NO: PB88-247598 $10.00

Swanson G [1995]. Occupational cancer surveillance: new approaches, final performance report.
GRANT NO: R01-OH-02067. 25 pp.
NTIS NO: PB95-269890 $19.50

Swift D [1986]. Fiber deposition in human upper airway model, final performance report.
GRANT NO: R01-OH-01605. 4 pp.
NTIS NO: PB89-130728 $6.50

Timblin C, Janssen Y, Mossman B [1995]. Transcriptional activation of the proto-oncogene c-jun by asbestos and H2O2 is directly related to increased proliferation and transformation of tracheal epithelial cells. Cancer Research *55*(13):2723-2726.
GRANT NO: K01-OH-00146

Treadwell M [1997]. Asbestos induced alteration in endothelial cell function.
GRANT NO: R03-OH-03267. 8 pp.
NTIS NO: PB97-206296 $10.00

Treadwell M, Fava R, Hunt J, Krieser R, Barchowsky A [1996]. Expression and activity of urokinase and its receptor in endothelial cells exposed to asbestos.
GRANT NO: R03-OH-03267. 30 pp.
NTIS NO: PB97-206304 $19.50

Treadwell M, Mossman B, Barchowsky [1996]. Increased neutrophil adherence to endothelial cells exposed to asbestos. Toxicology and Applied Pharmacology *139*(1):62-70.
GRANT NO: R03-OH-03267

Treuting E, ed. [1979]. Occupational health nursing.
GRANT NO: A14-OH-00010. 190 pp.
NTIS NO: PB83-103580 $38.00

University of Missouri [1981]. Missouri workshops on asbestos treatment in schools.
GRANT NO: R18-OH-01054. 9 pp.
NTIS NO: PB84-240555 $10.00

Valic F, Beritic-Stahuljak D, Rossiter C, Skuric Z, Zuskin E [1983]. Early detection of health hazards due to asbestos exposure, final report.
GRANT NO: PL-480-02-009-3. 215 pp.
NTIS NO: PB83-244434 $41.00

Valic F, Beritic-Stahuljak D, Rossiter C, Skuric Z, Zuskin E [1989]. Early detection of health hazards due to asbestos exposure, final report.
GRANT NO: PL-480-02-009-3. 47 pp.
NTIS NO: PB89-215669 $21.50

Zhang Y, Lee T, Guillemin B, Yu M, Rom W [1993]. Enhanced IL-1beta and tumor necrosis factor-alpha release and messenger RNA expression in macrophages from idiopathic pulmonary fibrosis or after asbestos exposure. Journal of Immunology *150*(9):4188-4196.
CA NO: U60/CCU-206153

2. CONTRACT REPORTS

Contract reports are generated primarily from a contractual agreement between NIOSH and a non-governmental organization. They typically describe scientific research conducted by that organization for NIOSH.

Chen C, Katt R, Kent M, Hamrick M, Perry W, Smith D, Spooner C, Moore E [1983]. Technological feasibility of controlling asbestos and silica at mines and mills, final report, volumes 1 and 2.
CONTRACT NO: 210-81-4104. 299 pp. (Vol. 1), 248 pp. (Vol. 2)
NTIS NO: PB83-244343 (Vol. 1) $49.00
NTIS NO: PB83-244350 (Vol. 2) $44.00

Cooper W, Black R, Popendorf W, Gaffey W [1975]. Study of sheet metal workers, final report.
CONTRACT NO: 099-71-0055. 41 pp.
NTIS NO: PB83-101220 $21.50

Kaplan S, Gaffey W [1981]. Miners exposed to amphibole mineral, a retrospective cohort mortality study.
CONTRACT NO: 210-76-0200. 50 pp.
NTIS NO: PB87-163754 $21.50

Nicholson W, Daum S, Lorimer W, Velez H, Lilis R, Selikoff I, Miller A, Anderson H, Fischbein S, Holstein E, Rom W, Rosenman K, Todaro J, Cheng W, Li V, Tarr D [1982]. Investigation of health hazards in brake lining repair and maintenance workers occupationally exposed to asbestos.
CONTRACT NO: 210-77-0119. 130 pp.
NTIS NO: PB83-220897 $31.00

Polakoff P [1980]. Bay area asbestos surveillance project (BAASP), final report.
CONTRACT NO: 210-78-0113. 68 pp.
NTIS NO: PB87-105466 $21.50

Sunderman F [1975]. Electromotive interactions of metals in asbestos carcinogenicity.
CONTRACT NO: 099-72-0024. 46 pp.
NTIS NO: PB82-152943 $21.50

Wertheimer A, Trainer M [1979]. Development of an automated fiber counting instrument.
CONTRACT NO: 210-77-0067. 45 pp.
NTIS NO: PB83-113142 $21.50

Whisnant R [1975]. Evaluation of image analysis equipment applied to asbestos fiber counting.
CONTRACT NO: 210-75-0080. 57 pp.
NTIS NO: PB83-109702 $21.50

Wingert K [1973]. Asbestos - beryllium study, final report.
CONTRACT NO: 099-71-0017. 7 pp.
NTIS NO: PB82-230988 $10.00

PART III

OTHER INFORMATION ON ASBESTOS

ASBESTOS
CAS # 1332-21-4

Agency for Toxic Substances and Disease Registry — September 1996

This fact sheet answers the most frequently asked health questions about asbestos. For more information, you may call 404-639-6000. This fact sheet is one in a series of summaries about hazardous substances and their health effects. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

SUMMARY: Exposure to asbestos usually occurs by breathing contaminated air in workplaces that make or use asbestos. Asbestos is also found in the air of buildings containing asbestos that are being torn down or renovated. Asbestos exposure can cause cancer and other serious lung problems. This substance has been found in at least 58 of the 1,430 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is asbestos?
(Pronounced ăs-bĕs'təs)

Asbestos is the name that's used for a group of six different fibrous minerals (amosite, chrysotile, crocidolite, and the fibrous varieties of tremolite, actinolite, and anthophyllite) that occur naturally in soil and rocks in some areas. Asbestos fibers vary in length and may be straight or curled.

Asbestos fibers are resistant to heat and most chemicals. Because of this, asbestos fibers are used for a wide range of manufactured goods, mostly roofing shingles, ceiling and floor tiles, paper products, asbestos cement products, friction products (automobile clutch, brake, and transmission parts), textiles, packaging, gaskets, and coatings.

What happens to asbestos when it enters the environment?

- Asbestos can enter the air and water from the weathering of natural deposits and the wearing down of manufactured asbestos products, such as brake pads.
- Small fibers may remain suspended in the air for a long time before settling. Larger fibers tend to settle more quickly.
- Asbestos fibers aren't able to move through soil and they aren't broken down to other compounds in the environment. Therefore, they can remain in the environment for decades or longer.
- Asbestos fibers may build up in animals.

How might I be exposed to asbestos?

- Breathing low levels in air.
- Breathing higher levels in air while working in industries that make or use asbestos products or near a building that contains asbestos products and is being torn down or renovated.
- Breathing higher levels in air near an asbestos-related industry or near an asbestos-containing waste site.
- Drinking water containing asbestos from natural sources or from asbestos-containing cement pipes in drinking water distribution systems.

How can asbestos affect my health?

Asbestos mainly affects the lungs. Changes in the membrane surrounding the lung are quite common in workers exposed to asbestos. These are also sometimes found in people living in areas with high levels of asbestos in the air, but effects on breathing usually aren't serious.

ATSDR Internet home page via WWW is http://atsdr1.atsdr.cdc.gov:8080/atsdrhome.html

Breathing very high levels of asbestos may result in a slow buildup of scar-like tissue in the lungs and in the membrane that surrounds the lungs. This disease is called asbestosis, and is usually found in asbestos workers and not in the general public. People with asbestosis have shortness of breath, often along with a cough and sometimes heart enlargement. This is a serious disease and can eventually lead to disability or death.

How likely is asbestos to cause cancer?

The Department of Health and Human Services (DHHS) has determined that asbestos is a known carcinogen.

It is known that asbestos causes cancer in people. There are two types of cancer caused by exposure to high levels of asbestos: cancer of the lung tissue itself and mesothelioma, a cancer of the membrane that surrounds the lung and other internal organs. Both of these are usually fatal. These diseases don't develop immediately, but show up only after many years.

Interactions between cigarette smoke and asbestos increase your chances of getting lung cancer. Studies of workers suggest that breathing asbestos can increase the chances of getting cancer in other parts of the body (stomach, intestines. esophagus, pancreas, kidneys), but this is not certain.

People who are exposed to lower levels of asbestos may also have an increased risk of developing cancer, but the risks are usually small and are difficult to measure.

It is not known whether ingesting asbestos causes cancer. Some people who had been exposed to asbestos fibers in their drinking water had higher-than-average death rates from cancer of the esophagus, stomach, and intestines. However, it isn't known whether this was caused by asbestos or by something else.

Is there a medical test to show whether I've been exposed to asbestos?

Chest X-rays cannot detect asbestos fibers, but can detect early signs of lung disease caused by asbestos. Other tests (lung and CAT scans), are also useful in detecting changes in the lungs.

Tests exist to measure asbestos fibers in urine, feces, mucus, or material rinsed out of the lung. However, low levels of asbestos fibers are found in these body fluids in nearly all people, so higher-than-average levels can only show that you have been exposed to asbestos, not whether you will experience any health effects.

Has the federal government made recommendations to protect human health?

In 1989, the EPA banned all new uses of asbestos; uses established before this date are still allowed. The EPA has established regulations that require school systems to inspect for damaged asbestos and to eliminate or reduce the exposure by removing the asbestos or by covering it up. The EPA has set a limit of 7 million fibers per liter (MFL) as the concentration of long asbestos fibers that may be present in drinking water.

Glossary

Carcinogen: A substance that can cause cancer.

CAS: Chemical Abstract Service.

MFL: Million fibers per liter.

CAT scan: A medical test in which a computer makes a 3-dimensional image of a body organ.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological profile for asbestos. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns. For more information, contact: Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop E-29, Atlanta, GA 30333, Phone: 404-639-6000, FAX: 404-639-6315. ATSDR Internet home page via WWW is http://atsdr1.atsdr.cdc.gov:8080/atsdrhome.html

CANCER FACTS
National Cancer Institute • National Institutes of Health

Questions and Answers About Asbestos Exposure

1. What is asbestos?

"Asbestos" is the name given to a group of minerals that occur naturally as masses of strong, flexible fibers that can be separated into thin threads and woven. These fibers are not affected by heat or chemicals and do not conduct electricity. For these reasons, asbestos has been widely used in many industries. Four types of asbestos have been commonly used:

- Chrysotile, or white asbestos (curly, flexible white fibers), which accounts for about 90 percent of the asbestos currently used in industry;
- Amosite (straight, brittle fibers that are light gray to pale brown in color);
- Crocidolite, or blue asbestos (straight blue fibers); and
- Anthophyllite (brittle white fibers).

Chrysotile asbestos, with its curly fibers, is in the serpentine family of minerals. The other types of asbestos, which all have needle-like fibers, are known as amphiboles.

Asbestos fiber masses tend to break easily into a dust composed of tiny particles that can float in the air and stick to clothes. The fibers may be easily inhaled or swallowed and can cause serious health problems.

2. How is asbestos used?

Asbestos has been mined and used commercially in North America since the late 1800s, but its use increased greatly during World War II. Since then, it has been used in many industries. For example, the building and construction industry uses it for strengthening cement and plastics as well as for insulation, fireproofing, and sound absorption. The shipbuilding industry has used asbestos to insulate boilers, steampipes, hot water pipes, and nuclear reactors in ships. The automotive industry uses asbestos in vehicle brakeshoes and clutch pads. More than 5,000 products contain or have contained asbestos, some of which are listed below:

- Asbestos cement sheet and pipe products used for water supply and sewage piping, roofing and siding, casings for electrical wires, fire protection material, chemical tanks, electrical switchboards and components, and residential and industrial building materials;

- Friction products, such as clutch facings; brake linings for automobiles, railroad cars, and airplanes; and industrial friction materials;

- Products containing asbestos paper, such as table pads and heat-protective mats, heat and electrical wire insulation, industrial filters for beverages, small appliance components, and underlying material for sheet flooring;

- Asbestos textile products, such as packing components, roofing materials, heat- and fire-resistant clothing, and fireproof draperies; and

- Other products, including ceiling and floor tile; gaskets and packings; paints, coatings, and sealants; caulking and patching tape; and plastics.

In the late 1970s, the U.S. Consumer Product Safety Commission banned the use of asbestos in wallboard patching compounds and gas fireplaces because these products released excessive amounts of asbestos fibers into the environment. In addition, asbestos was voluntarily withdrawn by manufacturers of electric hair dryers. These and other regulatory actions, coupled with widespread public concern about the hazards of asbestos, have resulted in a significant annual decline in U.S. use of asbestos: Domestic use of asbestos amounted to about 560,000 metric tons in 1979, but it had dropped to about 55,000 metric tons by 1989.

3. What are the health hazards of exposure to asbestos?

Exposure to asbestos may increase the risk of several serious diseases:

- Asbestosis—a chronic lung ailment that can produce shortness of breath and permanent lung damage and increase the risk of dangerous lung infections;

- Lung cancer;

- Mesothelioma—a relatively rare cancer of the thin membranes that line the chest and abdomen; and

- Other cancers, such as those of the larynx and of the gastrointestinal tract.

4. Who is at risk?

Since the early 1940s, millions of American workers have been exposed to asbestos dust, including many of the 4.5 million men and women who worked in shipyards during the peak shipbuilding years of World War II. Health hazards from asbestos dust have been recognized in workers exposed in shipbuilding trades, asbestos mining and milling, manufacturing of asbestos textiles and other asbestos products, insulation work in the construction and building trades, brake repair, and a variety of other trades. Demolition workers, drywall removers, and firefighters also may be exposed to asbestos dust. As a result of Government regulations and improved work practices, today's workers (those without previous exposure) are likely to face smaller risks than did those exposed in the past.

Although it is known that the risk to workers increases with heavier exposure and longer exposure time, investigators have found asbestos-related diseases in some shipyard workers exposed to high levels of asbestos fibers for only brief periods (as little as 1 or 2 months). Even workers who may not have worked directly with asbestos but whose jobs were located near contaminated areas have developed asbestosis, mesothelioma, and other cancers associated with asbestos exposure.

Generally, workers who develop asbestos-related diseases show no signs of illness until many years after first exposure. For example, the time between first exposure to asbestos and the appearance of lung cancer is generally 15 years or more; a lag of 30 to 35 years is not unusual. The lag period for development of mesothelioma and asbestosis is even greater, often as long as 40 to 45 years.

There is also some evidence that family members of workers heavily exposed to asbestos face an increased risk of developing mesothelioma and perhaps other asbestos-related diseases. This risk is thought to result from exposure to asbestos dust brought into the home on the shoes, clothing, skin, and hair of workers.

5. How great is the risk?

Not all workers exposed to asbestos will develop diseases related to their exposure. In fact, many will experience no ill effects.

Asbestos that is bonded into finished products such as walls, tiles, and pipes poses no risk to health as long as it is not damaged or disturbed (for example, by sawing or drilling) in such a way as to release fibers into the air. When asbestos particles are set free and inhaled, however, exposed individuals are at risk of developing an asbestos-related disease. Once these nearly indestructible fibers work their way into body tissues, they tend to stay there indefinitely.

The risk of developing asbestos-related diseases varies with the type of industry in which the exposure occurred and with the extent of the exposure. In addition, different

types of asbestos fibers may be associated with different health risks. For example, results of several studies suggest that crocidolite and amosite are more likely than chrysotile to cause lung cancer, asbestosis, and, in particular, mesothelioma. Even so, no fiber type can be considered harmless, and proper safety precautions should always be taken by people working with asbestos.

6. How does smoking affect risk?

Many studies have shown that the combination of smoking and asbestos exposure is particularly hazardous. Cigarette smokers, on the average, are 10 times as likely to develop lung cancer as are nonsmokers. For nonsmokers who work with asbestos, the risk is about five times greater than for those in the general population. By contrast, smokers who also are heavily exposed to asbestos are as much as 90 times more likely to develop lung cancer than are nonexposed individuals who do not smoke. Smoking does not appear to increase the risk of mesothelioma, however.

There is evidence that quitting smoking will reduce the risk of lung cancer among asbestos-exposed workers, perhaps by as much as half or more after at least 5 years without smoking. People who were exposed to asbestos on the job at any time during their life or who suspect they may have been exposed ***should not smoke***. If they smoke, they should stop.

7. Who needs to be examined?

Individuals who have been exposed (or suspect they have been exposed) to asbestos dust on the job or at home via a family contact should inform their physician of their exposure history and any symptoms. A thorough physical examination, including a chest x-ray and lung function tests, may be recommended. Interpretation of the chest x-ray may require the help of a specialist who is experienced in reading x-rays for asbestos-related diseases. Other tests also may be necessary.

As noted earlier, the symptoms of asbestos-related diseases may not become apparent for many decades after exposure. If any of the following symptoms develop, a physical examination should be scheduled without delay:

- Shortness of breath;
- A cough or a change in cough pattern;
- Blood in the sputum (fluid) coughed up from the lungs;
- Pain in the chest or abdomen;

- Difficulty in swallowing or prolonged hoarseness; and/or
- Significant weight loss.

8. What are the treatments for asbestos-related diseases?

The key to successful treatment of asbestos-related diseases lies in early detection. The health problems caused by asbestosis are due mainly to lung infections, like pneumonia, that attack weakened lungs. Early medical attention and prompt, aggressive treatment offer the best chance of success in controlling such infections. Depending on the situation, doctors may give a vaccine against influenza or pneumococcal pneumonia as a protective measure.

Treatment of cancer is tailored to the individual patient and may include surgery, anticancer drugs, radiation, or combinations of these therapies. Information about cancer treatment is available from the National Cancer Institute-supported Cancer Information Service, whose toll-free telephone number is 1-800-4-CANCER.

9. How can workers protect themselves?

Employers are required to follow regulations dealing with asbestos exposure on the job that have been issued by the Occupational Safety and Health Administration (OSHA), the Federal agency responsible for health and safety regulations in the workplace. Regulations related to mine safety are enforced by the Mine Safety and Health Administration (MSHA). Workers should use all protective equipment provided by their employers and follow recommended work practices and safety procedures. Workers who are or who have been exposed to asbestos should not smoke cigarettes.

Workers who are concerned about asbestos exposure in the workplace should discuss the situation with other employees, their union, and their employers. If necessary, OSHA can provide more information or make an inspection. Area offices of OSHA are listed in the "United States Government" section of telephone directories' blue pages (under "Department of Labor"). If no listing is found, workers may call or write to one of the OSHA regional offices listed on page 9. Mine workers may contact MSHA's Office of Standards, Variances, and Regulation at Room 627, 4015 Wilson Boulevard, Arlington, VA 22203; the telephone number is 703-235-1910.

The National Institute for Occupational Safety and Health (NIOSH) is another Federal agency that is concerned with asbestos exposure in the workplace. The Institute conducts asbestos-related research, evaluates work sites for possible health hazards, and makes safety recommendations. In addition, NIOSH distributes publications on the health effects of asbestos exposure and can suggest additional sources of information. The address is Office of Information, National Institute of Occupational Safety and

Health, 4676 Columbia Parkway/Mailstop C-19, Cincinnati, OH 45226. The toll-free telephone number is 1-800-35-NIOSH (1-800-356-4674).

10. What should people who have been exposed to asbestos do?

It is important for exposed individuals to:

- Stop smoking;
- Get regular health checkups;
- Get prompt medical attention for any respiratory illness; and
- Use all protective equipment, work practices, and safety procedures designed for working around asbestos.

11. Will the Government provide examinations and treatment or pay for such services? What about insurance coverage?

Medical services related to asbestos exposure are available through the Government only for certain groups of eligible individuals. In general, exposed individuals must pay for their own medical services unless they are covered by private or Government health insurance. Medicare may reimburse people with symptoms of asbestos-related diseases for the costs of diagnosis and treatment (following review of medical procedures for appropriateness). General and specific information about benefits is available from the Medicare office serving each state; for the telephone number of the nearest office, call 1-800-772-1213.

People with asbestos-related diseases also may qualify for financial help, including medical payments, under state workers' compensation laws. Because eligibility requirements vary from state to state, workers should contact the workers' compensation program in the state where the last exposure occurred. (The telephone number may be found in the blue pages of a local telephone directory.)

If exposure occurred during employment with a Federal agency (military or civilian), medical expenses and other compensation may be covered by the Federal Employees' Compensation Act. Workers who are or were employed in a shipyard by a private employer may be covered under the Longshoremen and Harbor Workers' Compensation Act. Information about eligibility or how to file a claim is available from the U.S. Department of Labor, Office of Workers' Compensation Programs, Room S-3229, 200 Constitution Avenue NW, Washington, DC 20210; the telephone number is 202-219-7552.

Retired military personnel and their eligible dependents may receive health care at any Department of Defense medical facility, Department of Veterans Affairs (VA) hospital, or Public Health Service hospital. Where no Federal facility is available, civilian facilities may be used under the Civilian Health and Medical Program for the Uniformed Services. Those over age 65 may be covered by Medicare. Former members of the military who believe they may have a service-related medical problem may inquire about care at a VA facility or telephone the local VA office.

Workers also may wish to contact their international union for information on other sources of medical help and insurance matters. One organization, the Asbestos Victims Special Fund Trust, provides financial assistance to asbestos victims who have not received workers' compensation or compensation through legal avenues. Information is available from the Trust at Suite M-11, 1500 Walnut Street, Philadelphia, PA 19102; the telephone number is 1-800-447-7590.

12. Is there a danger of nonoccupational exposure from products contaminated with asbestos particles?

Asbestos is so widely used that the entire population has been exposed to some degree. Air, beverages, drinking water, food, drug and dental preparations, and a variety of consumer products all may contain small amounts of asbestos. In addition, asbestos fibers are released into the environment from natural deposits in the earth and as a result of wear and deterioration of asbestos products.

The U.S. Environmental Protection Agency (EPA) regulates the general public's exposure to asbestos in buildings, drinking water, and the environment. The EPA's Toxic Substances Control Act (TSCA) Assistance Office can answer questions about toxic substances, including asbestos. Printed material is available on a number of topics, particularly on controlling asbestos exposure in schools and other buildings. The TSCA office can provide information about accredited laboratories for asbestos testing and can refer inquirers to other resources on asbestos. Questions may be directed to the TSCA Assistance Office, U.S. Environmental Protection Agency, 7408 M Street SW, Washington, DC 20024; the telephone number is 202-554-1404.

The Consumer Product Safety Commission (CPSC) is responsible for the regulation of asbestos in consumer products. The CPSC maintains a toll-free information line on the potential hazards of commercial products; the telephone number is 1-800-638-2772. In addition, CPSC provides information about laboratories for asbestos testing, guidelines for repairing and removing asbestos, and general information about asbestos in the home. Publications are available from the Office of Public Affairs, Consumer Product Safety Commission, 4330 East-West Highway, Bethesda, MD 20816; the telephone number is 301-504-0580.

The U.S. Food and Drug Administration is concerned with asbestos contamination of foods, drugs, and cosmetics and will answer questions on these topics. The address is Office of Consumer Affairs, Food and Drug Administration, HFE-88, 5600 Fishers Lane, Rockville, MD 20857; the telephone number is 301-443-3170.

13. What other organizations offer information related to asbestos exposure?

The American Lung Association and the American Cancer Society can provide information about lung disease, cancer, and smoking. Local chapters of these organizations are listed in telephone directories. Material about cancer and how to quit smoking is available by calling the National Cancer Institute-supported Cancer Information Service (CIS). The CIS, a program of the National Cancer Institute, provides a nationwide telephone service for cancer patients and their families, the public, and health care professionals. CIS information specialists have extensive training in providing up-to-date and understandable information about cancer and cancer research. They can answer questions in English and Spanish and can send free printed material. In addition, CIS offices serve specific geographic areas and have information about cancer-related services and resources in their region. The toll-free number of the CIS is 1-800-4-CANCER (1-800-422-6237).

Occupational Safety and Health Administration (OSHA)
Regional Offices

Region I (serves Connecticut, Massachusetts, Maine, New Hampshire, Rhode Island, and Vermont)	First Floor 133 Portland Street Boston, MA 02114 617-565-7164
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U.S. Department of Labor
Program Highlights

Fact Sheet No. OSHA 93-06

BETTER PROTECTION AGAINST ASBESTOS IN THE WORKPLACE

What Is Asbestos?

Asbestos is a widely used, mineral-based material that is resistant to heat and corrosive chemicals. Typically, asbestos appears as a whitish, fibrous material which may release fibers that range in texture from coarse to silky; however, airborne fibers that can cause health damage may be too small to see with the naked eye.

Who Is Exposed?

An estimated 1.3 million employees in construction and general industry face significant asbestos exposure on the job. Heaviest exposures occur in the construction industry, particularly during the removal of asbestos during renovation or demolition. Employees are also likely to be exposed during the manufacture of asbestos products (such as textiles, friction products, insulation, and other building materials) and during automotive brake and clutch repair work.

What Are the Dangers of Asbestos Exposure?

Exposure to asbestos can cause asbestosis (scarring of the lungs resulting in loss of lung function that often progresses to disability and to death); mesothelioma (cancer affecting the membranes lining the lungs and abdomen); lung cancer; and cancers of the esophagus, stomach, colon, and rectum.

What Protections Are Mandatory?

The U.S. Occupational Safety and Health Administration (OSHA) has issued revised regulations covering asbestos exposure in general industry and construction. Both standards set a maximum exposure limit and include provisions for engineering controls and respirators, protective clothing, exposure monitoring, hygiene facilities and practices, warning signs, labeling, recordkeeping, and medical exams.

Nonasbestiform tremolite, anthophyllite, and actinolite were excluded from coverage under the asbestos standard in May 1992.

Here are some of the highlights of the revised rules, published in the Federal Register June 20, 1986; and on Sept. 14, 1988:

— Permissible Exposure Limit: In both general industry and construction, workplace exposure must be limited to 0.2 fibers per cubic centimeter of air (0.2 f/cc), averaged over an eight-hour work shift. The excursion or short-term limit is one fiber per cubic centimeter of air (1 f/cc) averaged over a sampling period of 30 minutes.

— Exposure Monitoring: In general industry, employers must do initial monitoring for workers who may be exposed above the "action level" of 0.1 f/cc. Subsequent monitoring must be conducted at reasonable intervals, in no case longer than six months for employees exposed above the action level.

In construction, daily monitoring must be continued until exposure drops below the action level (0.1 f/cc). Daily monitoring is not required where employees are using supplied-air respirators operated in the positive pressure mode.

— Methods of Compliance: In both general industry and construction, employers must control exposures using engineering controls, to the extent feasible. Where engineering controls are not feasible to meet the exposure limit, they must be used to reduce employee exposures to the lowest levels attainable and must be supplemented by the use of respiratory protection.

— Respirators: In general industry and construction, the level of exposure determines what type of respirator is required; the standards specify the respirator to be used.

— Regulated Areas: In general industry and construction, regulated areas must be established where the 8-hour TWA or 30-minute excursion values for airborne asbestos exceed the prescribed permissible exposure limits. Only authorized persons wearing appropriate respirators can enter a regulated area. In regulated areas, eating, smoking, drinking, chewing tobacco or gum, and applying cosmetics are prohibited.

Warning signs must be displayed at each regulated area and must be posted at all approaches to regulated areas.

— Labels: Caution labels must be placed on all raw materials, mixtures, scrap, waste, debris, and other products containing asbestos fibers.

— Recordkeeping: The employer must keep an accurate record of all measurements taken to monitor employee exposure to asbestos. This record is to include: the date of measurement, operation involving exposure, sampling and analytical methods used, and evidence of their accuracy; number, duration, and results of samples taken; type of respiratory protective devices worn; name, social security number, and the results of all employee exposure measurements. This record must be kept for 30 years.

— Protective Clothing: For any employee exposed to airborne concentrations of asbestos that exceed the PEL, the employer must provide and require the use of protective clothing such as coveralls or similar full-body clothing, head coverings, gloves, and foot covering. Wherever the possibility of eye irritation exists, face shields, vented goggles, or other appropriate protective equipment must be provided and worn.

In construction, there are special regulated-area requirements for asbestos removal, renovation, and demolition operations. These provisions include a negative pressure area, decontamination procedures for workers, and a "competent person" with the authority to identify and control asbestos hazards. The standard includes an exemption from the negative pressure enclosure requirements for certain small scale, short duration operations provided special work practices prescribed in an appendix to the standard are followed.

— Hygiene Facilities and Practices: Clean change rooms must be furnished by employers for employees who work in areas where exposure is above the TWA and/or excursion limit. Two lockers or storage facilities must be furnished and separated to prevent contamination of the employee's street clothes from protective work clothing and equipment. Showers must be furnished so that employees may shower at the end of the work shift. Employees must enter and exit the regulated area through the decontamination area.

The equipment room must be supplied with impermeable, labeled bags and containers for the containment and disposal of contaminated protective clothing and equipment.

Lunchroom facilities for those employees must have a positive pressure, filtered air supply and be readily accessible to employees. Employees must wash their hands and face prior to eating, drinking or smoking. The employer must ensure that employees do not enter lunchroom facilities with protective work clothing or equipment unless surface fibers have been removed from the clothing or equipment.

Employees may not smoke in work areas where they are occupationally exposed to asbestos.

— Medical Exams: In general industry, exposed employees must have a preplacement physical examination before being assigned to an occupation exposed to airborne concentrations of asbestos at or above the action level or the excursion level. The physical examination must include chest X-ray, medical and work history, and pulmonary function tests. Subsequent exams must be given annually and upon termination of employment, though chest X-rays are required annually only for 45 and over workers whose first asbestos exposure occurred more than 10 years ago.

In construction, examinations must be made available annually for workers exposed above the action level or excursion limit for 30 or more days per year or who are required to wear negative pressure respirators; chest X-rays are at the discretion of the physician.

Where Can I Get More Information?

Copies of the general industry asbestos standard Part II, Health Standards, Stock Number 869-017-00110-4, $16.00) and the construction industry standard (Stock Number 869-017-00112-1, $14.00) are available from the Superintendent of Documents, Government Printing Office, Washington, DC 20402-9325, or telephone 202-783-3238. These standards are also available on CD-ROM (Stock Number 729-013-00000-5, $88.00) by subscription for four updates per year or a single disk for $28.00.

Two pamphlets summarizing the rule are also available: (in single copies) "Asbestos Standard for General Industry" and "Asbestos Standard for Construction Industry," and can be obtained by sending a self-addressed mailing label to the OSHA Publications Office, Room N-3101, Washington, D.C. 20210, telephone 202-219-4667 or from any local OSHA office.

Questions about the standards can be answered by any local OSHA office or by OSHA regional offices located in Boston, New York, Philadelphia, Atlanta, Chicago, Dallas, Kansas City, Denver, San Francisco, and Seattle.

All local OSHA offices have available for loan slide programs on the general industry and construction asbestos standards. Training on asbestos and other safety and health hazards is conducted at the OSHA Training Institute, 1555 Times Dr., Des Plaines, IL 60018, telephone 708-297-4810; tuition is charged.

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