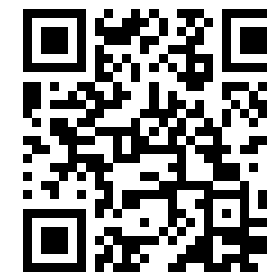
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Prevention Branch

Division of Cancer Control and Rehabilitation

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# FOREWORD

There is increasing realization that asbestos may be present across the entire spectrum of human exposure--in our air, water, food, drugs, cosmetics; in our homes and, most importantly, in our workplaces. There is also developing awareness that several types of cancer may be the consequence of exposure to this material, especially for certain segments of the population.

Recognizing our responsibility to transfer information to the scientific community and the public, the Division of Cancer Control and Rehabilitation, National Cancer Institute, is making available this document on asbestos and cancer. Several of the more important objectives of the document are to: (1) present both current and historical evidence of the carcinogenic potential of asbestos; (2) examine potential exposure of the public; (3) describe current intervention and control technology; and (4) discuss the possible prevention roles of various individuals and groups in the community. Dissemination of information is a fundamental prerequisite to the formulation and implementation of action programs for effective cancer control and prevention at the federal, state, and local levels.

This document represents work of individuals in many different research fields, and the contributions of all these individuals are acknowledged.

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# Chapter I
# INTRODUCTION

Because of their unique combination of resistance to heat and chemical attack, high tensile strength, and flexibility, fibrous asbestos minerals have long been used by man. The early Greek geographer, Pausanias, speaks of golden lamps made about 430 B.C. with incombustible wicks of "Carpathian flax." The Romans used asbestos as cremation clothes to conserve the ashes of deceased persons of rank. The French emperor, Charlemagne, had a tablecloth of asbestos and is reputed to have impressed his enemies by passing it through fire to clean it.

Today, asbestos is found in thousands of commercial products including heat-resistant textiles, reinforced cement, special filters for industrial chemicals, thermal insulation, floor tiles, gaskets, and brake linings. More than 725,000 metric tons (800,000 short tons) of asbestos have been consumed in the United States alone in producing these products in each of several recent years. As a consequence of man's utilization of asbestos, coupled with the natural occurrence of the mineral, asbestos fibers are found in the air we breathe, the food we eat, and the water we drink.

## A Hazard to Human Health

The fact that asbestos is a hazard to man's health was recognized quite early. In the first century, both Pliny the Elder, the Roman naturalist, and Strabo, the Greek geographer, wrote of a sickness of the lungs in slaves whose occupation was the weaving of asbestos into cloth. However, the association of asbestos with chronic respiratory disease had to be rediscovered in the modern era. A series of case reports was followed by an epidemiologic study published in London in 1930,[1] 52 years after large-scale mining of asbestos had begun with the opening of a mine at Thetford, Quebec, Canada. The cancer-producing potential of asbestos was not established until 1949, when a report was published describing an excess of cancer of the lung and pleura among individuals dying from asbestosis.[2]

It is now clear that among asbestos workers, there is, in addition to the risk of asbestosis, a greatly increased risk of death from lung cancer and from pleural and peritoneal mesothelioma, malignancies that are seldom found in the general population.[3,4] Moreover, asbestos has been linked with gastro-intestinal, oropharyngeal, and laryngeal cancer.

According to the U.S. Public Health Service, one million persons now living in the United States either work or have worked in the



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asbestos product manufacturing industry.[5] This figure does not include those employed in mining and milling asbestos; those whose work may involve installation, modification, or repair of asbestos products; persons exposed indirectly to asbestos in the course of their work such as at shipyards or in the construction industry; persons living in the neighborhood of an asbestos product factory; or consumer users of asbestos materials.

## What is "Asbestos?"

To the mineralogist, asbestos is the generic name for a group of naturally occurring hydrated mineral silicates of the amphibole or serpentine series that are characterized by fibers or bundles of fine single crystal fibrils. Naturally occurring asbestos fibers typically have length-to-width ratios of the order of 100 and higher.

Included in this definition are the following minerals:

- Chrysotile
- Crocidolite
- Amosite, and
- The fibrous varieties of anthophyllite, tremolite, and actinolite.*

All of these minerals may occur in a nonfibrous form, in which case they are not classified as asbestos. Commercially, chrysotile is the form of asbestos used most. Crocidolite, amosite, and anthophyllite also have some commercial significance.

It is important to note that identification of asbestos fibers is relatively simple with macroscopic samples that clearly show the fibrous nature and other unique characteristics of these minerals. Positive identification is based on morphology, crystallographic structure, color, hardness, optical properties, and appearance. However, in the case of microscopic samples, positive identification becomes increasingly difficult, even when special microanalytical techniques are used.

The identification of asbestos is complex because many of the minerals that are chemically almost identical to different varieties of asbestos (e.g., grunerite to amosite, serpentine to chrysotile) exhibit

---

*Crocidolite, amosite, anthophyllite, tremolite, and actinolite are derived from the amphibole series and may be referred to as "amphiboles."



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perfect prismatic cleavage (the ability to break along well-defined crystallographic planes), so that physical degradation often leads to the formation of minute cleavage fragments that are chemically as well as physically indistinguishable from asbestos fibers. Recent comprehensive studies at the U.S. Bureau of Mines have concluded that there is currently no absolute way to distinguish between finely divided asbestos and certain other minerals of similar composition.[6] Some minerals other than asbestos that may exhibit fibrous structure are listed in Appendix A.

On the other hand, recent biological studies suggest that, in terms of carcinogenic activity, mineral shape and size may be more important than chemical nature.[7] The question of asbestos carcinogenicity can, therefore, be viewed as part of a broader issue--i.e., tissue modification caused by mineral fibers.

## Occurrence of Asbestos

All forms of asbestos develop through several stages of geologic processes (paragenesis) from parent rocks that are transformed into asbestos. Parent rocks of asbestos minerals include basic constituents normally found in ultramafic, dolomitic, or limestone rocks. Transformation may occur under localized conditions of temperature and pressure which lead to recrystallization of other in-situ minerals (metamorphism). It may occur as a result of the action of hot mineral solutions that can dissolve or otherwise alter some minerals to form others (hydrothermal processes).

In all of the commercial asbestos deposits, the geologic conditions have been favorable to the development of fibers of sufficient quality and concentration to warrant their extraction. (The same conditions may operate in other mineral deposits but yield asbestos fibers that are too disseminated or scarce to be of commercial interest.)

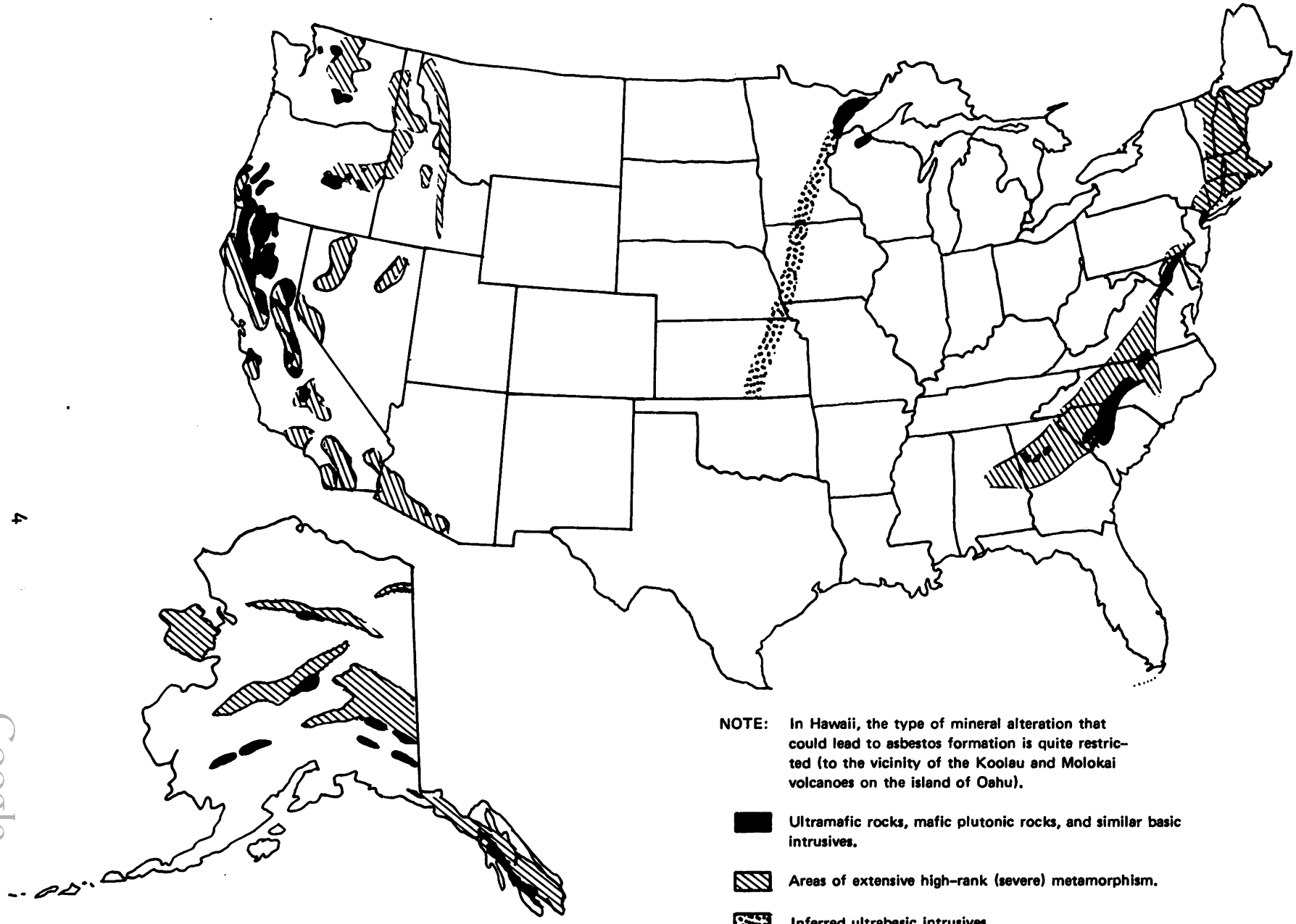
Minerals and rocks that can contain asbestos are listed in Appendix A. A favorable mineral association is not a sufficient condition for the formation of asbestos--it will form only if special structural and other geologic conditions are met. If asbestos presence is, in fact, established, the minerals and rocks listed in Appendix A would be significant sources of contamination for humans, since they include important raw materials for industry and are mined in commercial quantities, worldwide.

The areas of the United Stated having basic geologic constituents associated with the formation of asbestos are shown in Figure 1. Again, this graphic does not necessarily depict actual asbestos occurrence but,



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SOURCES: "Tectonic Map of North America," U.S. Geological Survey; SRI International

FIGURE 1 DISTRIBUTION OF ULTRABASIC AND METAMORPHIC ROCK FORMATIONS IN THE UNITED STATES



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rather, is an overview of the areas that are more likely than others to provide the geologic setting associated with formation of asbestos.

Although deposits of asbestos may be found throughout the United States, asbestos is commercially mined and milled at only five locations, in the states of California, Arizona, and Vermont. (Most of the asbestos consumed in the United States is imported, nearly all of it from Canada.) In addition to commercial mining and milling of asbestos, human-created occurrences of asbestos fiber may result from the mining and milling of mineral ores associated with asbestos; from the inadvertent disturbance of asbestos deposits by activities such as farming and road building; from the transportation of asbestos ore, milled fiber, products, and wastes; from the manufacture, use, repair, and demolition of asbestos-containing products; and from the disposal of asbestos wastes. All are discussed in succeeding chapters of this monograph.

## Regulation of Asbestos-Fiber Emissions

A recommendation for limiting exposure to asbestos in U.S. industry was made in 1938 by the U.S. Public Health Service.[8] The recommended limit, an airborne concentration of less than 5 million particles per cubic foot, was formally recognized in 1964--as a guideline issued by the Bureau of Labor Standards. No legal regulations were established until passage of the Occupational Safety and Health Act of 1970 and establishment of the Occupational Safety and Health Administration (OSHA).

### Occupational Exposure

OSHA regulations apply directly to all private employers, including federal government contractors, but not to federal, state, or local government agencies. Federal agencies are required to establish their own occupational safety and health programs consistent with the standards of the Act and subsequent OSHA regulations. States are encouraged by the Act to develop programs and regulations, for private employers, that are at least as effective as OSHA regulations and can, under these conditions, assume responsibility for enforcing standards--at the time of this writing, 24 states have programs of their own.[9] The current OSHA limit on occupational exposure to asbestos fibers is an 8-hour time-weighted average of 2 fibers per milliliter, no longer than 5 micrometers, with a length-to-width ratio of at least 3:1, detected by a method using phase-contrast (optical) microscopy.

The permissible levels of occupational exposure to asbestos contained in all federal regulations (and one proposal) are summarized in tabular form in Appendix B. Also, the provisions of these regulations for method of compliance, monitoring, medical surveillance, education, and the keeping of records are summarized there.



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## Emissions to Air and Water, Disposal of Solid Waste, Transportation

A national air emission standard for asbestos, first published in 1973,[10] requires either the institution of specified air-cleaning methods or else no visible emissions (except water) to be released to the outside air. The standard applies to the milling of asbestos (but not to adjacent storage depots); manufacturing or processing of specified products; renovating or demolishing certain buildings (but not ships) containing more than a specified amount of friable asbestos insulation; and to wastes containing commercial asbestos or products of asbestos mining and milling. Friable or spray-on insulating materials, except when applied to equipment or machinery, must contain no commercial asbestos; however, spray-on paints, decorative materials, and weatherproofing are not regulated.

Under the terms of guidelines and standards promulgated in 1974 and 1975, certain asbestos-manufacturing operations were to achieve, by July 1, 1977, wastewater effluent limitations requiring application of the best practicable control technology currently available; and by July 1, 1983, except for operations involving solvent recovery, there is to be no discharge of wastewater pollutants to navigable waters.[11]

Federal agencies are directed by two Executive Orders to monitor, evaluate, and control their activities so as to protect and enhance the quality of the environment and to conform to air and water quality standards of the Clean Air Act and the Federal Water Pollution Control Act.

## Food, Drugs, Consumer Products

The Food and Drug Administration (FDA) has reviewed several commercial practices that may result in asbestos contamination of food and drugs and, in January of 1976, revoked approval for use in foods of sodium chloride (salt) produced by the electrolytic diaphragm process.[12] In the absence of more-reliable data on background concentrations of asbestos in water and the contribution of asbestos filters to levels of asbestos found in ingestible products, the FDA has not regulated the use of asbestos filters for filtering edible foods, beverages, and nonparenteral drugs.[13] The agency has, however, enacted regulations to limit asbestos and other fibrous materials in parenteral drugs.[14] The FDA has approved (1) the use of asbestos as a component of various types of food-contact articles in which contamination of food is not



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likely to occur, and (2) the use of talc, in which asbestos is a possible impurity, in cosmetics.*

The Consumer Product Safety Commission has banned general-use garments containing asbestos. The use of asbestos in special garments such as fire-fighting suits is permitted, but only if they are constructed so that asbestos fibers will not become airborne under normal conditions of use.[16]†

## Constraints on Monitoring and Measuring Asbestos Levels

As will be brought out later in this monograph, it is simply not known what attributes of asbestos it is that constitute a health hazard--size, shape, mass, type etc.--nor is it known what amount is hazardous or over what period of time. The U.S. federal occupational standards are, perforce, an attempt to optimize what is known about the health hazard with the technical and economic practicalities of measuring the substance. For example, by using phase-contrast optical microscopy, rather than the higher resolution electron microscopy, it is not possible to count all the fibers and not always possible to even distinguish between asbestos and other fibers (as noted previously); electron microscopy, on the other hand, is time-consuming and expensive. The difficulties of measuring and identifying are of such proportions that there are extreme variations in the measurement of a sample, both within a laboratory and among laboratories.

As a consequence, the reader should always bear in mind the possible grossness of measurements referred to throughout this document, even when they are expressed in terms that bespeak of precision--e.g., the decimal point. At the same time, the fact remains that asbestos is a human carcinogen and that, as such, is a suitable subject for treatment in this document, the prime purpose of which is helping man control cancer. The constraint on monitoring and measuring asbestos is such, nevertheless, that it is the subject of more extensive treatment in Appendix C of this document.

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*Talc is listed as a "generally recognized as safe" (GRAS) substance for use in paper, paperboard, and cotton food packaging materials; as an anticaking agent for forms used in molding various food shapes and in chewing gum base; and to coat polished nonenriched rice, as a free-flow agent, and as a vehicle for enrichment formulas.[15] An FDA ruling on talc as a direct food or drug additive has been deferred until an acceptable analytic method can be developed.[14]

†Full attainment of the desired result of regulation inevitably requires adequate means of enforcement. Legal powers of enforcement as well as de facto enforcement practices differ widely among the various government agencies having statutory authority. See Reference 17 for an example of divergence of enforcement practice in just one regulatory agency from what is required by law.



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Summarized in the next four chapters are: production and uses of asbestos fiber and fiber-containing products; the biological effects, carcinogenic and noncarcinogenic, of exposure to asbestos; occupational and nonoccupational exposures and exposure levels. In the final three chapters, strategies and programs for the control of the health hazard represented by human exposure to asbestos are set forth.



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## Chapter II
## PRODUCTION AND USE OF ASBESTOS FIBERS AND PRODUCTS

Although some exposure of humans to asbestos fibers in air and water is always possible as a result of the weathering of asbestos-containing rock, it is man's large-scale commercialization of the mineral that has engendered greater health risk. In this chapter, the major facets of such commercialization in the United States are reviewed--consumption and production volumes; categories of manufactured goods; and processes for mining, milling, transporting, and manufacturing asbestos and asbestos-containing products.*

### U.S. Consumption of Asbestos Fibers

During the five years ending in 1975 the amount of asbestos fiber apparently consumed† in the United States averaged some 800,000 tons annually, although between 1974 and 1975 apparent consumption declined 27% from 856,000 to 629,000 tons.‡ It is expected that consumption will have recovered to about 820,000 tons in 1976. The sharp decline between 1974 and 1975 reflects not only a market affected by a sharp recession, but also a substantial interruption of supply associated with work stoppages, a landslide at a major mine, and a serious fire at an asbestos mill in Quebec, Canada. The estimated U.S. apparent consumption of asbestos fiber for the 1971-75 period is shown in Table 1.

It can be seen in Table 1 that most of the asbestos fiber used in the United States is imported (about 90%). Nearly all of this imported asbestos is chrysotile fiber that comes from Canada (96.5% in 1974). The

---

*This chapter includes information obtained through a special survey of industry carried out in the Fall of 1976 by the Mineral and Metals Center, SRI International.

†"Apparent" consumption--i.e., production plus imports, plus net shipments from government stockpiles if any, less exports. Apparent consumption figures do not take into account any changes in inventory levels of manufacturers and therefore differ from "actual" consumption figures (an example of which is found in Table 4 in this chapter).

‡Volumes in this chapter are expressed in short tons (2,000 pounds) in accordance with industry practice.



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Table 1

APPARENT U.S. CONSUMPTION OF ASBESTOS FIBER
(Thousands of Short Tons)

| | 1971 | 1972 | 1973 | 1974 | 1975 |
|---|---|---|---|---|---|
| Production | 131 | 132 | 150 | 113 | 100 |
| Imports | 681 | 736 | 792 | 776 | 575 |
| Stockpile releases | 10 | 13 | 7 | 29 | 4 |
| Exports | 54 | 59 | 66 | 62 | 50 |
| Apparent consumption | 768 | 822 | 883 | 856 | 629 |

Source: U.S. Bureau of Mines, Commodity Data Summaries, 1976.

Republic of South Africa accounted for some 3% of the U.S. imports in 1974 (crocidolite and amosite fibers), and a number of countries supplied the remainder. A substantial portion of Canadian output is produced there by U.S. companies that manufacture asbestos products.

It should be noted that in addition to importing both crude and milled fibers, the United States imports products manufactured from asbestos. In 1974, for example, the value of products exported from Canada to the United States was as shown below:[1]

| | Canadian Dollars (millions) |
|---|---|
| Brake linings and clutch facings | $0.9 |
| Building materials | 3.7 |
| Other products | 3.2 |
| Total | $7.8 |

However, the value of imported products is not large compared with U.S. domestic shipments, which, for example, totalled $742.6 million in 1972.[2]

The U.S. Bureau of Mines has estimated that U.S. demand for asbestos in the year 2000 will range between 1.0 million and 1.8 million tons.[3] It is also possible, however, that substitution of other



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materials for asbestos could result in a lower level of demand. This substitution could occur for a number of reasons such as:

- More rapid price increases for asbestos than for its competitive products.
- Health and safety considerations.
- Lack of availability (an industry expert has estimated that by 1980 supply could fall short of potential world demand by about 500,000 tons, or about 10% of world demand).

## U.S. Production and Producers of Asbestos Fibers

The volume and value at the mines of asbestos fiber production in the United States during recent years is shown in the tabulation that follows:

| | Production (Thousands of Short Tons) | Value Per Ton | Total Value (Millions of Dollars) |
|---|---|---|---|
| 1971 | 131 | $ 93 | $12.2 |
| 1972 | 132 | 102 | 13.5 |
| 1973 | 150 | 121 | 18.2 |
| 1974 | 113 | 158 | 17.9 |
| 1975 | 100 | 182 | 18.2 |

The decline in production volume from the all-time peak of 150,000 tons in 1973 resulted primarily from the closing or reduction in output of several mines such as GAF Corporation's Lowell Vermont mine (acquired by the Vermont Asbestos Group), Pacific Asbestos Corporation's mine near Copperopolis, California (later reopened by Calaveras Asbestos Limited), and the Christie mine of Coalinga Asbestos Company, Inc., at Coalinga, California (a 15,000-ton-per-year mine that has remained closed). The average domestic production of only 125,000 tons annually during the 1971-75 period is a relatively small fraction of the nearly 800,000 tons consumed annually in the United States during those years. As a result of an increase in the production volume and average value per ton in 1976, the value of U.S. asbestos fiber production could be in the range of $25-30 million in 1976 (11-13% of the mine/mill value of all asbestos likely to be consumed during the year).

U.S. asbestos mines and mills, their locations, output, and employment are shown in Table 2. The current total mill capacity is on the



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Table 2

OPERATING AND NONOPERATING ASBESTOS MINES AND MILLS IN THE UNITED STATES

| In Operation as of Late 1976 | Type of Mining | Mine Name | Mine Location | Mill Location | Fiber Type | Annual Production | Employment |
|---|---|---|---|---|---|---|---|
| Atlas Asbestos Corp. (California) | Open pit | Santa Cruz | 30 mi. NW of Coalinga | At mine | Short | 30,000 tons | 30 |
| Jaquays Mining Co. (Arizona) | Under-ground | El Dorado | 32 mi. N of Globe | Globe | Short and spinning | 3,000 tons | 35 |
| Union Carbide Corp. (California) | Open pit | Santa Rita | Eastern San Benito County | King City (55 mi. W of mine) | Short | 35,000 tons | 68[a] |
| Vermont Asbestos Group (Vermont) | Open pit | Lowell | Belvidere Mountain | At mine (15 mi. from Hyde Park) | Short to medium and spinning | 40,000 tons | 208 |
| Calaveras Asbestos Ltd. (California) | Open pit | Pacific Asbestos | Copperopolis | At mine | Short to medium | 35,000 tons | 175 |
| Suspended Operations | | | | | | | |
| Coalinga Asbestos Co. (Division of Johns-Manville Corp.) (California) | Open pit | | 30 mi. NW of Coalinga | At mine | Short | | |
| Asbestos Mfg. Co. (Arizona) | Under-ground | | Near Globe | Globe | Short and spinning | | |
| Metate Asbestos Co. (Arizona) | Under-ground | | Near Globe | Globe | Short and spinning | | |
| Powhatan Mining Co. (North Carolina) | Open pit | | Burnsville, N.C. | Baltimore, Md. | (Anthophyllite) | | |

[a]Mill employment only--deposit is mined by independent contractor on an intermittent basis.

Source: SRI--industry contacts and trade journals.



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order of 143,000 tons per year (all in the form of chrysotile fibers). California mines and mills account for about 70% of total U.S. output.

It is difficult to predict changes in U.S. asbestos output because such changes will be a function of costs, including environmental costs, in the United States as compared with costs in Canada and other sources of supply. Neither current nor projected costs are published.

## End Uses of Asbestos

The high tensile strength, flexibility, heat and chemical resistance, and favorable frictional properties of asbestos fiber make it adaptable to a large number of uses. Depending on the length of fibers and other characteristics, asbestos can be:

Carded, spun, or woven

Used as structural reinforcement of materials such as cement, plastic and asphalt

Laid and pressed to form paper.

Some authorities, such as the U.S. Bureau of Mines, state that there are more than 2,000 discrete uses of asbestos; others, such as the Asbestos Information Association and Canada's Department of Energy, Mines and Resources, suggest that there are upwards of 3,000 uses. A selected few of the many applications are shown in Table 3.

The properties of asbestos fibers determine the uses to which the fibers are put. Properties of major importance are length distribution, bundle diameter distribution, harshness, tensile strength, and surface activity. Other considerations include color and content of iron and dust. The relative-strength standard developed for chrysotile asbestos by the mining industry in Quebec is a convenient basis for delineating some major uses. For example:

- Long fibers (Groups 1 and 2* and 3) are used in textiles, electrical insulation, filtration media, and maximum-strength asbestos cement products.

- Medium length fibers (Groups 4, 5, 6) are used as reinforcing fillers in asbestos cement products, in friction materials such as brake linings and clutch facings, in paper, and in pipe covering.

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*No. 1 crude is 3/4 inch staple and longer (to nearly 6 inches). No. 2 crude is 3/8 to 3/4 inch staple. Other groups are milled fibers.



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Table 3

SELECTED ASBESTOS PRODUCTS AND THEIR END USES

| Floor Tile | Gaskets and Packings | Friction Products | Paints, Coatings and Sealants | Asbestos-Reinforced Plastics | Asbestos Cement Pipe |
|---|---|---|---|---|---|
| Office floors | Valve components | Clutch/transmission components | Automotive/Truck body coatings | Electric motor components | Chemical process piping |
| Commercial floors | Flange components | Brake components | Roof coatings and patching compounds | Molded product compounds for high-strength/weight uses | Water supply piping |
| Residence floors | Pump components | Industrial friction materials | | | Conduits for electric wires |
| | Tank sealing components | | | | |

| Asbestos Textiles | Asbestos Paper | Asbestos Cement Sheet |
|---|---|---|
| Packing components | Gas vapor ducts for corrosive compounds | Hoods, vents for corrosive chemicals |
| Gasket components | Fireproof absorbent papers | Chemical tanks and vessel manufacturing |
| Roofing materials | Table pads and heat protective mats | Portable construction buildings |
| Commercial/industrial dryer felts | Heat/fire protection components | Electrical switchboards and components |
| Heat/fire protective clothing | Molten glass handling equipment | Residential building materials |
| Clutch/transmission components | Insulation products | Molten metal handling equipment |
| Electrical wire and pipe insulation | Gasket components | Industrial building materials |
| Theater curtains and fireproof draperies | Underlayment for sheet flooring | Fire protection |
| | Electric wire insulation | Insulation products |
| | Filters for beverages | Small appliance components |
| | Appliance insulation | Electric motor components |
| | Roofing materials | Laboratory furniture |
| | | Cooling tower components |

Source: Asbestos Information Association/North America.



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- Short fibers (Groups 7, 8) are used as reinforcing filters in plastics, floor tile, and asphalt and in paints and oil-well drilling muds.

The consumption pattern for asbestos fiber in the United States is shown in Table 4. The construction industry--including new building, renovation, and maintenance--accounts for an estimated 70%-80% of total U.S. consumption. The first four uses shown in Table 4, which are construction-industry related, account for only 65% of the total use, but a portion of some of the other uses is associated in various ways with the construction industry. Several other aspects of Table 4 are noteworthy:

- Chrysotile fiber accounts for a very high proportion of total asbestos use (94% in 1974).
- About 98% of the crocidolite is used in the production of asbestos cement pipe, because of (1) its hardness, brittleness, and high tensile strength, which add to the rigidity of the end product, and (2) its superior filtration qualities, which enhance the drainage of water, permitting the cement to dry more rapidly.
- Asbestos cement pipe and sheets account for a large proportion of total use (38% in 1974).
- A very large proportion of total asbestos use is accounted for by the shorter-length fibers (Quebec Grade 7 chrysotile alone accounted for nearly 40% of the total use in 1974).

The transportation industry uses about 14% of all the asbestos consumed. The applicance industry uses some 5%-6% of the total.[5]

## Handling of Fiber and Products During Their Production

Producing the many asbestos-containing products that are used involves mining asbestos ore; milling, or hand-separating, the fibers from the ore and from each other; transporting both ore and fiber; and manufacturing the products themselves. These four general processes are reviewed briefly in the sections that follow.

### Mining

Most asbestos ore is mined in surface operations. Of the five U.S. mines in operation, four are surface mines and one is underground.* Each operating mine is associated with a mill that processes the ore.

*For a description of each mine and a discussion of the mining, milling, and dust-control procedures, see National Environmental Research Center, Characterization and Control of Asbestos Emissions from Open Sources, North Carolina, September, 1974.



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Table 4

MAJOR U.S. END USES OF ASBESTOS FIBER, BY TYPE AND GRADE
1974
(Short Tons)

| | Chrysotile | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Group 1 & 2 | Group 3 | Group 4 | Group 5 | Group 6 | Group 7 | Group 8 | Total Chrysotile | Crocidolite | Amosite | Anthophyllite | Total Asbestos | Percent of Total |
| Asbestos cement pipe | - | - | 138,800 | 41,900 | 300 | 4,200 | - | 185,200 | 36,400 | 1,100 | 200 | 222,900 | 26.4% |
| Asbestos cement sheet | - | - | 10,900 | 15,000 | 52,000 | 11,800 | - | 90,500 | - | 4,300 | - | 94,800 | 11.2 |
| Flooring products | - | - | - | 49,000 | - | 104,500 | - | 153,500 | - | - | - | 153,500 | 18.1 |
| Roofing products | - | - | 3,700 | 11,100 | 12,700 | 46,300 | - | 73,800 | - | 1,700 | - | 75,500 | 8.9 |
| Packing and gaskets | 100 | 1,900 | 7,500 | 7,500 | 1,400 | 10,300 | - | 28,700 | 100 | - | - | 28,800 | 3.4 |
| Insulation, thermal | - | 100 | 900 | 100 | 3,400 | 2,800 | - | 7,300 | - | 1,800 | - | 9,100 | 1.1 |
| Insulation, electrical | - | - | 400 | 1,600 | - | 2,700 | - | 4,700 | - | - | - | 4,700 | 0.6 |
| Friction products | - | 5,600 | 1,300 | 29,500 | 6,300 | 36,600 | 300 | 79,600 | - | - | 200 | 79,800 | 9.4 |
| Coatings and compounds | - | - | 100 | 400 | 300 | 36,700 | 400 | 37,900 | - | - | - | 37,900 | 4.5 |
| Plastics | 400 | 1,000 | 900 | 800 | - | 7,500 | 6,300 | 16,900 | 200 | - | 700 | 17,800 | 2.1 |
| Textiles | 700 | 14,200 | 3,900 | 800 | - | 800 | - | 20,400 | - | - | - | 20,400 | 2.4 |
| Paper | - | - | 5,500 | 400 | 23,900 | 33,300 | - | 63,100 | 200 | - | - | 63,300 | 7.5 |
| Other | - | 300 | 1,100 | - | 2,300 | 32,700 | - | 36,400 | 400 | 500 | - | 37,300 | 4.4 |
| Total | 1,200 | 23,100 | 175,000 | 158,900 | 102,600 | 330,200 | 7,000 | 798,000 | 37,300 | 9,400 | 1,100 | 845,800 | |
| Percentage of Total | 0.1% | 2.7 | 20.6 | 18.8 | 12.1 | 39.3 | 0.8 | 94.4 | 4.4 | 1.1 | 0.1 | | 100.0% |

Source: U.S. Bureau of Mines: Mineral Yearbook (preprint), 1974.



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In three cases, the mine and mills are at the same location, but two mines send their ore by truck to mills 32 and 55 miles away. (See Table 2.) The methods used to mine asbestos ore in the United States are described below.

Area strip mining, as practiced in the California operations, entails removal of the ore by earth-moving equipment from shallow deposits--in one instance without even the need for blasting. Generally, a shallow overburden with low concentrations of asbestos fiber must be removed.

Open pit mining, as practiced in the Vermont operation, is similar to area strip mining operations except that to follow the fiber veins, the workings are much deeper. Blasting and removal of ore occur primarily on the sides of the pit along terraces that spiral down around the sides of the pit toward the bottom.

Underground mining, as practiced in the Arizona operation, entails following the veins of ore with shafts, galleries, and drifts, using blasting and earth-moving equipment. This operation is followed by transporting ore to the surface, where it is processed further.

At the typical asbestos mine, coarse ore is crushed by a jaw or gyratory crusher to a size that can be accommodated by the mill. Oversize rock is separated by rotating cylindrical trommel screens and is crushed in a secondary crusher, usually of a conical type. The ore streams are conveyed to driers--rotary kilns in larger installations--where moisture in the ore (up to 30% by weight) is removed. The dried ore is then stored, with large amounts being held to allow for variations in fiber demand and mine production over time. Prior to milling, dried ore is conveyed to an additional crushing step.

## Milling

Milling, done primarily by hammer mills (fiberizers) or crushers, serves to free the fibers from the rock and separate the fibers from each other. In general, longer-length fibers in the final mix bring higher prices. Hence, it is desirable to hold the mechanical working or fibers to a minimum since, although the fibers have very high tensile strength, they are so fine that they are easily broken.

The most expensive grades of fiber are not mechanically milled at all; rather, they are hand-separated ("cobbed") from the surrounding rock into bundles of relatively long fibers with lengths of 3/4" or more. Such fiber is valued for manufacturing asbestos textiles.

The solution to the problem of maximizing the recovery of fibers other than hand-cobbed, in all but one of the asbestos mills in the United States, is to use mechanical means to free the fiber bundles from the rock, but to use air aspiration systems to separate and convey the


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fibers. In these systems, the ore is shaken on progressively finer screens through which small rocks and fiber bundles pass for further treatment while larger rocks are retained for further crushing or for conveyance to tailing dumps. The fibers freed are removed by the flow of air through powerful suction hoods. Separated fibers are caught in dry cyclones and conveyed to screens that separate them according to size. After sizing, the fibers are sent to bins for storage. Subsequent operations include removing the fibers from the storage bins, blending in fibers of different sizes to produce the desired final shipping specification, and bagging for shipment.

The one exception to the air-aspiration milling system is found in a California mill that processes the loose fiber ore by a wet-separation system.

Transportation

Conveyors and trucks are used at mine and mill sites to move ore from mines to mills.

Asbestos fibers typically are shipped from the mills in 100-pound, multiwall, paper or plastic bags.* (One producer reports the use of a stronger, woven, polyvinyl bag for some shipments.) Bags are pressure-packed to reduce bulk, damage, and dust. It is customary to tape ripped or punctured bags when the damage is discovered; yet the fact that the fibers are tightly packed may often prevent them from escaping even if the damaged area is not repaired. The technology is now available to pack asbestos even more tightly--i.e., to form blocks with twice the density of fiber shipped in conventional bags, a 100-pound block having a volume of about one cubic foot.

Palletizing is used almost universally, with the bags glue-locked to each other or shrink-wrapped to the pallet (wrapped with a film of plastic which is then shrunk) to stabilize the load. Much of the asbestos shipped is further unitized by being loaded into sealed railroad boxcars or shipping containers. Sometimes the asbestos is made into pellets, which, rather than being packed in bags, are loaded on and off railroad boxcars by gravity flow through pipes.

The majority of Canadian chrysotile fiber is shipped into the United States in conventional sealed railroad boxcars in which the contents are protected by inflated-rubber bags. The modern damage-free bulkhead cars are being used as they become available. The cars are routed directly from the Canadian mills to the U.S. manufacturing plants. Smaller proportions of chrysotile imports from Canada are received in containers, either by rail or by ship via the Atlantic

*Some manufacturers can add the bags, along with the fibers, to their product mix without even opening them.



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Ocean. All fiber from South Africa, the source of all U.S. imports of crocidolite, arrives by ship in containerized bags at Gulf Coast ports.

U.S. exports of fiber, virtually all of which are from California and destined for Mexico, Central America, and the Far East, leave the mills by both rail and truck. Containerized ocean shipments leave from ports such as Stockton, Sacramento, Oakland, and San Francisco. Although 100-pound bags are by far the most widely used in the industry, one of the California mills, for the convenience of its customers, ships all of its fibers in bags weighing from 10 to 50 pounds. One company plans to shrink-wrap each paper bag individually, for added protection, since some users buy in less-than-pallet lots. A representative of one of the world's largest shippers of asbestos reports that about 2% of the bags shipped sustain some damage.

## Manufacture of Asbestos-Containing Products

Production processes used in the consumption of asbestos are highlighted below for selected products.*

Asbestos cement products use the largest amount of asbestos of any product category (about 317,000 tons, or 38% in 1974). Specific products include wallboard, pipe, shingles, and blocks. Advantages of the products over their nonasbestos counterparts are better tensile strength, strength-to-weight ratio, strength under heat stress, resistance to acid, and smoothness of finished surface (critical to ensure laminar flow in pipe used for transport of liquids).

Asbestos fiber (primarily chrysotile, but also others to a limited extent) is mixed, either wet or dry, with portland cement and silica in proportions ranging from 10% to 70% of the total material. If the mixing is done dry, the mixture is generally metered in a flat layer onto an open surface, where the requisite water is applied by overhead spray. The resulting layer, much thinner than the final product, is then wound onto mandrels in a spiral mat (for pipe) until the requisite thickness is built up, or is layered flat (for wallboard or shingle forms). The same winding or layering process may be used for wet-mixed products, or the mixture may be cast. Finishing processes for the dried cement products vary with performance requirements and type, and may include grinding, drilling, sawing, or cutting.

Asbestos can be made into the full range of textile products--from nonwoven lap and felt, through yarn and cord, to woven cloth, rope, and tube. The asbestos fibers required for textiles are significantly different from those used for other asbestos products; they must be quite

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*For a more detailed discussion of the manufacture of asbestos-containing products, see U.S. Environmental Protection Agency, "Development Document for Effluent Limitations, Guidelines and Standards of Performance: Asbestos Manufacturing," Washington, D.C., 1973.



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long to be spinnable. Spinnable fiber is sometimes obtained by textile producers in hand-cobbed, "crude," form--i.e., as unopened, rock-free fiber blocks or bundles--since, as noted previously, it is difficult to protect fiber length during milling operations.

If the fibers are received as crude, they are opened in edge (knife) mills into small bundles and then milled into extremely fine flexible fibers. The resultant fibers, as well as being more delicate and breakable, are also more "floatable," leading to a greater emission potential per unit weight. Once the fibers have been adequately opened and fluffed, they may be blended with up to 20% of a cellulosic fiber such as cotton, the specific material chosen depending upon the application of the final product. The subsequent processes, such as carding, lapping, roving, spinning, and weaving or braiding (as required) are all performed on equipment essentially identical to standard textile machinery.

Asbestos is used in vinyl and asphalt floor tiles as a filler and reinforcement to provide strength and stability without reducing flexibility and compressibility. Very short fibers are used, comprising 8-30% of the total weight. In the case of vinyl tile, for example, polyvinyl chloride resin serves as the binder, limestone and other materials are used as fillers, and pigments and chemical stabilizers make up the rest of the typical mix. The manufacturing process is typically a 24-hour operation that includes weighing, mixing, heating to about 150°C, decorating, calendering, cooling, waxing, stamping, inspecting and packaging.

Friction products and gaskets typically contain 30-80% asbestos, generally in some sort of organic binder. In friction products, the asbestos is used for its unique combination of strength, compaction characteristics, friction properties, and stability at high temperatures. Asbestos is used in these products in two different ways: (1) the asbestos, as loose fiber, is mixed with the binder; or (2) the asbestos, as either matted or woven textile, is impregnated with the binder. The low total volume of the latter process is because it is used only in special situations, generally in gaskets, where dimensional stability and elasticity are of significance.

Asbestos paper has essentially the same properties as the usual cellulose-based paper, except that it has superior thermal insulation properties and fire resistance. It is used primarily as building paper (roofing and flooring) although it has been reported that the above-mentioned qualities also find use in high-quality-bond document papers. Asbestos paper is made using the same processes as those used for standard woodpulp papers, but the raw materials mix, by weight, might be 70-90% asbestos fibers (typically short in length), china clay and starch (or sodium silicate) as the binders, plus other constituents that provide special properties.



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# Chapter III
# BIOLOGICAL EFFECTS OF ASBESTOS FIBERS

As noted in the Introduction to this monograph, asbestos fibers are known to cause cancer. In this chapter, (1) a summary of what is known about the disposition of these fibers in the body is followed by (2) a description of the carcinogenic effects of the fibers, based on human studies as well as animal experimentation; also included is (3) a discussion of the noncarcinogenic effects of asbestos fibers.

## Disposition of Fibers in the Body

Technical difficulties associated with the assay of biological tissues for asbestos have limited the amount of information available about the disposition of asbestos. Almost all data have come from animal experiments in which asbestos was monitored by electron microscopy or radiotracers. Asbestos fibers typically enter the body by inhalation or ingestion.

### Inhaled Asbestos

The disposition of asbestos fibers entering the respiratory tract is not fully understood. Certainly some fibers are ultimately deposited in the airways and lung tissue. Some could also be expectorated or conveyed to the gastrointestinal tract by airway clearance mechanisms and possibly some to the pleural and peritoneal cavities via lymphatic drainage.

Of asbestos fibers found at autopsy in human lungs, a majority are less than 5 μm in length;[1,2] seldom do they exceed lengths of 200 μm or diameters of 3.3 μm.[3] One autopsy study of persons with occupational exposure demonstrated that all asbestos fibers examined in the lung were less than 0.5 μm in diameter.[2] This preponderance of small fibers in part reflects their ability to remain suspended in air for longer periods than larger fibers, but it is also a function of their deposition and clearance characteristics once they enter the respiratory tract. It is also possible that some fibers may be fragmented as the result of biological activity within pulmonary tissues.

Longer fibers are screened more effectively by the nasal hairs. Inside the upper respiratory tract, fibers are deposited through the forces of gravitational sedimentation and impaction at points where the air stream changes direction; and these depositions depend largely on



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fiber diameters.[3,4] In the small airways, especially at airway branch points, the collision cross-sectional area, which is a function of fiber length, is of greater importance.[3] As a result of these obstacles, a greater proportion of fibers reaching the gas-exchange surface of the alveoli may be small, compared with fibers entering the upper respiratory tract.

Studies with mammalian cells in culture indicate that these shorter fibers (usually less than 5 μm) may be engulfed by alveolar macrophages and transported to lymphatic channels or the mucociliary blanket for excretion. Longer fibers may be only partially engulfed or may be engulfed by several macrophages at once.*[7]

Data from autopsies of humans have suggested that some fibers may enter alveolar lymphatic channels and be carried to hilar and mediastinal lymph nodes.[8] Numerous fibers can be found in the pleura, a serous membrane which covers the surface of the lungs, thoracic diaphragm, and chest wall; how they gain access to the pleura is not known. Generally, the concentration of fibers in the pleura is less than in the lung itself, but in some areas of the pleura, fiber concentrations similar to those within the lung tissue have been observed.[9]

## Ingested Asbestos

Most asbestos fibers entering the gastrointestinal tract are probably excreted with the feces. Although it has been reported in one study that there is little evidence of asbestos fibers[10] penetrating the walls of the gastrointestinal tract, there have been animal studies showing such penetration. In a study reported in 1965, chrysotile fibers were found in the lining of the colon in rats fed a diet containing a massive amount (6%) of chrysotile asbestos.[11] In another study, fibers of amosite asbestos suspended in saline, when placed into an isolated segment of rat jejunum in vivo, were found penetrating and within the jejunal wall.[12] One recent study of rats fed 250-300 mg of asbestos per week for a year did indicate that if penetration of the gastrointestinal tract lining does indeed occur, the number of

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*In an experiment with rats, about one-third of inhaled asbestos (crocidolite) was deposited on the surface of the respiratory tract. Half the amount deposited at inhalation was found immediately afterward in the gastrointestinal tract, nose, pharynx, and larynx; clearance from these latter respiratory tissues to the gastrointestinal tract was practically complete within an hour. Of the remaining crocidolite deposited in the lungs, one-quarter had been evacuated at the end of the month.[5] In another study of rats exposed to amphibole asbestos for six months, 41%-74% of the asbestos found in the lungs immediately after exposure had been removed within 18 months.[6]



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penetrating fibers would be very small (90% probability of less than 1500 fibers).[13]

## Injected Asbestos

Asbestos injected into the bloodstream is rapidly removed and deposited in various tissues, with highest concentrations observed in lungs, liver, and spleen.[14,15] Limited evidence suggests that asbestos in the blood may be transported across the placenta of rats.[16]

In mice, asbestos injected subcutaneously migrates along lymphatic pathways from the injection sites. Fibers accumulate in lymphoid tissues, particularly in regional lymph nodes, and are usually contained within macrophages. Small numbers of fibers may be found in the spleen, pleura, liver, kidneys, and brain.[17]

## "Asbestos Bodies"

Approximately 10% to 30% of the fibers retained by human lungs (usually longer than 5 μm) become coated with mucopolysaccharide and hemosiderin to form yellow-to-brown rod-shaped structures with clubbed ends, often beaded along their length.[18] These structures were first called "asbestos bodies," but now they are frequently referred to by the more general term, "ferruginous bodies," since identical structures may result from the coating of fibers other than asbestos. It has been hypothesized that this coating, laid down by engulfing macrophages, renders the fibers biologically less active.

It is thought that a certain balance is achieved between the formation of asbestos bodies and their dissolution or excretion.[19,20]

Asbestos bodies found in the sputum are strong presumptive evidence of asbestos exposure. Occupational exposure as brief as one day and as long ago as ten years has been shown to produce sputa containing asbestos bodies.[20] Asbestos bodies in lung smears or tissue (unlike those in sputum) are commonly found among residents of urban areas who may never have been exposed to asbestos in the workplace.[21-23]

Asbestos bodies or fibers have been detected in extra-pulmonary tissues of persons occupationally exposed to asbestos: in tonsils, thoracic and abdominal lymph nodes, pleura, peritoneum, liver, spleen, pancreas, kidney, adrenals, and small intestine. The numbers found appear to be far fewer than in the lung.[8,24]

## Carcinogenic Effects--Human Studies

The many observations, both case reports and epidemiologic studies, of cancerous effects among humans exposed to asbestos fibers could be



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looked at from a number of points of view, but the ones used here are: types of cancer; fiber type; dose-response relationship; latency; age at first exposure; smoking; indirect occupational exposure; household exposure; and residential exposure. The section on human carcinogenic effects is followed by a summary discussion of animal experiments and asbestos-related cancer.

High rates of lung cancer have been observed in asbestos workers exposed to all commercial asbestos types. Among some groups of asbestos workers, approximately 20% of all deaths are caused by lung cancer where the proportion of deaths expected from this cause would be only about 5%.[25] Pleural and peritoneal mesotheliomas* are a frequent occurrence among occupational groups exposed to chrysotile, crocidolite, and amosite. Estimates for certain occupational groups suggest that as many as 8%-11% of deaths may be due to mesothelioma, a relatively rare cause of death in the general population.[26] Some occupational groups exposed to asbestos have, furthermore, demonstrated an excess of other cancers, especially of the larynx and gastrointestinal tract.

Asbestos exposure leading to an excess of cancer may occur among groups exposed indirectly, as in shipyard workers or in groups mining other minerals that may contain asbestos as a contaminant. Mesothelioma also has occurred among persons living in the homes of asbestos workers or in the vicinity of asbestos facilities.

Both cigarette smoking and occupational asbestos exposure individually increase the risk of lung cancer but, together, they act to produce a risk of lung cancer that exceeds the sum of their separate risks.

## Evidence of Lung Cancer

The evidence that asbestos is a cause of lung cancer is overwhelming. Lung cancer was first linked with exposure to asbestos in 1935, when three cases of asbestosis and carcinoma of the lungs were found at autopsy in asbestos textile workers.[27,28] Other case reports followed. In 1949 the Chief Inspector of Factories of England and Wales examined 225 deaths from asbestosis or in which asbestosis had been proved at autopsy. Cancer of the lung or pleura was found in 31, or 13.2%. This was not characteristic of other pneumoconioses; among 6,884 deaths with silicosis at autopsy, for example, only 91, or 1.32% had cancer of the lung or pleura.[29]

Further evidence implicating asbestos in the etiology of lung cancer came from a matched-pair case-control study published in 1954. Upon classifying by 5 years or more employment in occupations

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*Mesothelioma is a term used to refer to a tumor made up of cells from the pleura or peritoneum.



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involving asbestos exposure (steam fitters, boilermakers and asbestos workers), 10 patients with lung cancer but only one control had been so employed.[30]

In a study reported one year later, 11 of 113 workers employed in an asbestos textile factory in the United Kingdom for at least 20 years were found to have died from lung cancer. By applying to this group age-specific mortality rates for lung cancer in the general population, 0.8 deaths would have been expected. The ratio of observed to expected deaths, therefore, was greater than 13.[31] Continued study of workers at this factory has shown a reduced risk of lung cancer, although still two- to three-fold in excess, among workers first employed after 1933, when regulations for control of asbestos exposure in the United Kingdom had gone into effect.[32,33] Numerous other studies have independently confirmed an increased risk of lung cancer in various occupational groups exposed to asbestos.[25,34-39]

## Evidence of Mesothelioma

There is also overwhelming evidence that asbestos is a cause of pleural and peritoneal mesothelioma. Cases have been described in persons with occupational and nonoccupational exposures. The first case report of a pleural neoplasm related to asbestos exposure appeared in 1933.[40] Additional cases were noted in the 1940s,[41-43] and in 1954 a peritoneal tumor was reported.[44] However, it was not until 1960, with the publication of a series of cases from South Africa, that the association between mesothelioma and asbestos exposure was generally recognized. Of 33 South African patients with mesothelioma, 32 gave a history of occupational exposure to asbestos or residence in a crocidolite mining area.[45] Subsequently, a plethora of individual case reports, case series, case-control studies, and studies of the mortality of occupational groups have related the occurrence of mesothelioma to a history of exposure to asbestos.[25,39,46-53] The ratio of pleural to peritoneal tumors varies considerably in different studies, but peritoneal tumors seem to be associated with heavier exposures and with asbestosis.[54-56]

## Laryngeal Cancer

The evidence casually linking asbestos and laryngeal cancer is highly suggestive. In a study of 119 patients with squamous carcinoma of the larynx and age- and sex-matched controls, 33 with laryngeal cancer, but only 3 controls, gave a history of occupational exposure to asbestos.[57,58] This difference was striking, and the suggested increased risk has since been confirmed by similar case-control studies,[59] and studies of occupational cohorts.[60,61]



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## Digestive System Cancer

An excess risk of cancers of the digestive system attributable to occupational asbestos exposure has been suggested by a number of epidemiological studies.[25,34,35,60,62-69] A major problem with these studies has been the inclusion of peritoneal mesothelioma cases among all observed cases, making it difficult to document an increased risk of any one digestive system cancer independent of that for mesothelioma.

In occupational cohort mortality studies where peritoneal mesotheliomas were separated from other cancers of the digestive system, excess cancers of some sites have been observed. Among 933 amosite asbestos factory workers who were first employed between 1941 and 1945, there were 11 deaths from stomach cancer by 1971 (vs. 4.58 expected); 15 deaths from cancer of the colon or rectum (vs. 7.05 expected), and none from cancer of the esophagus (vs. only 1.23 expected).[25] Of 1.779 deaths through 1974 among a cohort of 17,800 insulation workers there were observed 15 deaths from oropharyngeal cancer (vs. 7.87 expected); 14 deaths from cancer of the esophagus (vs. 5.35 expected); 18 from stomach cancer (vs. 11.23 expected), and 47 from cancer of the colon or rectum (vs. 28.63 expected).[25,60] Other studies of groups exposed to asbestos examining the risk of these specific cancers after excluding mesotheliomas are needed to further elucidate the role of asbestos in cancers of the digestive system.

## Other Cancers

Studies of women asbestos workers have suggested possible increases in cancers of the ovary and uterine cervix. Among a group of female English asbestos worker, 4 and possibly 6 deaths from ovarian cancer were observed while only 2.1 had been expected.[55] Investigators from the Soviet Union have reported significantly increased rates of cervical cancer among older female asbestos workers; however, the numbers of persons in the study populations were not indicated.[35] Findings in these reports need confirmation elsewhere.

## Association of Effects with Fiber Type

There are few studies of persons exposed to a single type of asbestos, and the studies that are available often lack information on potential confounding factors such as cumulative exposure, smoking history, and physical characteristics of airborne fibers. It is therefore exceedingly difficult to assign a scale of relative pathogenicity to different types of asbestos. For example:

- Crocidolite mined in the Northern Cape Province of South Africa and in Western Australia is frequently associated with pleural mesotheliomas, whereas fewer cases have been reported for crocidolite from the South African Transvaal.



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It has been proposed that this apparent difference in risk may relate to a difference in physical characteristics of fibers from these areas--crocidolite fibers from the Transvaal are thicker and longer.[70]

- As asbestos proceeds from mine to mill to manufacturing plant, it can become subdivided (and fractured), producing fibers of smaller diameter and length, with possibly greater or smaller attendant biological risk.

- Asbestos thought to be of one type may be "contaminated" with asbestos of another type. Lung tissue from men employed in Canadian chrysotile mining has been observed to contain tremolite and other amphiboles, often more than the amount of chrysotile.[71]

All types of commercial asbestos have been related to high rates of lung cancer in asbestos workers, and in occupational groups exposed to chrysotile, crocidolite, and amosite, pleural and peritoneal mesotheliomas have been observed.[72] Studies of anthophyllite miners in Finland have shown a slight excess of lung cancer, but no mesotheliomas.[36,37] The lack of mesotheliomas may be due to the small size of the cohort studied; however, none has been reported from the communities in the mining area.[73]

There is some information suggesting that chrysotile may not be as hazardous as other types of asbestos.[38,47,74-78] The mortality experience of a cohort of workers employed at an asbestos paper and millboard manufacturing plant that used only chrysotile asbestos would seem to bear this out.[79]

Pleural and peritoneal tumors, as well as excess lung cancer, have been found in the mortality experience of workers who had mined and milled New York State talc.[80,81] This talc may contain large quantities of tremolite asbestos as well as smaller amounts of anthophyllite and chrysotile. In Italy, where the talc is reportedly uncontaminated, mining and milling has not been associated with mesothelioma or excess lung cancer.[82]

## Dose-Response Relationship

Evidence that the risk of developing cancer is related to the degree of exposure to asbestos by some quantitative estimate strengthens the basis for assuming asbestos to be of causal importance. A precise dose-response relationship is difficult to establish for any environmental exposure and no less so for asbestos. However, attempts have been made for asbestos and there is some evidence suggesting that dose



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as measured by severity of exposure and duration of employment relates to rates of cancer in groups occupationally exposed to asbestos.

One study concluded that within an age cohort for which data were most accurate there was an increasing mortality due to lung cancer with increasing duration of employment.[63] However, rates for individuals employed the longest were lower than those in the category of next greatest duration of employment; persons employed longest might more likely be those whose occupational exposure to asbestos was less intense or who were themselves less susceptible to cancer and respiratory diseases (for example, persons who do not smoke).

An investigation of mortality among workers at an asbestos textile factory in England found markedly reduced mortality from lung cancer, and from other diseases in the presence of asbestosis, with reduction in length of exposure before 1933.[32] Moreover, the risk of lung cancer and mesothelioma among workers at a Londom asbestos textile and insulating materials factory was independently found to be related to the severity and duration of exposure.[39,55,83]

The respiratory cancer mortality (includes deaths due to pleural mesothelioma in addition to cancers of the lung and larynx) of a group of retired asbestos workers was categorized according to cumulative dust exposure. "Exposure" was the product of job-characteristic dust levels in millions of particles per cubic foot (mppcf) and number of years on the job, summed across all jobs held (thus giving cumulative exposure in mppcf-years). The result is shown in Table 5. It can be seen that there is a clear gradient of increasing Standard Mortality Ratio (SMR), or ratio of observed to expected deaths x 100, with increasing cumulative dust exposure.[78]

Table 5

DEATHS FROM RESPIRATORY CANCER BY CUMULATIVE DUST EXPOSURE

| Total Dust Exposure (mppcf-yr) | Number of Men | Respiratory Cancer Deaths | | |
|---|---|---|---|---|
| | | Observed | Expected | SMR |
| Under 125 | 533 | 15 | 9.0 | 166.7 |
| 125-249 | 305 | 12 | 4.8 | 250.0 |
| 250-499 | 328 | 17 | 5.2 | 326.9 |
| 500-749 | 126 | 9 | 1.8 | 500.0 |
| 750 and over | 56 | 5 | 0.9 | 535.6 |

Source: Chapter Reference 78.



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## The Cancer Latency Period

From all available evidence, the period between first exposure to asbestos and death from lung cancer appears to be related to intensity of exposure.[84] Among workers at an English asbestos textile and insulating materials factory, an excess mortality from lung cancer was demonstrated following a latency of 15 years for those with heavier exposures, whereas an excess did not appear until 25 years from onset of exposure for those whose exposures were less intense.[39,61,83]

Fifteen years is probably the minimum latency period for asbestos-related lung cancer. An excess of lung cancer deaths first appeared among a group of heavily exposed amosite asbestos workers 15 years after onset of exposure,[85] and in a large cohort of insulation workers followed from 1967 through 1974 (135,000 person-years of observation), no applicable increase in mortality from lung cancer was observed before 15 years had elapsed from onset of exposure. The peak increase occurred about 30-35 years after onset of exposure.[25]

In 85% of one series of mesothelioma cases, death occurred more than 25 years after first exposure to asbestos, with a range of 3.5 to 53 years.[86] Another investigator reported a mean latency period of 37 years,[87] and among a large cohort of asbestos workers, most deaths from pleural and peritoneal mesotheliomas occurred 30-35 years after first exposure.[25]

## Incidence of Cancer and Age at First Exposure to Asbestos

A characteristic of many cancers is a marked increase in incidence with advancing age. It is generally acknowledged that the higher incidence of cancer in older persons is not necessarily because their tissues are more predisposed to cancer, but because of the usually long period between initial exposure and the appearance of diagnosable tumors.[88] In fact, with regard to susceptibility of body tissue to cancer, it has been hypothesized, from experimental animal evidence, that the tissues of younger people may be more susceptible to carcinogens but that, conversely, older people may be more susceptible to cancer because of a less efficient immune surveillance system.[89]

In a study of the relative incidence of lung cancer according to age at first exposure to asbestos, data were obtained on 117 men who were exposed for more than 20 years and who were followed for an average of 13 additional years. A greater incidence of lung cancer was observed among men first exposed at older ages. For those first exposed under age 25, the annual lung cancer incidence was 26% of the rate for all ages; for first exposure between ages 25 and 29 it was 165%; at age 30+ it was 195%. These incidence rates were corrected to account for variation in the duration of exposure, duration of survival after first exposure, and for the fact that some lung cancers may have been due to



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nonoccupational causes more common among older men. The data suggested that susceptibility to asbestos-related cancer may increase with age.[88,89]

It seems unlikely, however, that age per se is a principal factor in determining frequency of cancer, and it is not clear whether age itself plays a part that is independent of exposure duration and time elapsed since first exposure.

## Smoking and Asbestos-Related Cancer

There is strong evidence that cigarette smoking alone is sufficient to cause lung cancer. Many studies attempting to examine the effect of cigarette smoking on the increased risk of lung cancer observed in groups exposed to asbestos have been deficient in one or more of these respects:

- Follow-up periods have been too short to allow accurate computation of risk after the necessary 15-20 year latency.
- Nonsmoking asbestos-exposed groups have been very small.
- Smoking habits have not been completely ascertained.
- Smoking-adjusted mortality rates from the general population have not been used for comparison.

A few studies have found that cigarette smoking was insufficient to account for the increased risk of lung cancer among asbestos workers. This has been generally accepted as evidence that asbestos can act independently to cause lung cancer, a view that has been corroborated by animal experiments and by some evidence suggesting an increased risk among nonsmoking asbestos workers.[62]

An investigation of two groups of asbestos workers--one with a high dust exposure and a high respiratory cancer mortality, the other with a lower dust exposure and a lower respiratory cancer mortality--found that their smoking habits were similar.[78] This implies that high doses of asbestos can account for higher mortality among smokers. Another investigation, a case-control study of lung cancer patients, revealed an enhanced risk of lung cancer with asbestos exposure, whatever the number of cigarettes smoked.[62]

In a study of the combined effects of asbestos exposure and smoking, the smoking habits of 1,334 male and 482 female asbestos factory workers were examined in relation to mortality from lung cancer over a 10-year period. Among 955 smokers with severe asbestos exposure, 41 lung cancer deaths were observed, while only 11.3 were expected for smokers from the general population. Among 161 never-smoking asbestos workers with



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severe asbestos exposure, 1.7* lung cancers deaths were observed, while only 0.2 were expected for nonsmokers in the general population. There were approximately five times as many person-years of observation in the smoking group compared with the nonsmoking group, but about 24 times as many lung cancer deaths among the smokers.[90]

In another study, smoking histories were obtained from 11,657 of a cohort of 17,800 insulators. In 9,591 workers with a history of cigarette smoking, 248 lung cancer deaths were observed, compared with 59.5 expected. Among 609 workers who had smoked pipes and/or cigars, two lung cancer deaths were observed and 1.24 were expected. Of 1,457 workers who never smoked regularly, four lung cancer deaths were observed, while 1.08 were expected.[25] Expected deaths were based on approximate smoking-specific U.S. death rates.

Further analyses of group data have been performed to examine how cigarette smoking and asbestos might be acting together.[91,92] On a statistical basis it appears that these two independent causes of lung cancer interact positively. In the general population, cigarette smokers have a 10-15-fold excess risk of lung cancer. One study observed an 8-fold excess of lung cancer among smoking asbestos workers compared with smokers in the general population, but the excess was 92-fold when compared to the general population of nonsmokers.[92] This suggests that the combined effect of smoking and asbestos exposure is greater than the simple sum of their separate effects. The relation of this statistical interaction to the pathogenesis of lung cancer is uncertain. However, it seems clear that, due to the important enchancement of risk by one cause complementing the other, the increased risk of lung cancer in groups exposed to asbestos may be concentrated among those who also smoke.

There is very little evidence that cigarette smoking increases the frequency of developing pleural mesothelioma after asbestos exposure, and no evidence exists that smoking increases the risk of peritoneal mesothelioma. Of 9,951 insulation workers with a history of cigarette smoking, there were 23 deaths from pleural mesothelioma and 47 deaths from peritoneal mesothelioma, with none expected. Among 1,457 workers who never smoked regularly, there were two deaths from pleural mesothelioma, eight deaths from peritoneal mesothelioma, and none expected.[25]

## Cancer from Incidental Occupational Exposure

Persons who do not work with asbestos directly, but who may be incidentally exposed to asbestos while working, may suffer excesses of cancer. A 10% random sample of shipyard workers, widely distributed

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*This is an adjusted figure to compensate for missing smoking information for the deceased.



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throughout the various trades, revealed that some had pleural plaques and some had pulmonary fibrosis, and retrospective and prospective cohort mortality studies have revealed a 2.5-fold excess risk of lung cancer as well as a number of mesotheliomas among workers with pleural plaques. Even those without X-ray evidence of exposure to asbestos had a slightly increased risk of lung cancer and a few mesotheliomas. A study of sheetmetal workers also revealed a small excess of lung cancer and one mesothelioma.[93-95]

### Disease in Workers' Households

There are presently 37 reported cases of mesothelioma in persons whose presumed exposure to asbestos was limited to living in the homes of asbestos workers.[96] However, no comprehensive studies of the distribution of mesothelioma by age and sex among contacts of asbestos workers in their homes have been conducted.

The majority of reported mesotheliomas related to household exposure have been in women, perhaps because they are more likely to be exposed to asbestos by washing the garments of asbestos workers.[96,97] Thirty-five percent of examined family contacts of asbestos workers were found to have radiological abnormalities characteristic of asbestotic disease.[72,96]

### Cancer in the Neighborhood of Asbestos Facilities

On the basis of numerous anecdotal reports, indirect assessments, and case-control studies, there seems little doubt that both pleural and peritoneal mesotheliomas may result from some types of residental exposure to asbestos.[45,48-50,74,86,98-103] However, there have been no adequate population-based studies, and an accurate estimate of risk, where occupational and household exposure are definitely excluded, cannot be made. On the basis of one case-control study of mesothelioma patients, relative risk of mesothelioma was estimated at 2.1 for residentially exposed, and 4.3 for occupational exposed, persons.[49]

Two studies have been made of the possible effects of increased asbestos contamination of drinking water in Duluth, Minnesota, due to the disposal of taconite tailings into Lake Superior. No carcinogenic effects have been noted, but the period of observation was short relative to the probable latency period of environmentally-induced cancer.[104,105]

## Carcinogenic Effects--Animal Studies

All commercial types of asbestos--chrysotile, crocidolite, amosite, anthophyllite--have been found to be carcinogenic when tested in mice, rats, hamsters, and rabbits. A brief review of evidence derived from



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experimental observations is presented below for its value in corroborating known human cancer risks, predicting other effects, and increasing the plausibility of certain hypotheses on causal mechanisms. A more detailed treatment of these and other relevant studies in animals can be found in Appendix D.

Intrapleural or intraperitoneal injection of asbestos has produced sarcomas* and mesotheliomas. Laboratory animals have not been known to develop mesotheliomas spontaneously, so that finding even a single such tumor in an experiment may be significant. Rats and rabbits receiving intrapleural injections of crocidolite developed pleural mesotheliomas, as did rats receiving chrysotile.[106] For both these fiber types the carcinogenic response appeared to be dose-related in another study with rats.[107] Mesotheliomas have also been induced in rats by Russian chrysotile[108] and in hamsters by various types of asbestos fibers.[109]

Peritoneal mesotheliomas were observed in rats following intraperitoneal injections of chrysotile and crocidolite but not amosite.[106] Rats that received intraperitoneally chrysotile milled to 99% <3 μm also developed peritoneal tumors.[110] Mesotheliomas were induced in rats inoculated with crocidolite[111] and in mice inoculated with chrysotile, crocidolite, or glass fiber.[112]

Lung carcinomas and pleural mesotheliomas have followed from the inhalation of asbestos. Rats exposed to various doses of chrysotile, crocidolite, and amosite have developed malignant tumors of the lung and of the mesothelium.[106,113-115] Among these studies, adenocarcinoma, squamous cell carcinoma, and fibrosarcoma were reported. Among groups of rats that were exposed with varying durations to five different types of asbestos fibers, all of which produce asbestosis, a dose-response relationship for malignancies was suggested.[6] In this study it was observed that as little as one-day exposure was sufficient to produce tumors.

Few data exist about the effects of asbestos administered orally. One study found that oral administration of asbestos filter material to rats led to an increased incidence of malignant tumors.[116] Carcinomas of the lung, kidney, and liver, as well as reticulum-cell sarcomas, were found.

The available evidence from animal studies relating asbestos type to differences in carcinogenic potency is limited and inconsistent. This may be due to the predominant influence of size and shape of fibers, which may vary from one study to another for each asbestos type. Mineral fibers other than asbestos, but of similar size, can produce mesotheliomas in rats after intrapleural or intraperitoneal injection.[107,117,118] Although the carcinogenic mechanism involving fibers has not

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*A sarcoma is a malignant tumor derived from mesodermal tissue.



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been entirely elucidated, a reasonable hypothesis is that it may be related to morphologic characteristics.

There are few studies in which fiber size alone has been varied and adequately recorded (diameter as well as length). Furthermore, the preparation of fibers for experimental purposes may alter mineral properties.[119]* However, smaller fibers are thought to be more active in producing tumors.

## Noncarcinogenic Effects of Asbestos

Noncarcinogenic effects of asbestos exposure were noted several decades before the association between asbestos and cancer was recognized. In 1906, deaths among asbestos textile workers from penumoconiosis were reported in England and France.[121,122] These were the first reports in modern times of the diffuse interstitial fibrosis of the lung associated with asbestos exposure--"asbestosis."

### Asbestosis

Asbestosis, which is characterized by a diffuse interstitial fibrosis of the lung, is one of the many dust-related lung diseases that are terminal pneumoconioses. However, unlike some of the other pneumoconioses, asbestosis does not predispose to the development of pulmonary tuberculosis, nor does evidence suggest that it is causally related to emphysema and chronic bronchitis.[123]

#### Clinical Findings

The signs and symptoms of asbestosis, listed below, are no different from those for other forms of diffuse interstitial fibrosis; there are no pathognomonic features.

Symptoms:

- Breathlessness on exertion
- Cough, usually dry, but may be productive
- Chest tightness or pain

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*Ball milling of chrysotile, for example, can result in decreased crystallinity and changes in interlayer branching and surface hydroxyl configuration. These alterations have been accompanied by decreased hemolytic activity.[120]



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Signs:

- Decrements in lung function (decrease in lung volume and flows)
- Radiographic abnormalities (chest)
- Rales, basilar
- Restricted chest motion
- Clubbing of fingers
- Cyanosis
- Cor pulmonale (right ventricle hypertrophy)
- Pleural effusion.

The earliest and most prominent clinical finding--breathlessness on exertion--rarely becomes apparent until after at least a decade of exposure. Thus, by the time this "early" clinical finding appears, the underlying disease process is well under way. As the disease progresses, breathlessness may be present even at rest.

The most characteristic physical sign exhibited by the patient with asbestosis is the presence of dry, crackling sounds (rales), heard on auscultation at the lung bases and in the axillae during inspiration. As fibrosis progresses, rales become more widespread and occupy a greater part of the inspiratory cycle.

Clubbing of the fingers is usually a late feature of asbestosis and is not found consistently. Cyanosis of the skin and mucous membranes of the mouth and tongue may also occur in the later stages of the disease.

Radiographic Abnormalities

Radiographic features of asbestosis are similar to those of other forms of diffuse interstitial fibrosis of the lung, except for the frequency of pleural changes, expecially pleural plaques (which should always signal the possibility of asbestos exposure). Radiographic diagnosis of asbestosis is based on the presence of small, irregular, or round opacities distributed prominently in the lower lung fields. The earliest changes often occur bilaterally in the cosophrenic angles. Short, horizontal, linear septal lines (Kerley B-lines), which are believed to represent lymphatic obstruction,[54] may also be present. With time, the abnormal shadows gradually spread upward into the middle and upper zones of the lung fields and become increasingly coarse and blotchy. In more advanced cases, a "honeycomb" pattern may be present,



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and the outline of the diaphragm and heart may become blurred and "shaggy." Pleural changes are likely to be present as well, perhaps in as many as 50% of the cases.

## Pulmonary Function Changes

The interstitial fibrosis associated with asbestos exposure is accompanied by changes in pulmonary function characteristically observed with interstitial fibrosis from other causes as well. Thus, while these changes are not unique to pulmonary asbestosis, they provide useful diagnostic information when interpreted together with other evidence such as exposure history, signs and symptoms, and chest radiographic findings. Changes in pulmonary function considered most characteristic of asbestosis are:

- General reduction of lung volume, especially of vital capacity (VC)
- Decrease in pulmonary flow rates such as indicated by forced expiratory volume in one second ($FEV_{1.0}$)
- Impaired alveolar-capillary diffusing capacity, reflected by reduced oxygenation of the arterial blood and increased alveolar-arterial $PO_2$ gradient (alveolar-capillary block syndrome).

Although it is usually claimed that airway obstruction is rarely a major feature of asbestosis,[123-125] one investigator has pointed out that epidemiologic studies of lung function have been unable to clarify the relationship between obstructive airway disease and asbestos exposure. She advised that, until further evidence becomes available, "an open mind should be kept in this regard."[54]

## Asbestosis and Cancer

On the basis of case reports, an association between asbestosis and lung cancer was suspected as early as the 1930s. More than a decade later, two authors[126,127] reported that, among British asbestos workers, carcinoma of the lung and pleura had been found in about 15% of deaths either caused by asbestosis or in which asbestosis had been proved present at autopsy.* By the period 1961-1963, figures from the British Ministry of Labour showed that approximately 50% of patients certified as suffering from asbestosis (in contrast to exposed to

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*It remained for another investigator[31] to show that the actual risk of dying from lung cancer was increased on the order of 10-fold over the general population for male asbestos workers employed for 20 years or more between 1922 and 1953.



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asbestos) died of (or with) lung cancer.[128*] Although carcinoma of the lung is often found in the presence of asbestosis, there appears to be no scientific evidence that the two lesions are interrelated, except that they both may be classified as diseases that are causally associated with exposure to asbestos.

With regard to another asbestos-related cancer, two reviewers of the subject have noted that mesothelioma of the pleura and peritoneum has often been associated with even low levels of asbestos exposure for brief periods in the remote past.[54,123] There appears to be no regular correlation between severity of asbestosis and occurrence of mesothelioma. In fact, it is unusual to find significant pulmonary interstitial fibrosis (asbestosis) with pleural mesothelioma. However, prominent asbestosis is frequently observed in association with tumors of the peritoneum,[54,123] and peritoneal tumors generally appear to be associated with heavier asbestos exposure.[45,130] One reviewer noted that this appears to support the notion of retrograde lymphatic spread of asbestos fibers from the lung to the abdominal lymphatics resulting from thoracic lymphatic obstruction due to advanced pulmonary fibrosis.[54]

## Asbestos Pleural Effusion

Benign pleural effusion, which usually occurs in the presence of some degree of parenchymal asbestosis, is another clinical manifestation of disease due to asbestos exposure. Among a series of 57 patients with asbestosis or asbestos exposure, 12, or 21%, were found to have "asbestos pleural effusion"--i.e., a pleural effusion in an individual with a history of occupational exposure to asbestos in the absence of any other disease known to cause pleural effusion.[131] A number of individuals diagnosed as having had asbestos pleural effusion have subsequently developed mesothelioma.[132,133]

## Pleural Calcification, Diffuse Fibrosis, and Plaques

Asbestos-related plaques occur as discrete, elevated, grey-white lesions on the inner surface of the rib cage and on the diaphragm.

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*The reason for this apparent increase in the percentage of deaths in which asbestosis and lung cancer were found on autopsy is not clear. One explanation may be that the cases described by earlier authors[126,127] received much of their asbestos exposure in the early quarter of the centruy, when it was likely that airborne concentrations of asbestos were high. As a result, many of these cases may have succumbed to asbestosis at an early age--before lung cancer, with its long latent period, could develop.[128,129]



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Microscopically, the plaques consist primarily of connective tissue, often containing deposits of calcium. They do not interfere with pulmonary function to any significant extent, nor do they necessarily indicate the presence of pulmonary fibrosis.

An association between pleural abnormalities and exposure to asbestos--both occupational and nonoccupational--has been clearly demonstrated.[54,123,134-139] Accordingly, their presence should always alert the examiner to the possibility of asbestos exposure. In this regard, it may be 20 to 40 years after exposure to asbestos that pleural calcifications appear radiographically.[135]

Although pleural plaques do not, themselves, appear to be precursors of malignant disease, a retrospective death-certificate study of 408 shipyard workers with pleural plaques showed that the risk of developing bronchial carcinomas was increased by a factor of 2.4.[95] Three cases of mesothelioma also occurred in this series. In a prospective study from the same shipyard, 235 men with radiographic evidence of pleural plaques were found to have a 2.4-fold increased risk of bronchial carcinoma. Among 70 deaths, 13 were due to mesothelioma, an excess of obvious significance.[94]

## Asbestos Warts

Asbestos warts, or corns, are of minor health significance, but they are an indication of exposure to asbestos. They are caused when asbestos fibers penetrate the skin and are most often found on the hands and forearms.[140] The warts may have a pinpoint, black center and are often tender to pressure. Unless removed by excision, they may persist for years.

## Animal Studies--Evidence of Noncarcinogenic Effects

Exposure by inhalation to any of the four commercial asbestos types may result in fibrosis of the lungs in animals as well as in humans.[141] Under experimental conditions a fibrogenic response to inhaled asbestos has been reported in rats, hamsters, guinea pigs, rabbits, and monkeys.[142-144]

Outside the laboratory, pulmonary fibrosis in the presence of fibers and asbestos bodies has been demonstrated in baboons, donkeys, and wild rodents living in the vicinity of crocidolite mines or mills.[145] Pulmonary asbestosis has also been reported in a dog kept as a rat catcher in a London asbestos factory.[146] There are marked interspecies differences in susceptibility to asbestosis. Tissue reaction in rats, rabbits, and monkeys is typically less severe than in hamsters and guinea pigs.[142,147]



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Subcutaneous injection of asbestos or injection into the pleural or peritoneal cavities produces a fibrotic reaction. Thickening of the pleural, pericardial, and peritoneal membranes has been reported, with formation of adhesions and granulomas as well as pulmonary and mediastinal abscesses.[17,148,149]

Neither shape nor chemical composition is sufficient to explain the fibrosing effects of asbestos. Fibrosis has been induced with a variety of fibrosis as well as nonfibrosis mineral dusts. Some investigators feel that fibers are more fibrogenic than nonfibrous particulates and that fibrogenic reaction increases with increasing fiber length. However, the role of fiber length is difficult to evaluate without simultaneously taking into account possible effects of diameter and aspect ratio (length/diameter).

On injection, chrysotile heated to 400°C caused fibrotic reaction in the pleura of mice equivalent to that of unheated chrysotile, but fibrosis production diminished as temperatures increased above 400°C (at 600°C, 800°C, 1000°C). Asbestos in dust from automobile brake linings, which are subjected both to high temperature and mechanical grinding, resembled chrysotile that had been heated to 800°C or more and then ground.[1]

Effects of asbestos that may be related to fibrogenesis have been observed *in vivo* in mammalian tissues as well as in cell culture. Biochemical changes, including stimulation of anaerobic metabolism, and physical effects such as damage to cell membranes and chromosomes have been noted.*[150-158]

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*Such observations may shed light on the mechanisms by which asbestos induces fibrosis. For example, it has been postulated that during phagocytosis, asbestos causes damage to the membranes of macrophage lysosomes, which are cell organelles that contain lytic enzymes. Subsequent intracellular release of these enzymes may injure or kill the macrophages, resulting in release of a fibrogenesis-stimulating factor.[159]



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## Chapter IV
## OCCUPATIONAL EXPOSURES

There is little recent data in the published literature on exposures to asbestos, and it is difficult to assess whether what has been published is typical. Furthermore:

- Occupational asbestos concentrations are commonly reported as optical-microscope-visible fibers per milliliter greater than 5 μm long, and these may account for only a small fraction of the total electron-microscope-visible fibers
- It is not known whether small differences in fiber counts actually reflect differences in fiber levels or, if they do, whether they indicate a change in the risk of incurring asbestos-related diseases (see Appendix C).

Nevertheless, quantitative air sampling data from representative occupations is provided in this chapter in an attempt to indicate the range of exposures found in three workplace situations: asbestos mining and milling, production and processing of asbestos products, and utilization of asbestos-containing products.

Fewer than 600 persons in the United States are employed in mining and milling asbestos.* However, industries that manufacture asbestos products or that make use of them provide jobs for millions. These industries may be categorized as primary, secondary, or consumer according to whether they produce manufactured goods from raw asbestos fiber, process asbestos manufactured products to make other products, or utilize a finished product containing asbestos without additional modification. Over 37,000 persons are employed in the manufacture of primary asbestos products; 300,000 are employed in secondary asbestos industries; and millions more work in the asbestos consumer industries--over 185,000

*Many more may be exposed to asbestos as a contaminant during the mining and milling of other minerals.



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of them in shipyards and almost 2 million in automotive sales, service, and repair.[1]

## Exposures in Mining and Milling

Sources of asbestos exposure in mining and milling include blasting, crushing, transporting, and drying ore; air-aspiration milling; and disposing of waste. Time-weighted-average (TWA) levels of fiber in mining reported in 1973 ranged from 0.5 to 2.8 optical-microscope-visible (o-m-v)* fibers per milliliter, with an average of 0.9. Much higher concentrations were found in milling, where exposures ranged from 6.0 to 12.1 fibers per milliliter.[2]

In addition to mining and milling of asbestos, the mining and milling of other mineral ores that may contain asbestos as an impurity is a potential source of asbestos exposure. For example, concentrations of asbestiform fiber in one hard-rock gold mine were reported in 1976 to average 0.25 o-m-v fibers per milliliter, ranging up to 2.8,[3] and fiber counts of 8 to 260 per milliliter were recorded in a talc mining and milling operation, according to a 1973 report.[4]

## Exposures in the Asbestos Products Industries

For all segments of the primary industry, manufacturing begins with fiber receiving and warehousing. Levels of airborne fiber in these areas have ranged from 0.2 to 2.5 o-m-v fibers per milliliter and are typically about 1.0.† Levels at the upper limit of the range reflect damaged shipments, careless unloading or ineffective housekeeping. The most important factor influencing asbestos exposure at this step of production is the condition of the bags in which asbestos is shipped.

Next, asbestos fibers are introduced into the process. Bags of asbestos are usually cut open and dumped manually, either into open hoppers or into bag opening enclosures. This activity can result in relatively high exposures if hooding is inadequate or lacking. Disposal of the emptied bags may also add to airborne fiber levels. In four of the seven major primary industries, highest TWA exposures occur in fiber introduction and have ranged from 0.3 to 10.0 o-m-v fibers per milliliter, typically somewhat above 2 fibers.

Exposures in mixing and blending depend upon how dry the materials are that are being mixed, the intensity of agitation, and the effectiveness of ventilation. Typical TWA values in the past have been

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*Greater than 5 micrometers in length.

†Data in this section are from Chapter Reference 1 (published in March 1976).



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approximately 2.2 fibers per milliliter, with a range of 0.2 to 10.0 fibers per milliliter. Sometimes asbestos is dumped directly from the bags into mixing or blending tanks, augmenting the usual exposures found at this step.

Once the asbestos fibers are engulfed by a medium that prevents them from becoming airborne, exposures drop. This may occur at the step of mixing and blending--as in the production of floor tile, paper, and cement pipe--or in a subsequent step.

Exposure levels in formulation operations have ranged from 0.2 to 22.0 fibers per milliliter, with an average level of 1.8 fibers per milliliter. This wide variety of exposures is due to the number of different processes represented by that stage.

Finishing operations vary significantly from one type of industry to another, but usually include machining (i.e., cutting, drilling, grinding) the rough product to specification. The mechanical energy imparted during machining causes asbestos fibers to break loose and become airborne. Average TWA levels of exposure in these operations have ranged from 0.1 to 8.0 fibers per milliliter, with a mean of 1.6.

In the last two production steps--inspection, and storage and shipping--asbestos exposures are usually the result of airborne dust generated by other operations, which may drift through the plant to these areas or adhere to the products themselves, becoming airborne during handling. Exposures are typically less than 1 fiber per milliliter.

Exposure levels in the major asbestos industries are summarized in Table 6 and are discussed below.[1]

## Friction Products

Friction products may contain 30% to 80% asbestos, and TWA exposures in the industry vary widely (0.1 to 15.0 o-m-v fibers per milliliter), averaging 2 fibers per milliliter for most operations. Greater concentrations may occur in preforming operations, ranging from 0.5 to 22.0 fibers per milliliter and typically about 4 fibers per milliliter. The elevated exposure levels found in these operations result from manual handling of the dry preform mix (asbestos fibers and metal reinforcing materials in an organic matrix), which is conveyed in open carts, scooped by hand, weighed, and poured into a block mold for mechanical pressing into the shape of the finished product.

Other operations that may yield high asbestos levels include fiber introduction, mixing of the dry preform, and finishing. During finishing, the products are trimmed, drilled, sanded, ground, and sawed to specification. Exposures vary with work practices and equipment. For example, exposures at ventilated radial grinders are below 2 fibers per



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Table 6

EXPOSURE TO AIRBORNE ASBESTOS IN SELECTED ASBESTOS PRODUCT MANUFACTURING INDUSTRIES

| | Asbestos Concentrations (Time-Weighted Average in Fibers/ml)[a] | | | | | Number of Employees | |
|---|---|---|---|---|---|---|---|
| | Most Operations | | Operations with Highest Levels | | | | |
| | Typical | Range | Typical | Range | Name of Operation(s) | Production Workers | Total |
| **Friction Products** | | | | | | | |
| Primary | 2 | 0.1-15.0 | 4 | 0.5-22.0 | Forming or Rolling | 4,900 | 7,300 |
| Secondary | | 2.5-6.5 | | | | | 34,500 |
| **Asbestos Paper** | | | | | | | |
| Primary | 1 | 0.75-2.7 | 2 | 0.3-2.8 | Fiber Introduction | 1,100 | 4,500 |
| Secondary | | 1.0-3.5 | | | | | 198,000 |
| **Asbestos-Reinforced Plastics** | | | | | | | |
| Primary | 1 | 0.2-2.5 | 2 | 0.5-3.0 | Fiber Introduction | 900 | 2,600 |
| Secondary | | 0.5-2.0 | | | | | 11,000 |
| **Cement Pipe** | 1.5 | 0.25-3.5 | 2 | 0.6-4.5 | Finishing | 1,600 | 2,400 |
| **Cement Sheet** | | | | | | | |
| Primary | 2 | 0.3-8.7 | 3 | 0.9-8.4 | Dry Mixing, Sanding | 600 | 1,300 |
| Secondary | | 1.0-6.0 | | | | | 24,000 |
| **Floor Tile** | 1 | 0.5-4.3 | 4 | 0.9-4.3 | Fiber Introduction | 2,900 | 6,700 |
| **Textile** | | | | | | | |
| Primary | 4 | 0.25-10 | 4 | 2.0-10 | Carding | 2,400 | 3,700 |
| Secondary | | 2.0-6.0 | | | | | 7,500 |
| **Paints, Coatings and Sealants** | 1 | 1.0-2.5 | 2.5 | 1.5-8.0 | Fiber Introduction | 350 | 3,000 |

[a] Optical-microscope-visible fibers, 5 μm long or longer.

Sources: Daley AR, Zupko AJ, Hebb JL: Technological feasibility and economic impact of OSHA proposed revision to the asbestos standard. Roy F. Weston Environmental Consultants-Designers, March 1976 (prepared for: Asbestos Information Association of North America).



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milliliter, but chamfers and backgrinders may cause exposures of 5-8 fibers per milliliter.[5] If the finished products have not been cleaned of adherent particulates, exposures may be high during inspection (4-7 fibers per milliliter).

## Asbestos Paper

Exposure levels in the asbestos paper industry vary with the asbestos content of the manufactured product, which can range from 5% to virtually 100%. Typical TWA concentrations in the primary industry average 1 o-m-v fiber per milliliter (range 1-3 fibers per milliliter) except in fiber introduction and stock preparation (wet blending), where concentrations typically average 1-3 fibers per milliliter and range up to 10. The lower concentrations are achieved in plants which use disintegrating, pulpable bags, thus obviating bag opening, dumping, and disposal.

Since papermaking is a wet process, little asbestos dust exposure is realized after fiber introduction until the product is dried. Exposure concentrations may then be elevated somewhat by the manual handling and mechanical modification (slitting, calendering, converting, etc.) needed to prepare paper sheet according to specifications.

## Asbestos-Reinforced Plastics

The asbestos content of reinforced plastic is relatively small,[1] and reported exposures in both primary and secondary industries are lower than in most other segments of the asbestos industry. TWA concentrations during most operations range from 1.0 to 2.5 o-m-v fibers per milliliter. Fiber introduction, dry blending, and handling of the blended mixture and preform are sources of moderate levels of exposure. After the preform has been remelted, the asbestos is bound tightly in the polymer matrix, reducing the potential for airborne release. Fibers may still break free, however, during finishing.

## Asbestos-Cement Pipe and Sheet

Asbestos-cement products may contain 10% to 70% asbestos. Although more asbestos is used in manufacturing asbestos-cement pipe and sheet than any other primary asbestos products, relatively few workers are employed in these branches of the industry (3,600 total, 2,200 in production).[1]

In asbestos-cement pipe factories, TWA fiber levels range from 0.5 to 4.5 o-m-v fibers per milliliter, averaging about 1.5. In the



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manufacture of asbestos-cement sheet, some fiber-introduction and dry-mixing operations may yield higher exposure levels (0.3 to 8.7 fibers per milliliter) than in the manufacture of asbestos-cement pipe, because fiber may be introduced directly into the dry mixer. Once fibers are engulfed by the cement mortar during wet mixing, there is little opportunity for them to become airborne until finishing. Higher levels (averaging above 2 fibers per milliliter) may be found during cutting and machining in cement pipe factories; and sheet trimming and sanding present the highest levels of exposure in the asbestos-cement-sheet process, generally about 2.5 and 3.0 fibers per milliliter, respectively.

### Floor Tile

Asphalt or vinyl asbestos floor tile contains 8% to 30% asbestos. Airborne TWA asbestos concentration ranges from 0.5 to 5 o-m-v fibers per milliliter. Typical concentrations are approximately 1 fiber per milliliter, except for fiber introduction, where concentrations of 4 fibers per milliliter are common. Once the asbestos is engulfed by the agglomerated plastic during the later phases of mixing, the potential for exposure is reduced significantly.

### Asbestos Textiles

Levels of exposure in primary and secondary production of asbestos textiles vary directly with the asbestos content of the manufactured products but generally are higher than in any other asbestos industry besides milling. A typical TWA concentration of airborne fiber--i.e., for most operations--in the primary textile industry is 4 o-m-v fibers per milliliter (range 0.1 to 22.3 fibers per milliliter) except in the carding operation, where the typical concentration exceeds 5 fibers per milliliter (range 6.1 to 27.3). High asbestos exposures in the asbestos textile industry result from the processing of dry--or, at best, partially damp--fibers, which are easily dispersed into the atmosphere. During carding, the vigorous manipulation of the dry fibers to separate and align them accounts for the particularly high concentrations observed at this step--even in the face of intensive efforts to achieve effective ventilation.

The liquid dispersion method of asbestos textile manufacture, in which asbestos fibers are mixed in water with chemical dispersing agents, results in much lower exposure levels (less than 1 fiber per milliliter) than in conventional plants.[6] Moreover, the use of asbestos textiles



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made from dispersed yarns results in significantly lower asbestos exposures to the user than from conventionally manufactured products.[7]

## Exposures in the Utilization of Asbestos-Containing Products

With two notable exceptions, the insulation trades and clutch and brake installation and repair, levels of exposures to workers in the consumer industries are generally very low. But although exposure levels are low, the vast majority of workers in the asbestos consumer industries, it is surmised, are less aware of the health hazards of asbestos than are workers in the production industries and may not utilize basic control methods to minimize risk.

### Insulation Trades

Exposures in the insulation trades vary widely, but they include the highest occupational exposures and control is difficult. In the early 1970's, there were approximately 36,000 insulation installers[8] employed largely in insulating industrial equipment, commercial buildings, and ships. It is difficult to obtain characteristic exposure levels for these workers due to the many different insulating materials and conditions of work.

The asbestos content of materials in use ranges from 10% to almost 100%. Asbestos substitutes are gaining in use as regulations over the use of asbestos become increasingly more restrictive.

Jobs performed by insulation workers can be classified into six categories:

- Prefabrication: materials are precut and shaped using hand or power saws either on the job or at the contractor's shop (10% of time).
- Application: Materials are fitted, hammered, or carved and attached to surfaces by wiring or gluing (40% of time). Some materials used to be sprayed applied, but this practice has been virtually eliminated in recent years.
- Finishing: Materials are coated with asbestos-containing cement, resin, asbestos or cotton cloth, or petroleum based sealer (30% of time).
- "Rip-out": Removal of old or unusable materials in the process of reinsulating (10% of time).
- Mixing: Mineral wool, asbestos, fiber glass, and cement or glue are mixed in buckets or troughs separately or in combination (5% of time).
- Miscellaneous: Cleaning up, transporting materials (5% if time).



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Percent of time at each task is highly variable, of course, and intended only as a rough guide.[9]

Highest concentrations encountered by insulation workers have occurred during "rip-out" or removal of old asbestos insulations. In a 1968 report on air samples collected on a ship during removal of sprayed asbestos coatings, removal of 100%-asbestos lagging, and subsequent cleanup were said to average 248 o-m-v fibers per milliliter, 62-159 fibers per milliliter, and 353 fibers per milliliter, respectively; in comparison, the application of pipe lagging containing 15% asbestos resulted in exposures of 5-60 fibers per milliliter, and cutting and drilling incombustible board prior to installation yielded exposures of 0.7-4.5 fibers per milliliter.[10] Levels of 30-100 o-m-v fibers per milliliter have been reported ruing application of spray asbestos insulation.[11] Nearby workers may be exposed to elevated levels of asbestos as the result of the activities of insulators--especially in shipbuilding, where work often goes on in enclosed poorly ventilated spaces.

## Brake and Clutch Repair

There are almost 2 million persons employed in automotive sales, service and repair, of whom 900,000 are said to be frequently exposed to asbestos from automotive brake and clutch repair.[1] (Note that this figure does not include persons who repair other kinds of brakes and clutches.)

Asbestos exposures were determined for specific brake servicing operations including blowing-out automobile brake drum assemblies, grinding used truck brake linings, and bevelling new truck brake linings. Average peak o-m-v asbestos air concentrations for these activities based on sampling within 10 feet of the operator were 10.5, 3.75, and 37.3 fibers per milliliter, respectively.[12]

In a similar study, mean concentrations found 3-5 feet, 5-10 feet, and 10-20 feet from an operator blowing dust out of brake drums were 16.0, 3.3, and 2.6 fibers per milliliter. Grinding truck brake shoes gave average concentrations of 4 fibers per milliter, and bevelling produced an average count of 37 fibers per milliliter. Measurable concentrations (0.1 fibers per milliliter) were found at distances up to 75 feet from the blowing-out operation (14 minutes after), 60 feet from grinding, and 30 feet from bevelling, indicating that other garage employees besides those directly involved in brake and clutch repair are at risk.[13] Another study estimated the time-weighted average exposure for brake mechanics to be 0.8 o-m-v fibers per milliliter.[14]

## Installation of Floor Tile, Roofing, and Siding

There is limited information relating to levels of exposure during installation of asphalt or vinyl asbestos floor tile. Because asbestos



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fibers are firmly imbedded in the tiles, installation per se is unlikely to be a source of important asbestos exposure. It is accepted practice, however, to sand old asphalt or vinyl tile floors before installing new covering. Conventional belt sanders with coarse grit are used to sand the tiles, and, normally, 240 to 250 square feet of tile can be sanded per hour. One report states that levels of 1.2 and 1.3 o-m-v fibers occurred during a simulation of normal sanding activities over a short-term sampling period.[15] It is likely that fiber levels fluctuate significantly depending on the age and condition of the tile being sanded, grade of sandpaper, speed of the sander, size of the workspace, ambient humidity, and quality of ventilation.

Installing asbestos roofing and siding should result in exposures of lesser magnitude since these operations are performed outside.

## Use of Spackling, Patching, and Taping Compounds

Asbestos may be a primary component of spackling, patching, and taping compounds used in wallboard construction to finish joints and repair damage, or it may be a contaminant of talc, limestone, or other rock used as raw material. Used mostly in the construction industry, the compounds are also used by persons doing their own construction and repair, and intermittent exposure to asbestos may occur during mixing, application, and sanding (finishing).

To determine possible exposure during application, air samples were collected at various jobs and sites. Peak airborne asbestos concentrations measured during such operations as hand sanding, pole sanding, mixing of dry spackle with water, and sweeping-up averaged 2.3 to 47.2 optical-microscope-visible fibers per milliliter.[16]

All exceed the current OSHA occupational standard for an 8-hour time-weighted-average (2 fibers per milliliter), and many exceed the permissible ceiling (10 fibers per milliliter).

## Wearing Asbestos Garments

Tests of wearing asbestos garments have indicated that breathing zone concentration can exceed 2 o-m-v fibers per milliliter. At one plant where hoods, coats, mittens, and leggings were worn, concentrations of airborne asbestos fibers ranged from 9.9-26.2 fibers per milliliter, and the 8-hour time-weighted-average concentration was 4.7 fibers per milliliter.[17]

Exposures from wearing fire-fighting helmets also have been measured. A new helmet with an unlined asbestos cover, an identical older helmet, and a helmet covered with aluminized asbestos cloth produced breathing zone concentrations of 2.3, 1.4 and 0.0 o-m-v fibers per milliliter, respectively.[18]



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# Chapter V
# NONOCCUPATIONAL EMISSIONS AND EXPOSURES

Persons not employed in asbestos-related occupations are exposed to asbestos fibers that originate from natural sources or from man-created sources such as the manufacture and use of asbestos products. Such asbestos may be inhaled--as, for example, in an office building in which the air is contaminated by asbestos insulation--or it may be ingested with water, food, and drugs. As would be expected, the further one gets from the occupational environment, the fewer data there are on such exposures--termed "nonoccupational" exposures. However, the available data are reviewed here to provide at least some indication of possible general environmental contamination. (See Appendix C for a discussion of the difficulties in measuring asbestos contamination.)

## Asbestos Emissions from Natural Sources

Rock that contains asbestos can be disturbed by natural means, such as weathering or landslides, or inadvertently by such human interventions as road building, construction, and tilling of the soil. In such cases, free asbestos fiber may be deposited onto soil or enter air and water,[1] thereby contributing to levels of contamination in the ambient air and in water as discussed in this chapter.

The occurrence of rock formations that could possibly contain asbestos was discussed in Chapter I of this monograph, and the geographical distribution of such rock formations in the United Stated is shown in Figure 1. If the primary areas of source rock are looked at in conjunction with high population density, the most critical areas for emissions from natural sources appear to be eastern Pennsylvania, southeastern New York, southwestern Connecticut, and greater Los Angeles and San Francisco.

## Asbestos Emissions from Human-Created Sources

Human-created sources of nonoccupational exposures to asbestos include the mining and milling of asbestos; the transportation of asbestos materials and products; the manufacture, installation, use, and demolition of asbestos products; and the disposal of wastes.

Some gross estimates of annual emissions in the United States from asbestos mining and milling, manufacturing, use of asbestos



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products, and disposal of wastes have been made and are shown in Figure 2. Although these estimates are uncertain, by at least an order of magnitude, several important conclusions are indicated:

- Asbestos is preponderantly disposed to land, least to water.
- Most of the asbestos disposed to land is consumer waste, which is more likely to be disposed to uncontrolled waste dumps and handled by persons unaware of the hazards.
- Disposal to land is an important source of atmospheric asbestos and, because of proximity to urban populations, may be even more significant to health than the emissions to air that come from mining and milling.

## Redistribution and Fate of Asbestos in the Environment

Because asbestos is exceptionally resistant to thermal and chemical degradation, it persists in the environment and can be widely redistributed by both natural forces and human means. The magnitude of this redistribution is governed by an extraordinarily complex set of factors which include the height of the emission source, the rates of air and water flow, fiber diameter, rain, thermal air inversions, electrostatic forces, agglomeration of particles, and the density of vehicular traffic on asbestos-containing landfill, to name only a few.

### Redistribution by Air

If, for example, asbestos is emitted to air as part of a "large" agglomerated particle, it will settle to earth relatively quickly and thereby have a limited potential for environmental contamination; thus, concern over the relatively large quantities of asbestos emitted to air from mines is somewhat attenuated by knowledge that the mining processes tend to produce relatively large particles. At the same time, however, an appreciable fraction of the large mass of asbestos discharged by mills is in the form of free fibers that may remain in the atmosphere for long periods of time, travel great distances, and expose many people. Studies of atmospheric pollution in the area surrounding asbestos mines and mills in Finland showed small amounts of asbestos dust as far away as 27 kilometers.[2]

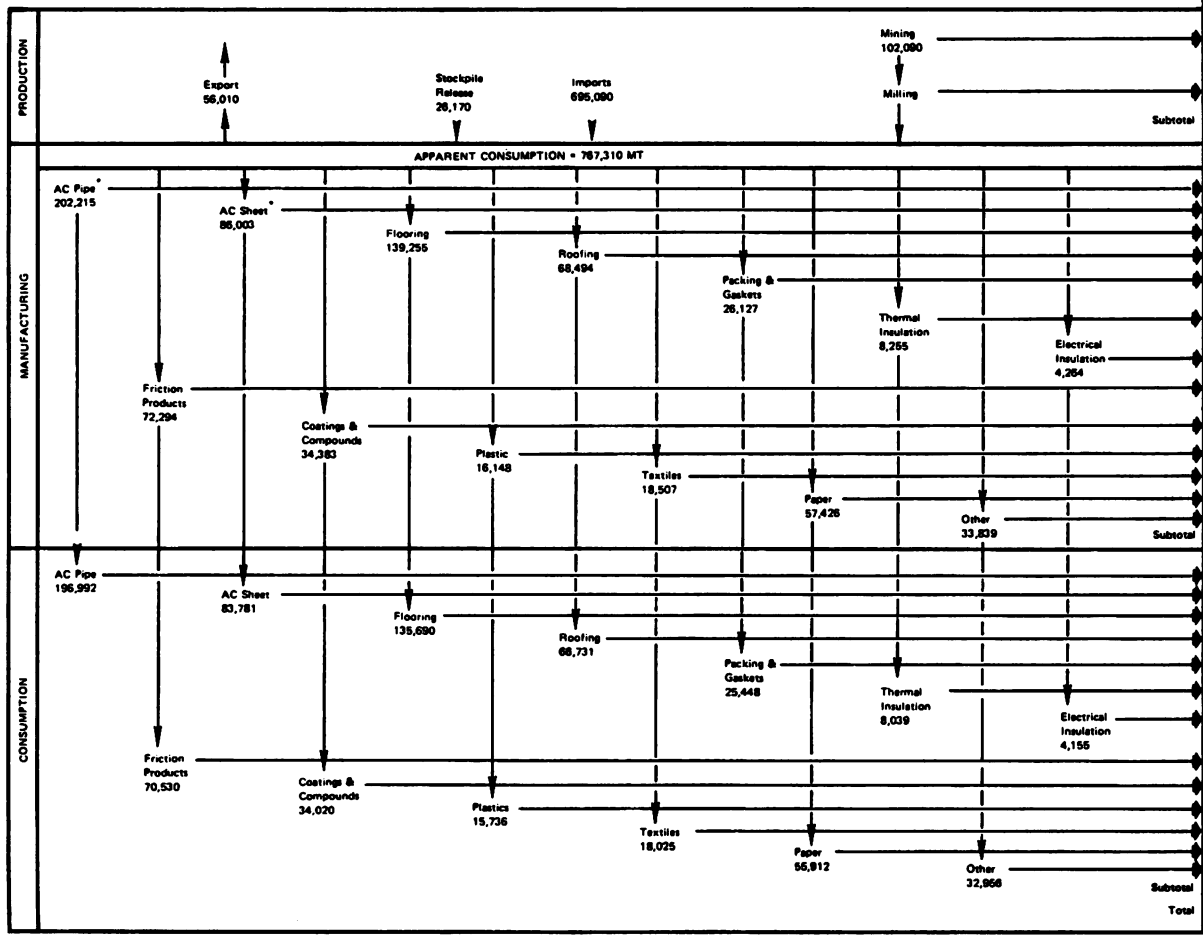
A simplified calculation of "drift distance" for two sizes of asbestos fibers was made for this monograph using the method of Cowherd[3] and a terminal settling velocity as determined according to Harris.[4] Fibers were presumed to be injected at a height of 50 feet (15.2 meters)


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ASBESTOS DISPOSALS AND EMISSIONS

| | Total | Disposed to Land[a] | Emissions To Air: From Process | Emissions To Air: From Disposal | Emissions To Air: Total | Emissions To Water |
|---|---|---|---|---|---|---|
| Mining | 6,701 | 6,313 | 373 | –[b] | 373 | 15 |
| Milling | 13,681 | 12,827 | 1,039 | –[b] | 1,039 | 15 |
| Subtotal | 20,382 | 18,940 | 1,412 | –[b] | 1,412 | 30 |
| AC Pipe | 5,223 | 5,056 | 20 | 101 | 121 | 47 |
| AC Sheet | 2,222 | 2,150 | 9 | 43 | 52 | 20 |
| Flooring | 3,585 | 3,481 | 14 | 70 | 84 | – |
| Roofing | 1,763 | 1,712 | 17 | 34 | 51 | – |
| Packing & Gaskets | 679 | 653 | 13 | 13 | 26 | – |
| Thermal Insulation | 216 | 206 | 4 | 4 | 8 | 2 |
| Electrical Insulation | 109 | 107 | – | 2 | 2 | – |
| Friction Products | 1,854 | 1,810 | 18 | 36 | 54 | – |
| Coatings & Compounds | 363 | 344 | 2 | 17 | 19 | – |
| Plastic | 412 | 404 | – | 8 | 8 | – |
| Textiles | 482 | 463 | 3 | 9 | 12 | 7 |
| Paper | 1,514 | 1,436 | 29 | 29 | 58 | 20 |
| Other | 883 | 846 | 17 | 17 | 34 | 3 |
| Subtotal | 19,295[c] | 18,667 | 146 | 383 | 529 | 99 |
| AC Pipe | 34,078 | 34,053 | 5 | 20 | 25 | •• |
| AC Sheet | 8,288 | 8,278 | 2 | 8 | 10 | – |
| Flooring | 171,684 | 171,666 | 1 | 17 | 18 | – |
| Roofing | 68,371 | 68,360 | 1 | 10 | 11 | – |
| Packing & Gaskets | 16,051 | 16,022 | – | 4 | 4 | 25 |
| Thermal Insulation | 2,294 | 2,285 | 8 | 1 | 9 | – |
| Electrical Insulation | 2,475 | 2,475 | – | – | – | – |
| Friction Products | 63,958 | 63,930 | 18 | 10 | 28 | – |
| Coatings & Compounds | 29,687 | 29,683 | – | 4 | 4 | – |
| Plastics | 14,312 | 14,309 | – | 3 | 3 | – |
| Textiles | 13,322 | 13,301 | 18 | 3 | 21 | – |
| Paper | 40,031 | 40,018 | 6 | 7 | 13 | – |
| Other | 32,328 | 32,320 | 2 | 4 | 6 | – |
| Subtotal | 494,877[d] | 494,700 | 61 | 91[d] | 152[d] | 25 |
| Total | 534,554 | 532,307 | 1,619 | 474 | 2,083 | 154 |

[a] Based on 1974 data.

[b] Asbestos cement.

[c] i.e., quantities of asbestos refuse that reach the land; emissions from disposal do occur (and are accounted for in this chart under "Emissions").

[d] All emissions considered to be "process" emissions.

[e] Subtracting the manufacturing and consumption disposals and emissions—514,172 tons—from total apparent consumption yields a figure of 253,138 tons as "net addition to asbestos products in use" for the year.

[f] Includes an estimated 15 tons emitted from incineration of solid waste.

SOURCE: SRI International

FIGURE 2 DISPOSALS AND EMISSIONS OF ASBESTOS FROM ASBESTOS PRODUCTION, MANUFACTURING, AND CONSUMPTION IN THE UNITED STATES[a] (Metric Tons)

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into a constant crosswind of 10 mph (4.5 meters/second) with no net effect of turbulence. The locality was assumed to be rural with a "roughness height" equivalent to that of a wheat field. A small fiber, one with a diameter of 0.1 micrometers and length of 10.0 micrometers, under such conditions would drift 1120 kilometers; a large fiber, 1.0 micrometer in diameter and 50 micrometers long, would drift 13.3 kilometers.

## Redistribution by Water

Asbestos borne by water can also travel considerable distances. Studies of Lake Superior, reported in 1974, indicate that asbestos particles can move several hundred miles or more.[5] Another report, made in 1976, shows that high river flows in surrounding regions have resulted in unusually high fiber counts in the Philadelphia and Atlanta water supplies.[6]

Water samples taken from wells located in areas containing asbestos rock have shown elevated concentrations of asbestos. A well at Malvern, Pennsylvania, drilled in a belt of serpentine rock, had an asbestos content of up to 0.157 micrograms per liter, in contrast with a well at Glendale, Arizona, in an area known not to have such rock, which had an asbestos content of 0.023 micrograms per liter or less.[7]

## The Ultimate Fate of Asbestos Fibers

Very little has been reported on the ultimate fate of asbestos fibers once they are released to the environment. While it is known that fibers can be readily subdivided by mechanical means into fibrils of submicron diameter, it has not been established if fibers are subdivided by natural means. It does seem likely, however, that natural forces such as erosion, grinding, abrasion, moisture, and temperature gradients would cause their eventual subdivision.

All types of asbestos resist prolonged attack by strong alkalis. However, it has long been known that hydroxyl groups of chrysotile, in contrast with other asbestos types, will react with weak acids and even water, causing magnesium and silicon to be released from the crystal lattice.[8-10] Generally, despite some degradation, it is felt that the fibrous morphology is retained.[8] Thus, to a limited extent, chrysotile may undergo decomposition through reaction with water and acid present in the environment.

Temperatures required for thermal decomposition of asbestos are seldom attained in the natural environment. With chrysotile, dehydration occurs at about 100°C, and full dehydroxylation is achieved at


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800°C.[8] Thermal decomposition of amphibole asbestos occurs at somewhat higher temperatures.

## Exposure to Airborne Asbestos

As one would expect, airborne asbestos can be found in the vicinity of asbestos mines, mills, manufacturing facilities, and waste dumps. But elevated levels of fibers also may be found near concentrations of braking vehicles, in buildings in which asbestos spray products have been used, and in cars and homes of asbestos workers who have contaminated them with dust brought from the work area on clothing, body, or equipment. Asbestos may be inhaled by persons who install their own asbestos roofing or flooring, or who repair such items as automobile brakes and clutches, home heating and plumbing systems, wires for toasters and waffle irons, or the walls of their homes.

Other situations with possible exposures to airborne asbestos include the use of roads and driveways surfaced with asbestos-bearing gravel or paving, humidifiers charged with water containing high levels of asbestos, talcum powders, paints containing asbestos, and cigarettes with asbestos filters (reportedly no longer used in U.S. cigarettes). Even powdered pâpier maché mixes, which are widely used in elementary schools, have been found to contain 50% or more asbestos.[11]

These exposures may be of a one-time or intermittent nature, but, because of the cumulative permanence of a small portion of inhaled asbestos, they contribute to a person's total risk.

Asbestos is also found as a contaminant in the ambient air, and this source is treated first in the review of exposure sources that follows.

### Exposure from Ambient Air

The large majority of the U.S. population does not live in areas that have elevated levels of atmospheric asbestos due to asbestos mining, milling, manufacturing, or construction. Nor are most people involved in part-time installation or repair of asbestos products. Ambient air, therefore, constitutes the basic source of atmospheric exposure for the population at large.

There is a paucity of atmospheric asbestos-concentration data, due, in part, to the cost and difficulty of obtaining it. Moreover: data have sometimes been reported as mass of asbestos per volume of air (most often the case)* while sometimes as concentration of fibers;

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*Appendix E contains data from several studies.



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usually only chrysotile asbestos has been measured; and the procedures for measuring, and hence the precision of the measurements, have varied widely. The Environmental Protection Agency is currently developing standardized measurement procedures, but until these are implemented and substantially more data are accumulated, atmospheric asbestos levels will be largely unknown for most geographical areas.

The scant data available for ambient levels of asbestos in rural air include a reported range of 0.01-0.1 nanograms per cubic meter,[12] and levels of 40-100 electron-microscope-visible fibers per cubic meter found in a remote area of California.[13]

Average reported concentrations of asbestos in urban air vary between 0.09 and 70 nanograms per cubic meter[14,15] and, from one study, between 0 and 2,400 electron-microscope-visible (2.4 o-m-v) fibers per cubic meter.[13] Fiber readings in one study of air at Silver Bay, Minnesota--readings that are not typical, since the locale is near a taconite milling operation--ranged up to 150,000 electron-miscroscope-visible fibers per cubic meter.[16]

## Exposures from Asbestos Mining, Milling, and Product Manufacture

Asbestos fibers are released in mining and milling during removal of overburden and preparation of ore bodies for strip and open-pit mining, as well as during drilling blasting to remove ore. Other emissions occur from ore piles and waste dumps that are exposed to wind due to disturbance by bulldozers. Drying, crushing, grinding, and screening of the ore result in the release of fibers. In this connection, the large volume of air required for air-aspiration milling (seven to ten tons of process air for every ton of fiber produced),[17] together with the length of time that asbestos fibers can remain suspended in air, generates a significant potential for emissions.

Baghouses are the predominant engineering control measure used to remove airborne dust from effluent air streams at mills and manufacturing plants. A study in 1974 showed that baghouses using specific filter materials, and when properly designed, operated, and maintained, have a collection efficiency of over 99.99% for fibers greater than 1.5 micrometers in length.[18] However, the collection efficiency for fibers less than 1.5 microns in length was approximately 98%. Since generally there are an enormous number of short fibers, a considerable quantity of fibers can be released to the atmosphere even after passage through the best available baghouses. Fibers removed that are not reintroduced into the production process must be disposed of outside the plant and may result in emissions to air.

A 1974 field survey on the air concentrations of asbestos fibers in the vicinity of an asbestos mill at Coalinga, California, showed 100 million electron-microscope-visible fibers per cubic meter within 500 meters from the mill tailings pile.[18] No data for farther distances


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from the source are reported. A 1974 EPA report showed atmospheric concentrations of 2 to 106 micrograms per cubic meter within about one kilometer of the Vermont mine-mill complex.[19] Average readings were about 30 micrograms per cubic meter. At another site, about 1.5 kilometers from the plant, the readings were .012 to 0.180 micrograms per cubic meter, with an average of about .096 micrograms per cubic meter.

In 1972, some atmospheric sampling was conducted at the asbestos mill located near King City, California.[20] One sampling station located 100 meters downwind of the source showed concentrations on the order of 100 million fibers per cubic meter, and, due to unusual wind conditions, concentrations on the order of 10 million e-m-v fibers per cubic meter were recorded at a station located 500 meters upwind from the source. Another atmospheric sampling, conducted within 3 milometers of the mill during August and November of 1974, showed concentrations of up to 1 million fibers per cubic meter downwind and 10,000 fibers per cubic meter upwind.[21] Elevated concentrations (4,500 fibers per cubic meter) were found out to the farthest station (3 km). These samples were obtained with an 0.8 micron-pore-sized Nuclepore filter, whereas the 1972 samples were collected with a Millipore filter. Hence, it would appear that the Nuclepore filter failed to collect a substantial number of fibers smaller than one micron and that the 1974 data may have underestimated the total number of fibers present. The King City mill is unique in that it uses a wet process; hence, it is believed that most of the fibers in the atmosphere come from the ore pile and tailings dump.

Dispersion of asbestos emitted to the atmosphere depends upon fiber length, topography, meteorological conditions, and the emission source itself. Wind speed and temperature stratification are important factors. As asbestos travels in the atmosphere, gravity and rain remove it from the atmosphere, and the process of agglomeration can be a significant determinant of how many fibers will be present. Because most of these factors are different at each site, detailed estimates of asbestos concentration in the atmosphere at each site require individual, specialized, calculations.

## Exposure from Transportation of Materials Containing Asbestos

Movement of asbestos ore from mine to mill in open trucks, often over roads paved with mill tailings, may contribute to the overall contamination of the environment.* However, three of five mills operating

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*The national asbestos air emission standard[22] prohibits the surfacing of roadways with wastes containing commercial asbestos or tailings from asbestos mining and milling, except for temporary roadways in the area of an ore deposit. The use of wastes that may contain noncommercial asbestos as a contaminant has not been regulated.



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in the United States are located at the mines, the remaining two are separated by short, rural, distances (32 and 55 miles).

Shipment of milled asbestos fiber, usually in bags, can result in emissions when bags are broken, but such emissions are minimized by pressure packing and unitization as described in Chapter II of this monograph. If bags are reused, either in the asbestos industry or elsewhere, they may become a source of contamination.[23] When milled asbestos is pelletized and transported in sealed railroad cars (see Chapter II) the potential for emission is no doubt reduced considerably.

Emissions of fibers could occur during the shipment of manufactured products, but they would be negligible, since most manufactured products contain asbestos tightly bound in a matrix. A more important emission source, perhaps, would be the transporting of asbestos-containing solid wastes in open vehicles through urban areas. Also, the transportation of other asbestos-bearing ores, such as talc and taconite, and their products may result in environmental emissions.

## Exposure from Asbestos Manufactured Products

Most asbestos is incorporated into finished products where the fibers are bound in a matrix (e.g., asbestos-cement pipe and sheet, flooring and roofing products, and friction products), and this reduces the possibilities for air contamination. Yet, by the application of sufficient energy, fibers may be dislodged from even tightly bound materials; automobile brake linings are an example.

Clearly, there are opportunities for human nonoccupational atmospheric exposure during installation, use, and repair of asbestos products. However, since there are so many products that use asbestos or materials that may be contaminated with asbestos, it would be next to impossible to estimate human exposure for each product type. In the paragraphs that follow, some data and information are presented for automotive friction materials and spray asbestos.

### Automotive Friction Materials

Friction materials used in automotive brake linings, disk pads, and clutch facings contain an average concentration of 50% chrysotile asbestos by weight.[24] It has been estimated that about 118 million pounds of asbestos are used annually to produce brakes in the U.S. and, assuming a 15% grinding and milling loss, approximately 103 million pounds of asbestos are actually incorporated into brakes; similarly, it has been estimated that 4.5 million pounds are incorporated annually into automotive clutch facings.[25] However, since brakes and clutches are usually repaired before they are completely worn out, and since some working automobiles are scrapped, not all this automotive asbestos


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will be released to the atmosphere. Hence, the estimate of how much actually is worn away annually is 74 million pounds, but only a small amount of which is released as fibrous material.[25]

Tests performed on brake linings have indicated that under conditions of normal usage, considerable alteration of the asbestos occurs. One study has reported that most of the dust collected from brake drums is nonfibrous and is similar in appearance to thermally degraded asbestos, and it was suggested that the temperature at the points of contact of brake linings and drum actually reaches levels at which thermal degradation of asbestos can occur.[24]

Three research studies of asbestos emissions from brake linings give estimated percentages of free fiber at 1% or less, 0.3%, and less than 0.02%, respectively.[25,26,27] In the first of these studies, it was estimated that annually in the United States there are 239,340 pounds of asbestos fiber emissions from cars, buses, and trucks and that, of this amount, 204,952 pounds drop out on the roadway; 7,655 pounds become airborne, and 26,733 pounds are retained within the brake and clutch housings.*[25] These atmospheric emissions are of greatest concern in ubran areas near traffic routes with high volumes of braking vehicles.

Electron and light microscopy were used in a recent study to analyze the number and size of asbestos fibers collected from air at four Los Angeles freeway loop sites and from upwind ambient air controls within 200 feet of the freeway.[13] Concentrations of chrysotile asbestos at the four freeway sites were low, generally in the range of 0 to 12,000 electron-microscope-visible fibers per cubic meter, and they did not differ significantly from concentrations of chrysotile in the matched upwind ambient air samples (0 to 9,000 fibers per cubic meter). Concentrations of amphibole asbestos did not differ between freeway or controls (1200 fibers per cubic meter). There was no correlation of asbestos fiber concentrations in the freeway samples with number or speed of motor vehicles passing by during the sampling periods, nor was there a correlation with wind direction or velocity.

Measurements were also made of chrysotile and amphibole asbestos at the San Francisco Bay Bridge toll plaza, and the concentrations there

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*There is apparently an error in data used in this study from "Brake Emissions: Emission Measurement from Brake and Clutch Linings from Selected Mobile Sources," March 1973 EPA (NTIS #68-04-0020). Total emissions should have been 239,340 pounds and, therefore, the other emission figures have been scaled up accordingly in this monograph.



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were found to be 1,400 electron-microscope-visible fibers of both types per cubic meter. This compared with an average San Francisco Bay Area atmospheric chrysotile concentration of 500 fibers per cubic meter.[13]

## Spray Asbestos

From 1958 through 1973, spray materials containing 10% to 30% asbestos by weight were used extensively to fireproof girders, spandrels, and decking of high-rise office buildings, and use of spray asbestos for decorative and acoustical purposes dates from the mid-1930s.[15,28] Erosion of such spray materials alone may cause asbestos fibers to enter building air, but the materials might also be damaged and dislodged--as, for example, by workmen repairing fixtures inside the space between a ceiling and the floor above. In large office buildings, air is often returned to the ventilation system through these spaces.

A recent study of public buildings in which asbestos sprays had been used showed that there were elevated levels of asbestos within the buildings, as compared with the air outside. Also, the difference between inside and outside air was greater in the case of fibrous-sprayed buildings than in the case of buildings sprayed with cementitious asbestos.[28]

Flaking of sprayed asbestos from ceilings has been reported inside schools, libraries, dormitories, and warehouses.[19,29,30,31] Air concentrations may range from 0.02 optical-microscope-visible fibers per milliliter under quiet conditions to 4.0 per milliliter during dry dusting.[31]

Prior to implementation of federal regulations on asbestos-containing fireproofing materials,* data were obtained at various building sites in lower Manhattan where such materials were being sprayed.[32] Generally, average atmospheric concentrations within one-quarter mile of a construction site were at least twice the background level. Current spray materials that contain 1% asbestos or less may be

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*Spray-on materials used to insulate or fireproof structures, pipes, and conduits must contain less than 1% asbestos on a dry-weight basis, and, for the spray application of material containing more than 1% asbestos used to insulate or fireproof machinery or equipment, no visible emissions are permitted.[22] Spray-on paints, decorative materials, and weatherproofing are not regulated.



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expected to result in no more than one-tenth of the elevated asbestos air concentrations that occurred previously.

## Exposures from Disposal of Asbestos Products and Wastes*

Solid wastes produced from the manufacture and use of asbestos-containing products and from demolition† can be emission sources, and in the past, these waste materials were often disposed of without regard to their emission potential. Moreover, their disposal may result in the mingling of asbestos-containing wastes with municipal wastes in open dumps, thus creating a long-term emission source.

Industrial asbestos wastes include process wastes such as dust, slurries, waste from overspraying, and mill tailings; waste collected by air control equipment (e.g., the dust from sawing, grinding, drilling, etc. that is vented to control devices); scrap; and emptied asbestos shipping bags. This latter item, the bags in which the milled fiber is received, is an asbestos-containing waste common to almost all manufacturers of asbestos products. If the bags are not shredded and incorporated directly into the product mix, they are incinerated or disposed to landfill, sometimes sealed in plastic bags. Occasionally, they may be treated like nonasbestos wastes and result in exposures to unknowing handlers.[23]

Water may become polluted with asbestos fibers in manufacturing, particularly in the paper and cement product industries, and during use in wet cyclones for cleaning exhaust gases from factories. The slurry waste from such processes may be directed either into settling ponds and the water recirculated (the dried waste disposed to land), or it may be dumped directly into convenient sewers, rivers, or lakes.[33] In either case, but especially in the latter, it can contribute to contamination of the environment with asbestos.

The results of a survey of waste disposal methods used by asbestos products manufacturers show that 37% of 97 plants surveyed used dumps for wastes and that 13.4% used landfills. (The remainder reuse,

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*See also Reference 19.

†For years, asbestos has been incorporated into material such as insulation, cement sheet, roofing, and floor tiles and used in constructing industrial and commercial buildings and ships. (For the most part, single-family residences contain only small amounts of asbestos insulation.) When such buildings and ships are demolished, areas of loosened asbestos, especially from insulating materials, are open to the ambient air and can emit fibers. Obviously, demolition will continue to be a source of emissions in the future, requiring control measures.



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sell, store or wet-slurry their wastes.) Of greatest concern from the standpoint of emissions are those wastes that are disposed to uncovered dumps.

Asbestos mills generate vast quantities of waste. Whereas a large manufacturing waste disposal site may have a surface area of 12,000 square meters (about 3 acres), a large mill tailings disposal site may be 400,000 square meters (about 100 acres). Mill waste may contain from less than 1% asbestos by weight, as in the case of the Vermont mill, to over 30% asbestos, in some California operations.

Obviously, there also are opportunities for asbestos emissions from the disposal of nonindustrial wastes, which constitute the majority of asbestos wastes disposed to land (Figure 3). Most notable of these wastes are those generated by the renovation and demolition of ships and buildings, which may contain large amounts of friable asbestos insulation as well as many other asbestos products.

Three studies of asbestos concentrations in air near asbestos waste dumps, all conducted prior to the establishment of EPA standards in the fall of 1975, have been published.[13,19,34] It is apparent from these studies that atmospheric asbestos concentrations in the vicinity of waste disposal sites, often in urban areas, are considerably higher than background concentrations--perhaps 10 to 1,000 times higher--and may even approach occupational levels. (The revised EPA asbestos standard that went into effect after these data were recorded may help reduce emissions.)[35]

## Exposures of Asbestos Workers' Families

Families of persons employed in the asbestos industry may be subjected to asbestos contamination that augments their exposures from other sources. Workers may bring asbestos fibers home on their skin or clothing or on equipment such as lunch boxes and automobiles. Atmospheric concentrations of asbestos in the homes of asbestos workmen have been reported to be 100-500 nanograms per cubic meter,[28] concentrations similar to estimated concentrations in the vincinity of asbestos mines and mills and much higher than the 0.09 to 70 nanograms per cubic meter reported in some U.S. cities.*[14,15]

## Exposure to Asbestos in Drinking Water

Drinking water is one of the possible routes by which humans are exposed to asbestos. Contamination of drinking water may be due partly

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*It is known that occupants of households of asbestos workers have elevated rates of asbestosis and mesothelioma.[36]



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to erosion from natural deposits of serpentine and other asbestos-containing materials found throughout the United States, as noted previously in this chapter ("Redistribution and Fate of Asbestos in the Environment"). Substantial contamination may also result from improper disposal of asbestos wastes. These wastes may be effluents that are directly discharged into water systems, or they may be released to the atmosphere or disposed of on land and subsequently join the water system.

Another potential contaminator of drinking water is the piping and pumping of municipal water distribution systems. About 200,000 miles of asbestos-cement pipes are used to carry water to U.S. consumers, and the pipes provide a source of asbestos fibers from leaching and erosion.[7,38] Gaskets and insulation used in treatment and in pumps are other possible contaminators.*[6]

The major difficulties in determining the asbestos content of water are discussed in Appendix C. Furthermore, most of the analyses that are available are for "grab" samples--samples of a few liters of water that are taken from one source at one time. The degree to which grab samples represent the characteristics of an entire municipality's water supply over location and time could be questioned--some municipalities receive their water from several sources, and seasonal and climatological variations can change the asbestos content of water.†

For this monograph, data on the concentration of asbestos in drinking water were extracted from 9 different published studies, representing 105 water supplies in the United States.[6,7,39-45] (See also Appendix E.) An effort was made to select data only for finished drinking

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*A study of asbestos concentrations in two water systems using asbestos-cement pipes revealed average increases of 0.074 and 0.004 micrograms of asbestos respectively per liter of water at the tap.[7] Laboratory tests conducted by the EPA[37] and Johns-Manville[7] on sections of asbestos-cement pipe also have shown increased concentrations of fiber in water. However, a recent (February 1978) report showed no significant release of chrysotile asbestos from asbestos-cement pipe exposed to the action of "moderately agressive" water.[38]

†In San Francisco, water from other sources is supplemented by water from a reservoir that is located in a chrysotile rock area. (Recall also the study mentioned previously that showed higher asbestos fiber counts in Philadelphia and Atlanta water supplies during periods of high river flows in the surrounding regions.) Further, a study published in 1974 found that the amount and mineralogical nature of suspended solids in the Duluth water supply is most evident when heavy rainfalls are followed by an increase in the amount of suspended solids resulting from river runoff and shore erosion.[39]



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water--some of the samples were taken from taps in the water supply system; others were taken at municipal water stations. Also, only studies in which electron-microscope measurements were made are included. The cities in the studies were not selected to be representative of U.S. cities; indeed, some were selected because asbestos was suspected to be in their drinking water.

Chrysotile fibers, amphibole fibers, or both were found to be present in 56 of the 105 water supplies. Fiber concentrations in individual samples varied between the lower limit of detection and 130 million electron-microscope-visible fibers per liter (for only two cities did the samples range over 40). Mass asbestos concentrations in individual samples varied between below-detectable and 800 micrograms per liter (for only three cities did the samples range over 60).*

## Exposure to Asbestos in Foods and Drugs

Asbestos contents of food and drugs have not been well-established to date, and the FDA has no regulations concerning the content of asbestos in foods and nonparenteral drugs.

Foods may be contaminated during their agricultural phase from asbestos in air and soil and from asbestos impurities in talc used as a pesticide vehicle. Uptake of contaminated water by plant roots, as well as deposition of contaminated water directly onto leafy surfaces by sprinkler irrigation, are possibilities, but no published literature has been found to substantiate them. One instance of accidental contamination of food in the course of transportation has been documented.[46]

Processed foods may become contaminated with asbestos either from water used in their preparation or from the use of asbestos filters, adhesives, rubber, and resins in processing and packaging.[47] Foods for which asbestos filters are used may include beer, wine, liquors, fruit juices, sugar, lard, and vegetable oil.[48] Asbestos filters have also been employed to process cider, condiments, drinking water, mouthwashes, syrups, tonics, and vinegar.[49]

The authors of one study found between 1.1 and 6.6 million electron-microscope-visible fibers per liter in U.S. and Canadian beer, and between 1.7 and 12.2 million fibers per liter in Canadian soft drinks. They also reported between 1.8 and 11.7 million fibers per liter in wines from various parts of the world.[50] In another study, between 13.1 and 24.0 million fibers per liter were found in one

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*Some authors reported their data as fibers per liter, some reported micrograms per liter, and some reported both.



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manufacturer's gin; the water used in the processing of gin contained between 3.3 and 8.7 million fibers per liter.[51]

Any estimate of the quantity of asbestos consumed with food must be purely speculative since virtually no concentration data have been reported. The FDA has reported that under test and market conditions certain foods contained less than 10 ppb asbestos.[52]

Asbestos filters also have been used in the processing of drugs and blood plasma. But since April 1975, as noted in Chapter I, the FDA has disapproved their use in preparing parenteral drugs and biologics. Asbestos filters may be used, however, in the manufacture of nonparenteral drugs and their ingredients.

Talc, which may contain asbestos as an impurity, has been used in the manufacture of medicinal capsules and tablets and has found various other uses in medicine and dentistry.[53-55] Also, talc may be added to processed foods, either directly as an ingredient or indirectly as a form release agent or as a constituent of packaging materials.[56-66]



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# Chapter VI
# CONTROL OF THE ASBESTOS HAZARD--
# PHYSICAL CONTROL

Three program approaches for controlling the adverse health effects of asbestos fibers in man's environment are presented in this and the following two chapters. These approaches are:

- Physical control of human exposure to asbestos fibers--i.e., reducing the contact between man and the fibers
- Medical surveillance--measures taken by physicians and other health-care personnel in behalf of asbestos-exposed persons
- Education--ultimately, of course, of persons exposed to asbestos fibers so that physical and medical control measures will be of maximum effectiveness, but also education of persons who are in a position to motivate those who are exposed, and other persons with responsibilities for asbestos-control measures.

Reducing the extent of contact between asbestos fibers and man can be accomplished by actions discussed in this chapter. Such actions include: (1) engineering of the materials, processes, and facilities for utilizing asbestos commercially; (2) administrative measures that relate to the persons who might be exposed; (3) improvement of work practices; (4) control of emissions and asbestos wastes; and (5) control during transportation of asbestos raw materials and products. The chapter includes a section on the application of control measures in several specific manufacturing and consumer industries.

## Engineering Measures

Control of airborne asbestos fiber by engineering methods is not greatly different from the control of other solid particulate matter having a similar aerodynamic size, though some special problems and technologies may be involved. Engineering control methods include:

- Enclosure
- Exhaust ventilation
- Isolation



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- Plant design
- Treatment of asbestos
- Substitution of alternative materials.

## Enclosure

Unless borne by wind, dust particles, even when impelled at extremely high velocities, travel only a short distance in air--a few inches at the most. This behavior is particularly true of asbestos fibers, due to their low mass and aerodynamic characteristics. Because of the short travel distance, a good start toward control of asbestos dust generated by machine operations is enclosure or hooding. However, enclosures must often have openings, to permit manual operation of a machine or so that the machine may otherwise carry out its function. Hence, there must be a continual inflow of air into the unit at sufficient velocity to prevent the escape of asbestos dust. For many operations, an intake velocity of 200 feet per minute at the face of an enclosure is adequate. Enclosures for high-velocity wheels or operations involving very large parts require special care in design.

## Exhaust Ventilation

Adequate exhaust ventilation, with negative pressure, must be provided for enclosed areas to remove airborne fibers. The design of a proper exhaust system is critical and should be accomplished by persons who are experienced in the principles of air movement and other important aspects of ventilation-system design.

Dusts for asbestos operation carry air at velocities ranging from 3,000 to 5,500 feet per minute. The lower velocities are used where relatively small concentrations of well-divided fibers must be carried, such as in the dust-control systems at textile plants. The higher velocities are used where space is restricted and where larger pieces of material must be carried, as in dust systems for machines that cut and shape brake shoes or asbestos-cement pipe. Most systems, including low-pressure penumatic conveying systems, function well at velocities of 4,000 to 4,500 feet per minute.

All parts of asbestos control systems should be maintained under negative pressure to prevent leakage of dust into the plant from loose joints and open seams, as well as to ensure adequate collection at hood faces. Another major consideration in a plant's ventilation system is the provision of make-up air--the amount of make-up air introduced should slightly exceed the amount of air exhausted. A mechanical air



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supply system, rather than "natural" room ventilation, is preferred and, in most cases, necessary to achieve the necessary ventilation performance.

Many specifications for duct contruction design may be found in Industrial Ventilation issued by the American Conference of Governmental Industrial Hygienists.[1]

Even with a ventilation system that is designed and built according to good engineering principles, periodic measurements must be made to determine that the system is adequately balanced and performing according to design. Such measurement include:

- Static pressure
- Air flow
- Supply, capture, and conveying velocities
- Fan performance.

Frequent measurements are essential, since plugging and wear can cause variations in the balance and, hence, efficiency of the system.

## Isolation

"Dirty" operations in the asbestos industry are frequently isolated to minimize human exposure to asbestos fibers. For some operations, particularly vibrating screens and bagging at asbestos mills, such engineering measures as ventilation and enclosure have not been able to reduce airborne asbestos levels below the two-fiber time-weighted-average limit, thereby necessitating isolation.

In addition to reducing fiber levels through a plant, there are other advantages in isolating asbestos-fiber-emitting operations and restricting access to them: (1) an employee working at a dusty operation is not so likely to relax his adherence to restrictive work practices if he is separated from fellow employees who are working freely at operations that present no exposure hazard; also, isolation can (2) reduce costs for local exhaust ventilation, and (3) make for more efficient housekeeping.

Unloading and storage of asbestos is another candidate for isolation because of dust from bags that are inevitably perforated by in-transit shifting or careless loading. Such bags should be repaired--or the asbestos rebagged--and vacuum-cleaned within the boxcar before being



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transferred to the warehouse. Also, of course, careful unloading is required to minimize bag breakage.

Bag-opening stations, as well as being enclosed and ventilated, should be isolated from other operations in the mixing or compounding area. The emptied bags themselves should be "isolated" by being rolled up within the opening station's ventilation hood and put into either a clean, sealed bag, a shredder, or a tube that conveys emptied bags to an isolated, enclosed, central collection point from which they are ultimately disposed of in sealed containers. (In some industries, bags can be mixed with other material and submersed into the manufacturing process.)

## Plant Design

Not often is industry afforded the opportunity of designing and constructing a plant to specifications that place such hazard control measures as isolation and dust control at the forefront of priority. As a rule, process flow efficiency, quality control, and cost factors receive higher priority. But enlightened management is aware that efficiency, product quality, and cost savings do not necessarily conflict with design for health and safety.

An ideal design for isolating a dusty work area is one in which entry can be made only through locker and clothes-change rooms. Two locker rooms, separated by a shower room should be provided--one for street clothes, the other for work clothes and protective equipment. Interposing a shower bath between the two locker rooms makes taking a shower at the end of a shift more likely. (See Figure 3.)

The contaminated change room should be under negative pressure, with the exhaust air directed to a suitable collecting system. Air flow between the two locker rooms should be toward the contaminated room. If connecting doors between change rooms and shower are self-closing and well sealed, it may be possible to use the separating shower room as an air lock.

Some other important considerations in designing for asbestos fiber control are:

- Engineering a dusty operation so that it can be handled by as few employees as possible.
- Including a protected observation area next to an isolated work area so that entry of supervisory personnel and visitors can be kept to a minimum.



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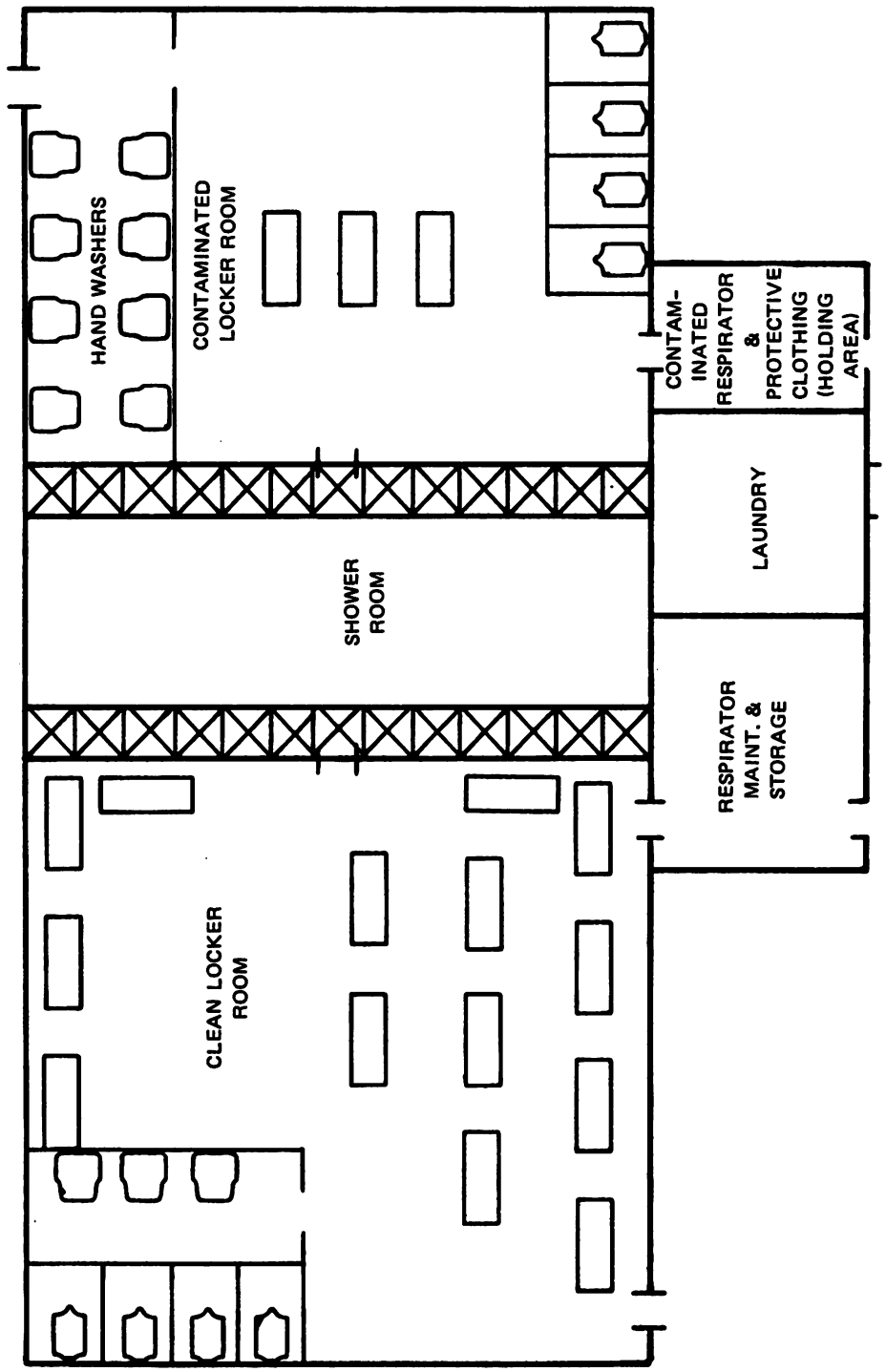



FIGURE 3 LOCKER ROOM, SHOWER ARRANGEMENT



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- Planning the layout so that airflow into hoods, enclosures, and other exhaust equipment is not disturbed by drafts from fans, windows, and doors. Off-set doors with indirect, right-angle entries help to deflect and diffuse incoming air currents.

- Constructing interiors such that beams, pipes, and ledges do not serve as areas where airborne asbestos can settle.

## Treatment of Asbestos

There are various methods of treating loose asbestos to reduce fiber emissions. One of the most effective is wetting. Although not applicable to operations that will not tolerate moisture, such as the manufacture of friction products, wetting can be used in mining and milling and in the construction industry.

At present there is one asbestos mill in the United States that utilizes wet processing. At this mill, asbestos is separated from heavier rock through a series of flotation devices. It is then collected between pillow filters and extruded and dried in pellet form, or it is fluff-dried and packaged as loose fibrous material. Dust control is needed only for pelletizing and bagging the finished product.

Use of pelletized asbestos, which can be pumped pneumatically into enclosed railroad cars and unloaded through gravity release at its destination, has been tried in the manufacture of friction products and textiles but with little success. These products require long fibers, but pelletizing breaks fibers into shorter, unusable, lengths. The shorter fiber length of pelletized asbestos should present less of a problem in the manufacture of other asbestos-containing products.

A recent method of treatment to reduce airborne fibers in the asbestos textile industry is the application of polymer to asbestos yarn.[2] Such a coating, however, is not useful where the inherent surface characteristics of asbestos fibers are required, particularly when the fibers are to be bound in a matrix with other materials.

Treatment of asbestos with anti-dusting agents may be helpful. These agents are viscous liquids that are applied to dry asbestos by spraying or mixing. The fibers are then dried at room temperature. This procedure retains the performance criteria of untreated asbestos.[3]

## Substitution

Although the use of asbestos is well entrenched in many important applications, it is likely that substitution will play some future role in reducing the health hazards from asbestos. For example, one of



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the largest asbestos users, the U.S. Navy, has issued an "Instruction" that asbestos not be used in construction, overhaul, repair, and maintenance where suitable alternative materials have been designated.

Materials that have been investigated as possible alternatives to asbestos include:

| | | |
|---|---|---|
| Fibrous glass | Steel wool | Rock wool |
| Kaolin wool | Slag wool | Exfoliated vermiculite |
| Silica | Cellulose | Potassium titanate |
| Ceramics | Sintered metals | Carbon fibers |

Few alternative materials have proved as satisfactory as asbestos due to lack of strength, heat resistance, flexibility, or durability, or because of cost. Moreover, since attention has been drawn to the possibility that inhaled fibers other than asbestos may be carcinogenic,[4] it is essential that the toxicity of proposed asbestos substitutes be evaluated.

In certain industrial processes where asbestos is used as a binder, less toxic materials have been substituted with little effect on the quality of the product. This has been the case in the manufacture of rubber, plastics, and various adhesives and cements. Similarly, less asbestos might be used in paints, coatings, caulks, sealants, and joint fillers.

Satisfactory asbestos substitutes have been developed for a variety of reinforced plastics and resins and for insulating materials. For critical insulating applications, however, the strength and heat resistance of asbestos cannot be duplicated economically.

Not many replacements have been found for asbestos in paper products in which the heat resistance, chemical inertness, and electrical and insulating properties of asbestos are highly valued--products such as mill board, roofing felts, pipe coverings, fine quality electrical and insulating papers, and asbestos-latex flooring felts. Glass cloth, felt, and thread have found limited application in roofing and flooring underlayments, but in the majority of these paper products, asbestos fibers are still used.

Two of the more successful asbestos substitutes, soda-lime-silica and high silica glass fibers, are viewed briefly below.

### Soda-Lime-Silica Glass Fibers

Soda-lime-silica glass filaments, made by highly refined processes involving the use of platinum dies, are of high quality and



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uniform size. Some are less than one-half micron in diameter and are adaptable to highly specialized uses, such as weaving into fabrics.

Glass fibers will not burn, but they will soften and coalesce at temperatures which vary according to the composition of the glass. Their heat and moisture resistance is limited by the organic film applied to them during manufacture to improve processing and to reduce breakage during subsequent plying and weaving operations. (Without such a coating, the fibers are more brittle and self-abrasive.) High-temperature properties are impaired to some extent, but the fibers will withstand temperatures up to 1,200°F.

Exposure of very fine glass fibers to water vapor results in relatively rapid deterioration, making them less resistant than asbestos to the effects of steam and moisture. Attempts to use them in place of asbestos in asbestos-cement products have been unsuccessful because of a chemical reaction, between the glass and cement, that decomposes the fibers.

Glass fibers are efficient thermal insulators in types of equipment, such as stoves and refrigerators, where conditions are not corrosive. Their high tensile strength, greater thermal stability compared with organic fibers, and electrical resistance make them suitable for electrical insulation. Glass fiber is used in conjunction with asbestos, or as an optical alternative material in Navy shipboard cable.[5]

A glass-asbestos cloth designed during World War II to extend the supply of asbestos textile fibers has continued in use as a covering on thermal insulation applied to piping on naval vessels. It is woven with a plied yarn, having one strand each of glass and asbestos.

Glass fabrics or combined glass-asbestos fabrics have some advantage over asbestos textile products because they are lighter and stronger, but they are generally less resistant to flexure, abrasion, and chemical action. Fabrics made of interwoven glass and asbestos yarns are being made in many weights and colors for use as theater curtains and fireproof draperies.

As a substitute for asbestos in friction equipment, glass fiber has, in general, given unsatisfactory results, chiefly because of the poor abrasive characteristics of glass.

## High-Silica Glass Fibers

Glass fibers approximating vitreous silica in composition are superior to soda-lime-silica glass fibers in resistance to the



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action of water vapor and high temperature. They are difficult to manufacture, however, because fused silica is extremely viscous at its melting point (1,725°F).

## Administrative Measures

Administrative measures are a complement to engineering control of the hazard in the workplace. Possible administrative measures include (a) limiting the number of employees exposed, (b) limiting the duration of exposure for any given person, (c) restricting smoking and eating in the workplace, and (d) smoking cessation programs.*

### Limiting the Number of Employees Exposed

The number of employees exposed to excessive airborne concentrations of asbestos may be limited by:

- Restricting access to contaminated areas (this measure can also involve engineering design; hence, also see "Isolation" and "Plant Design" further on in this chapter)
- Reducing to a minimum the number of persons handling asbestos
- Conducting particularly dusty operations during shifts where the number of persons in the plant is at a minimum.

Smaller numbers of continuously exposed employees are more easily and effectively trained, controlled, and protected than a larger group that is only casually and occasionally exposed.

### Limiting the Duration of Exposure for Any Given Person

As noted previously, the OSHA standard for asbestos is an 8-hour time-weighted average of no more than 2 fibers, greater than 5

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*Another administrative measure, which has been used in the British dyestuffs industry in connection with cancer control,[6] would be to give preference to job applicants who are of an advanced age and who have not previously been occupationally exposed to asbestos. The reasoning here is that the risk of developing asbestos-related cancer during the employees' lifetimes will be somewhat reduced. However, while this measure may have some basis in theory with regard to lung cancer, it does not take into account other asbestos-related diseases or even the possible aggravation of adverse health conditions not normally considered to be asbestos-related. Moreover, there are other ethical and economic considerations that would need to be explored. Still another possible administrative measure--giving preference to job applicants who do not smoke (because of the greater risk to smokers)--would appear to be subject to the same considerations.



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micrometers in length per milliliter of air; also concentrations must never exceed 10 fibers per milliliter. This means that during any single shift, employees may be exposed to airborne asbestos levels above 2 fibers per milliliter so long as such excursions are compensated for by equivalent reductions in exposure, except that in no instance can the exposure exceed 10 fibers per milliliter.

> As an example: employees on a shift can be exposed for 4 hours to an airborne asbestos level of 4 fibers per milliliter, which is equivalent to a 2-fiber exposure for 8 hours, as long as they are subject to zero exposure for the remainder of the day. The remaining four hours of the shift would be covered by employees who had received zero exposure during the first half of the 8-hour day. Or, for example, a worker could be exposed to an airborne level of 9 fibers per milliliter for 1 hour, as long as he was exposed to a level of no more than 1 fiber per milliliter for the remaining 7 hours of the shift.

Control of exposure using averaging is deficient in a number of respects since, in effect, it is assumed that: (1) exposure levels will remain constant, not fluctuate; (2) the worker receives no asbestos exposures, on or off shift, other than those associated with his work; (3) 2 fibers per milliliter greater than 5 microns in length is that level at which, for practical purposes, there is zero physiological response; and (4) the submicron fibers present but not counted are unimportant form a toxicity standpoint.* Other assumptions are that a sufficient work force is available for required alternation of personnel, and that economics (increased payroll) is not a factor.

## Restrictions on Smoking and Eating

For any toxic materials, a strong corporate stand should be established against the practice of eating, drinking, or smoking on the job. These activities should be restricted to a designated, clean location visited only after established decontamination procedures have been followed. If such action represents a change in policy, the change should be clearly and frankly explained as a step being taken to provide a safe work environment.

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*It is noted in a recent publication that asbestos fibers longer than 5 μm having a length-to-diameter ratio greater than 3 (using 430x phase-contrast light microscopy) account for approximately 2% of all asbestos fibers present in industrial settings and that, hence, every fiber greater than 5 μm in length corresponds roughly to an actual fiber count of 50.[7]



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## Smoking Cessation Programs

Because of the interaction of tobacco smoking and working around asbestos in producing lung cancer, and since smoking is a health hazard in its own right, a smoking cessation program might be undertaken as a cancer control measure. Unfortunately, however, the various programs that have been tried have rarely brought about significant long-term cessation, although smoking rates have been reduced. It is possible that recent moves of federal and state governments to restrict smoking in public places* and public-service media campaigns to educate the public on the hazards of smoking could improve the efficacy of cessation programs.

Various approaches to smoking cessation are discussed in Appendix E of this monograph with a view to providing a broad perspective of the available options. If it is decided that a smoking cessation program should be undertaken, the discussion should be of help to the health-care worker in selecting an appropriate program.

## Work Practices, Including Housekeeping and Use of Personal Protective Equipment

Changes in work practices often may be the most cost-effective way of reducing occupational exposure to asbestos. Some of the ways in which work practices may be modified include:

- Mixing asbestos mortar in closed polyethylene bags rather than in mortar boxes or buckets.
- Maintaining central fabrication shops from which insulation material is sent to the field for installation with minimal on-site cutting or sawing.
- Permitting power tools to be used only in central shops.
- Using single-point cutting and chipping tools, rather than saws or cutting equipment using abrasion.
- Jettisoning polyethylene bags into the product mix when possible.
- Substituting vacuuming for the blowing off of machines and equipment with compressed air.
- Good housekeeping (as discussed in more detail below).
- Use of personal protective equipment (also discussed below).

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*Since 1976, 19 states have passed a total of 23 antismoking ordinances that restrict smoking in public places of recreation, waiting rooms of health facilities, and restaurants.



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## Housekeeping

Good housekeeping is essential to reducing levels of airborne asbestos. Waste materials such as rejects, scrap, shavings, or other debris should be picked up and placed in plastic bags. At the end of a shift, these bags should be taped shut, labeled as to the hazard contained therein, and disposed of.

Asbestos dust on floors, ledges, equipment, overheads, and other plant surfaces can become airborne when disturbed by drafts or work activity, and it should be removed. Sweeping is not the way to remove it, however, because the fine fibers are entrained into the air and deposited on remote ledges, pipes, and other inaccessible surfaces. Nor is wet mopping a satisfactory way of cleaning, since it tends only to spread the dust around. Vacuum cleaning is the recommended method--preferably a central vacuum system.

## Personal Protective Equipment

The control measures previously discussed in this chapter can significantly reduce exposure to asbestos fibers, and they must be employed first. If, however, these measures are not sufficient, or if an unexpected event creates a potential for exposure greater than the maximum permitted, personal protective equipment will be necessary. To reiterate, respirators and protective clothing must always be available, but they should never serve as a replacement for engineering control measures.

### Respirators

Respirators may always be necessary during the cleaning or repair of exhaust ductwork or during manual shakedown of collection bags in baghouses. Also, this form of protection may be the only feasible method of controlling asbestos exposures during the removal of thermal insulation or the application of some asbestos products.

The use of respirators is not to be taken lightly, however. Since the devices place a burden on the respiration of the wearer--and, at best, are a nuisance--a determination must be made as to whether an individual can use the equipment and perform whatever work it is that he is assigned to do. Factors that must be considered in such a determination include: physiologic/physical ones such as oxygen needs for the task, hair, beards, and glasses; medical conditions that may be present such as pulmonary or cardiovascular disease; and psychological disposition toward wearing a respirator.

The type of respirator needed--for example, powered versus man-powered--will be dictated by the preceding factors as well as by the


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concentration of airborne asbestos fiber.[8] (The concentration of fiber should always be rechecked whenever there are significant changes in process, control, worksite, or climate.) Respirators require proper fitting, maintenance, and cleaning to be effective.

The elements of an acceptable respirator program are set forth by the American National Standards Institute (New York) in ANSI Standard Z88.2-1969 "American National Standards Practices for Respiratory Protection."

## Protective Clothing

Special clothing, not to be worn outside the workplace, should be worn by all asbestos workers--not only to protect them, but to curtail exposure of other persons.

The most satisfactory basic protection is afforded by coveralls, preferably made of cotton-polyester material--cotton alone cannot be used, because static build-up causes fibers to adhere to the cloth tenaciously. Disposable paper coveralls, although they are comparatively inexpensive and they eliminate the potential for exposure of laundry workers, are easily torn or perforated by body movement, chemical action, or sparks. Moreover, some types of paper suits are hard-finished and nonporous, almost airtight, and they can lead to heat stress if worn over street clothes. The coverall garment should be one-piece, without pockets, cuffs, or rolled edges, and with adequate closures for necessary openings. Coveralls should be clean each day and must never be worn away from the plant.

A head covering is also required, and lightweight paper surgical-type caps are satisfactory. Hard hats do not prevent the accumulation of fibers in the hair, and, where the hats are required, a paper cap should be worn under them.

Foot coverings, in the form of canvas booties, rubber galoshes, or safety shoes, are also needed. Either form is satisfactory as long as they are not worn away from the plant.

Street clothes and personal effects should be kept in a "clean conditions" room, and work clothes and protective equipment should be kept in a "contaminated locker" room. (See Figure VII-1.) When personnel leave a restricted work area they should enter the "contaminated locker" room and remove clinging asbestos fibers by using a vacuum equipped with a filtered exhaust system. Protective equipment should be removed


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(respirators last) and deposited in the "contaminated locker" room lockers. The worker should then shower and put on his personal outer clothing in the "clean conditions" room.

If laundering of work clothing is done by an outside laundering service, rather than in the plant, the laundry service should be advised in writing of the asbestos hazard. When collected for laundering, the clothing should be vacuum-cleaned, dampened, packed in plastic bags, sealed, and clearly marked "Asbestos Contaminated Clothing--Wet Before Handling."

## Control in Specific Manufacturing and Consuming Industries

Following are some observations on exposure control as it relates specifically to several major asbestos manufacturing and using industries and to demolition and rip-out. Generally speaking, control of asbestos exposure outside of mining, milling and manufacturing industries is difficult--persons at risk are less aware of the hazard and of proper work practices, and, moreover, engineering controls and personal protective equipment may be lacking.

### Asbestos Textile Production

Control of asbestos exposure during production of asbestos textiles has presented a greater problem than during the manufacture of other asbestos products. In part, this may be due to the use of machiner that was originally designed for processing other, less toxic, fibrous materials.

Fiber preparation--which involves fluffing, grading, beating, and combing and which generates heavy concentrations of dust--is followed by the mixing of asbestos with another material such as cotton or rayon. Dust from these operations must be controlled by enclosures and ventilation.

Carding, twisting, spinning, weaving, and braiding of fibers are dry processes which, by their physical arrangement, are very difficult to enclose and ventilate, and it appears that current technology cannot eliminate excessive exposure from these operations. Control of temperature and humidity and general room ventilation have been used to reduce exposures. It is usual practice to keep the ventilated room at a slightly positive pressure to help maintain proper temperature and humidity, and since the rate of air change is often as frequent as


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one complete change every 6 minutes, it is necessary to recycle the air before cleaning.

### Asbestos Paper

Since much of the paper-making process involves wet materials, little dust is created. Drying is usually accomplished by passing the wet sheet over steamheated rollers, which gradually remove the moisture. The low-pressure, high-exhaust-volume hoods that are used to collect the water vapor and to transport it away from the drying paper also serve to remove any asbestos dust that may be released during the drying operation.

Bulk packaging of paper products by winding them on spools, reels, or beams is a dry operation, but local exhausts and vented area hoods are effective dust-control measures for this operation.

In general, the use of hydropulpers with pulpable bags; proper ventilation rates; and the control measures mentioned previously will serve to maintain asbestos exposure in paper making at acceptable levels.

### Asbestos-Cement Pipe and Sheet

The prime point for application of control procedures in the manufacture of asbestos-cement pipe and sheet are at the mixing vat into which dry asbestos is introduced and in which the asbestos is agitated and at the final stage where the finished product is cut, machined, buffed, and packaged. (The addition of water, sand, and cement curtails dust exposure in the interim stages.) Local exhaust ventilation with carefully designed enclosures are essential for proper dust control at the critical exposure points.

### Automotive Brake and Clutch Repair

The greatest exposure to asbestos during repair of automotive brakes and clutches occurs when brake drums are cleaned by blowing them with compressed air and when brake linings are ground and beveled. Brake drums should be vacuumed instead of blown out. Grinding and beveling operations will have to be controlled by enclosure and exhaust ventilation.

### Construction

Control of exposure to asbestos fibers in building construction is difficult since few operators are sufficiently localized to permit the



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use of enclosures or exhaust ventilation. General room ventilation coupled with respiratory protection are the only means of preventing excessive exposure to fibers that may occur during necessary on-site sawing or shaping of asbestos products such as insulation; mixing and application of asbestos patching, taping, or spackling compounds; and sanding and finishing of spackling, tape, or floor tile.

Generally, the application of floor tile, roofing, and siding does not require extraordinary control procedures, since asbestos fibers are securely embedded in the product and little or no dust is created. A vacuum device installed on the periphery of a sanding wheel to evacuate dust and fibers would help maintain low levels of exposure and recharging of fibers into the air from the work area can be controlled by careful housekeeping.

Much of the exposure in the building industry has been reduced by substitituions for asbestos-containing materials, and further emphasis on using alternative materials is anticipated as awareness of the health hazard of asbestos fibers increases.

## Demolition and Rip-out of Asbestos-Containing Insulation

The potential for exposure to asbestos fibers during demolition of ships and buildings and during rip-out of asbestos thermal insulation is high. Insulating materials that contain asbestos have been used less and less in recent years but, because there is so much asbestos material already in place and because dust control during demolition and rip-out is difficult, these operations will remain potentially hazardous for years to come. The following measures will help to reduce the hazard:

- Airborne dust can be reduced considerably by soaking the insulation--nonabsorbent surfaces punctured to permit water to be introduced, and absorbent surfaces soaked by a fine, low-pressure water spray so that dust does not arise from the impingement of the water upon the surface.
- Insulation should be removed, if possible, by sawing or cutting with tools fitted with dust collecting devices rather than by tearing away.
- Materials removed should not be allowed to fall to the ground but should instead be placed in bags for disposal.
- Slurries of waste that fall must not be allowed to dry--they should be removed while still wet.
- If possible, a high-capacity exhaust system should be employed at the work site.
- Where wetting and exhaust ventilation are not possible, control efforts should be directed toward isolating the hazardous operations.



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## Control of Emissions to the General Environment

### Air Pollution Control

Methods for controlling asbestos emissions to community air are similar to those for controlling any particulate matter, with some variations due to the special characteristics of asbestos. Since operations that generate asbestos fibers are usually conducted under negative pressure, careful cleaning of the air in the ventilating system will adequately control general air pollution. However, while the air-cleaning methods mentioned below may be adequate for many asbestos-related activities, they are not practical for use in demolition or rip-out. Control of general air pollution in this case is limited to use of water sprays.

The most useful control method is fabric filtration, and design parameters for successful systems have been published.[9-12] The efficiency of fabric filter units used to collect asbestos fibers ranges from 95% to 99.9% on the basis of weight. These units operate dry, and removing the filters and packaging them for disposal can be a dusty operation, requiring the use of personal protective equipment.

Wet collectors--wet dynamic scrubbers and Venturi-type collectors--range in efficiency from 50% to 90%. The fibers collected are in a slurry and may pose a water pollution problem. Usually, the slurry is filtered and the wet fibers disposed of in a suitable container.

Mechanical collectors (cyclones) generally operate with the same efficiency range as wet scrubbers, the actual efficiency depending on the size, design, and energy expended. Although the fibers collected are dry, many of them are fractured, thereby limiting their further use. Because the fibers are dry, personal protective equipment may be required when they are removed from the collectors for disposal. Because they require no power other than to move air and they have no parts to wear out other than the collector shell,[2] mechanical collectors are economical to operate and maintain.

Electrostatic precipitation has proved to be less effective than other air-pollution-control means for asbestos fibers--yielding, at the best, 70% efficiency.

### Water Pollution Control

Until recently, little attention was directed toward the waste waters associated with asbestos manufacturing, and there is virtually no published information on such waters. The number of plants is not large, and the volumes of wastes have been relatively small. Furthermore: a significant amount of process water in manufacturing operations is recirculated, most plants have some form of waste treatment, and many plants are situated where they can discharge the process waste



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waters to municipal sewers. Recently, however, increased concern over asbestos fibers in the air has resulted in (1) conversion of some dry processes into wet ones and (2) use of water sprays to control dust from mining and from piles of tailings or gob (low-grade ore), thereby increasing the potential for water pollution.

## Waste Water Treatment Processes

The standard processes for removing suspended-solid wastes from water have been found to be satisfactory for removing asbestos fibers:

- Pretreatment--removal of oil, grease, and the larger aggregates of solid matter.
- Primary treatment--sedimentation and chlorination
- Secondary treatment--biological processes such as aerated lagoons
- Tertiary treatment[13]

Tertiary treatment--sometimes referred to as "advanced waste water treatment" or "physical-chemical treatment"--is required if the effluent from secondary treatment is not considered satisfactory.[15]

The several means of removing suspended solids in tertiary treatment including microstraining, diatomaceous earth filtration, chemical clarification, and deep-bed, granular media filtration.

In microstraining, the waste water is strained through a woven mesh screen on the surface of a rotary drum that revolves on its horizontal axis. As the drum rotates, the solids are strained out of the water and to a position from which they are removed from the drum.

Diatomaceous earth filtration is a form of mechanical separation that utilizes diatomaceous earch, a finely powdered filter-aid that is built up (coated) on a supporting medium, to trap solid material. The fibers removed are mixed in with the filter medium, and both must be disposed of.

In the chemical clarification process, chemicals such as aluminum, iron, or calcium oxides are added to the water to coagulate fine solids. Coagulation is followed by a flocculation phase, in which particulates are aggregated into larger dumps that will settle. This process if followed by sedimentation, in which the flocs that have been previously formed are allowed to settle to the bottom of a settling tank. While some of the solid material will have been removed by sedimentation, the material remaining must be removed by filtration, usually carried


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out in beds of a porous medium such as sand or coal, or a combination of media such as sand and coal, or sand, coal, and garnet.

## Control of Asbestos Fibers in Potable Water Supplies

The extent of asbestos in the nation's water supplies has not been established conclusively, and it has not been established to what extent ingested asbestos might be harmful to humans. In the meantime, it is the opinion of some researchers that oral intake of asbestos should be reduced as much as possible.[14,15]

One method of reducing asbestos concentrations in potable water involves minor modification of standard coagulation/filtration techniques as practiced in most water-treatment plants. Preliminary results show that the number of asbestos fibers could be consistently reduced to below-detectable limits (<20,000 fibers per liter). Even simple filtration systems have been shown to be partially effective and could prove useful as a low-cost interim measure in areas of high fiber concentration.[15]

The results of one study indicate that both alum and polyelectrolyte coagulation optimize fiber removal and that they can be used with sand filters. This is a definite advantage, since sand filters are used in the majority of filtration plants in North America, and Europe as well.[16]

During 1974, two diatomite-filter pilot plants operating at 10 gallons per minute removed over 80% of amphibole asbestos fiber from a drinking water source. The turbidity of the finished water was 0.05 or 0.06 FTU (Formazin Turbidity Units), with over 95% of the fiber removed. Removal of amphibole fiber seemed to be significantly better than that of chrysotile fiber.[17] The pilot plant study suggests the following conclusions applicable to the filtration of Lake Superior Water at Duluth:

- Several filter operating conditions can result in 95% to 98% removal of asbestiform fibers. Conditions providing the best filtered water would involve the use of either alum-coated Hyflow Super Cel (or equivalent grade) as body feed and precoat, or alum-coated C-512 filter aid (or equivalent grade) as precoat plus a continuous coagulant feed of Cat-Floc B polymer to the filter influent water.
- The operating data indicate that vacuum diatomite filters are significantly more expensive as a means of producing filtered water and that they would be very difficult to operate under conditions of high turbidity.



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- A least-cost-design analysis of several alternative plants suggests that a plant designed for a 20-year life and for a turbidity of 1.9 FTU equalled or exceeded only 5% of the time could produce 30,000,000 gallons of water per day at a cost of 5.56¢ per 1000 gal. It is suggested in the study that the best protection against price increases for filter-aid would occur if the filtration plant were designed for a water turbidity of about 2.5-3.5 FTU.

## Waste Disposal

The greatest hazard associated with asbestos solid waste is the potential for air emissions arising from improper handling and from improper final disposal. At each step in the handling of the solid waste material--whether the waste is to be concentrated, isolated, disposed of, reused, or otherwise treated--hazards to the waste handlers may arise. Hence, asbestos-containing wastes must be treated with the same respect accorded asbestos products and their production.

The most desirable general waste management options, in order of priority, are:[18]

- Waste reduction--by reducing the amount of asbestos used, substituting less-hazardous material, and making the process more efficient.
- Waste separation and concentration--segregating hazardous and nonhazardous wastes.
- Waste recovery--reusing the material.
- Secure ultimate disposal--disposal to landfill in a way that precludes future reentrainment.

The most important aspects of controlling asbestos solid waste are discussed in the paragraphs that follow: (a) identification, (b) separation, (c) secure transport, and (d) secure ultimate disposal.

### Identification

Obviously, there can be no control measures directed at asbestos solid waste if asbestos has not been identified as part of the solid waste stream issuing from a plant--identification and then tracking asbestos-containing materials as necessary.



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Separation*

The source of asbestos-containing waste having been identified, that waste should be separated from nonhazardous wastes, taking care to prevent exposures to workers during separation. For holding small quantities of dry asbestos wastes, the most generally satisfactory containers are heavy-gauge, impervious plastic bags. Asbestos may also be disposed of as a slurry, provided the slurry does not dry between collection and disposal.

Secure Transport

Waste must be transported to the ultimate disposal site without producing emissions. In practice, this means that community (public or private) disposal services cannot be relied upon unless they are aware of the hazard and of proper handling procedures, and unless they can provide closed conveyance to the ultimate disposal site. If waste is disposed of on the premises of a plant, employees of the facility will likely be familiar with handling precautions.

The integrity of the waste container must be maintained. Care must be taken not to rip or tear plastic bags, and more-permanent waste containers such as cans or bins must have tight-fitting lids that will not come off during transit.

Secure Ultimate Disposal

The only disposal of asbestos waste that can be considered "secure ultimate disposal" is depositing it in a site that is covered with a layer of nonasbestos-containing waste or earth that is at least 15 centimeters deep if an adequate vegetative cover is also established and maintained, or 60 centimeters deep if there is no such vegetative cover. Emissions from waste may also be controlled by (1) maintaining a resinous or petroleum-based dust-suppression cover at the site, or (2) by wetting the waste with water and sealing it in an impermeable container before disposal.[19]

Covering waste with soil and planting vegetation does not require as much care as is needed for maintaining a disposal site with dust-suppression agents; hence, it is the more desirable control method in most cases.

Control During Transportation

The transportation of asbestos ore from mine to mill is generally not a significant source of airborne asbestos fibers, although private

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*See Chapter IV, Occupational Exposure, for additional details and applications.



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mine-mill roads may be paved with tailings from which fibers can be liberated when trucks pass by. Some of these emissions can be reduced by tarring, sprinkling, or treating the roads with dust-suppressing chemicals. EPA regulations permit no visible emissions from mine or mill roadbeds. Manufactured asbestos products not firmly embedded in a matrix,* such as asbestos textiles or spray asbestos materials, should be either:

- Transported and stored in enclosed, impermeable, sealed areas
- Wetted and covered with tarpaulin or similar material to prevent drying
- Appropriately packaged--packaging that is leakproof and durable enough to withstand abrasion or puncture during normal handling, and that bears easily visible labels that warn persons about the hazard.

Areas used to transport or store asbestos not appropriately packaged should not be used at the same time to transport or store other goods. Such areas should be posted with signs warning of the asbestos hazard. Before the areas are used to transport or store goods, accumulations of asbestos fiber should be removed thoroughly, preferably with a vacuum cleaner having a high-efficiency filter.

Personnel working in or near areas used to transport or store regulated asbestos that is not appropriately packaged should follow the protective procedures outlined earlier in this chapter.

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*Manufactured products containing asbestos firmly embedded in a matrix--asbestos cement, asphalt, plastics, and the like--may not reasonably be expected to result in asbestos air concentrations that are high enough to warrant packaging.



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# Chapter VII
# CONTROL OF THE ASBESTOS HAZARD--
# MEDICAL MANAGEMENT

A means of limiting, in the industrial population, diseases that can be somehow related to asbestos is to recognize during a preemployment examination persons who have increased risk for these diseases by virtue of other attributes they possess and to recommend that they not be hired for jobs involving asbestos exposure. Those workers necessarily exposed to asbestos should be enrolled in a medical monitoring program. In this way, hopefully, asbestos-related diseases, disorders predisposing to these diseases, and conditions likely to be aggravated by asbestos exposure can be detected early enough so that removal from exposure or medical intervention may successfully limit their course. Nevertheless, asbestosis may progress even after removal from asbestos exposure, and screening programs for the early detection and treatment of lung cancer have not yet proved to be more than marginally beneficial.

## Composition of the Workforce

The extent to which discovering and not hiring certain high-risk persons for jobs involving asbestos exposure can be put into effect in any particular situation will be limited, inevitably, by social and political considerations. Each industrial physician must develop his own program for dealing with these matters in relation to preemployment examination policy.

The desired net effect of the employment screening approach is to assemble and maintain a workforce whose combined average risk of developing disease, with exposure to asbestos, is less than what it would be were it to include persons whose individual risks, even without occupational asbestos exposure, are relatively higher. The approach would be expected to be most effective in relation to those diseases for which evidence of a causal role of asbestos is most convincing and the magnitude of the problem is greatest--lung cancer, mesothelioma, and asbestosis. Summarized below are some considerations for applying control



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over the industrially exposed population by excluding from employment certain individuals on medical grounds.

## Lung Cancer

Lung cancer is the most frequent of cancers related to asbestos exposure and today exacts the heaviest mortality of any asbestos-related disease. Several risk factors for lung cancer have been identified:

- Cigarette smoking greatly increases the mortality from lung cancer, and, based on present knowledge, it is the most important single risk factor.
- A history of lung cancer in a first-degree relative--parents, siblings, offspring--has been shown to confer almost the same magnitude of risk of lung cancer for the general population as cigarette smoking, and, together, both factors are synergistic.[1]
- Prior occupational exposure to carcinogens affecting the lung also may reasonably be expected to enhance the risk of lung cancer in an asbestos worker. Such carcinogens include arsenic; chloromethyl ethers; chromium; coal tar, petroleum, and by-products; creosote; mustard gas; nickel; radium; and uranium.[2]
- Nonmalignant respiratory disease may increase the risk of lung cancer. Pulmonary fibrosis has been noted in association with lung cancer in persons with scleroderma[3,4] and certain rare hereditary diseases of the lung such as fibrocystic pulmonary dysplasia[5,6] and congenital cystic disease of the lung.[7] Several reports have associated an excess risk of lung cancer with chronic bronchitis, after taking into account differences in smoking habits.[8-11] However, such studies have often employed broad smoking categories and have neglected such important variables as duration of smoking and extent of inhalation.[12] Perhaps most convincing was the finding of a substantial excess mortality attributed to nonmalignant respiratory diseases among nonsmoking blood relatives of lung cancer cases that was not present among case spouses.[13] And significantly higher rates of impaired forced expiration have recently been found among never-smoking relatives of patients with lung cancer or chronic obstructive pulmonary disease when compared with neighborhood controls.[14] Experiments with animals indicate that individuals with asbestosis may be at increased risk of lung cancer, regardless of level and duration of asbestos exposure.[15]

Possible laboratory measures of lung-cancer risk include inducible levels of aryl hydrocarbon hydroxylase and the condition of exfoliated cells in sputum. Aryl hydrocarbon hydroxylase (AHH) is an inducible



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enzyme thought to be responsible for converting hydrocarbon carcinogens such as are found in tobacco smoke to an active form. One team of investigators has reported that levels of inducible AHH activity in human tissues appear to be genetically regulated and that patients with bronchogenic carcinoma have higher levels of inducible activity than controls.[16,17] Inducible enzyme levels, therefore, might indicate among cigarette smokers those individuals at greatest risk of developing lung cancer. At present, however, there are numerous difficulties with the assay technique even in the most experienced laboratories, and the method is not ready for general use.[18] Furthermore, the association between levels of inducible AHH activity and cancer has not yet been firmly established.[19]

Examination of exfoliated cells found in sputum has been used to assess the state of the human tracheobronchial tree. Cancer has been observed to develop after a series of gradual cytological changes occurring over several years. These changes have been categorized into stages of regular squamous cell metaplasia, various degrees of a typical squamous cell metaplasia, carcinoma in situ, and invasive carcinoma.[20] The more severe the cellular changes, the greater is the likelihood of developing lung cancer.[21] Cytological examination of sputum can be used to complement the use of risk factors listed above since, at any one time, the condition of exfoliated cells must reflect the interrelated effects of all operating risk factors, age, and latency. A great limitation to the widespread use of this technique at present is the paucity of laboratories capable of accurate cytologic interpretation of sputum samples.[22]

Persons with any of the risk factors for lung cancer or whose sputum examination reveals cytopathology of severity equal to or greater than moderate atypical squamous cell metaplasia would best not be hired for jobs involving asbestos exposure.*

## Mesothelioma

Little is known about susceptibility to mesothelioma, an important cause of mortality in asbestos workers. Persons who have had an

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*The evidence that asbestos is causally related to laryngeal cancer is highly suggestive, but not as overwhelming as it is for lung cancer and mesothelioma. Therefore, the exclusion from employment of individuals with other risk factors for this disease would probably be less beneficial. It should be noted, however, that except for family history, the risk factors identified for lung cancer generally apply to cancer of the larynx. Risk of laryngeal cancer has, in addition, been correlated with alcohol consumption.[23-25] This cancer is rare, and most susceptible persons will have been screened from employment in efforts to exclude persons with elevated risk of lung cancer.



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asbestos-related pleural effusion may be at great risk,[26] and further asbestos exposure should be prevented.

### Asbestosis

Exclusion from employment of those persons most susceptible to disease would likely prove more efficient for asbestosis, for which exposure to asbestos is a necessary factor, than for lung cancer or mesothelioma, for which it is not. However, despite suggested effects of cigarette smoking,[27] immunological response,[28,29] and specific HL-A antigens,[30] risk factors for asbestosis have not been established,[31] and there is to date no method for identifying from medical testing or questioning those individuals who are more susceptible to asbestosis.

Because of the potential adverse effects of further respiratory disability from asbestosis in individuals with existing chronic respiratory disease, it would be prudent not to hire such persons for jobs involving asbestos exposure.

## Early Detection and Treatment of Asbestos-Related Diseases

In general, treatment of asbestos-related diseases, even when detected early, is far from satisfactory. Cure is rarely possible, and oftentimes death or severe disability supervenes despite the best of efforts. Furthermore, programs for early detection and treatment run the risk of instilling a false sense of security, which may detract from more primary efforts to prevent disease by controlling exposures. This must be kept in mind during the subsequent discussion. A suggested protocol for preemployment and follow-up medical examinations of asbestos workers is given in Table 7.

### Lung Cancer

Screening programs which rely on roetgenograms and symptom questionnaires at intervals of six months have been notably unsuccessful in improving the changes of survival from lung cancer.[32, 33] The Philadelphia Pulmonary Neoplasm Research Project reported a 5-year survival rate of only 12% of individuals whose tumors were detected within 6 months of a negative roentgenogram, as against 4% in those whose tumors were detected more than 6 months afterward.

A semiannual screening program conducted among residents of Veterans Administration domiciliaries and consisting of stereoroentgen films, questionnaires, and sputum cytology slides reported a 3-year postoperative survival of only 12%.[21] This study documented a considerable



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Table 7

MEDICAL EXAMINATIONS FOR ASBESTOS-EXPOSED WORKERS

Preemployment

Questionnaire: medical history, family history, history of smoking* and consumption of alcoholic beverages, occupational history

Physical Examination: concentrating on the oral cavity, chest, and abdomen and including a digital examination of the rectum

Spirometry: including measurements of vital capacity, forced vital capacity, and forced expiratory volume at one second

Chest X-ray: posteroanterior and lateral views (14 x 17 inches)

Sputum Cytology

Follow Up

Nonsmokers, Ex-Smokers, and Smokers Who do not Inhale

- No More Than Mild Atypical Sputum Cytopathology: a yearly questionnaire, spirometry, chest X-ray, and sputum cytology
- More Than Mild Atypical Sputum Cytopathology: a yearly questionnaire and spirometry; chest X-ray and sputum cytology every 4 months
- 40 Years Old and Older, At Least 20 Years from Onset of Asbestos Exposure: add fecal occult-blood testing and an examination of the oral cavity every 6 months

Smokers Who Inhale

- Less than 15 years from Onset of Asbestos Exposure:
  -No more than mild atypical sputum cytopathology--a yearly questionnaire, spirometry, chest X-ray, and sputum cytology
  -More than mild atypical sputum cytopathology--a yearly questionnaire and spirometry, chest X-ray and sputum cytology every 4 months
- 15-20 Years from Onset of Asbestos Exposure:
  -No more than mild atypical sputum cytopathology--a yearly questionnaire and spirometry; chest X-ray and sputum cytology every 6 months
  -More than mild atypical sputum cytopathology--a yearly questionnaire and spirometry; chest X-ray and sputum cytology every 4 months
- More Than 20 Years from Onset of Asbestos Exposure:
  -Less than 40 years old--a yearly questionnaire and spirometry; chest X-ray and sputum cytology every 4 months
  -40 years old and older--add fecal occult-blood testing and an examination of the oral cavity every 6 months.

*Since smoking is such an important risk factor, breath should be sniffed for tobacco odor; and in situations where the reliability of smoking histories is in doubt, levels of expired air carbon monoxide or serum thiocyanate may be used to distinguish cigarette smokers from nonsmokers.[53]

Sources: Protocol modified from the Mt. Sinai School of Medicine, Environmental Sciences Laboratory Pulmonary Surveillance Program for Asbestos Exposed Workers.



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amount of inter- and intra-observer variability in the interpretation of sputum smears and chest X-ray films, but noted that the addition of positive and suspect sputum cytopathology increased the sensitivity of the screening method by about 50% without significantly compromising its specificity.[21,34,35]

Currently, large detection and follow-up programs are under way at the Mayo Foundation, Johns Hopkins University, and Memorial Sloan-Kettering Cancer Center to evaluate the efficacy of sputum cytologic examinations, chest X-rays, and questionnaires administered every four months. At the Mayo Lung Project each chest X-ray is reviewed by three physicians individually; and check roentgenography for patients having follow-up examinations at the Mayo Clinic itself consists of poster-oanterior (PA) stereoroentgenograms and 350-kV PA views, as opposed to conventional PA and lateral films.[36,37]

In the presence of normal chest X-rays, if a single sputum speci-ment contains frankly cancerous cells or if repeated specimens from the same individual contain markedly atypical cells, procedures to localize a tumor are set into motion. Detailed radiologic and radio-isotope studies are undertaken, and a thorough otolaryngologic examina-tion is made to rule out cancer of the upper respiratory tract. If localization is not achieved, a meticulous endoscopic investigation follows utilizing fiberoptic bronchoscopy, succeeded if necessary by bronchographic studies.[36]

Initial results from the Mayo Lung Project suggest that no more than a third of detected cases of lung cancer may be expected to sur-vive five years or more. However, more observation is needed in order to determine actual rates of survival. Roentgenographically occult tumors, which are likely to be centrally placed, were generally smaller and had a better postoperative prognosis. Most newly diagnosed lung cancers (64%) were detectable by chest X-ray alone, and only 13% of cancers detected after an initially negative screen were first noted as the result of clinical symptoms.[36,37]

Whatever the outcome of the current detection and follow-up pro-grams, certain limitations will apply:

- Some individuals should be ineligible for screening because of inability to tolerate pulmonary resection or unwillingness to undergo operation if it becomes necessary.[38-40] To screen such persons not only would be wasteful, but also might ultimately contribute to lowering the morale of other par-ticipants in the screening program--that is, whether or not the death of a screening participant was due to inability or unwillingness to be operated on, it might be regarded by others as a failure of the program.



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- A considerable number of persons may discontinue participation in the screening program because of retirement, termination of employment, or other reasons. Since the incidence of lung cancer increases with age[21] and has been found in one study to be higher among screening dropouts,[38] there is reason to believe that persons who discontinue participation in a screening program may in fact be at greater risk.
- A certain proportion of screening examinations will be found to be incomplete or technically unsatisfactory.[21]
- Once a cancer has been detected, there will inevitably be a delay until localization and operative resection. The greater this delay, the less the potential benefit that can accrue from the screening program.
- It will be difficult to extrapolate from results obtained at premier centers of medical care to likely results in an average industry program. (The difficulty in finding laboratories capable of accurate sputum cytologic diagnosis has already been mentioned.)
- The considerable financial expense of a screening program and the drain on available time of medical care personnel should not be overlooked.
- Despite screening, some cancers will not be detected early; and despite early detection, certain cancers will be inoperable or have a poor prognosis.
- A not-inconsiderable percentage of persons who will have been successfully operated on to remove a lung cancer will develop a second tumor.[36]

Despite these limitations, medical screening remains the only means of assisting the unfortunate individuals destined to develop asbestos-related diseases and should not be abandoned. The use of sputum cytology to distinguish persons of heightened susceptibility to lung cancer for removal from asbestos exposure or for motivation to quit smoking deserves to be tested. Screening examinations remind the workers of the health hazards of his job and may be used to enlist his cooperation in improving work practices as well as in changing detrimental personal habits.

It is suggested that programs for early detection of lung cancer in asbestos workers consist of periodic sputum cytologic examinations, chest X-rays, and symptom questionnaires administered according to a schedule which varies with age, risk of lung cancer, and time elapsed since first exposure to asbestos (see Table 7). Available medical facilities that are competent in localizing and resecting lung cancer should be identified in advance of the program to minimize time taken from detection of cancer to operation. Emphasis must be placed on proper check roentgenography, since chest X-rays alone should detect



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the majority of lung tumors;[21,37] sputum cytology is more effective in detecting centrally placed tumors[36] whereas asbestos workers may more likely develop peripheral lung cancers (adenocarcinomas).[41] Each chest film must be read independently by more than one physician especially trained to detect early lung cancer and qualified in the ILO/U/C classification of radiographs of pneumoconioses.[42]

## Laryngeal Cancer

Asbestos workers with clinical symptoms of hoarseness or pain or soreness of the throat should be referred to an ear, nose, and throat specialist for a detailed otolaryngologic examination of the upper respiratory tract.

## Mesothelioma

At present, mesotheliomas are uniformly fatal. Neither radical surgery, radiation, nor chemotherapy prolongs survival; in fact, these modes of treatment may be harmful. Since no useful therapy is available, screening for early detection beyond what may be done to detect lung cancer is of no clinical value. Invasive diagnostic procedures should be kept to a minimum and management restricted to the relief of pain and breathlessness.[43,44]

## Cancers of the Alimentary Tract

Fecal occult-blood testing has been used an an annual screening device to detect colorectal cancer in asymptomatic men and women 40 years old and older.[45] Positive tests have been obtained from persons with cancer of the stomach, small intestine, colon and rectum, and, to a lesser extent, from persons with benign gastrointestinal lesions.[46*]

Persons with positive results should be referred to a gastroenterologist for further studies, which may include endoscopy, cytology, biopsy, and radiology. Early detection and excision of a colorectal and gastric cancer, it has been noted, may result in 5-year survival rates as high as 90%, compared with overall national averages of 40% and 10%, respectively.[49,50]

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*False positives can be reduced considerably by using guaiac-impregnated filter paper slides in conjunction with a diet high in residue (to stimulate bleeding from existing lesions) and free of red meat and high peroxidase foods (e.g., horseradish and beets). Vitamins and aspirin-containing medications should also be avoided.[45,47,48]



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## Asbestosis

Periodic comparative chest X-rays and pulmonary function tests (see Table 7) will improve the chances of detecting early asbestosis. Many abnormalities, however, are nonspecific, and it will be difficult to determine if these reflect early asbestosis or are merely related to smoking or aging.[51] Pleural thickening or plaques in an asbestos worker must always be suspected as evidence of a biological effect related to inhaled asbestos.[52] Persons with early asbestosis or with pleural thickening should be removed from asbestos exposure and referred to a chest physician for careful follow up.



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## Chapter VIII
## CONTROL OF THE ASBESTOS HAZARD--EDUCATION

Asbestos was the subject of the first occupational safety and health standard issued by the Department of Labor, following passage of the OSHA Act of 1970. But although the standard has been amended since (to change the allowable airborne exposure concentration), no specific directive has been included regarding education or training of supervisors or employees.

However, in standards subsequently promulgated for a number of carcinogens--including vinyl chloride, the subject of another control monograph by the National Cancer Institute*--there are particular requirements specified for employee training and indoctrination in, for example, the following: nature of the carcinogenic hazard, nature of the operation involving the carcinogenic agent; recognition of conditions that may release the agent; purpose and application of decontamination practices; emergency practices and procedures and the employee's specific role in them; and purpose and application of the medical surveillance program.[1] It would appear that a comparable mandate should exist for work with asbestos.

### Goals of Education

Implicit in the fundamental goal of controlling the human carcinogenic hazard from asbestos are several goals of education--increased knowledge of:

- Work processes involving asbestos and the potential for fiber emissions
- The physical characteristics of asbestos, so as to understand its dispersion and potential for inhalation
- Diseases that may result from exposure to asbestos fibers and how these diseases are manifested

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*The reader is advised to read the section on "Educational Control" in the cancer control monograph Vinyl Chloride, since some of the information presented there complements the material presented here with regard to the asbestos-control situation. Among the topics discussed there are "Understanding 'Risk;'" review of a National Academy of Sciences study on informing workers and employers about occupational cancer; and an outline of an on-going vinyl chloride education program that embodies some of the approaches discussed here with regard to asbestos.



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- The concept of "risk"
- Reasons for, and methods of, environmental monitoring
- The purpose and nature of engineering and work practice control methods as discussed in the previous chapter (exhaust ventilation, personal protective devices and clothing, personal hygiene, etc.)
- The elements of medical surveillance and the reasons for it
- The role of related factors in disease production, such as smoking.

## Modes of Education--The Written and the Spoken Word

Of the two modalities employed in delivering health messages, the written word and the spoken word, the written word is far less effective. It lacks the elements of concern, warmth, dedication, and personal interest of the instructor, and there is no opportunity for question and discussion. Furthermore, the efficacy of the written word depends on the reading skills of the reader. However, if one must reach hundreds of employees at once the written word might have to be used but, even then, it should be as a reinforcement to the oral mode of communication.

One good opportunity for oral communication is when the physician and an employee review the results of the employee's periodic medical examination. Attention is then focused on one individual--one who is, at the same time, full of foreboding and apprehensiveness, nurtured by misunderstanding, folk beliefs, inadequate information, and rumor. The physician at this time can clarify medical terms, give meaning to test results, and suggest changes in work habits and life styles.

Most oral communication will take place at group education sessions. In some employment jurisdictions, such sessions may be required by labor-management contracts or by law.[2] The subjects for presentation may be mandated; selected solely by the occupational health staff; or, perhaps most effective, chosen through joint consultation of health personnel with department heads, plant manager, and trade union officials.

Education must go beyond a pro forma attempt to meet mandated requirements. As recommended by a special committee of the National Research Council,[2] provision must be made for the worker to acquire more information than is provided by the "package" of educational material. He, the worker, must be assured that his questions will be answered, if not during the information session, then later by telephone,



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letter, or consultation with a member of the occupational health staff or someone equally knowledgeable.

An excellent forum for group health education is during the orientation of new employees. The occupational health staff should use the occasion to discuss the purposes of the preplacement medical examination recently completed, to describe available health services, and to explain engineering controls, work practices, and use of personal protective equipment. Pursuit of a health problem in the future will be facilitated by this initial introduction.

## The Educators

Persons from a variety of backgrounds may be involved in the education effort--physcians, nurses, health educators, industrial hygienists, safety specialists, and others.

### Physicians

Physicians that teach usually do so in institutions of higher learning, where they can remain comfortable with a technical lexicon and oral shorthand. It takes greater effort and more time to describe medical disorders and risk factors to a layman in terms he can understand, yet this must be the charge of the physician, particularly the specialists in occupational medicine pulmonary disease.

Pulmonary disease specialists, in particular, as they become more knowledgeable about asbestos-related diseases, must inform their colleagues in the medical community of diseases. For in spite of a growing body of pertinent literature, many physicians remain uninformed about, and unsuspecting of, asbestos-related diseases. Many chest physicians are members of lung associations, and such membership can provide many opportunities for the education of fellow medical practitioners as well as lay community leaders. Also, when necessary, the pulmonary disease specialist could assist the occupational physician in educational programs for industry.

### Nurses

Health education has long been recognized as a primary function of the occupational health nurse and an area in which she can make a considerable contribution. The nurse often has a closer relationship with the employee than does the physician and therefore may have greater influence on changing the employee-patient's behavior. Whereas the advice of the medical director might be interpreted as the biased word



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of management, a skilled nurse utilizes every patient visit as an opportunity for presenting material relevant to health behavior.

### Health Educators (Communication Specialists)

Professionally trained health communicators may be found in the larger occupational health programs of industry. As full-time personnel in the medical facility, they have been able to learn the mission of the organization, to become acquainted with work processes, to identify problems or concerns peculiar to the work population, and to absorb the "shop patois." This background puts the health educator in an excellent position to communicate successfully with the employee.

### Industrial Hygienists

The industrial hygienist is an integral part of the occupational health staff. Whereas the occupational physician has expertise in the recognition, evaluation, and control of diseases relating to environmental hazards in the workplace, the industrial hygienist is trained to recognize, evaluate, and control the environmental hazards themselves. One aspect of this responsibility is the education of the working community. The industrial hygienist informs workers about measures to control physical hazards and motivates them to do what they can to minimize personal exposure and to assist in improving the work environment.

### Union Health and Safety Specialists

In recent years, a small but increasing number of trade unions have appointed full-time staff persons in the area of occupational safety and health. These individuals may be extremely influential, because they know the issues and supporting data well and can communicate information to their locals by means of periodic letters, memoranda, newsletters, or reprinted presentations.[12]

### Industrial Safety and Other Training Specialists

Ever since the birth of the safety movement in the United States, specialists in accident prevention have conducted educational programs for employees. Although most of these efforts have related to physical trauma, some slight redirection could channel them toward the prevention of asbestos-caused disease.

In addition to training in safety, there may be company training programs in other subject matter, from management skills to technical



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craft apprenticeship information. Training specialists who conduct such programs might also assist in health-hazard education.

### Science/Medical Writers

Medical writers on the staffs of large daily newspapers and other periodicals might be asked to a plant to be made familiar with the content of a medical surveillance program, to be apprised of health survey results, and to be updated on general occupational health activities. If a good working relationship evolves, chances will be improved that reports are in-depth and accurate, and the result could be yet another avenue for education of the worker.

## Target Groups for Education

As indicated, there are many target groups within industry for whom instruction in prevention of asbestos disease is needed. The language in the OSHA occupational safety and health act of 1970 direct training toward both the employer and the employed and, in addition, there are trade union officials, retirees and other former workers, workers' families, and miscellaneous persons in the community at large.

### Managerial and Supervisory Personnel

Managers should be as familiar, or even more familiar, with the risks of the materials they ask their workers to handle and with preventive measures, medical surveillance, and the other areas of knowledge listed at the beginning of this chapter under "Goals of Education."

There is often an unusual climate in which to educate managers about asbestos-related disease. That is, a number of executives and line managers have risen from the ranks, so that at the inception of their careers they, themselves, worked with asbestos fiber. Hence, any occupational health procedure draws special attention because such managers know of the latency period before manifestation of disease. Their concern is as great as that of newly employed mechanics' helpers, while, at the same time, their literacy level is probably higher. However, while they may know that the fiber is hazardous, they might not be as familiar with aspects of the problem as they should be, and they should receive as much medical and industrial hygiene information as they can absorb.

Because he is at the level of management closest to the worker, a well-informed supervisor should be particularly effective in


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modifying the behavior of workers since he will be in a position to motivate rather than commanding or invoking the sanction of regulations.

## Workers in the Asbestos, as Well as Other, Trades

While workers in the asbestos trades are obvious targets of educational efforts, there are other workers who may be exposed to asbestos and who, therefore, should be informed of the asbestos hazard and how to minimize it. These workers include those who dismantle ships and buildings, welders, painters, electricians, carpenters, marine machinists, shipfitters, machinists, and automotive brake and clutch repairmen.

## Retirees and Other Former Workers

Because of the long delay in manifestation of asbestos-related disease, it is necessary to maintain contact with retirees and with persons who have left work with asbestos to obtain employment elsewhere. The asbestos worker cannot be permitted to depart with the misapprehension that if he no longer has contact with that material he is no longer at risk of development asbestos-related diseases.

Exit interviews provide an opportunity to emphasize the need for life-time contact and to stress the need for continued medical surveillance. Wherever possible, former asbestos workers should be included in plant programs of health education, smoking cessation, and medical surveillance.

## Workers' Families

In addition to learning about asbestos-related disease, the families of asbestos workers should be informed about the importance of smoking cessation and about the potential contamination by fibers brought home on clothing, equipment, lunchboxes, and automobiles, and in the form of souvenir ore samples. Also, it has been observed that wives who visit with their employee-spouses at the plant physician's office often ask more astute, more penetrating questions than their mates. Communication is good with mixed audiences, and the program of prevention will be strongly reinforced by informed families.

## Occupational Health Professionals

Because there are so many substances, combinations of substances, industries, levels of industry (raw materials, intermediate manufacturing, finished product manufacturing and distribution), occupational



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health personnel are not all-knowing about all hazardous substances currently in use. For example, with the possibility of asbestos insulation in innumerable buildings being removed during renovation or demolition, it is possible that the health personnel of a company or institution not in the asbestos industry may suddenly be faced with the inauguration of a project to protect personnel against the untoward effects of fiber inhalation. Finally, the ubiquity of asbestos is such that it could be present in a plant without a company's health personnel's knowledge--i.e., through the reworking of asbestos-containing parts or raw materials manufactured elsewhere. Information on the asbestos hazard will sharpen the health professional's index of suspicion, and he will seek out the material's presence throughout the workplace.

## Assessment of Education's Value

Every health education program should be validated as to its worth. While a rigorous evaluation may not be possible, there are means by which the effectiveness of an education program might be judged. Although for a work force as a whole, some workers are retiring and some workers are leaving for other employment, small groups can be tested informally before and after various education segments in which they participated as follows:

- Compliance in engineering controls and safe work practices--without giving great visibility to the check-off procedure, workers could be observed prior to and after the educational program to determine if there is a change in their use of engineering control devices or in their compliance with safe work practices.
- Compliance in medical surveillance programs--workers often report for chest radiography or pulmonary function testing only after innumerable telephone calls and notices, and improved compliance rate would indicate some success in education.
- Basic knowledge appropriate to target group--groups can be pre- and post-tested for retention of common knowledge expected of workers in daily contact with asbestos. Differences in reading ability, cultural differences and anxiety levels will influence test results, but there should be at least some rough indication of added information or altered behavior.


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# APPENDICES

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Appendix A

ASBESTOS-RELATED AND -ASSOCIATED MINERALS

Minerals Other Than Asbestos that May Exhibit Fibrous Structure

| Mineral Species[a] | Morphological Characteristics |
|---|---|
| Pyrophyllite | Sometimes occurs as radiating needlelike crystals in feldspars, kyanite, and in quartz veins from reef mines. |
| Vermiculite | Normally exhibits a flaky structure, but fibrous varieties have been reported. |
| Attapulgite (a clay) | Commonly crystallizes in well-defined fibrous forms. |
| Lepidolite (a mica) | Normally fibrous or granular when present as overgrowths on muscovite micas. |
| Minnesotaite (a talc) | Commonly fibrous rather than platy. |
| Chamosite | Sometimes occurs in the form of minute fibrous crystals, associated with sedimentary iron formations (ironstones) |
| Halloysite (a clay) | Frequently occurs with kaolinite and is characterized by elongated tubular crystals, similar to needles. |
| Holmquistite (an amphibole) | Commonly occurs in aggregates and sometimes displays an asbestiform texture. |
| Richterite (an amphibole) | Commonly exhibits a fibrous crystallographic habit similar to asbestos. |

[a]The first three have important industrial applications; the others are found in connection with commercial mineral operations, where they are normally considered an impurity.



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## Minerals and Rocks Possibly Associated With Asbestos

| Mineral or Rock | Uses |
|---|---|
| Talc[a] | Ceramic applications (whiteware, wall tiles, electrical porcelain); extender for paints and pigments; lubricant and filler for cosmetics, and in the preparation and packaging of foods. |
| Phlogopite | A member of the mica family, used in a number of electric and electronic applications, such as insulation in aircraft sparkplugs, and as a thermal insulator.[b] |
| Chlorite | A common constituent of metamorphic rocks; due to its green coloration, is used in construction for aesthetic purposes. |
| Kaolinitic clays | Used in ceramics (whitewares, thermal insulators); as a filler or extender in the rubber, paint, and plastics industries; in the paper industry to impart gloss, opacity, brightness, and printability; and in medications. |
| Bentonitic clays (Montmorillonite, Fuller's earth) | Drilling muds; as a binder for iron ore pelletizing; in filtering applications; and a filler for paints, cosmetics, pharmaceuticals, and ceramics. |
| Vermiculite | Used primarily in the construction industry as an insulator but is also employed in the agricultural and horticultural industries as a soil conditioner. |
| Taconite and similar metamorphic iron deposits | A primary source of iron for the steel industry. |
| Magnesite and Brucite | Used as a raw material for magnesia-containing refractories, fluxes, and miscellaneous chemicals and can be used in the production of magnesium metal. Brucite is often associated with magnesite and, as such, is used as a source of magnesia. |
| Marble[c] | Metamorphic carbonate rock used for polished stone and other applications in construction and for sculpture |

[a] Also known as steatite or soapstone.

[b] Most phlogopite is imported into the United States. Its occurrence in this country is resticted mostly to small, noncommercial deposits and as an impurity in certain marbles.

[c] The term "marble" is sometimes used indiscriminately to describe other rocks that can be polished and that have a pleasing appearance; such rocks include the so-called verde antique or serta green, some onyxs, and slates. Verde antique (serta green) is commonly a serpentine and is therefore likely to include chrysotile contaminants.

Sources: W.A. Deer, et al. "Rock-Forming Minerals," Longmans Green & Co., 1961
N.W. Hendry, "The Geology, Occurrences and Major Uses of Asbestos," New York Academy of Sciences, 132, Dec. 31, 1965.
Malcolm Ross, "Geology, Asbestos and Health," Environmental Health Perspectives, 9, 1974, pp. 123-4.
Robert L. Bates, "Geology of the Industrial Rocks and Minerals," Dover Publications, Inc., N.Y., 1969.



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# Appendix B

## FEDERAL REGULATIONS OF OCCUPATIONAL EXPOSURE

### Definitions and Airborne Levels

| Federal Organization[a] | Scope | Definitions | 8-Hr Time-Weighted-Average (Fibers/ml) | Ceiling (Fibers/ml) |
|---|---|---|---|---|
| (1) Occupational Safety and Health Administration, Dept. of labor | Regulation | Longer than 5μ using the membrane filter method at 400-450x magnification with phase contrast illumination | 5[b]<br>2[c] | 10 |
| (2) Occupational Safety and Health Administration | (Proposal) Does not apply to construction work | Same as OSHA regulation but adds: length to diameter ratio of at least 3:1; maximum diameter 5μ | 0.5 | 5 |
| (3) Mining Enforcement and Safety Administration Dept. of Interior | Metal and nonmetallic open pit mines; sand, gravel, crushed stone operations; underground mines | Same as OSHA regulation | 5 | 10 |
| (4) Bureau of Mines Dept. of Interior | Surface work areas of underground and surface coal mines | Longer than 5μ; length/width ratio at least 3:1 in 20 randomly selected fields using phase contrast microscopy at 400-450x magnification | 2 | |
| (5) Executive Office | Head of each federal agency must maintain an occupational safety and health program meeting requirements of the Occupational Safety and Health Act of 1970; assure periodic inspections and prompt abatement of unsafe or unhealthful working conditions; provide training programs; keep records as prescribed by Secretary of Labor. Secretary of Labor is to issue guidelines to agencies, evaluate and assist each program, and report to President annually. | | | |

[a] Sources: (1) Code of Federal Regulations (CFR), Title 29, Part 1910.93a; (2) CFR, Title 29, Part 1910.1001; (3) CFR, Title 30, Parts 55.5, 55.6, 55.7 (1975); (4) CFR, Title 30, Part 71.202 (1976); (5) Executive Order #11807, Feb. 3, 1975.

[b] July 1972.

[c] July 1976.



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## Appendix B (continued)

### Summary of Provisions for Compliance

- Occupational Safety and Health, Dept. of Labor--Regulation

| | |
|---|---|
| Methods of Compliance | Specified engineering methods: Including local exhaust ventilation of all tools producing asbestos fibers in excess of exposure limits. |
| | Personal protective equipment: Respirators--when technically not feasible to reduce exposure by engineering methods or work practice and in emergencies; personnel rotation preferred to respirators; respiration selection procedures. |
| | Special clothing: Whole body clothing, head and foot coverings, gloves for airborne levels exceeding limits. Laundering by management. Personal protective equipment used when spraying, demolishing, or removing asbestos insulation. (For excessive levels, change rooms and separate clothes lockers provided to prevent contamination of street clothes from work clothes.) |
| | Work practices: Wet wherever possible; no asbestos cement, mortar, coating, grout, plaster or similar material removed from container unless wetted, enclosed, ventilated; waste which may produce air levels above limits must be disposed of in closed containers. |
| Monitoring | Personal and environmental air samples at least every 6 months if exposures may reasonably be foreseen to exceed limits. Employees must be notified of excessive exposure and of action taken. |
| Medical | Preplacement, annual and termination chest X-ray (PA, 14"x17"), respiratory disease history pulmonary function tests including FVC, $FEV_{1.0}$. Physician determines ability to tolerate respirator. |
| Education | Caution signs, labels. |
| Records | Exposure levels retain 3 years. Medical records retain 20 years. |

- Occupational Safety and Health Administration, Dept. of Labor--Proposal

| | |
|---|---|
| Methods of Compliance | Specified engineering methods: (Same as regulation.) |
| | Personal protective equipment: (Same as regulation.) |
| | Special clothing: Whole body clothing, head and foot coverings, gloves, for airborne levels exceeding limits. Laundering by management. Personal protective equipment used when spraying, demolishing, or removing asbestos insulation. |
| | Regulated areas: Areas where exposures may exceed limits must restrict access to authorized persons; maintain a daily roster of persons entering; prohibit eating, drinking, smoking, and |



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Summary of Provisions for Compliance

chewing nonfood items. Persons entering area must wash hands, face, and forearms prior to eating, drinking, smoking, and must shower at the end of the workshift. Clothes lockers and showers shall be arranged to separate regulated and uncontaminated areas. Separate storage facilities for street clothes and work clothes shall be provided.

Monitoring — Personal and environmental air samples monthly if above limit; every 3 mos. if below limit. If under limit twice consecutively from 5 days to 3 mos. apart, no need to sample unless reason to believe exposure levels have changed. Measurements of the effectiveness of mechanical ventilation every 3 mos.

Medical — X-rays, history, tests--same. Sputum cytology required for employees 45 yrs old or older with 10 or more yrs exposure. Physician's written opinion if any medical condition may place person at increased risk of impairing health from asbestos exposure and recommended limitations of exposure or use of personal protective equipment.

Education — Adds training to include asbestos exposures, controls, use and limits of respirators, purpose of medical surveillance, OSHA standard.

Records — Exposure levels, medical records, regulated area rosters: retain 40 yrs or yrs employed + 20, whichever greater. Records of employee training and mechanical ventilation performance: retain 3 yrs. All records to be transferred to successor or OSHA if plant changes hands or closes.

- Mining Enforcement and Safety Administration, Dept. of Interior

  Specified engineering methods including wet methods, exhaust, and dilution ventilation.

  Respiratory protective devices when exceed exposure limits, engineering methods not feasible, or when necessary by the nature of the work; respirator selection and maintenance. (Does not apply to coal mines.)

- Executive Office

  See page B-1.



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## Appendix C

## MONITORING AND MEASURING ASBESTOS CONTAMINATION

Although the role of asbestos as a cause of cancer and other diseases is clear, the mechanisms underlying disease causation are not well understood, and this impedes efforts to measure and, subsequently, control human exposures to the substance. As if the technical and economic difficulties of measuring asbestos concentrations--the subject of this chapter--were not enough, two other facets of the hazard are murky: (1) Which attribute(s) of asbestos it is that actually causes disease and which therefore must be measured accurately; and (2) the amount of asbestos that is hazardous, and over what period of time.

The attributes of asbestos that have been implicated in various hypotheses on asbestos carcinogenesis include: *

- Size and shape of individual fibers[1]
- Number of fibers[2]
- Total mass of asbestos[3]
- Type of asbestos[2]
- Trace metal content[4]
- Trace organic content[5]
- Surface charge[6]
- Surface adsorptive characteristics[6]

Because of the inordinate difficulties of separating the last four attributes from nonasbestos "background", these four are not suitable indices of exposure for routine monitoring situations. In addition, hypotheses based on trace metal or organic content have fallen out of favor. Because there is no consensus on the attribute(s) that cause cancer and, therefore, no consensus on the appropriate indicator of risk from environmental exposure to asbestos, the "best" monitoring and measuring method will be that which permits the most detailed characterization of exposure through measurement of the first four variables--size and shape of individual fibers; number of fibers; total mass of asbestos; and type of asbestos.

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*References will be found at the end of this Appendix.



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In addition to the limitations just mentioned, it has not yet been established whether the time-course of exposure, or dose rate, is significant in determining carcinogenic risk.[7] This leaves open the possibility that it may be just as important to determine peak exposures as to determine time-weighted average exposures.

Because of these limitations, and because of the technical difficulties and costs of measurement discussed below, the method of measuring must be chosen judiciously to fit the circumstances: occupational versus general environment; measurement of asbestos concentrations in water versus those in air; monitoring the environment at large versus determining exposures to certain individuals. Also, the methods chosen may differ according to the fiber type that is of concern.

## Monitoring Devices and Methods

The most desirable means of monitoring in general, would be one in which a device could measure directly the asbestos concentration in air, water, or in a bulk sample of material. Such a device, which might be used for continuous monitoring, is not available for most situations, however.

### Some Limited-Application Monitoring Devices

Some devices for measuring general particulate contamination of fluids--based upon the piezoelectric balance,[8] Beta-attenuation,[9] or light-scattering principles[10]--may be suitable for monitoring applications where the asbestos concentration is a known, constant, and relatively large fraction of the general particulate contamination.[11] They are not appropriate for such purposes as asbestos-pollution monitoring in community air, where the total particulate loading will be several orders of magnitude greater than the asbestos loading.[12] Nor are they appropriate in surveys of industrial sites, where the asbestos/total loading ratio and the size of airborne asbestos fibers may fluctuate markedly.[2] Thus, continuous monitoring is generally not practicable, and sampling will almost always be necessary.



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Monitoring by Membrane Filtration

The major method of monitoring in use for environmental media is membrane filtration. The membrane filter method is directly applicable to air, stack gases, water, food liquids, and other fluid media. With solid media, it can be used as a final concentration step for fibers that remain after some preliminary digestion procedure.

The procedure most commonly used is a single-step filtration, in which all suspended particulate material in the medium is entrapped, together with the suspended asbestos. It is also possible, however, to utilize a dual-filter set-up in which the first (coarser) filter entraps the larger material while allowing much of the smaller material (including most of the asbestos) to pass through to be collected on the second (finer) filter for analysis.[12] Although this dual-filter method has some advantages for applications in which the asbestos concentration is low and concentrations of other particulate material are high, the method cannot be generally recommended because as filter loading increases, the collection characteristics change causing an increasing amount of asbestos fibers to be trapped on the first filter.

The filter best-suited for use in most environmental sampling for asbestos is a mixed cellulose-ester membrane filter (similar to those manufactured by the Millipore Corporation), with a mean nominal pore size of 0.45 or 0.8 μm. These filters will remove, from water, 99.9% of asbestos fibers present[13] and are estimated to be "...almost 100% efficient..." for removal of asbestos fibers from air.[14]

For some applications, the "Nuclepore" membrane filters manufactured by General Electric are most appropriate.[12] They are more suitable for scanning electron microscopy of collected samples because of their relatively flat surface and their relative stability in an electron beam. However, they are difficult to use in direct environmental monitoring, because they have a high pressure drop at normal sampling rates and a



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tendency to develop static charges, which makes them difficult to handle after sampling.*

Measuring Asbestos in Air

It is most probable that inhalation of airborne asbestos is attended by greater carcinogenic risk than is ingestion (as discussed in Chapter III). Hence, it is most important that present knowledge of exposure to airborne asbestos, as a function of time for various population groups, be refined so that those groups at greatest risk can be ascertained.

The biologically effective concentration and deposition characteristics of the particles available for inhalation (in the breathing zone of the individual) are the critical parameters to be measured. The biologically effective concentration will be some complex function of fiber number, size, shape, mass, and type of asbestos best correlated with disease. Deposition characteristics (deposition in the airways of of man) are complex and are discussed elsewhere in this monograph (Chapter III). Suffice it to say that the diameter of the individual fiber is the principal controlling variable in deposition, but length is important enough that both should be measured, although this is difficult in practice.

Measuring Asbestos in Water

The sampling of water to determine asbestos content requires special care if the results are to be representative of potential human exposures. As with air, there are seasonal effects on sources of asbestos and on the distribution of asbestos in a given body of water. Stream flow rates, thermal stratification, sedimentation, and differing source flow rates may all have an effect, as may (for drinking water) the characteristics of the distribution system.

The Environmental Measurements Advisory Committee of the EPA has recently been studying the question of measuring asbestos in drinking water. As part of this effort, the Athens (Georgia) Environmental Research Laboratory has developed a preliminary method for such assessment.

*For discussions of membrane filtering that are much more thorough, see References 14, 20, 24, 25, and 26.



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In part, the guidelines given there include:

> "It is beyond the scope of this procedure to furnish detailed instruction for field sampling; the general principles of sampling waters are applicable. There are some considerations that apply to asbestos fibers, a special type of particulate matter. These fibers are small, and in water range from .1 μm or more. Because of the range of size there may be a vertical distribution of particle sizes. This distribution will vary with depth depending upon the vertical distribution of temperature as well as the local meteorological conditions. Sampling should take place according to the objective of the analysis. If a representative sample of a water supply is required a carefully designed set of samples should be taken, representing the vertical as well as the horizontal distribution, and these samples composited for analysis.
>
> The sampling container shall be a clean polyethylene, screw-capped bottle capable of holding at least one liter. The bottle should be rinsed at least two times with the water that is being sampled prior to sampling. (Note: Glass vessels are not suitable as sampling containers.) A minimum of approximately one liter of water is required and the sampling container should not be filled. It is desirable to obtain two samples from one location."[15]

When the bulk water sample has been collected, it is filtered through a membrane filter and the filter prepared further for analysis.

Measuring Asbestos in Food

The most critical, and yet unresolved issue in measuring asbestos in food is a method to be used in preparing solid or semi-solid food for separation of any asbestos that may be contained. At present, no such generally suitable method exists. Surface contamination (e.g. on rice) may be measured relatively easily by straightforward washing and filtration techniques. For liquid foods and beverages, simple filtration, with membrane filters, is often appropriate, followed by treatment to remove organic co-contamination of the filter surface.[16]



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Analysis of Samples--Some Technical and Economic Constraints

The most valid analytical methods for complete assessment of asbestos contamination are those methods based upon electron microscopy. This is particularly true for assessment of general community exposure from air or water. In some cases, where the fiber-size distributions of asbestos exposures are constant and already known from electron microscopy, the use of optical microscopy, or the methods of continuous monitoring referred to earlier, may be justified for routine surveillance.

Technical Constraints

Individual asbestos fibers can be identified by phase-contrast optical microscopy. However, without using electron microscopy it is not always possible to distinguish asbestos fibers from amorphous inorganic (fibrous glass or mineral wool), natural organic (plant or animal), or synthetic organic (nylon, orlon, etc.) fibers. Moreover, in most monitoring situations the majority of fibers will be below the resolution of the phase-contrast microscope. Complete characterization and identification of such fibers requires the following information:[17, 18]

- Dimensions and morphology
- Electron diffraction pattern
- Elemental composition

For samples such as community air and water, for which more than one fiber type cannot be ruled out a priori, obtaining this information is no simple matter. Each fiber must be identified separately by using electron microscopes,[30] equipped with X-ray fluorescence and selected area electron diffraction capabilities, for the determination of elemental composition and crystallographic characteristics.

With any of the analytical methods, compromises are necessary because of the relatively "heroic" sample-preparation methods required, especially for transmission electronic microscopy. Many of these methods--involving such techniques as ashing, multiple transfers of liquid resuspensions, and filtrations--tend to subdivide fibers into fibrils. Thus, it is often difficult to reconstruct the original size distribution.



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For samples in which the concentration of asbestos fibers is low and the size distribution small, size distribution analysis may be unreliable because only a few fibers will be seen.

### Economic Constraints

The equipment needed for complete analyses in a continuous monitoring program will require a highly skilled and experienced operator and extensive technical support. The minimum initial capital investment required for new equipment will be in the range of $100,000-$250,000, and an annual operating budget of nearly $100,000 will be required. This will cover the expense of purchasing and installing a scanning/transmission electron microscope, and the salaries, fringe benefits, and overhead expenses for a microscopist and two or three technicians.

For an uncomplicated membrane filter sample--when only one type of fiber is present, and other particulate material is not excessive--the number and mass of fibers and their size distributions may be obtained for a cost of a few hundred dollars per sample. If the sample is more complex, the costs may run to several thousand dollars, and the sample may require several weeks for analysis.[19]

Some progress has been made in automating the counting and sizing of fibers, from photomicrographs,[20,21] but these automated methods are based only upon recognition of fibrous morphology and are therefore unsuitable for evaluation of "mixed" exposure by fiber type.

## Reproducibility of Measurements

Because of the difficulties of measuring asbestos-fiber contamination, there are large variations in the measurement of asbestos in samples of air and water, both within and among laboratories. Several replicability and duplicability studies are summarized below.

### Asbestos in Water

Although the attainment of an intralaboratory precision of ±30% in analysis of water has been reported,[18] it is not uncommon for results



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from duplicate samples analyzed by competent workers in independent laboratories to vary by more than an order of magnitude.

In a reproducibility study reported in 1975, seven "competent" laboratories showed readings of below-detection-limit to 9 million fibers per liter for a water sample known to contain 7.5 million fibers per liter. In another case where five replicate analyses were made in each of two laboratories on an identical sample, one laboratory showed readings of 0.79 to 4.0 million fibers per liter, while the other showed readings of 8. to 29. million fibers per liter.[15]

In another study, reported in 1976, investigators evaluated variation among and within nine laboratories that analyzed samples of water from five different sites on Lake Superior. The averages for the nine laboratories varied from 1.6 million to 78.7 million fibers per liter. The within-laboratory coefficient of variation (standard deviation divided by the average) ranged from about 50% to 100%.[22]

Asbestos in Air

There is also considerable variability in measurements of atmospheric asbestos concentrations, especially of ambient air, both within and among laboratories. Data on duplicate analyses of ten ambient air samples from measurements by the Mount Sinai Department of Environmental Medicine and the California Department of Health are given in Table C-1 Differences between the two laboratories may be two orders of magnitude or more. No one laboratory's readings were consistently higher. The Mount Sinai group also conducted replicate analyses of four samples. These results, shown in Table C-2, show differences of less than an order of magnitude. The authors of the study in which these findings were reported state that an individual value may be accurate within a factor of two or three of a sample mean.[23]

The inaccuracy in values obtained from a specific analysis can result from several circumstances: (1) statistical variation in the number of fibrils found in given grid squares, (2) a much greater variation in volume of these fibrils, (3) incomplete dispersal of chrysotile bundles, (4) variability in the amount of material lost during processing, and (5) low-level contamination of the sample at various points during its preparation and analysis.



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Table C-1

DUPLICATE ANALYSIS OF TEN AMBIENT AIR SAMPLES

| Sample Number | Asbestos Concentration (nanograms/meter$^3$) Mount Sinai | Calif.Dept. of Health |
|---|---|---|
| 74-000-003 | 0.6 | 120.0 |
| 74-000-012 | 44.0 | 0.4 |
| 74-000-023 | 0.0 | 13.0 |
| 74-000-032 | 2.7 | 0.0 |
| 73-003-038 | 2.6 | 0.0 |
| 73-003-046 | 7.3[a] | <800.0 |
| 73-003-054 | 7.3 | 0.0 |
| 73-003-064 | 21.0 | 0.0 |
| 74-000-110 | 46.0 | 14.0 |
| 74-000-119 | 1.4 | 2.2 |

[a]With an annotation: "Disregard, only 2 fibers observed."

Source: Reference 23 (end of this Appendix).



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Table C-2

REPLICATE ANALYSIS OF FOUR AMBIENT AIR SAMPLES

| Sample Number | Asbestos Concentration (nanograms/meter$^3$) 1st Analysis | 2nd Analysis | Average |
|---|---|---|---|
| 73-003-038 | 0.0 | 5.3 | 2.6 |
| 73-003-046 | 7.6 | 7.1 | 7.3 |
| 73-003-054 | 3.2 | 11.4 | 7.3 |
| 73-003-064 | 12.0 | 30.0 | 21.0 |

Source: Reference 23 (end of this Appendix).

Although the phase-contrast microscopic method has been demonstrated to be useful for evaluation of occupational asbestos exposures, and a coefficient of variation (for sampling and analysis) of 22% has been reported for trained technicians,[24] it should be recognized that there may be a two-fold variation between results (for reference samples) from experienced and novice microscopists for amosite, and a four-fold variation for chrysotile.[25] It is also important to recognize that the fraction of fibers in a sample of airborne asbestos that will be visible in using this method will vary by type of asbestos and also (within each type) by the particular industrial process being evaluated.

Conversions Between Counts by Different Methods and Between Number of Fibers and Mass.

Because of the large variations in measurements, it is generally not possible to convert counts by the phase-contrast optical method to equivalent electron microscopic counts, except within very wide margins of error. Indications of the error that may be introduced by uncritical acceptance of any constant factor for conversion may be found in (a) a study of size distributions for airborne asbestos exposures in



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various asbestos processes[2] and (2) a comparison of optical and electron microscopic methods applied to samples of air taken from inside public buildings.[23] In the latter study, variations by factors of 20-30, and no apparent correlation, were found.

Nor can counts of visible dust particles by the midget impinger method be equated with fiber levels determined by phase-contrast microscopy, as illustrated in another study which gives measurements of both methods for one job at four asbestos textile plants[26] Within a plant, asbestos levels increased with midget impinger counts; but if comparisons are made across plants, it is clear that the limits of uncertainty are approximately an order of magnitude.

In a 1974 study, there were differences of several orders of magnitude between fiber/mass ratios in emission streams of asbestos processing plants and mills, making any attempt to apply a constant conversion factor between number and fibers and mass uncertain at best.[26]

Some important conclusions can be drawn:

- The ratios of electron-microscope-visible to optical-microscope-visible fibers vary among plant emissions, the workplace, and the general environment, as well as within each of these categories.
- No universal ratios or factors for conversion of optical microscope results to electron microscope results exist. In this monograph, when needed, a factor of 50 has been used to convert occupational fiber levels from optical-microscope-visible to electron-microscope-visible fibers. The factor is based on a conservative estimate that 2% of total fibers are optical-microscope-visible.
- No single factor for conversion of mass emissions to fiber emissions exists.
- Fiber/mass ratios differ markedly from the occupational to the general environments, and they also differ markedly within each environment. The number of electron-microscope-visible fibers per nanogram might well range from 100-10,000. Based on the observed data available and for convenience sake, 1000 fibers per nanogram



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has been used in this monograph for atmospheric levels near plants, and 250 fibers per nanogram for ambient urban air.

- The monitoring method chosen will be different for each application but must depend upon electron microscopy as a reference method.
- Because mass of asbestos can be directly calculated from asbestos type and electron-microscope-visible fiber size and number, it is recommended that these indices be recorded whenever possible. The fiber size distribution can be completely stated by the geometric means and standard deviations of fiber length and diameter, assuming that fiber length and diameter vary independently of each other.

## REFERENCES


1. Stanton MF, et al: Carcinogencity of fibrous glass: pleural response in the rat in relation to fiber dimension. J Nat Cancer Inst 58(3): 587-603, 1977.

2. Gibbs GW, Hwang CY: Physical parameters of airborne asbestos fibers in various work environments--preliminary findings. Am Ind Hyg Assn J 36(6):459-466, 1975.

3. SRI International: Personal communication with Nicholson WJ, October 1976.

4. Cralley LJ, Keenan RG, Lynch JR: Exposure to metals in the manufacture of asbestos textile products. Am Ind Hyg Assn J 28(5):452-461, 1967.

5. Harrison JS, Roe FLC: Studies of carcinogenesis of asbestos fibers and their natural oils. Ann NY Acad Sci 132:439-450, 1965.

6. Gorski CH, Stettler LE: The surface energetics of asbestos minerals. Am Ind Hyg Assn J 35(6):345-353, 1974.

7. Cooper WC (Ed.): The Need for and Feasibility of Controls of Asbestos Air Pollution. National Academy of Sciences, Washington, D.C., 1971.

8. Sem GJ, Tsurubayaski K: A new mass sensor for respirable dust measurement. Am Ind Hyg Assn J 3(11):791-800, 1975.

9. Almich B, Soloman M, Carson GA: A theoretical and laboratory evaluation of a portable direct-reading particulate mass concentration instrument. U.S. Dept. Health, Education and Welfare, Public Health Service, CDC, NIOSH, July 1975.




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Published by AsbestosClaims.Law
asbestosclaims.law/library/25239/145

10. Mercer, TT: Aerosol Technology in Hazard Evaluation, Academic Press, New York, 1973.

11. Harwood CF: Asbestos Air Pollution Control. Chicago, State of Illinois Institute for Environmental Quality, 11 EO Document #71-8, 1971.

12. Spurney KR, et al: The sampling and electron microscopy of asbestos aerosol in ambient air by means of Nuclepore filters. J Air Pollut Control Assoc 26(5): 496-498, 1976.

13. Manalan DA: Asbestos removal by membrane filter. Presented at the Spring meeting of the Parenteral Drug Association, Inc., Dorado Beach, Puerto Rico, April 5, 1974.

14. Lynch JR, Ayer HE: Measurement of asbestos exposure. J Occup Med 10(1): 21-24, 1968.

15. Anderson CH: A Preliminary Interim Procedure for Determining Fibrous Asbestos. Athens, GA, U.S. Environmental Protection Agency, Environmental Research Laboratory, July 28, 1976.

16. McGrath PP, Ewell JB: Application of electron microscopy to the problem of particulate contaminants in food, drugs and biologicals. Scanning Electron Microscopy 1976 (Part III). Proceedings of the Workshop on Techniques for Particulate Matter Studies in SEM. Chicago, ITT Research Institute, April 1976.

17. Pooley FD: The identification of asbestos dust with electron microscop microprobe analysis. Ann Occup Hyg 13(3): 181-186, 1975.

18. Beaman DR, File DM: Quantiative determination of asbestos fiber concentration. Anal Chem 48(1): 101-110, 1976.

19. Langer AM: Approaches and constraints to identification and quantification of asbestos fiber. Environ Health Perspect 9: 133-136, 1974.

20. Harness I: Airborne asbestos dust evaluation. Ann Occup Hyg 16(4): 397-404, 1973.

21. Pavlidis T, Steiglitz K: The automatic counting of asbestos fibers in air samples. Presented at the 3rd Joint Conf on Pattern Recognition, IEEE Computer Society, Nov. 8-11, 1976.

22. Brown, Jr. AL, Taylor WF, Carter RE: The reliability of measures of amphibole fiber concentration in water, Environ Res 12(2): 150-160, October 1976.

23. Nicholson WJ, et al: Asbestos Contamination of the Air in Public Buildings. Research Triangle Park, NC, U.S. Environmental Protection Agency Pub. #450/3-76/004, Oct. 1975.



Digitized by Google


Published by AsbestosClaims.Law
asbestosclaims.law/library/25239/146

24. Leidel NA, Bayer SG, Zumwalde RD: Membrane Filter Method for Evaluating Airborne Asbestos Fibers. U.S. Public Health Service, NIOSH, TN 84, 1973.

25. Beckett ST, Attfield MD: Inter-laboratory comparisons of the counting of asbestos fibers. Ann Occup Hyg 17(2):85-96, 1974.

26. Ayer HE, Lynch JF, Fanney JH: A comparison of impinger and membrane filter techniques for evaluating air samples in asbestos plants. Ann NY Acad Sci 132:274-287, 1965.

27. Harwood CF, Siebert P, Blazsak TP: Assessment of Particle Control Technology for Enclosed Asbestos Sources. Research Triangle Park, NC, U.S. Environmental Protection Agency Pub #650/2-74/008, Oct 1974.



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Appendix D

ANIMAL STUDIES RELATED TO CARCINOGENIC EFFECTS OF FIBERS

Inhalation

Mice

Hybrid mice ($AC/F_1$) were exposed to a commercial preparation of chrysotile dust at a concentration of 150-500 mppcf 40-60 hours per week for 17 months and were sacrificed after exposure. A high incidence of multiple pulmonary adenomas* was observed in the exposed group (45.7%, or 58/127), than in controls (36.0%, or 80/222).[160+]

Rats

White rats, some of whom had received an intratracheal application of caustic, (reportedly to impede mucociliary clearance), were exposed to chrysotile dust 30 hours per week for 62 weeks at a mean concentration of 86 $mg/m^3$. Of 72 rats surviving 16 months or more, 25 developed malignanat thoracic tumors (adenocarcinomas, squamous cell carcinomas, fibrosarcomas, and a mesothelioma). The incidence of animals with cancer was twice as great among caustic-treated survivors (15/31 or 48%) as among those who had not been treated (10/41 or 24%). There were no tumors in 39 caustic-treated and untreated control animals.[113]

Squamous carcinomas of the lung were found in 2 of 31 Charles River CD rats surviving exposure to crocidolite at a concentration of 49 $mg/m^3$ 16 hours a week for two years. No malignant tumors were observed among rats exposed to chrysotile or amosite, but 5 of 40 exposed to chrysotile developed multiple pulmonary adenomas.[106]

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*Benign tumors of the lung.

+Chapter III references--see Apendix H.



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Groups of 69 Charles River CD rats were exposed to crocidolite, amosite, or chrysotile at mean concentrations of about 50 $mg/m^3$ 16 hours per week for two years. Among the group exposed to crocidolite, four developed malignant lung tumors (squamous cell carcinomas and an adenocarcinoma). Three of the group exposed to amosite developed thoracic cancers (a broncoalveolar carcinoma, a fibrosarcoma, and a pleural mesothelioma). Two pulmonary carcinomas (a squamous cell and a papillary carcinoma) and one pleural mesothelioma developed in the animals exposed to chrysotile.[115]

In a similar experiment, exposure for six months to two years produced a 5% incidence of thoracic malignancies among groups exposed to chrysotile and amosite, but a 14% incidence among the group exposed to crocidolite. While mass concentrations of the three types of asbestos were all about 50 $mg/m^3$, counts of optically visible fibers were 54, 864, and 1105 million per cubic meter, respectively.[114]

In another study, C/D Wistar rats were exposed to amosite, anthophyllite, crocidolite, and chrysotile (Canadian or Rhodesian) at a concentration of 12 $mg/m^3$ respirable dust 7 hours per day for varying lengths of exposure. All fiber types produced asbestosis, which progressed after removal of the rats from the dust. Lung carcinomas and pleural mesotheliomas were found in groups exposed to each of the four fiber types, and a single peritoneal mesothelioma was observed among animals exposed to crocidolite. Forty percent of animals exposed for two years developed malignant tumors of the lung and pleura. No such cancers were found among control animals.[6]

A dose-response relationship between asbestos and cancer was noted--with increasing duration of exposure there was an increasing incidence of observed malignancies. As little as one-day exposure was sufficient to produce lung carcinomas and pleural mesotheliomas. There was significantly more asbestosis (taking severity of asbestosis and length of survival into account) among animals with lung tumors than among those without tumors. In addition, among groups exposed for only one day, there were



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significantly more lung tumors among animals with asbestosis than among those without asbestosis.[6]

## Intratracheal Injection

### Rats

White rats received 1, 2, 3, 4, or 6 intratrachael injections of 3.5 mg chrysotile in aqueous suspension. Among 16-month survivors, 3 of 19 developed pulmonary adenocarcinomas. Two of these had received 4 intratracheal injections, and one had received 6 injections.[113]

After administration of 3 injections of 2 mg chrysotile containing 0.14 mg benzo(a)pyrene (a carcinogen in cigarette smoke) at monthly intervals, or a single injection of 2 mg chrysotile and 5 mg benzo(a)pyrene, lung papillomas, epidermoid carcinomas, reticulosarcomas, and pleural mesotheliomas were noted in 6/21 and 6/11 animals, respectively, within 28 months. No tumors occured in 49 rats given 3 monthly injections of 2 mg chrysotile alone or in 19 rats administered a single dose of 5 mg benzo(a)pyrene.[108]

### Hamsters

Eight pulmonary adenomas, 9 tracheobronchial papillomas, and 6 pulmonary carcinomas developed among 31 LVG/LAK hamsters receiving a dose of 4.5 benzo(a)pyrene and 12 mg chrysotile over a period of 12 weeks. No tumors were observed in 17 animals administered chrysotile alone. Among 34 receiving benzo(a)pyrene alone, 9 tracheobronchial papillomas and 1 pulmonary carcinoma were reported.[161]

The effect of intratracheal instillation of asbestos with oral or subcutaneous administration of nitrosodiethylamine (NDEA) was studied in Syrian golden hamsters. Aqueous NDEA was administered per gastric tube twice weekly for 20 weeks (total dose 60 mg), or 3.5 mg aqueous NDEA was injected subcutaneously once a week for 12 weeks (total dose, 42 mg). One mg chrysotile asbestos in polyglucin suspension was injected intratracheally once weekly for 6 weeks (total dose, 6 mg), beginning one month after the start of NDEA treatment. After 8-10 months of 21/52



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and 14/51 animals developed benign and malignant pulmonary tumors among the groups receiving asbestos and oral NDEA or subcutaneous NDEA, respectively. Among control groups receiving oral NDEA, subcutaneous NDEA, or asbestos alone, only 1/50, 3/47, and 0/50 developed pulmonary tumors.[162]

## Intrapleural Injection

### Mice

Two of 75 BALB/c mice receiving intrapleural inoculations of 10 mg crocidolite in aqueous suspension developed pleural tumors, but no tumors were observed among 75 mice injected with 10 mg chrysotile.[163]

### Rats

A single dose of 20 mg of crocidolite, amosite, anthophyllite, or chrysotile from various sources administered to CD Wistar rats produced an incidence of pleural mesothelioma ranging from 19% to 70%. Yet as little as 0.5 mg chrysotile or crocidolite was sufficient to induce mesotheliomas.[107]

Various asbestos types have induced mesotheliomas following intrapleural injection in Osborne-Mendel, Sprague-Dawley, CFY and Wistar/Alderly ICI rats.[107,108,161,163] The carcinogenic response to chrysotile and crocidolite appeared to be dose-related.[107,117]

Oil-extracted asbestos gave results similar to untreated samples,[107,164] thus casting doubt upon the hypothesis that natural oils and waxes,[165] contaminant oils from the milling process[166,167] or organic materials originating from storage in plastic or jute bags[168] contribute to the carcinogenicity of asbestos.

It has also been suggested that metal contaminants added during processing of fiber play a role in asbestos carcinogenesis.[113] Subsequent experiments using rats[169,170] have demonstrated that (1) treatment with acid, base, and ethylene diamine tetra-ascetic acid to remove metal contaminants; (2) selecting asbestos samples from among interior fibers not exposed to contaminating hammermills; or (3) using different


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samples of the same asbestos type containing different quantities of trace metals did not produce different tumor yields. By way of contrast, heating samples for two hours at 900-1000°C resulted in a substantial loss of carcinogenic activity in rats.[169]

Fiber diameter and length, in addition to shape, may be important determinants of carcinogenic potency. Eight subsamples of U.I.C.C. (Union Internationale Contre le Cancer) standard reference Canadian chrysotile were milled individually to a finer powder than the standard mixture. Injection of each subsample intrapleurally resulted in a higher yield of mesotheliomas in Wistar rats than did the pooled reference material. The highest tumor yield of all was produced by a separate superfine chrysotile prepared by water sedimentation of Grade 7 commercial asbestos. Carcinogenicity was correlated with number of fibers less than 0.5μm in diameter and greater than 10μm long.[107]

On the other hand, pulverizing asbestos before applying it to the pleura of Osborne-Mendel rats by means of a coated glass pledget reduced carcinogenicity. Fibers of diameter less than 0.2μm and length 5-10μm, which were present in greater numbers in pulverized specimens, were considered to be less effective carcinogens than fibers of greater size.[118] By contrast, after intraperitoneal injection (see "Intraperitoneal Injection" below), fibers less than 5μm long were fully potent in inducing tumors. It must be kept in mind that prolonged milling may alter the crystalline structure of asbestos and thus influence biological effects.

Hamsters, Guinea Pigs, and Rabbits

Pleural mesotheliomas developed in Golden Syrian hamsters after intrapleural injection of 10 mg chrysotile, amosite, anthophyllite, or crocidolite. An apparent dose-response relationship was observed for chrysolite, with 0, 4, and 9 mesotheliomas induced among 50 animals by injection of 1, 10, Or 25 mg, respectively. Prolonged milling, which reduced the majority of fibers to submicroscopic dimensions, eliminated carcinogenic effects and greatly reduced fibrogenic activity.[109]



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One pleural tumor was observed among 50 guinea pigs inoculated with 10 mg crocidolite in an aqueous suspension, but none were observed in a similar group inoculated with chrysotile.[171]

Of 3 rabbits surviving at least 12 months following injection with 16 mg crocidolite, 2 developed pleural mesotheliomas, one at 22 and one at 24 months.[106]

Intraperitoneal Injection (Mice and Rats)

Peritoneal mesotheliomas occured in 20 of 60 BALB/c mice within 18 months of injection with 10 mg crocidolite.[171]

Peritoneal mesotheliomas were reported in Charles River CD, Sprague Dawley, and Wistar rats following intraperitoneal injection of crocidolite[106,111,171,172] and in Charles River CD and Wistar rats following injection of chrysotile.[106,110,172] Although the incidence of peritoneal tumors among groups of 40 animals was virtually identical, the time from injection to observation of the earliest tumor was 276 days for 25 mg chrysotile as opposed to 343 for 6 mg chrysotile.[173] Tumors were produced by four 25 mg injections of powdered chrysotile (99.8%) of the fibers less than 5μm long), although the incidence was somewhat less than for standard chrysotile (12/37, or 32%, vs. 18/33, or 55%).[112]

Subcutaneous Injection (Mice and Rats)

CBA mice developed injection-site sarcomas and pleural and peritoneal mesotheliomas (2+5/17, 2+2/13, 1+1/12) after three sets of bilateral inguinal injections of 10 mg crocidolite, amosite, or chrysotile at intervals of 5 weeks. No tumors were observed among 15 controls injected with saline.[174] The investigators, however, have been unable to duplicate their findings and believe that, in their experiments, asbestos either was injected directly into the pleural or peritoneal cavities or ulcerated into these cavities through the overlying tissues.[175]

A single local tumor was observed following injection of 75 mg chrysotile to 33 Wistar rats.[112]



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Oral Administration (Rats and Hamsters)

One gastric leiomyosarcoma developed among 32 Wistar SPF rats fed 100 mg/day chrysotile 5 days per week for 100 days in a 6-month period. No tumor occurred among 16 controls.[176]

Among 42 animals examined after an average of 441 days on a diet containing 50 mg/kg body weight per day asbestos filter material (52.6% chrysotile asbestos), 12 malignant tumors were observed: 4 kidney carcinomas, 1 lung carcinoma, 3 reticulum cell sarcomas, and 4 liver cell carcinomas. Seven benign tumors including 1 lung adenoma, 2 cholangiomas, 2 stomach papillomas, and 2 mammary fibroadenomas were also noted. Among 49 untreated controls surviving an average of 702 days, 2 liver cell carcinomas and 5 mammary fibroadenomas occurred. The increased incidence of malignant tumors in the group fed asbestos filter material was statistically significant ($p < 0.01$)[116]; however, the meaning of this study as regards asbestos is uncertain since asbestos comprised only half of the filter material administered.

Ten rats fed a diet containing chrysotile, 5% by weight, for 21 months did not develop malignancies. Groups of 28-35 rats fed 10 mg chrysotile or crocidolite in butter once weekly for 16 or 18 weeks developed no malignant lesions which, when compared with a control group, could be related to the ingested asbestos.[10]

No tumors of the gastrointestinal tract were observed in groups of 45 hamsters fed a diet containing 1% chrysotile or amosite by weight for their lifespans.[177]

## Carcinogenicity of Other Mineral Fibers

Mineral fibers other than asbestos have been shown to be carcinogenic by the intrapleural or intraperitoneal routes of administration--but only when of a diameter similar to that of asbestos fibers (less than 5μm).

In groups of 32 rats, mesotheliomas occurred in 18 of those injected intrapleurally with fibrous brucite or "nemalite," which may be contaminated with chrysotile, in 3 of those injected with a ceramic fiber,



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and in 1 each of those injected with fibers of barium sulfate, glass powder, and aluminum oxide.[107] In studies by another team of investigators, the following results were noted: intraperitoneal injection of palygorscite (attapulgite) and nemalite--3 doses of 25 mg and 4 doses of 25 mg, respectively--resulted in tumors in about 75% of Wistar rats (26/34, 25/34). Gypsum, although fibrous, gave a low tumor yield (3/35), perhaps because it dissolves in tissue. Non-fibrous dusts (pectolite, sanidine, talc, biotite, hematite) produced few or no tumors. A dose-response relationship was observed for glass fibers--intraperitoneal injection of 2 mg, 10 mg, and 2 doses of 25 mg results in tumors in 27% (21/73), 53% (44/77), and 71% (55/77), respectively. No tumors appeared among 72 control animals.[117,118]



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## Appendix E

## AIR AND DRINKING WATER ASBESTOS CONCENTRATIONS FROM SOME PUBLISHED STUDIES

### Table E-1

### Atmospheric Concentrations of Asbestos In Some U.S. Urban Areas

| | Concentration (Nanograms/$m^3$) | | Source (Chapter V Reference) |
|---|---|---|---|
| | Average | Range | |
| Berkeley, CA | 6.8 | 2.1-12 | 67 |
| Boston, MA | 5.0 | | 67 |
| Chicago, IL | 24 | 9.5-200 | 67 |
| Dayton, OH | 6 | 0.4-11 | 14 |
| Frankfort, KY | 0.09 | 0.02-0.15 | 14 |
| Houston, TX | 5 | 4-6 | 14 |
| Los Angeles (freeway) | 27[a] | | 13 |
| Los Angeles (control) | 43[a] | | 13 |
| New York City, NY | 13.2 | 8.2-41 | 67 |
| Manhattan, NY | 30[a] | 8-65 | 32 |
| Brooklyn, NY | 19[a] | 6-39 | 32 |
| Bronx, NY | 12[a] | 2-25 | 32 |
| Queens, NY | 9[a] | 3-18 | 32 |
| Staten Island, NY | 8[a] | 5-14 | 32 |
| Pittsburgh, PA | 4 | 2-8 | 14 |
| Philadelphia, PA | 70[a] | 45-100 | 15 |
| Port Allegany, PA | 15[a] | 10-20 | 15 |
| Ridgewood, PA | 20[a] | 15 | 15 |
| San Francisco, CA | 25 | 8.7-68 | 67 |
| Washington, DC | 21 | 1.6-40 | 14 |

[a]Identified as chrysotile asbestos by the authors.



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Table E-2

Asbestos Concentrations in Drinking Water of Some U.S. Communities

| | Concentration | | | | Source |
|---|---|---|---|---|---|
| | Millions of Fibers per Liter | | Micrograms per Liter | | (Chapter V Ref.) |
| | Mean | Range | Mean | Range | |
| Birmingham, AL | BDL | | | | 40 |
| Montgomery, AL | | BDL-.12(C) | | | 40 |
| Tuscaloosa, AL | 0.45(C) | | | | 40 |
| Fairbanks, AK | BDL | | | | 40 |
| Anchorage, AK | 0.07(A) | | | | 40 |
| Glendale, AZ | | | 0.010 | 0-0.075 | 7 |
| Globe, AZ | | | 0.114 | | 7 |
| Yuma, AZ | 0.12(C) | | | | 40 |
| Jonesboro, AR | NSS | | | | 40 |
| Little Rock, AR | 0.27(C) | | | | 40 |
| Van Buren, AR | 40 | | | | 45 |
| Alameda County, CA | 0. | | | | 43 |
| Burlingame, CA | 0. | | | | 43 |
| Contra Costa County, CA | 0. | | | | 43 |
| Long Beach, CA | | | 0.618 | | 7 |
| Livermore, CA | 0. | | | | 43 |
| Millbrae, CA | 0. | | | | 43 |
| Pittsburg, CA | NSS | | | | 40 |
| Redding, CA | 0.5 | | | | 45 |
| Redwood City, CA | 0.0 | | | | 43 |
| Sacramento, CA | NSS | | | | 40 |
| San Diego, CA | | | 0.835 | | 7 |
| San Francisco, CA | 0. | | 0. | | 6 |
| San Francisco, CA | 1.54(C) | | | | 40 |
| San Francisco, CA | 0.6(C) | 0.2-1.(C) | | | 43 |
| San Jose, CA | 0. | | | | 43 |
| Weaverville, CA | 4.5 | | | | 45 |
| Boulder, CO | BDL | | | | 40 |
| Denver, CO | 0.007 | 0-0.056 | 0.042 | 0-0.333 | 6 |

BDL = Below detection limit (less than 50,000 fibers/l.)
NSS = Not significant (less than 5 fibers in 20 fields).
C = Chrysotile
A = Amphibole



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| | Concentration | | | | Source (Chapter V Ref.) |
|---|---|---|---|---|---|
| | Millions of Fibers per Liter | | Micrograms per Liter | | |
| | Mean | Range | Mean | Range | |
| New Haven, CT | NSS | | | | 40 |
| Stratford, CT | 0.38 | | | | 45 |
| Wilmington, DE | 0.29(C) | | | | 40 |
| Washington, DC | NSS | | | | 40 |
| Ft. Lauderdale, FL | NSS | | | | 40 |
| Melbourne, FL | 0.30(C) | | | | 40 |
| Miami, FL | BDL | | | | 40 |
| Atlanta, GA | 5.75 | 0.-12. | 0.192 | 0.-0.574 | 6 |
| Cairo, IL | NSS | | | | 40 |
| Chicago, IL | 0. | | 0. | | 6 |
| Kankakee, IL | 1.6 | | | | 45 |
| Indianapolis, IN | 0.18(C) | | | | 40 |
| Kansas City, KS & MO | 0. | | 0. | | 6 |
| Topeka, KS | NSS | | | | 40 |
| Witchita, KS | | | 1.006 | 0.417-1.593 | 7 |
| Ashland, KT | BDL | | | | 40 |
| New Orleans, LA | 0.88 | | | | 45 |
| Boston, MA | 3.98 | 0-10. | 19.55 | 0-35.7 | 6 |
| Bay City, MI | 1.2 | | 0.03(C) | | 41 |
| Eagle Harbor, MI | 0.13(A) | | 2. | | 42 |
| Marquette, MI | 0.16(A) | | 1. | | 42 |
| Midland, MI | 0.6 | | .001 | | 41 |
| Ontonago, MI | 0.24(A) | | 3.(A) | | 42 |
| Saginaw, MI | | | .0013 | | 7 |
| Beaver Bay, MN | 3.(A) | | | | 43 |
| Beaver Bay, MN | 12.4(A) | | 420 | | 42 |
| Cloquet, MN | 1.0(A) | | 60 | | 42 |
| Duluth, MN | 39.33(A) | 17-74.(A) | 13.3(A) | 2.7-27.(A) | 44 |
| Duluth, MN | 1.62(A) | | 110 | | 42 |
| Duluth, MN | 24 | 10.-35. | | | 41 |
| Duluth, MN | 2.3(A) | 1.-5.(A)<br>BDL-0.4(C) | | | 43 |
| Duluth, MN | | 1.1-120(A) | | | 40 |
| Duluth, MN | | | 190 | 30-800 | 39 |

BDL = Below detection limit (less than 50,000 fibers/1.)
NSS = Not significant (less than 5 fibers in 20 fields).
C = Chrysotile
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Table E-2 (continued)

| | Concentration | | | | Source |
|---|---|---|---|---|---|
| | Millions of Fibers per Liter | | Micrograms per Liter | | (Chapter V Ref.) |
| | Mean | Range | Mean | Range | |
| Grand Marias, MN | 0.02(A) | | 5. | | 42 |
| Grand Marias, MN | 0. | | 0. | | 44 |
| Silver Bay, MN | 0.26(A) | | 50. | | 42 |
| Silver Bay, MN | 2.(A) | | | | 43 |
| Two Harbors, MN | 1.95(A) | | 140 | | 42 |
| Two Harbors, MN | 2.5(A) | | | | 43 |
| Jackson, MS | NSS | | | | 40 |
| Independence, MO | | 0.36-0.58(C) | | | 40 |
| Kansas City, MO | 0.07(C) | | | | 40 |
| Springfield, MO | 0.30(C) | | | | 40 |
| St. Louis, MO | NSS | | | | 40 |
| Elizabeth, NJ | BDL | | | | 40 |
| Jersey City, NJ | 0.016(C) | | | | 40 |
| Manville, NJ | 0.82 | | | | 45 |
| Buffalo, NY | 0.13(C) | | | | 40 |
| Elmira, NY | NSS | | | | 40 |
| Glen Falls, NY | BDL | | | | 40 |
| New York, NY | 0. | | | | 6 |
| New York, NY | 0. | | | | 44 |
| Niagara Falls, NY | NSS | | | | 40 |
| Rochester, NY | BDL | | | | 40 |
| Cincinnati, OH | NSS | | | | 40 |
| Dayton, OH | NSS | | | | 40 |
| Muskogee, OK | BDL | | | | 40 |
| Tulsa, OK | BDL | | | | 40 |
| Bethlehem, PA | NSS | | | | 40 |
| Erie, PA | 0.07(C) | | | | 40 |
| Malvern, PA | | | 0.119 | 0.001-0.977 | 7 |
| Philadelphia, PA | 16.95 | 0.-130. | 0.143 | 0.-0.588 | 6 |
| South Pittsburg, PA | 0.21(C) | | | | 40 |

BDL = Below detection limit (less than 50,000 fibers/l.)
NSS = Not significant (less than 5 fibers in 20 fields).
C = Chrysotile
A = Amphibole



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Table E-2 (concluded)

| | Concentration | | | | Source (Chapter V Ref.) |
|---|---|---|---|---|---|
| | Millions of Fibers per Liter | | Micrograms per Liter | | |
| | Mean | Range | Mean | Range | |
| Newport, RI | | 0.4-1.0(C) | | | 40 |
| Providence, RI | | | 0.409 | 0.267-0.579 | 7 |
| Columbia, SC | 0.13(C) | | | | 40 |
| Greenville, SC | NSS | | | | 40 |
| Chattanooga, TN | 0.13(C) | | | | 40 |
| Chattanooga, TN | 4.7 | | | | 45 |
| Clarksville, TN | 0.09(C) | | | | 40 |
| Memphis, TN | | | 1.696 | | 7 |
| Nashville, TN | | 0.43-0.80(C) | | | 40 |
| Abilene, TX | BDL | | | | 40 |
| Amarillo, TX | 0.09(A) | | | | 40 |
| Dallas, TX | 0. | | 0. | | 6 |
| Houston, TX | 0. | | | | 43 |
| Brattleboro, VT | 0.11(C) | | | | 40 |
| Crystal Springs, VT | NSS | | | | 40 |
| Eden, VT | 0.08(C) | | | | 40 |
| Enosburg, VT | 0.05(C) | | | | 40 |
| Jericho-Underhill, VT | NSS | | | | 40 |
| North Troy, VT | | 0.98-2.2(C) | | | 40 |
| Quarry Hill, VT | NSS | | | | 40 |
| Richmond-Harrington, VT | NSS | | | | 40 |
| Charlottesville, VA | NSS | | | | 40 |
| Seattle, WA | 0.850 | 0.-1.9<br>BDL-1.812(A) | 0.303 | 0.-1.21 | 6 |
| Seattle, WA | | NSS-2.464(C) | | | 40 |
| Ashland, WI | 0.31(A) | | 20. | | 42 |
| Superior, WI | 4.(A) | | 1.4(A) | | 44 |
| Superior (Wells), WI | 0.03(A) | | 0.8(A) | | 42 |
| Cheyenne, WY | NSS | | | | 40 |
| San Juan, PR | NSS | | | | 40 |

BDL = Below detection limit (less than 50,000 fibers/l.)
NSS = Not significant (less than 5 fibers in 20 fields).
C = Chrysotile
A = Amphibole



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## Appendix F

## SMOKING CESSATION PROGRAMS

Although a number of different program strategies may reduce smoking rates, significant long-term cessation of smoking is rare. Most reviews indicate that the greatest rate of recidivism occurs between 1 and 5 weeks following treatment, when only 30% of those who had been abstinent at the end of treatment report continued abstinence. Follow-up at 3-18 months indicates approximate cessation rates of 20%-30%. One report noted 18% cessation at 5 years after treatment.[1]

Most smoking cessation programs have enrolled volunteers, who may have been motivated to quit on their own. Since 16% of persons who quit by themselves have been reported to remain abstinent one year,[2] the 20% to 30% rates of abstinence achieved 3 to 18 months after smoking cessation programs suggest that formal cessation programs may be only of modest benefit. Such programs, however, may be substantially beneficial for persons who might not have been able to quit on their own.

The discussion in this Appendix is intended to provide a broad perspective of the available options. There are several published works that provide a more comprehensive review of the subject.[3,4,5,6,7]

### Counseling by Health Professionals

When they advise patients to quit or reduce smoking, health professionals, particularly physicians, serve as agents of behavior change. One report described the results of a physician counseling program in which 100 surgical patients were seen for a brief interview during which the health hazards and financial burdens of smoking were discussed. After one year, 30% of males and 11% of females had stopped smoking. Following another such program, which included medical lectures, physical examinations, group discussions and films, the quit rate was 58%, and 29% were still abstinent a year later.[8,9]



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A number of health organizations have developed visual aids--pamphlets, films and posters--to assist physicians and others in counseling patients who smoke. These organizations include the American Lung Association, American Cancer Society, National Clearinghouse for Smoking and Health, and the American Heart Association. Titles of a number of the visual aids offered by these organizations are given in the National Cancer Institute's The Smoking Digest (in press).[10] Addresses of the organizations will be found in Table F-1.

Self-Help Program

A recent Gallup poll (1974) indicated that only 34% of smokers expressing a desire to quit were interested in attending cessation clinics. The majority of smokers who became abstinent quit without the use of formal smoking-cessation interventions.[10]

There appears, however, to be a great deal of interest in self-help manuals and kits as judged by the amount of such materials requested during antismoking campaigns.[11] A typical self-help manual includes a behavioral interpretation of smoking, a general explanation of the principles of behavior change, and specific instructions for implementing self-control procedures directed at smoking cessation.[12] For example, the smoker might be instructed to:

- List positive reasons for quitting
- Record the time each cigarette is smoked
- Note feelings and behavior prior to and during each smoke
- Reduce gradually the number of cigarettes smoked
- Impose circumstantial barriers to smoking, such as not carrying matches
- Change brands twice weekly, choosing each time a brand lower in tar and nicotine
- Increase physical activity
- Refrain from smoking for 48 hours
- Avoid situations most closely associated with smoking
- Find substitute behaviors for smoking

The effectiveness of self-help programs has not been established.



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Table F-1

POTENTIAL SOURCES OF ADDITIONAL INFORMATION

| | |
|---|---|
| Action on Smoking and Health (ASH)<br>2000 H Street, NW<br>Washington, D.C. 20006 | (202) 659-4310 |
| American Cancer Society<br>777 Third Avenue<br>New York, New York 10017 | (212) 371-2900 |
| American Health Foundation<br>Department of Behavioral Sciences<br>1370 Avenue of the Americas<br>New York, New York 10019 | (212) 489-8700 |
| American Heart Association<br>7320 Greenville Avenue<br>Dallas, Texas 75231 | (214) 750-5300 |
| American Lung Association<br>1740 Broadway<br>New York, New York 10019 | (212) 245-8000 |
| Canadian Council on Smoking and Health<br>343 O'Connor<br>Ottawa, Ontario K-2P-1V9 | (613) 236-6035 |
| Cancer Information Clearinghouse<br>7910 Woodmont Avenue<br>Suite 1320<br>Bethesda, Maryland 20014 | (301) 565-5955 |
| General Headquarters<br>5 Day Plan to Stop Smoking<br>Seventh Day Adventist Church<br>Narcotics Education Division<br>6840 Eastern Avenue, N.W.<br>Washington, D.C. 20012 | (202) 723-0800 |
| Kaiser Foundation Research Institute<br>1956 Webster Street, Room 310B<br>Oakland, California 94612 | (415) 645-5000 |
| National Association on Smoking and Health<br>4155 East Jewel Avenue<br>Denver, Colorado 80237 | (303) 753-0777 |

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Table F-1 (continued)

| | |
|---|---|
| National Clearinghouse for Smoking and Health<br>Public Health Service<br>U.S. Department of Health, Education & Welfare<br>Center for Disease Control<br>Atlanta, Georgia 30333 | (404) 633-3311<br>X 3235 - Tech. Info.<br>X 3145 - Public Info. |
| National Interagency Council on Smoking and Health<br>419 Park Avenue South<br>New York, New York | (212) 532-6035 |
| Occupational Health and Safety Administration<br>U.S. Department of Labor<br>Constitution Avenue and Third Street, N.W.<br>Washington, D.C. 20210 | (202) 523-7081 |
| Schick Center<br>4101 Frawley Drive<br>Fort Worth, Texas 76118 | (817) 268-1157 |
| Smoking and Health Information Program<br>P.O. Box 2003<br>Tyler, Texas 75710 | (214) 877-3011 |
| SmokEnders<br>Memorial Parkway<br>Phillipsburg, New Jersey 08865 | (201) 454-HELP |
| The Tobacco Institute<br>1776 K Street, N.W.<br>Washington, D.C. 20006 | (202) 457-4800 |
| Tyler Asbestos Workers Program<br>P.O. Box 2003<br>Tyler, Texas 75710 | (214) 877-3011 |

Source: Adopted from The Smoking Digest, National Cancer Institute (in press).



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## Group Therapy and Five-Day Plan

Various health organizations have sponsored community group smoking cessation clinics, which provide health information, encouragement, and group therapy. Groups typically involve 8-18 persons and a group leader, meeting once or twice weekly for a month. Participants in the group are informed of smoking risks, asked to describe why they smoke and to detail their smoking habits, and are then encouraged to follow one of several procedures for quitting. Estimates of the effectiveness of these programs after one year range from 18%-25%.[13,14] Higher success rates result from programs stressing formal long-term maintenance support.

One variant of group therapy is the Five-Day Plan, which consists of five daily meetings, 1-1/2 to 2 hours each. Up to several hundred volunteer participants may be treated at these sessions. Intervention strategies range from lectures and inspirational messages to fear-arousing stimuli and behavior modification procedures. One such program--a live-in clinic including lectures, exercise, and individual as well as group therapy--reported 21%-40% quit rates at a three-month follow-up.[7]

Isolated use of the nonspecific treatment factors characteristic of the smoking clinic approach (e.g., suggestion, high expectation of success in quitting) result in post-treatment cessation rates similar to those of clinics that use specific planned intervention strategies.[3,15] A follow-up of a group of 559 volunteers who had attended such a smoking clinic (pharmacologic agents, health education, and brief suggestions to use certain techniques for quitting) indicated that only 18% remained abstinent at five years.[1]

## Behavior Therapies

The behavior therapy approach assumes that problematic behavior is a function of a person's learned pattern of interacting with the environment. The goal is to teach more adaptive means of responding. The initial step is a "behavioral assessment," an evaluation that includes identification of antecedent events that trigger smoking; determination of thoughts and feelings that influence smoking behavior; analysis of



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personal smoking behavior (frequency, situations); and identification of the psychological consequences of smoking. A number of techniques may then be used to teach the individual new patterns of interacting with environmental smoking stimuli.[4,12] These techniques can be grouped into five major categories; systematic desensitization, punishment and aversive conditioning, stimulus control, reinforcement of nonsmoking, and multicomponent interventions.

Assuming that it is anxiety that elicits the urge to smoke, attempts have been made using relaxation techniques to desensitize smokers systematically to anxiety-evoking stimuli.[16,17,18] However, no substantial effect on smoking behavior has been reported.

A number of aversive conditioning techniques have been employed to modify smoking behavior. Loud noises or electric shocks have been coupled to smoking or to the urge to smoke, generally with little effect.[12]

Two techniques incorporate cigarette smoke as the aversive stimulus:

- Rapid smoking, which requires the individual to smoke rapidly and continuously, sometimes in conjunction with drafts of warm smoky air
- Satiation, which requires increasing cigarette consumption over a certain period of time (e.g., smoking double or triple the usual amount for a week)

In the context of a persuasive interpersonal relationship between patient and therapist, the rapid smoking technique appears to result in about 50% abstinence three to six months following termination.[12] While one report of a satiation program indicated 62% cessation four months following treatment,[19] other programs have not been as successful.[12] Before implementing the rapid smoking method, a medical screening should be required of participants due to the possible deleterious effects of increased carbon monoxide levels.[20]

Another behavioral technique is stimulus control. This may involve forbidding smoking in situations where smoking would habitually occur (e.g., no smoking while drinking coffee, watching TV, or following a meal) as well as gradually restricting the number of situations where smoking is permitted. While (a) there appears to be no clear cessation



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effect from using this method and (b) a high attrition has been reported frequently, reduction in the rate of cigarette consumption does result.[4]

The reinforcement of abstinence through the use of social or monetary incentives has been relatively successful. An example of a technique incorporating a monetary incentive is the use of a deposit made prior to initiation of the program. The money is returned in portions made contingent upon progressively longer periods of abstinence. Fifty percent cessation has been reported at six months as opposed to 24% in a group not employing the incentive.[21,22]

Multicomponent interventions have been designed by combining these techniques. A number of reports indicate that this approach may yield high abstinence rates (65% to 100% immediately; 55% to 65% after one year).[23,27]

Miscellaneous Individualized Techniques

There are a series of cigarette filters designed to assist smoking cessation by gradually reducing levels of inhaled tar and nicotine. The extent to which this method has been effective has not been evaluated.[10]

A diverse array of medications--including stimulants as well as tranquilizers, nicotine substitutes as well as antagonists, and anticholinergics--have been prescribed to assist smokers in overcoming withdrawal symptoms. With regard to actual cessation, however, none of these pharmacologic agents has shown any more promise than placebos.[28] In fact, several studies have suggested that placebo groups may have higher quit rates.[6]

Very little research is available on the effects of acupuncture as a smoking cessation tool. In one study, 50% of subjects were reported abstinent six weeks following auricular acupuncture.[29] This cessation rate is similar to those obtained using less invasive techniques, and, at the present time, the use of acupuncture does not appear to be justified.

Hypnotic techniques have included attempts to reveal personality conflicts assumed to be major underlying causes of the smoking habit as well as direct suggestions to quit.[30,31] While certain investigators



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have reported positive initial results,[32,33] there have been few controlled studies. Furthermore, initial successes have not been maintained at follow-up. One report of the results of a single session self-hypnotic treatment noted a cessation rate at one year of 20%.[34] Using this approach, another investigator has observed significantly higher rates of recidivism than with group counseling.[35]

Three issues related to smoking cessation--monitoring of smoking behavior, the "successful quitter," and gain of weight--are summarized briefly in Table F-2.

## Smoking Cessation Programs in Industry

Industry provides an ideal setting for smoking cessation programs. Industrial programs often have the advantages of:

- An existing system of occupational health care, which affords a means of careful health surveillance and follow-up
- An established network of communications, which permits the rapid dissemination of health information
- Peer and management interaction, which provides for social incentives
- Readily perceived benefits in terms of diminished illness and absenteeism, which can be assessed against program costs.

While the plant physician may wish to refer workers to smoking cessation programs outside the workplace, on-site programs reduce lost time and inconvenience. The local chapter of the American Cancer Society would be an excellent resource for information on available outside programs. (See also other information resources in Table F-1.)

A number of imaginative industry-based smoking cessation programs have been established, but as yet there have been no published reports of their effectiveness. Several programs have been described:[10]

> Intermatic Incorporated (Detroit) has a no-smoking "parimutuel" window where employees can bet up to $100 on their ability to stop smoking for a year. The company has contributed $1,000 to be divided among successful participants. Persons not remaining abstinent donate their bet to the American Cancer Society.



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Table F-2

MONITORING SMOKING BEHAVIOR, THE SUCCESSFUL QUITTER, AND WEIGHT GAIN

- Monitoring Smoking Behavior

  As with any intervention, monitoring of treatment effectiveness is critical. Although self-support has been the major measure used to date, it lacks precision for a variety of reasons which include the desire to please, denial of shortcomings, or inconsistent motivation to maintain accurate records over reporting intervals. Such objective measures as serum thiocyanate or expired-air carbon monoxide, along with self-support, might be useful for monitoring.[36]

- The Successful Quitter

  The successful quitter is likely to be a man, to be concerned about his health, to be older, to report fewer neurotic or psychosomatic symptoms, to smoke less and to have begun smoking at a later age, to have a supportive social milieu, and to have tried to quit on several previous occasions.[1,37] The behavior of spouses appears to be a significant factor. Smokers with nonsmoking spouses, spouses who also quit, or spouses who "made it easier to quit" were more likely to remain abstinent at 5-year follow-up.[1]

- Weight Gain

  A frequent concern expressed by those planning to stop smoking is whether or not they will gain weight. Indeed, three out of four persons in one study gained weight after giving up cigarettes.[38] Specific attention to the possibility of weight gain--i.e., a special dietary or behavioral weight management program--might possibly be used to good advantage in cessation programs.

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The Aluminaire Standard Glass Company (Phoenix) has established a program in which a dollar amount equivalent to what abstinent smokers would have spent for cigarettes is deducted from paychecks. At the end of one year the company matches the total deductions and pays the entire sum to the worker providing he has remained abstinent.

Sears Roebuck and Company (New York) encourages employees to take outside smoking cessation courses by rebating a portion of course fees to those remaining abstinent for six months or more.

An interesting program package including education and social monetary incentives was implemented at the Dow Chemical Company (Freeport, Texas) in collaboration with the American Cancer Society. Abstinence was rewarded by a dollar each week, and abstinent workers were enrolled in monthly and quarterly lotteries for prizes that included a boat and motor as well as cash awards. Ex-smokers were used to recruit program participants. For each recruited participant who remained abstinent for one month, the recruiter was awarded a chance in a lottery. (Recruiter incentive is thought to provide a useful source of social mobilization.)[39] Of 395 participants, only 15 (less than 4%) continued to smoke at program termination; however, the lack of adequate follow-up precludes an assessment of long-term effects.

While these programs are useful in providing rationale and motivation for smoking abstinence, some persons do not possess the skills needed to quit. It would be useful to assist such persons through the use of behavior therapies.

## REFERENCES


1. West DW, et al: Five year follow-up of a smoking withdrawal clinic population. Public Health 67:536-544, 1977.

2. Guilford J: Factors Related to Successful Abstinence from Smoking: Final Report. Los Angeles, American Institute for Research, 1966.

3. Bernstein DA: Modification of smoking behavior: An evaluative review. Psychol Bull 71:418-440, 1969.

4. Bernstein DA, McAlister A: The Modification of Smoking Behavior: Progress and problems. Addict Behav 1:89-102, 1976.

5. Hunt WA, Matarazzo JD: Three years later: recent developments in the experimental modifications of smoking behavior. Abnorma Psychol 81:107-114, 1973.

6. Schwartz JL: A critical review and evaluation of smoking control methods. Public Health Rep 84:489-506, 1969.




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asbestosclaims.law/library/25239/171

7. Schwartz JL, Rider G: Smoking cessation methods in the United States and Canada: 1969-1974. In: Steinfeld J, Griffiths W, Ball K, Taylor Rm,(eds.) Proceedings of the 3rd World Conference on Smoking and Health. DHEW Publication No. (NIH) 77-1413, 1977.

8. Handel S: Change in smoking habits in general practice. Postgrad Med J 49:479-681, 1973.

9. Delarue NC: A study in smoking withdrawal: the Toronto smoking withdrawal study centre: description of activities. Can J Public Health 64:2,5-19, 1973.

10. National Cancer Institute. The Smoking Digest: Progress Report on a Nation Kicking the Habit (in press).

11. Dubren R: Evaluation of a televised stop-smoking clinic. Pub Health Rep 92:81-84, 1977.

12. Lichtenstein E, Danaher BG: Modification of smoking behavior: A critical analysis of theory, research and practice. In Hersen M, Eisler RM, Miller PM, (eds.) Progress in behavior modification (vol 3) New York: Academic Press, 1976.

13. National Clearinghouse for Smoking and Health. Adult use of tobacco: 1975. HEW Public Health Service, 1976.

14. Schwartz JL, Dubitzky M: One-year follow-up results of a smoking cessation program. Can Public Health 59:161-165, 1968.

15. Lichtenstein E, et al: Comparison of rapid smoking, warm, smoky air, and attention placebo in the modification of smoking behavior. J Consult Clin Psychol 40:92-98, 1973.

16. Koenig KP, Masters J: Experimental treatment of habitual smoking. Behav Rese Ther 3:235-243, 1965.

17. Pyke S, McK Agnew N, Kopperud J: Modification of an overlearned maladaptive response through a relearning program: A pilot study on smoking. Behav Rese Ther 4:197-203, 1966.

18. Wagner JK, Bragg RA: Comparing behavior modification approaches to habit decrement-smoking. J Consult Clin Psychol 34:258-263, 1970.

19. Resnick JH: Effects of stimulus satiation on the overlearned maladaptive response of cigarette smoking. J Consult Clin Psychol 32:501-505, 1968.

20. Miller LC, et al: Potential hazards of rapid smoking as a technique for the modification of smoking behavior. N Engl J Med 297:590-592, 1977.

21. Tiche TJ, Elliott R: Breaking the cigarette habit: effects of the technique involving threatened loss of money. Paper presented at the annual meeting of the American Psychological Association, 1967.

22. Winett RA: Parameters of deposit contracts in the modification of smoking. Psychol Rec 23:49-60, 1973.



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asbestosclaims.law/library/25239/172

23. Harris MB, Rothberg C: A self-control approach to reduced smoking. Psychol Rep 31:165-166, 1972.

24. Chapman RF, Smith JW, Layden TA: Elimination of cigarette smoking by punishment and self-management training. Behav Res Ther 9:255-264, 1971.

25. Morrow J, et al: Elimination of cigarette smoking behavior by stimulus satiation, self-control techniques, and group therapy. Paper presented to the meeting of the Western Psychological Association, Los Angeles, April 1973.

26. Pomerleau OF, Ciccone P: Preliminary results of a treatment program for smoking cessation using multiple behavior and modification techniques. Paper presented to the meeting of the Association for Advancement of Behavior Therapy, Chicago, November 1974.

27. Tooley JT, Pratt S: An experimental procedure for the extinction of smoking behavior. Psychol Rec 17:209-218, 1967.

28. Gritz ER, Jarvik ME: Pharmacological aids for the cessation of smoking. In Steinfeld J, Griffiths W, Ball K, Taylor RM, (eds.) Proceedings of the 3rd World Conference on Smoking and Health, DHEW Publication No. (NIH) 77-1413, 1974.

29. Globglas A: Auricular acupuncture and the smoking habit. Nouv Presse Med p. 980, 1974.

30. Bryan WJ: Hypnosis and smoking. J Am Inst Hypn 5:17-37, 1964.

31. Johnston E, Donoghue JR: Hypnosis and smoking: A review of the literature. Am J of Clin Hypn 13:265-272, 1971.

32. Korger WS: Clinical and Experimental Hypnosis. Springfield: Thomas, 1963.

33. von Dedenroth TEA: The use of hypnosis in 1000 cases of "tobaccomaniacs." Am J Clin Hypn 10:194-197, 1968.

34. Spiegel H: A single-treatment method to stop smoking using ancillary self-hypnosis. Int J Clin Exp Hypn 18:235-249, 1970.

35. Shewchuk LA: A comparison of smoking cessation techniques: Initial success and eventual recidivism. Submitted to Public Health Reports, 1976.

36. Vogt TM, et al: Expired air carbon monoxide and serum thiocyanate as objective measures of cigarette exposure. Am J Public Health 67:545-549, 1977.

37. Pederson LL, Lefcoe NM: A psychological and behavioral comparison of ex-smokers and smokers. J Chronic Dis 29:431-434, 1976.

38. Hammond EC, Percy C: Ex-Smokers. NY State J Med 58:2956-2959, 1958.

39. Janis IL, Hoffman D: Facilitating effects on daily contact between partners who make a decision to cut down on smoking. J Pers and Soc Psychol 17:25-35, 1970.



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Appendix G

SOURCES OF EDUCATIONAL MATERIALS

## 1. Sources of Published Educational Materials

### Asbestos Information Association/NA

Materials: Brochures on work practices, fact books, reprints of scientific articles, conference proceedings, and films; reference library.

Address: 1835 "K" Street, N.W.
Washington, D.C. 20006

### Asbestos Information Committee

Materials: Brochures on work practices, health effects, and control procedures.

Address: 10 Wardour Street
London, W1V 3HG
England

### Asbestos Research Council

Materials: Brochures on work practices, ventilation, control procedures, protective devices, and disposal methods.

Address: P.O. Box 18 Cleckheaton
West Yorkshire, BD19 3UJ, England

### Congress of the United States

Materials: Pertinent Public Laws

Address: Document Room, Congress of the United States,
Washington, D.C.

### Johns-Manville Corporation

Materials: Brochures on work practices, health effects, films, slides with sound, slides with script, video tapes, newspaper, bulletins, letters, reports, and reprints of scientific articles.



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Address: Health, Safety and Environment Department
Ken-Caryl Ranch
Denver, Colorado 80217

National Institute of Occupational Safety and Health, U.S. Department of Health, Education and Welfare

Materials: "Criteria Document" on Asbestos; reports of occupational disease research and epidemiologic investigations.

Address: Robert A. Taft Laboratories
4676 Columbia Parkway
Cincinnati, Ohio 45226

National Safety Council

Materials: Reprints of articles, safety data sheets; reference library.

Address: 425 North Michigan Avenue
Chicago, Illinois 60611

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

Materials: Occupational Safety and Health Standard:
Subpart Z, Sec. 1910.1001, Asbestos
Brochures on OSHA 1970

Address: Occupational Safety and Health Administration
U.S. Department of Labor
Washington, D.C. 20210

Oil, Chemical and Atomic Workers International Union, AFL-CIO

Materials: Poster, slide-tape cassette presentation

Address: Citizenship-Legislative Department
1126-16th Street, N.W.
Washington, D.C. 20036

Quebec Asbestos Mining Association

Materials: Brochures on work practices, health effects, and control procedures.

Address: 5 Place Ville Marie
Montreal 113, Quebec, Canada



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## 2. Possible Sources of Published Educational Materials

American Association of Poison Control Centers

c/o Academy of Medicine of Cleveland
Poison Information Center
10525 Carnegie Avenue
Cleveland, Ohio 44105

American Medical Association

535 North Dearborn Street
Chicago, Illinois 60610

American Public Health Association

1015-18th Street, N.W.
Washington, D.C. 20036

Center for Science in the Public Interest

1757 "S" Street, N.W.
Washington, D.C. 20009

Companies mining asbestos ore

Consumer Federation of America

1012-14th Street, N.W., Suite 901
Washington, D.C. 20005

Consumers Union

256 Washington Street
Mount Vernon, New York 10550

Health Research Group

2000 "P" Street, N.W., Suite 708
Washington, D.C. 20036

Insulation Industry Hygiene Research Program

Environmental Sciences Laboratory
Mount Sinai School of Medicine of the City University
of New York
Fifth Avenue and 100th Street
New York, New York 10029



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Local affiliates of American Cancer Society, American Lung Association, and National Safety Council

Manufacturers or fabricators of asbestos products

Mining Enforcement and Safety Administration

U.S. Department of Labor
Washington, D.C. 20210

Scientist's Institute for Public Information

49 East 53rd Street
New York, New York 10022

SOURCE, Inc.

P.O. Box 21066
Washington, D.C. 20009

State Bureaus of Mines

State Departments of Health

State Workers' Compensation Authorities

Vitalograph Ltd.

8347 Quivira Road
Lenexa, Kansas 66215

Workers' Compensation insurance underwriters (casualty insurance companies)

3. Proceedings of Conferences

Public Information in the Prevention of Occupational Cancer.

Proceedings of a Symposium, December 2-3, 1976. Washington, D.C., National Academny of Sciences, 1977.

Proceedings of the Cancer in the Workplace Conference.

November 16, 1976. Piscataway, New Jersey, College of Medicine and Dentistry of New Jersey, 1977.



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asbestosclaims.law/library/25239/177

## Appendix H

## REFERENCES

### Chapter I


1. Merewether ERA, Price CW: Effect of Asbestos Dust on Lungs and Data Suppression in the Asbestos Industry. London, H.M. Stationery Office, 1930.

2. Merewether ERA: Asbestosis and carcinoma of the lung. In Annual Report of the Chief Inspector of Factories for the Year 1947. London, H.M. Stationery Office, 1949, p. 77.

3. Selikoff IJ: Cancer risk of asbestos exposure. In Symposium on the Origins of Human Cancer. Cold Spring Harbor, New York, Cold Spring Harbor Laboratory, September 7-14, 1976.

4. Newhouse, ML, Berry G: Predictions of mortality from mesothelial tumours in asbestos factory workers. Br J Ind Med 33:147-151, 1976.

5. Wagoner JK: Occupational carcinogenesis--The 200 years since Percivall Pott. Ann NY Acad Sci 271:1-4, 1976.

6. Minerals and their nonasbestos analogs. In Electron Microscopy of Microfibers, Mineral Fibers Session. University Park, Pennsylvania, Pennsylvania State University, August 23-25, 1976.

7. Stanton MF, Wrench C: Mechanisms of mesothelioma induction with asbestos and fibrous glass. J. Natl Cancer Inst 48(3):797-821, 1972.

8. Dreessen WC, et al: A Study of Asbestosis in the Asbestos Textile Industry. U.S. Treasury Department, Public Health Service. Public Health Bulletin No. 241, Aug 1938.

9. SRI International. Personal communication, Attorney John Hynan, Occupational Safety and Health Administration, November 18, 1976.

10. U.S. Code of Federal Regulations, Title 40, Part 61.22.

11. U.S. Code of Federal Regulations, Title 40, Part 427.

12. Federal Register 41(15):3286-3287, January 22, 1976.

13. Federal Register 40(51):11865-11869, March 14, 1975.

14. Code of Federal Regulations, Title 21, Parts 133.8 and 133.9.




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asbestosclaims.law/library/25239/178

15. U.S. Code of Federal Regulations, Title 21, Parts 121.101; and personal communication, Donald Miller, Food and Drug Administration, October 24, 1976.

16. U.S. Code of Federal Regulations, Title 16, Part 1500.17(7).

17. Nicholson WJ: Case study 1: asbestos--the TLV approach. Ann NY Acad Sci 271:152-169, 1976.

Chapter II

1. Canadian Mineral Yearbook. Dept. of Energy, Mines and Resources, Mineral Resources Branch, 1974.

2. U.S. Census of Manufactures, U.S. Dept. of Commerce, Bureau of the Census, 1972.

3. Mineral Facts and Problems. U.S. Dept. of Interior, Bureau of Mines, 1975.

4. Commodity Data Summaries. U.S. Dept of Interior, Bureau of Mines, 1976.

5. Asbestos Information Association of North America: Asbestos-general information. Washington, D.C., 1975.

Chapter III

1. Davis JMG, Conlam SW: Experimental studies on the effects of heated chrysotile asbestos and automobile brake lining dust injected into the body cavities of mice. Exp Mol Pathol 19:339-353, 1973.

2. Fondimare A, et al: Quantitative study of the deposition of asbestos in the lung and pleura of subjects with diverse exposures. Proceedings of the Symposium on the Pathology of Asbestos. Rouen, France, Oct 28, 1975.

3. Timbrell V: Inhalation and biological effects of asbestos. In Assessment of Airborne Particles (Mercer TT, Morrow PE, Strober W, eds.), Proceedings of the Third Rochester International Conference on Environmental Toxicity. Springfield, Illinois, CC Thomas, 1972, pp 429-445.

4. Gross P, DeTreville RTP: The lung as an embattled domain against inanimate pollutants. Am Rev Respir Dis 106:684-691, 1972.

5. Evans JC, et al: Studies on the deposition of inhaled fibrous material in the respiratory tract of the rat and its subsequent clearance using radioactive trace techniques. Environ Res 6:180-201, 1973.



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asbestosclaims.law/library/25239/179

6. Wagner JC, et al.: The effects of inhalation of asbestos in rats. Br J Cancer 29:252-269, 1974.

7. Allison AC: Experimental methods--cell and tissue culture: effects of asbestos particles on macrophages, mesothelial cells and fibroblasts. In Biological Effects of Asbestos (Bogoyski, et al., eds.). Lyon, France, International Agency for Research on Cancer, Scientific Pub. No. 8, 1973, pp 89-93.

8. Godwin ML, Jagatic J: Asbestos and mesotheliomas. Environ Res 3 (5-6):391-416, 1970.

9. Sebastien P, et al: Topographic distribution of asbestos fibres in human lungs in relation with occupational and nonoccupational exposure. 4th International Symposium on Inhaled Particles and Vapors. Edinburgh, 1976.

10. Gross P, et al: Ingested mineral fibers: do they penetrate tissue or cause cancer? Arch Environmen Health 29:341-347, 1974.

11. Westlake GE, Spjut HF, Smith MN: Penetration of colonic mucosa by asbestos particles, an electron microscopic study in rats fed asbestos dust. Lab Invest 14(11):2029-2033, 1965.

12. Storeygard AR, Brown AL: Penetration of the small intestinal mucosa by asbestos fibers. Mayo Clinic Proceedings 52:809-812, 1977.

13. Bolton RIE, Davis JMG: The short-term effects of chronic asbestos ingestion in rats. Ann Occup Hyg 19:121-128, 1976.

14. Cunningham HM, Pontefract RD: Asbestos fibers in beverages, drinking waters and tissues: their passage through the intestinal wall and movement through the body. J Assoc Offic Anal Chem 56:976-981, 1973.

15. Pontefract RD: Penetration of asbestos through the digestive wall in rats. Environ Health Perspect 9:213-224, 1974.

16. Cunningham HM, Pontefract RD: Placental transfer of asbestos. Nature 249:117-118, 1974.

17. Kanazawa K, et al: Migration of asbestos fibers from injection sites in mice. Br J Cancer 24:96-106, 1970.

18. Becklake MR, et al: Lung function profiles in the chrysotile asbestos fibers from injection sites in mice. Br J Cancer 24:96-106, 1970.

19. Ashcroft T, Heppleston AG: The optical and electron microscopic determination of pulmonary asbestos fibre concentration and its relation to the human pathological reaction. J Clin Pathol 26:224-234, 1973.

20. Farley ML, et al: Ferruginous bodies in sputa of former asbestos workers. Tyler, Texas, Texas Chest Foundation (unpublished), 1976.



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21. Gross P, deTreville RTP, Haller, MN: Pulmonary ferruginous bodies in city dwellers (a study of their central fiber). Arch Environ Health 19:186-188, 1969.

22. Bignon J, et al: Incidents of pulmonary ferruginous bodies in France. Environ Res 3:430-442, 1970.

23. Selikoff IJ, Hammond EC, Churg J: Carcinogenicity of amosite asbestos. Environ Health 25:183-186, 1972.

24. Langer AM, Mackler AD, Pooley FD: Electron microscopial investigation of asbestos fibres. Environ Health Perspect 9:63-80, 1974.

25. Selikoff IJ: Cancer risk of asbestos exposure, In Origins of Human Cancer (Hiatt HH, Watson JD, Winsten JA, eds.). Cold Spring Harbor, New York, Cold Spring Harbor Laboratory, 1977, pp. 1765-1784.

26. Newhouse ML, et al: Predictions of mortality from mesothelial tumours in asbestos factory workers. Br J Ind Med 33: 147-151, 1976.

27. Gloyne, SR: Two cases of squamous carcinoma of the lung occurring in asbestosis. Tubercle 17: 5-10-, 1935.

28. Lynch KM, Smith WA: Pulmonary asbestosis. III. Carcinoma of lung in asbestos-silicosis. Am J Cancer 24: 56-64, 1935.

29. Merewether ERA: Annual Report of the Chief Inspector of Factories for the Year 1947, London HMSO, 15-17, 1949.

30. Breslow L, et al: Occupations and cigarette smoking as factors in lung cancer. Am J Public Health 44: 171-181, 1954.

31. Doll R: Mortality from lung cancer in asbestos workers. Br J Ind Med 12:81-86, 1955.

32. Knox JF, et al: Mortality from lung cancer and other causes among workers in an asbestos textile factory. Br J Ind Med 25: 292-303, 1968.

33. Howard S, et al: A mortality study among workers in an English asbestos factory. (Submitted to Br J Ind Hyg) 1977.

34. Elmes PC, Simpson MJC: Insulation workers in Belfast, III, Mortality 1940-1966. Br J Ind Med 28: 226-236, 1971.

35. Kogan FM, Gulsenikova NA, Gulevskaya MR: The cancer mortality rate among workers in the asbestos industry of the Urals. Gig Sanit 37: 29-32, 1972.

36. Meurman LO, Kiviluoto R, Hakama M: Mortality and morbidity among the working population of anthophyllite asbestos miners in Finland. Br J Ind Med 31: 105-112, 1974.



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asbestosclaims.law/library/25239/181

37. Nurminen M: A study of the mortality of workers in an anthophyllite asbestos factory in Finland. Work Environ Health 9: 112-118, 1972.

38. McDonald JC, et al: The health of chrysotile asbestos mine and mill workers of Quebec. Arch Environ Hlth 28: 61-68, 1974.

39. Newhouse ML: Asbestos in the work place and the community. Ann Occup Hyg 16: 97-107, 1973.

40. Gloyne SR: The morbid anatomy and histology of asbestosis. Tubercle 14: 550-558, 1933.

41. Wedler HW: Asbestose und Lungenkrebs. Dtsch med Wschr 69:575-576, 1943.

42. Wedler HW: Über den Lungenkrebs bei Asbestose. Dtsch Arch Klin Med 191: 189-209, 1943.

43. Wyers H: That Legislative Measures Have Proved Generally Effective in the Control of Asbestosis, M.D. Thesis, University of Glasgow, U.K., 1946.

44. Leisher F: Primarer Deckzellentumor des Bauchfells bei Asbestose. Arch Gewerbepath Gewerbehyg 13: 382-392, 1954.

45. Wagner JC, Sleggs CA, Marchand P: Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. Br J Ind Med 17: 260-271, 1960.

46. Selikoff IJ, Churg J, Hammond EC: Relation between exposure to asbestos and mesothelioma. New Eng J Med 272: 560-565, 1965.

47. McDonald AD, McDonald JC: Etudes epidemiologiques sur les maladies dues a l'amiante au Canda. Rev Franc Mal Resp 4, Supp 2: 25-38, 1976.

48. Newhouse ML, Thompson H: Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. Br J Ind Med 22: 261-269, 1965.

49. McEwen J, et al: Mesothelioma in Scotland. Br Med J 4: 575-578. 1970.

50. Dalquen P, Dabbert AF, Hinz F: The epidemiology of mesothelioma: A preliminary report on 119 cases from the Hamburg area. Ger Med 15: 89-95, 1970.

51. Hain E, et al: Retrospective study of 150 cases of mesothelioma in the Hamburg area. Int Arch Arbetismed 33: 15-37, 1974.

52. Rubino GF, et al: Epidemiology of pleural mesothelioma in North-Western Italy (Piedmont). Br J Med 29: 436-442, 1972.



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asbestosclaims.law/library/25239/182

53. Zielhuis RL, et al: Pleura mesothelioma and exposure to asbestos; a retrospective case-control study in the Netherlands. Int Arch Occup Hlth 36: 1-18, 1975.

54. Becklake M: Asbestos-related diseases of the lung and other organs. Their epidemiology and implications for clinical practice. Am Rev Respir Dis, 114: 187, 1976.

55. Newhouse ML, et al: A study of the mortality of female asbestos workers. Br J Ind Med 29: 134-141, 1972.

56. Selikoff IJ, Hammond EC, Churg J: Mortality experiences of asbestos insulation workers, 1943-1968. In Pneumoconiosis (Shapiro HA, ed.), Proceedings of the International Conference, Johannesburg, 1969. Cape Town, Oxford University Press: 180-186.

57. Stell PM, McGill T: Asbestos and laryngeal carcinoma. Lancet 2: 416-417, 1973.

58. Stell PM, McGill T: Exposure to asbestos and laryngeal carcinoma. J Laryngol Otol 89:513-517, 1975.

59. Morgan RW, Shettigara PT: Occupational asbestos exposure, smoking, and laryngeal carcinoma. Ann NY Acad Sci 271: 309-310, 1976.

60. Daum SM, Seidman H, Selikoff IJ: Laryngeal and bucco-pharyngeal cancers in asbestos workers. In Proceedings of Third International Symposium on the Detection and Prevention of Cancer. New York, 1976.

61. Newhouse ML, Berry G: Asbestos and laryngeal carcinoma. Lancet 2: 615, 1973.

62. Martischnig KM, et al: Unsuspected exposure to asbestos and bronchogenic carcinoma. Br Med J (6063): 746-749, 1977.

63. Mancuso TF, El-Attar AA: Mortality pattern in a cohort of asbestos workers. A study based on employment experience. J Occup Med 9: 147-162, 1967.

64. Newhouse ML: Cancer among workers in the asbestos textile industry. In Biological Effects of Asbestos (Bogovski P, et al., eds.). Lyon, France, International Agency for Research on Cancer, Scientific Pub. No. 8, 1973, pp. 203-208.

65. Selikoff IJ: Epidemiology of gastrointestinal cancer. Environ Health Perspect 9: 299-305, 1974.



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asbestosclaims.law/library/25239/183

66. Selikoff IJ: Epidemiology of gastrointestinal cancer. Environ Health Perspect 9: 299-305, 1974.

67. Selikoff IJ, Hammond EC, and Churg J: Carcinogenicity of amosite asbestos. Arch Environ Health 25: 183-186, 1972.

68. Hammond EC, Selikoff IJ, and Churg J: Neoplasia among insulation workers in the United States with special reference to intra-abdominal neoplasia. Ann NY Acad Sci 132: 519-525, 1965.

69. Bradshaw E, Schonland M: Oesophageal and lung cancers in Natal African males in relation to certain socioeconomic factors. Br J Cancer 23(2): 275-284, 1969.

70. Timbrell V: Inhalation and biological effects of asbestos. In Assessment of Airborne Particles. Fundamentals, Applications and Implications to Inhalation Toxicity (Mercer TT, Morrow PE, Stober W, eds.). Springfield, Illinois, CC Thomas, 429, 1972.

71. Pooley FD: An examination of the fibrous mineral content of asbestos lung tissue from the Canadian chrysotile mining industry. Environ Res 12: 1281-1298, 1976.

72. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man, Asbestos 14: Lyon, France, International Agency for Research on Cancer, 1977.

73. Nurminen M: The epidemiologic relationship between pleural mesothelioma and asbestos exposure. Scand J Work Environ & Health 1: 128-137, 1975.

74. Wagner JC, et al: Epidemiology of asbestos cancers. Br Med Bull 27: 71-76, 1971.

75. Enterline PE, Henderson V: Type of asbestos and respiratory cancer in the asbestos industry. Arch Environ Health 27: 312-317, 1973.

76. McDonald JC: Cancer in chrysotile mines and mills. In Biological Effects of Asbestos (Bogovski P, et al., eds.). Lyon, France, International Agency for Research on Cancer, Scientific Pub. No. 8, 1973, pp 189-194.

77. McDonald JC, et al: Mortality in the chrysotile asbestos mines and mills of Quebec. Arch Environ Hlth 22: 677-686, 1971.

78. Enterline PE, De Coufle P, Henderson V: Mortality in relation to occupational exposures among retired asbestos workers. Br J Ind Med 30: 162-166, 1973.



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asbestosclaims.law/library/25239/184

79. Weiss W: Mortality of a cohort exposed to chrysotile asbestos. J Occup Med 19: 737-740, 1977.

80. Kleinfeld M, et: Mortality among talc miners and millers in New York State. Arch Environ Health 14: 663-667, 1967.

81. Kleinfeld M, Messite J, Zaki M: Mortality experience among talc miners and millers. J Occup Med 16: 345-349, 1974.

82. Rubino G, et al: Mortality study of talc miners and millers. J Occup Med 18: 186-193, 1976.

83. Newhouse ML: A study of mortality of workers in an asbestos factory. Br J Ind Med 26: 294-301, 1969.

84. Jones HB, Grendon A: Environmental factors in the origin of cancer and estimation of the possible hazards to man. Food Cosmet Toxicol 18: 251-268, 1975.

85. Seidman H, Lilis R, Selikoff IJ: In Short-term asbestos exposure and delayed cancer risk. Proceedings of Third International Symposium on the Detection of Cancer, New York, 1976.

86. Greenberg M, Lloyd Davies TA: Mesothelioma Register 1967-1968. Br J Ind Med 31: 91-104, 1974.

87. Lumley KPS: A proportional study of cancer registrations of dockyard workers. Br J Ind Med 33:108-114, 1976.

88. Doll R: Susceptibility to carcinogenesis at different ages. Gerontol Clin 4: 211-221, 1962.

89. Doll R: Cancer in aging: the epidemiological evidence. Dorn Memorial Lecture, 10th International Conference of the International Union Against Cancer. Houston, Texas, May, 1970.

90. Berry G, Newhouse ML, Turok M: Combined effect of asbestos exposure and smoking on mortality from lung cancer in factory workers. Lancet ii: 476-479, 1972.

91. Saracci R: Asbestos and lung cancer: an analysis of the epidemiological evidence on the asbestos-smoking interaction. Int J Cancer 20: 323-331, 1977.

92. Selikoff LJ, Hammond EC, Churg J: Asbestos exposure, smoking and neoplasia. JAMA 204: 106-112, 1968.

93. Cooper WC, et al: Study of Sheet Metal Workers--Final Report, Contract HSM-099-71-55, Washington, DC, US Dept Health, Education and Welfare, National Institute for Occupational Safety and Health, 1975.



Digitized by Google



Published by AsbestosClaims.Law
asbestosclaims.law/library/25239/185

94. Edge JR: Asbestos related disease in Barrow-in-Furness, Environ Res 11: 244-247, 1976.

95. Fletcher DE: A mortality study of shipward workers with pleural plaques. Br. J Ind Med 29: 142, 1972.

96. Anderson HA, et al: Household-contact asbestos neoplastic risk. Ann NY Acad Sci 271: 311-323, 1976.

97. Lillington GA, Jamplis RW, Differding JR: Conjugal malignant mesothelioma. N Eng J Med 291(11): 583-584, 1974.

98. Selikoff IJ, Churg J, Hammond EC: Asbestos exposure and neoplasia. JAMA 188: 22-26-, 1964.

99. McDonald AD, McDonald JC: Epidemiologic surveillance of mesothelioma in Canada. Can Med J 109: 356-362, 1973.

100. Bohlig H, Hain E: Cancer in relation to environmental exposure In Biological Effects of Asbestos (Bogovski P, et al., eds.). Lyon, France, International Agency for Research on Cancer, Scientific Pub. No. 8, 1973, pp. 217-221.

101. Yazicioglu S, et al: Asbestosis ve solunum sisteminin primer malign tumorleri arasindaki iliskiler uzerinde arastirma: 162 vak'anin dosyasi uzerinde retrospektif inceleme. Tuberkoloz ve Torask, Dergisinin 23: Mayis-Hazarin, Sayi 2-3, Ayri Baski, 1976.

102. Blot WJ, Fraumeni JF: Geographic patterns of lung cancer: industrial correlations. Amer J Epidemiol 103(6): 539-550, 1976.

103. Bohlig H, et al: Epidemiology of malignant mesothelioma in Hamburg. Environ Res 3: 365-372, 1970.

104. Masson TJ, McKay FW, Miller RW: Asbestos-like fibers in Duluth water supply. JAMA 228(8): 1019-1020, 1974.

105. Levy BS, et al: Investigating possible effects of asbestos in city water: surveillance of gastrointestinal cancer incidence in Duluth, Minnesota. Am J Epidemiol 103(4): 362-368, 1976.

106. Reeves AL, et al: Experimental asbestos carcinogenesis. Environ Res 4: 496-511, 1971.

107. Wagner JC, Berry G, Timbrell V: Mesotheliomata in rats after inoculation with asbestos and other materials. Br J Cancer 28: 173-185, 1973.

108. Shabad LM, et al: Experimental studies on asbestos carcinogenicity. J Natl Cancer Inst 52: 1175-1187, 1974.

109. Smith WE: Experimental studies on biological effects of termolite talc on hamsters. U.S. Bur. Mines Inf Cir (IC8639):43-48, 1974.



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asbestosclaims.law/library/25239/186

110. Pott F, Friedrichs KH: Tumoren der Ratten nach i.p. Injektion faserförmiger Staube. Naturwissenschaften 59: 318, 1972.

111. Maltoni C, Annoscia C: Mesotheliomas in rats following the intraperitoneal injection of crocidolite. In Characterization of human tumors (Maltoni C, Davis W, eds.) Proceedings of the Fifth International Symposium on the Biological Characterization of Human Tumors, Bologna, 4-6 April, 1973, 1:115

112. Pott F, Friedrichs KH, Huth F: Ergebnisse aus Tierversuchen zur kanzerogenen Wirking faserformiger Staube und ihre Deutung im Hinblick auf die Tumorentstehung beim Menschen. Zentralbl Bakteriol Hyg 162: 467-505, 1976.

113. Gross P, et al: Experimental asbestosis: The development of lung cancer in rats with pulmonary deposits of chrysotile asbestos dust. Arch Environ Health 15: 343-355, 1967.

114. Reeves AL: The carcinogenic effect of inhaled asbestos fibers. Ann Clin Lab Sci 6: 459-466, 1976.

115. Reeves AL, Puro HE, Smith RG: Inhalation carcinogenesis from various forms of asbestos. Environ Res 8: 178-202, 1974.

116. Gibel W, et al: Tierexperimentelle Untersuchungen über eine kanzerogene Workung nach Asbestfilter Material nach oraler Aufnahme. Arch Pathol 96: 245-250, 1973.

117. Stanton MF, Wrench C: Mechanisms of mesothelioma induction with asbestos and fibrous glass. J Natl Cancer Inst 48(3): 797-821, 1972.

118. Stanton MF: Some etiological considerations of fibre carcinogenesis. In Biological Effects of Asbestos (Bogovski P, et al., eds.) Lyon, France, International Agency for Research on Cancer, Scientific Pub. No. 8, 1973, pp. 289-294.

119. Occella E, Maddalon G: X-ray diffraction characteristics of some types of asbestos in relation to different techniques of communication. Med Lav 54(10): 628-636, 1963.

120. Langer AM, et al: Variation of some physical, chemical and biological properties of chrysotile asbestos subjected to prolonged milling. J Toxicol Environ Health (in press), 1977.

121. Murray M: In Report of the Departmental Committee on Compensation for Industrial Diseases. London, HMSO, 1907, pp. 127-128.



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Published by AsbestosClaims.Law
asbestosclaims.law/library/25239/187

122. Auribault M: Bulletin de l'Inspection du Travail, 1906, p 126.

123. Parkes WR: Asbestos related disorders. Br J Dis Chest, 67(4): 260, 1973.

124. Murphy, Jr. RJ, et al: Low exposure to asbestos gas exchange in ship pipe coverers and controls. Arch Environ Health 25:253-264, 1972.

125. Bader ME, et al: Pulmonary function and radiographic changes in 598 workers with varying duration of exposure to asbestos. Mt Sinai J Med 37(4): 492-500, 1970.

126. Merewether ERA: Asbestosis and carcinoma of the lung. In Annual Report of the Chief Inspector of Factories for the Year 1947. London, HMSO, p. 77, 1949.

127. Gloyne SR: Pneumoconiosis. A historical survey of necropsy material in 1205 cases. Lancet 1:810-814, 1951.

128. Buchanan WD: Asbestosis and primary intrathoracic neoplasms. Ann NY Acad Sci 132(1): 507+, 1965.

129. Selikoff IH, et al: Asbestosis and neoplasia. Am J Med 42(4): 487-496, 1967.

130. Davis JM: Histogenesis and fine structure of peritoneal tumors produced in animals by injections of asbestos. J Natl Cancer Inst 52: 1823, 1974.

131. Gaensler EA, Kaplan AI: Asbestos pleural effusion. Ann Intern Med 74: 178-191, 1971.

132. Elder JL: A study of 16 cases of pleurisy with effusions in ex-miners from Wittenoom Gorge. NZ J Med 2(3): 328-329, 1972.

133. SRI International. Personal communication, EA Gaensler, October 1976.

134. Navratil M., Trippe F: Prevalence of pleural calcification in persons exposed to asbestos dust, and in the general population in the same district. Environ Res 5: 210-216, 1972.

135. Selikoff IJ: The occurrence of pleural calcification among asbestos insulation workers. Ann NY Acad Sci 132(1): 351-367, 1965.

136. Kiviluoto, R: Pleural calcification as a roentgenologic sign of non-occupational endemix anthophyllite-asbestosis. Acta Radiol Supp 194, 1-67, 1960.



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asbestosclaims.law/library/25239/188

137. Burilkov T, Michailova L: Asbestos content of the soil and endemic pleural asbestosis. Environ Res 3: 443-451, 1970.

138. Zolov C, Burilkov T, Babodjov L: Pleural asbestosis in agricultural workers. Environ Res 1: 287-292, 1967.

139. Yazicioglu, S: Pleural calcification associated with exposure to chrysotile asbestos in southeast Turkey. Chest 70: 43-47, 1976.

140. Alden HS, Howell WM: The asbestos corn. Arch Dermatol Syphil 49: 312, 1944.

141. Vigliani EC: The fibrogenic response to asbestos. Med Lav 59(6-7): 401-410, 1968.

142. Wagner JC: Asbestosis in experimental animals. Br J Ind Med 20(1): 1-12, 1963.

143. Wehner AP, et al: Chronic inhalation of asbestos and cigarette smoke by hamsters. Environ Res 10(3): 368-383, 1975.

144. Holt PF, Mills J, Young DK: The early effects of chrysotile asbestos dust on the rat lung. J Pathol Bacteriol 87(1): 15-23, 1964.

145. Webster I: Asbestosis in non-experimental animals in South Africa. Nature (London) 197(4866): 506, 1963.

146. Schuster NH: Pulmonary asbestos in a dog. J Pathol Bacteriol 34: 751-757, 1931.

147. Holt PF: Experimental asbestosis with four types of fibers: importance of small particles. Ann NY Acad Sci 132: 87-97, 1965.

148. Burger BF, Engelbrecht FM: The biological effects of long and short fibers of crocidolite and chrysotile after intrapleural injection into rats. S Am Afr Med J 44(44): 1268-1270, 1970.

149. Davis JMG, Gross P: Are ferruginous bodies an indication of atmospheric pollution by asbestos? In Biological Effects of Asbestos (Bogovski P, et al., eds.). Lyon, France, International Agency for Research on Cancer, Scientific Pub. No. 8, 1973, pp. 238-242.

150. Rahman Q, et al: Biochemical changes caused by asbestos dust in the lungs of rats. Scan J Work Environ Health 1: 50-53, 1975.

151. Singh J, et al: Enzymatic changes in lung tissue of asbestotic guinea pigs. Environ Physiol Biochem 5: 267-272, 1975.



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asbestosclaims.law/library/25239/189

152. Sincock A, Seabright M: Induction of chromosome changes in Chinese hamster cells by exposure to asbestos fibers. Nature 257: 56-69, 1975.

153. Lavappa KS, Fu MM, Epstein SS: Cytogenic studies on chrysotile asbestos. Environ Res 10(2): 165-173, 1975.

154. Beck EG, Holt PF, Manojlovic N: Comparison of effects of macrophage cultures of glass fiber, glass powder, and chrysotile asbestos. Br J Ind Med 29: 280-286, 1972.

155. Bruch J: Response of cell cultures to asbestos fibers. Environ Health Perspect 9: 253-254, 1974.

156. Kaw JL, Zaidi SH: In vitro studies on the cytotoxic action of different varieties of absestos dust on macrophages. Acta Pharmacol Toxicol 36(3): 225-235, 1975.

157. Davies P, et al: Asbestos induces selective release of lysosomal enzymes from mononuclear phagocytes. Nature 251: 423-425, 1974.

158. Allison AC: Effects of silica and asbestos on cells in culture. In Inhaled Particles III (Walton WH ed.) Old Surry, England, Brothers Ltd, 1971, pp 437-445.

159. Harington JS, et al: Mineral fibers: chemical, physical-chemical and biological properties. Adv Pharmacol Chemother 12: 291-402, 1975.

160. Lynch KM: Pulmonary tumors in mice exposed to asbestos dust. Arch Ind Health 15: 207-214, 1957.

161. Smith WE, Miller L, Churg J: An experimental model for study of cocarcinogenesis in the respiratory tract. In Morphology of Experimental Respiratory Carcinogenesis (Nettesheim P, Hanna Jr. MG, Deathenge Jr. JW, eds.). U.S. Atomic Energy Commission Symposium Series 21: 299-316, 1970.

162. Vosamae A: Institute of Experimental and Clinical Medicine, Tullinn, Estonian SSR (RA/74/011). Abstract from International Agency for Research on Cancer, Lyon, France, 1976.

163. Davis JM: Histogenesis and fine structure of peritoneal tumors produced in animals by injections of asbestos. J Natl Cancer Inst 52: 1823, 1974.

164. Wagner JC, Berry G: Mesotheliomas in rats following inoculation with asbestos. Br J Cancer 23: 567-581, 1969.

165. Harington JS: Occurrence of oils containing 3:4-benzpyrene and related substances in asbestos. Nature 193 (4810): 43-45, 1962.



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Published by AsbestosClaims.Law
asbestosclaims.law/library/25239/190

166. Harington JS, Roe FJ: Studies of carcinogenesis of asbestos and their natural oils. Ann NY Acad Sci 132 (Part I): 439-450, 1965.

167. Roe FJC, Walters MA, Harington JS: Tumor initiation by natural and contaminating asbestos oils. Int J Cancer 1: 491-495, 1966.

168. Commins BT, Gibbs GW: Contaminating organic material in asbestos. Br J Cancer 23: 358-362, 1969.

169. Gross P, Harley RA, Jr: Asbestos-induced intrathoracic tissue reactions. Arch Pathol 96: 245-250, 1973.

170. Wagner JC: Studies of the carcinogenic effect of fibre glass on different diameters following intrapleural inoculation in experimental animals. NIOSH Symposium Occupational Exposure Fibrous Glass, Univ. of Maryland, MD, June 1974.

171. Davis JMG, Bolton RE, Garrett J: Penetration of cells by asbestos fibers. Environ Health Perspec 9: 255-260, 1974.

172. Shin ML, Firminger HI: Acute and chronic effects of intraperitoneal injection of two types of asbestos in rats with a study of the histopathogenesis and ultrastructure of resulting mesotheliomas. Am J Pathol 70(3): 291-313, 1973.

173. Pott F, Huth F, Friedrichs KH: Tumorigenic effect of fibrous dust in experimental animals. Environ Health Perspect 9: 313-315, 1974.

174. Roe FJ, et al: The pathological effects of subcutaneous injections of asbestos fibers in mice: migration of fibres to submesothelial tissues and induction of mesotheliomata. Int J Cancer 2: 628-638, 1967.

175. SRI International, Personal communication with FJC Roe, March 23, 1977.

176. Wagner JC, et al: Animal experiments with talc. In (Walton WC, ed) Inhaled Particles and Vapours, IV. New York, Pergamon, 1977.

Chapter IV

1. Daley AR, Zupko AJ, Hebb JL: Technological feasibility and economic impact of OSHA proposed revision to the absestos standard. Roy F. Weston Environmental Consultants-Designers, March 1976 (prepared for: Asbestos Information Association/North America).

2. Schutz LA, Bank W, Weems G: Airborne asbestos fiber concentrations in asbestos mines and mills in the United States. U.S. Bureau of Mines Health and Safety Program Technical Progress Report No. 72, June 1973.



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Published by AsbestosClaims.Law
asbestosclaims.law/library/25239/191

3. Dement JM, Zumwalde RD, Wallingford KM: Asbestos fiber exposures in a hard rock gold mine. Ann NY Acad Sci 271: 345-352, 1976.

4. Kleinfeld M, Messite J, Langer AM: A study of workers exposed to asbestiform minerals in commercial talc manufacture. Environ Res 6: 132-143, 1973.

5. Gidley MD, SRI International. Personal communication, 1977.

6. Curtis RA, Bierbaum PJ: Technological feasibility of the 2 fibers/cc asbestos standard in asbestos textile facilities. Am Ind Hyg Assoc J 36(2): 115-125, 1975.

7. Schneider T: Asbestos dust levels during work with cloths made from liquid dispersed chrysotile. Ann Occup Hyg 15: 425-426, 1972.

8. SRI International Chemical information data base, 1971.

9. Balzer JL, Cooper WC: The work environment of insulating workers. Am Ind Hyg Assoc J 29(3): 222-227, 1968.

10. Harries PG: Asbestos hazards in naval dockyards. Ann Occup Hyg 11: 135-145, 1968.

11. Reitze WB et al: Application of sprayed inorganic fiber containing asbestos: occupational health hazards. Amer Ind Hyg Assoc J 33(3); 178-191, 1972.

12. Lloyd JW: Communication regarding information indicating a potential health hazard for persons exposed to asbestos during the servicing of motor vehicle brake and clutch assemblies. National Institute for Occupational Safety and Health, Rockville, Maryland, August 1975.

13. Rohl AN, et al: Asbestos exposure during brake lining maintenance and repair. Environ Res 12: 110-128, 1976.

14. Castleman BC, et al: The hazards of asbestos for brake mechanics. Public Health Rep 90(3): 254-256, 1975.

15. Murphy RL, et al: Floor tile installation as a source of asbestos exposure. Am Rev Respir Dis 104: 576-580, 1971.

16. Rohl AN, et al: Exposure to asbestos in the use of consumer spackling, patching, and taping compounds. Science 189(4202): 551-553, 1975.

17. Gibbs GW: Fibre release from asbestos garments. Ann Occup Hyg 18: 143-149, 1975.

18. Lumley KPS: Asbestos dust levels inside firefighting helmets with chrysotile asbestos covers. Ann Occup Hyg 14: 285-286, 1971.



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asbestosclaims.law/library/25239/192

Chapter V

1. Asbestos: The need for and feasibility of air pollution controls. Natl Acad Sci, Washington, D.C., 1971.

2. Laamanen A, Noro L, Raunio V: Observations on atmospheric air pollution caused by asbestos. Ann NY Acad Sci 132(1): 240-254, 1965.

3. Cowherd C: The Impact of Fugitive Emissions of Fine Particles. Session III of the Symposium on Fugitive Emissions Measurement and Control. Hartford CT, EPA-600/2-76-246, May 1976.

4. Harris RL, Jr., and Frazer DA: A model for deposition of fibers in the human respiratory system. Am Ind Hyg Assoc J 37(2): 73-89, 1976.

5. United States of America vs. Reserve Mining Company. Court Proceedings No. 5-72, Civil 19, United States Court of Appeals, 8th Circuit, June 1974.

6. Stewart IM: Asbestos in the Water Supplies of Ten Regional Cities. U.S. Environmental Protection Agency, EPA-560/6-76-017, 1976.

7. American Water Works Association: A study of the problem of asbestos in water. Am Water Works Assoc J 66(9 pt.2): 1-22, 1974.

8. Harington JS, Allison AC, Badami DV: Mineral fibers: Chemical, physiochemical, and biological properties. Adv Pharmacol Chemother 12: 291-402, 1975.

9. Clark SG, Holt PF: Studies on the chemical properties of chrysotile in relation to asbestosis. Ann Occup Hyg 3: 22-29, 1961.

10. Reimschussel G. In Kramer JR, Mudroch O, and Tihor S: Asbestos in the Environment. McMaster University, prepared for Research Advisory Board, International Joint Commission and Environment Canada, June 5, 1974.

11. Aaronson T, Kohl G: Papiermaché. Environ 14(10): 25-26, 1972.

12. Thompson RJ, Morgan GB: Determination of Asbestos in Ambient Air. Presented at Identification and Measurement of Environmental Pollutants Symposium, Ottawa, Ontario, Canada, June 14-17, 1971.

13. Murchio JC, Cooper WC, DeLeon A: Asbestos Fibers in Ambient Air of California. California Air Resources Board, March 1973.

14. Henry WM, et al: Development of a rapid survey method of sampling and analysis for asbestos in ambient air. Columbus, Ohio, Battelle Laboratories, Contract No. CPA 22-69-110, February 29, 1972.



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asbestosclaims.law/library/25239/193

15. Selikoff IJ, Nicholson WJ, Langer AM: Asbestos air pollution. Arch Environ Health 25: 1-13, 1972.

16. Asbestos in the Great Lakes Basin, International Joint Commission, Great Lakes Research Advisory Board, 1975.

17. SRI International. Personal communication, representative of Johns-Manville Corp, 1976.

18. Harwood CF, Siebert P, Blaszak TP: Assessment of particle control technology for enclosed asbestos sources. IITRI, EPA-650/2-74-088, October 1974.

19. U.S. Environmental Protection Agency: Background information on national emission standards for hazardous pollutants - Proposed amendments to standards for asbestos and mercury. EPA-450/2-74-009a, 1974.

20. Wesolowski JJ, et al: Asbestos measured in the California environment. In Recent Advances in the Assessment of the Health Effects of Environmental Pollution, Vol. III. Commission of the European Communities, 1975.

21. John W, et al: Experimental Determinations of the Number and Size of Asbestos Fibers in Ambient Air. California Dept. of Health, ARB 3-688, 1976.

22. Federal Register. 38(66): 8829-8830, April 6, 1973.

23. Brodeur, P: The Expendable Americans. New York, Viking Press, 1973.

24. Harwood, CF: Asbestos air pollution from the wear of brake linings. IITRI, Chicago, April 1972.

25. Jacko MG, Du Charme RT, Somers JH: How much asbestos do vehicles emit? Auto Eng 81(6): 38-40, 1973.

26. Lynch JR: Brake Lining decomposition products. J Air Pollut Control Assoc 18(12): 824-826, 1968.

27. Anderson AE, et al: Asbestos emissions from brake dynamometer tests. SAE Trans, May 14-18, 1973.

28. Nicholson WJ, Rohl AN, Weisman I: Asbestos Contamination of the Air of Public Buildings. Mt. Sinai School of Medicine. U.S. Environmental Protection Agency, No. 450/3-76-001, 1973.

29. New York Times, January 4, 1977, p. 31.

30. Asbestos fallout. Eng News Rec 193(13): 41, 1974.



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31. Sawyer RN: Asbestos exposure in a Yale building. Environ Res 13: 146-169, 1977.

32. Nicholson WJ, Pundsack FL: Asbestos in the environment. In: Biological Effects of Asbestos (Bogovski, et al., eds.). Lyon, France, International Agency for Research on Cancer, Scientific Pub. No. 8, 1973, pp. 126-130.

33. Harwood CF: Asbestos air pollution control. Illinois Institute for Environmental Quality, PB-205208, 1971.

34. Harwood CF, Blaszak TP: Characterization and control of asbestos emissions from open sources. IITRI, EPA-650/2-74-090, September 1974.

35. Federal Register 39(208) 48292-48311, October 14, 1975.

36. Newhouse ML, Thompson H: Epidemiology of mesothelial tumors in the London area. Ann NY Acad Sci 132-579, 1965.

37. Millette JR: Analyzing for asbestos in drinking water. News of Environ Res in Cincinnati. U.S. Environmental Protection Agency, January 16, 1976.

38. Hallenbeck WH, et al: Is chrysotile asbestos released from asbestos cement pipe into drinking water? J Amer Water Works Assoc, February 1978, pp 97-102.

39. Cook PM, Glass GE, Tucker JH: Asbestiform amphibole minerals: Detection and measurement of high concentrations in municipal supplies. Science 85(154): 853-855, 1974.

40. Preliminary Assessment of Suspected Carcinogens in Drinking Water (Appendices). Interim Report to Congress, U.S. Environmental Protection Agency (June 1975).

41. Beaman DR, File DM: Quantitative determination of asbestos fiber concentrations. Anal Chem 48(1): 101-110, 1976.

42. Cook PM, Semi-Quantitative Determination of Asbestiform Amphibole Mineral Concentrations in Western Lake Superior Water Samples. In Advances in X-Ray Analysis, 18, 1975.

43. Cooper RC, Murchio JC, Preliminary Studies of Asbestiform Fibers in Domestic Water Supplies. Berkeley, University of California, AMRL-TR-74-125, Paper No. 5, 1974.

44. Nicholson, WJ: Analysis of amphibole asbestiform fibers in municipal water supplies. Environmental Health Perspectives 9: 165-172, 1974.



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45. SRI International. Personal communication, P Tobin, Environmental Protection Agency re Phase 2 data developed under contract with McCrone Associates, 1976.

46. FDA detains lima beans contaminated with asbestos after accident. Food Chemical News, September 24, 1973.

47. Code of Federal Regulations, Title 21, Section 121, Parts 2520, 2562, 2576, 2587, 1975.

48. Wolff AH, Oehme FW: Carcinogenic chemicals in food as an environmental health issue. J Am Vet Med Assoc 164(6): 623-629, 1974.

49. National Institute for Occupational Safety and Health (NIOSH), Report of Review Committee, NIOSH Pub No HSM-72-10267, 1972.

50. Cunningham HM, Pontefract RD: Symposium on industrial chemicals as food contaminants. J Assoc Off Anal Chem 56(4): 976-981, 1973.

51. Wehman HJ, Plantholf BA: Asbestos fibrils in beverages. PTI Gin. Bull Environ Contam Toxicol 11(3): 267-272, 1974.

52. Federal Register 40(51): 11866-11869, March 14, 1975.

53. Bernstein IL, Moteff J: Possible asbestos hazards in clinical allergy. J Allergy Clin Immunol 57(5): 489-492, May 1976.

54. Council on Dental Therapeutics, Council on Dental Materials and Devices: Hazards of asbestos in dentistry. J Am Dent Assoc 92: 777-778, April 1976.

55. SRI International. Personal communication with an industry source, 1976.

56. Schultz RZ, Williams CR: Commercial talc--Animal and mineralogical studies. J Ind Hyg 24: 75-79, 1942.

57. Kleinfeld M, Messite J, Langer AM: A study of workers exposed to asbestiform minerals in commercial talc manufacture. Environ Res 6: 132-143, 1973.

58. Cralley LJ, et al: Fibrous and mineral content of cosmetic talcum products. Am Ind Hyg Assoc J 29: 350-354, 1968.

59. Rohl AN, et al: Consumer talcums and powders: mineral and chemical characterization. J Tox Environ Health 2: 255-284, 1976.

60. Brobst DA, Pratt WP: United States Mineral Resources. U.S. Geological Survey Professional Paper 820, 1973.



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Published by AsbestosClaims.Law
asbestosclaims.law/library/25239/196

61. SRI International. Personal communication, R. Mickus, Rice Growers Association, October 1976.

62. SRI International. Personal communication, D. Miller, Food & Drug Administration, October 1976.

63. Blejer HP, Arlon R: Talc: A possible occupational and environmental carcinogen. J Occup Med 15(2): 92-97, 1973.

64. Eisenberg WV: Inorganic particle content of food and drugs. Environ Health Perspect 9: 183-191, 1974.

65. SRI International. Personal communication, Pitco, Ford Gum and Machine Co, October 1976.

66. Federal Register 40(51): 11866-11869, March 14, 1975.

67. Nicholson WJ, et al: Occupational and community asbestos exposure from wallboard finishing compounds. Bull NY Acad Med 51(10): 1180-1181, 1975.

Chapter VI

1. ACGIH: Industrial Ventilation--A Manual of Recommended Practice, published yearly.

2. U.S. Environmental Protection Agency: Asbestos: A review of selected literature through 1973 relating to environmental exposure and health effects. 29, 1976.

3. Smith, Winslow: Composition for inhibiting asbestos fiber dust, US Pat Off No. 3928000.

4. Stanton MF: Fiber carcinogenesis: Is asbestos the only hazard? J Natl Cancer Inst 52: 633-634, 1974.

5. US Bureau of Mines: Asbestos--a materials survey. Information Circular 7880, Washington, D.C.,

6. Engineering Equipment Users Association Handbook #33: Recommendations for Handling Asbestos, London, 1969.

7. Bruckman L, Rubino RA: Asbestos: rationale behind a proposed air quality standard. J Air Pollut Control Assoc 25(12): 1207-1215, 1975.

8. Federal Register 40(197), October 9, 1975, p 47662.

9. U.S. Environmental Protection Agency: Control Techniques for Asbestos Air Pollutants. USEPA Publ #AP-117, Research Triangle Park, NC, 27711, Feb. 1973.



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asbestosclaims.law/library/25239/197

10. Harwood CF, Siebert P, Blaszak TP: Assessment of Particle Control Technology for Enclosed Asbestos Sources. EPA Publ #EPA 650/2-74-088, USEPA, Research Triangle Park, NC. October 1974. (Also available as NTIS Publ #PH239926).

11. Harwood CF, et al: Asbestos emissions from baghouse controlled sources. J Am Ind Hyg Assoc 36(8): 595-603, Aug. 1975.

12. Seibert PC, Ripley TC, Harwood CF: Assessment of Particle Control Technology for Enclosed Asbestos Sources-Phase II. EPA Publ #EPA 600/2-76-065, USRPA, Research Triangle Park, NC, 27711, March 1976.

13. Krenkel PA: Waste treatment methodology. In Industrial Pollution (Sax, ed.), 1974.

14. Levy BS, et al: Investigating possible effects of asbestos in city water: surveillance of gastrointestinal cancer incidence in Duluth, Minnesota. Am J Epidemiol 103: 362-368, 1976.

15. Lawrence J, et al: Asbestos: its removal from potable water. Can Res Dev Nov/Dec: 29-30, 1973.

16. Lawrence J, et al: Removal of asbestos fibers from potable water by coagulation and filtration. Water Research 9: 397-400, 1975.

17. Baumann RE: Diatomite Filters for Asbestiform Fiber Removal from Water. Proc of Am Water Works Assoc, 95th Annual conf, Minneapolis, 1975.

18. Federal Register 40(6): 1873-1878, 1975.

19. U.S. Environmental Protection Agency: National emissions standards for hazardous air pollutants: Amendments to standards for asbestos and mercury. Federal Register 40(199): 48292-48302, Oct. 14, 1975.

20. Harwood CF, Ase P, and Stinson M: Study of the Effect of Asbestos Waste Piles on Ambient Air. In Proc of Symp on Fugitive Emission Measurement and Control. Washington D.C., Environmental Protection Agency, EPA Publ #EPA-600/2-76-246, p. 183-202, Sept. 1976.

Chapter VII

1. Tokuhata GK: Cancer of the lung: host and environmental interaction In Cancer Genetics (Lynch HT, Thomas CC, ed.), Springfield, Illinois, 1976.

2. Cole P, Goldman MB: Occupation. In Persons at High Risk of Cancer (Fraumeni Jr., F, ed.) New York, Academic Press, 1975, pp. 167-183.



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3. Montgomery RD, Stirling GA, Hamer NA: Bronchiolar carcinoma in progressive systemic sclerosis. Lancet 1: 486-487, 1964.

4. Godeau P, et al: Carcinome bronchioloalveolaire et sclerodermie. Sem Hop Paris 50: 1161-1168, 1974.

5. Koch BL: Familial fibrocystic pulmonary dysplasia: observations in one family. Can Med Assoc J 92: 801-808, 1965.

6. Swaye P, et al: Familial Hamman-Rich Syndrome: report of eight cases. Diseases Chest 55: 7-12, 1969.

7. McKusick VA, Fisher AM: Congenital cystic disease of the lung with progressive pulmonary fibrosis and carcinomatosis. Ann Int Med 48: 774-790, 1958.

8. Boucot KR, et al: Cigarettes, cough, and cancer of the lung. JAMA 196: 985-990, 1966.

9. Campbell AH, Lee EJ: The relationship between lung cancer and chronic bronchitis. Br J Dis Chest 57: 113-119, 1963.

10. Dean G: Lung cancer and bronchitis in Northern Ireland, 1960-2. Br Med J 1: 1506-1514, 1966.

11. Van der Wal AM, et al: Cancer and chronic non-specific lung disease. Scand J Resp Dis 47: 161-172, 1966.

12. Wynder EL, Fairchild, Jr. EP: The role of a history of persistent cough in the epidemiology of lung cancer. Am Rev Resp Dis 94: 709-720, 1966.

13. Tokuhata GK, Lilienfeld AM: Familial aggregation of lung cancer in humans. J Natl Cancer Inst 30(2): 289-312, 1963.

14. Cohen BH, et al: A common familial component in lung cancer and chronic obstructive pulmonary disease. Lancet ii: 523-526, 1977.

15. Wagner JC, et al: The effects of inhalation of asbestos in rats. Br J Cancer 29: 252-269, 1974.

16. Kellermann G, Luyten-Kellerman M, Shaw CR: Genetic variation of aryl hydrocarbon hydroxylase in human lymphocytes. Am J Hum Genet 25: 327-331, 1973.

17. Kellerman G, Shaw CR, Luyten-Kellermann M: Aryl hydrocarbon hydroxylase inducibility and bronchogenic carcinoma. N Engl J Med 289(18): 934-937, 1973.



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18. Shaw CR: The microsomal mixed function oxidases and chemical carcinogens. In Isozymes III Developmental Biology. San Francisco, Academic Press, Inc., 1975.

19. Paigen B, et al: Questionable relation of aryl hydrocarbon hydroxylase to lung cancer risk. N Eng J Med 297(7): 346-350, 1977.

20. Saccomanno G, et al: Development of carcinoma of the lung as reflected in exfoliated cells. Cancer 33(1): 256-270, 1974.

21. Lilienfeld A (Chairman), et al: An evaluation of radiological and cytological screening for early detection of lung cancer. A co-operative pilot study of the American Cancer Society and the Veterans Administration. Cancer Res 26: 2083-2147, 1966.

22. SRI International. Personal communication with G Saccomanno, November 1976.

23. Hammond EC: Tobacco. In Persons at High Risk of Cancer. (Fraumeni Jr., JF, ed.) New York, Academic Press, 1975.

24. Rothman KJ: Alcohol. In Persons at High Risk of Cancer. (Fraumeni, Jr., JF, ed.) New York, Academic Press, 1975.

25. Lynch HT: Miscellaneous problems, cancer, and genetics. In Cancer Genetics. Springfield, Illinois, CC Thomas, 1976.

26. SRI International. Personal communication, EA Gaensler, October 1976.

27. Weiss W: Cigarette smoking, asbestos, and pulmonary fibrosis. Am Rev Respir Dis 104: 223-227, 1971.

28. Turner-Warwick M: Immunology and asbestosis. In Biological Effects of Asbestos (Bogovski P, et al., eds.). Lyon, France, International Agency for Research on Cancer, Scientific Pub. No. 8, 1973, pp. 258-263.

29. Kang K-Y, et al: T-lymphocytes in asbestosis. N Engl J Med: 291: 735-736, 1974.

30. Merchant JA, et al: The HL-A system in asbestos workers. Br Med J 1: 189-191, 1975.

31 Becklake MR: Asbestos-related diseases of the lung and other organs, their epidemiology and implications for clinical practice. Am Rev Respir Dis 114: 187-227, 1976.

32. Brett GZ: The value of lung cancer detection by six-monthly chest radiographs. Thorax 23: 414-420, 1968.



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33. Boucot KR, Weiss W: Is curable lung cancer detected by semiannual screening? JAMA 224(10): 1361-1365, 1973.

34. Lilienfeld AM, Kordan B: A study of variability in the interpretation of chest X-rays in the detection of lung cancer. Cancer Res 26: 2145-2147, 1966.

35. Archer PG, et al: A study of variability in the interpretation of sputum cytology slides. Cancer Res 26: 2122-2144, 1966.

36. Fontana RS, et al: The Mayo lung project for early detection and localization of bronchogenic carcinoma, a status report. Chest 67: 511-512, 1975.

37. Fontana RS: Early diagnosis of lung cancer. Am Rev Resp Dis 116(3): 399-402, 1977.

38. Weiss W, Seidman H, Boucot KR: The Philadelphia pulmonary neoplasm research project: thwarting factors in period screening for lung cancer. Am Rev Resp Dis 111: 289-297, 1975.

39. Baker RB, et al: The detection and treatment of early lung cancer. Ann Surg 179(5): 813-818, 1974.

40. Grzybowski S, Coy P: Early diagnosis of carcinoma of the lung: simultaneous screening with chest X-ray and sputum cytology. Cancer 23: 113-120, 1970.

41. Whitwell F, Newhouse ML, Bennett DR: A study of the histological types of lung cancer in workers suffering from asbestosis in the United Kingdom. Br J Ind Med 31: 298-303, 1974.

42. International Labour Office: ILO-U/C classification of radiographs of pneymoconioses. Geneva, Switzerland, 1971.

43. Elmes PC: The natural history of diffuse mesothelioma. In Biological Effects of Asbestos (Bogovski P, et al., eds.). Lyon, France, International Agency for Research on Cancer, Scientific Pub. No. 8, 1973, pp. 267-272.

44. Elmes PC, Simpson MJC: The clinical aspects of mesothelioma. Quart J Med, New Series, XLV (179): 427-449, 1976.

45. Winawer SJ, et al: Feasibility of fecal occult-blood testing for detection of colorectal neoplasia: debits and credits. Cancer 40 (Sup 5): 2616-2619, 1977.

46. Stephens FO, Lawrenson KB: The pathologic significance of occult blood in feces. Dis Colon Rectum 13(6): 425-428, 1970.



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47. Greegor DH: Occult blood testing for detection of asymptomatic colon cancer. Cancer 28(1): 131-134, 1971.

48. Ostroe JD, et al: Sensitivity and reproducibility of chemical tests for fecal occult blood with an emphasis on false-positive reactions. Am J Dig Dis 18(11): 930-940, 1973.

49. Sherlock P, Winawer SJ: Modern approaches to early identification of large-bowel cancer. Am J Dig Dis 19(10): 959-964, 1974.

50. Prolla JC, Kobayashi S, Kirsner JB: Gastric cancer. Arch Intern Med 124: 239-246, 1969.

51. Selikoff IJ: The occurrence of pleural calcification among asbestos insulation workers. Ann NY Acad Sci 132: 351-367, 1965.

52. Vogt TM, et al: Expired air carbon monoxide and serum thiocyanate as objective measures of cigarette exposure. Am J Public Health 67: 545-549, 1977.

Chapter VIII

1. Code of Federal Regulations. Title 29, Chap. XVII, Subpart Z, Section 1910.1003-1910.1029.

2. Committee on Public Information in the Prevention of Occupational Cancer, Division of Medical Sciences, Assembly of Life Sciences, National Research Council: Informing Workers and Employers about Occupational Cancer. Washington, D.C., National Academy of Sciences, 1977, p. 26.

3. Felton JS: Health education--a responsibility of the occupational health professional. J Occpp Med 19: 346-350, May 1977.

4. Felton JS: Orienting the new employee in the services of the industrial medical department. Indust Med 16: 519-525, November 1947.

5. Felton JS: Orientation of the new employee by the health division of an atomic energy research laboratory--a four-year review. Indust Med and Surg 21: 107-110, March 1952.

6. Expert Committee on Health Education of the Public--First Report, Geneva, World Health Organization, October 1974, p. 22.

7. Scope, Objectives, and Functions of Occupational Health Programs (Revised December 1971). Chicago, American Medical Association, 1971.

8. Moser RH: Psychosemantics (On Speaking to Patients). In Diseases of Medical Progress: A Study of Iatrogenic Disease, Ed. 3. (Moser RH, ed.). Springfield, Illinois, CC Thomas, 1969, p. 812.



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9. Council on Occupational Health, American Medical Association: The Role of Medicine Within a Business Organization. JAMA 210: (Nov 24) 1969.

10. Joint Statement of the Role of the Registered Nurse in Employee Health Programs--California Medical Association, California Nurses' Association, California Hospital Association, in Joint Statements (of the Associations). Sacramento California Nurses' Association, 1972, p 27.

11. Samuels S: In Public Information in the Prevention of Occupational Cancer. Proceedings of a Symposium held in Washington, D.C. December 2-3, 1976. Washington, D.C., National Research Council, National Academy of Sciences, 1977, p. 147.

12. Mancuso, TF: Help for the Working Wounded. Washington, D.C., National Association of Machinists and Aerospace Workers, 1976.

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