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Occupational and Environmental Exposure to Asbestos

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Introduction

The usefulness of asbestos as an industrial material must be considered to understand the breadth of its consequent public health impact. Since its discovery as an indestructible material centuries ago, it has been used in countless applications, often because no identified substance rivals its engineering or commercial performance.

Asbestos applications result from its many unique physical attributes. Its high tensile strength stabilizes mixtures with concrete, asphalt, and plastic. Asbestos also offers a stable material for frictional use, that is, as a brake surface. Because of the length and pliability of its fibers, it has been incorporated into specially manufactured products, including gaskets, pads, fabric sheets, and asbestos paper with intrinsic properties of resistance and strength. Because it blocks heat transfer and is itself fireproof, it represents an ideal insulation material. Mixed into a slurry, it has been applied in economical fashion to building surfaces for fire protection and heat retention. In both its fabric and compacted-brick forms, it has been used to encase furnaces and kilns.

Economic advantages of asbestos must also be considered in explaining its widespread application. As a natural (mined) rather than a manufactured substance, it was more available and its use not as closely evaluated by producers or consumers. Present in natural deposits on several continents, it has remained easily available for construction and industrial exploitation by all nations, both industrialized and developing. Its production cost, as a truly raw material, has always been far less than substitute agents, which require manufacture and even technology licensing.

Historical Origin and Applications

Preindustrial Applications

The first recorded use of asbestos is as a wick material for oil lamps in ancient times. The material's name originates from the Greek for "inextinguishable" or "indestructible."[1] Woven into cloth, asbestos provided nearly miraculous resistance to fire, especially impressive for shrouds of the deceased whose cremation was open to public display.

Combining asbestos with clay and other malleable materials is also cited as one of the earliest applications of the material. In Finland in 2500 BC, asbestos was added to clay pots for greater strength. Asbestos as a fortifying additive remains its major present-day use as a component of cement, concrete, paint, vinyl, and tar mixtures, accounting for 70% of current applications worldwide.

The Modern Period

The past decades have witnessed a drastic change in America's patterns of asbestos use. Regulatory and health issues, rather than direct economic and engineering factors, now dominate. In the United States, regulatory concern regarding asbestos' use and continued presence has continually grown. A ban on the use of asbestos was proposed by the U.S. Environmental Protection Agency (EPA) in 1989 to prohibit the manufacture, importation, processing and distribution, and commerce of certain asbestos-containing products.[2] The provision also called for labeling requirements. However, in October 1991, the United States Court of Appeals for the 5th Circuit vacated and remanded most of the EPA Asbestos Rule. The legal implications of the court's decision forced the EPA to revise its rule under the Clean Air Act (CAA) and Toxic Substances Control Act (TSCA). The ban under the TSCA includes (1) corrugated paper, (2) roll board, (3) commercial paper, (4) specialty paper, (5) flooring, and (6) new uses of asbestos. Products not currently banned include asbestos cement products, roofing felt and coatings, asbestos cement shingle, millboard, asbestos automatic transmission components, clutch facings, friction materials, disc brake pads, and brake linings. Under the Clean Air Act most spray-applied surfacing asbestos containing materials that have more than 1% asbestos are banned, as are wet-applied and preformed asbestos pipe insulation.[3]

The Collegium Ramazzini, an international, nongovernmental organization that promotes public policy on occupational and environmental issues, has proposed an international ban on asbestos. The members believe substitutes for asbestos exist that are safer and note that many European countries have already banned asbestos use.[4] However, others have criticized their proposal arguing that the risks of continued asbestos use have been exaggerated and that health studies have not yet determined the risk of substitute materials.[5]

The late Dr. Irving J Selikoff, whose scientific, clinical, and public affairs careers are synonymous with asbestos and its health effects, categorized the societal impact of asbestos disease into three population "waves" of asbestos exposure and consequent clinical disease. Because of the well-documented latent interval for asbestos-related disease, the public health impact from each period of asbestos disease trails the period of exposure by 30 to 50 years.

The first wave of asbestos exposure comprises the workers whose activities actually generate asbestos for use, including the miners and packagers who transformed an ore into an industrial material. This exposure period, involving relatively few workers, extends from the initial use of the mineral into the early twentieth century. These workers, in countries where asbestos was first processed, Canada and South Africa, prompted the initial recognition of the diseases that required a latent period of decades to manifest themselves.[6]

The second wave of asbestos-induced disease represents the impact of the manufacturing and construction use of the material. The most important peak in Western society's exposure to asbestos occurred during the period of rapid economic expansion surrounding World War II. Intense and high-volume ship construction, structural insulation, and the industrial fabrication of asbestos-containing products created a huge cohort of exposed workers during the mid-twentieth century. The ensuing period of public health impact manifested itself from the 1970s through the 1990s.

The third wave of asbestos exposure and disease generates the most controversy and conjecture regarding both its size and the intensity of its public health impact. This comprises the cohort of citizens exposed to asbestos already in place. This population is likely to be exposed during the disruption of preapplied asbestos insulation in homes and commercial buildings. Specific groups exposed to the highest dose of the mineral during this phase include building maintenance workers, construction workers, electricians, custodians, and the workforce employed specifically for asbestos abatement. The public health impact is seen at the present time. Disasters such as the collapse of the World Trade Center on September 11, 2001, raise concerns regarding the release of in-place asbestos into the ambient air and possible health effects of such exposures (Figures 2-1 and 2-2).

Worldwide production of asbestos declined between 1980 and 2000, but worldwide use of asbestos remains sizable despite the increased recognition of its health consequences. In the United States, use of asbestos had markedly diminished even before the U.S. Environmental Protection Agency (EPA) ban in July 1989. Although the present rule still allows significant uses of asbestos, U.S. consumption of asbestos dropped from a high of 801,000 metric tons in 1973 to minimal amounts recently (Figure 2-3).

Occupational Exposure to Asbestos

Asbestos Processing

In the United States, for geological reasons, asbestos production has never been an important commercial enterprise. Even before restrictions for asbestos use, the combined workforce involved in mining and milling was known to be fewer than 600 people.[7] Mining creates exposure levels that are surprisingly low when compared to those of materials manufactured, averaging 0.9 fibers/ml.[1] Because of the way the ore is handled, the fibers remain consolidated and have not yet become individualized. In contrast, the subsequent operation of mineral refining and milling (usually designed to "open" the bundles

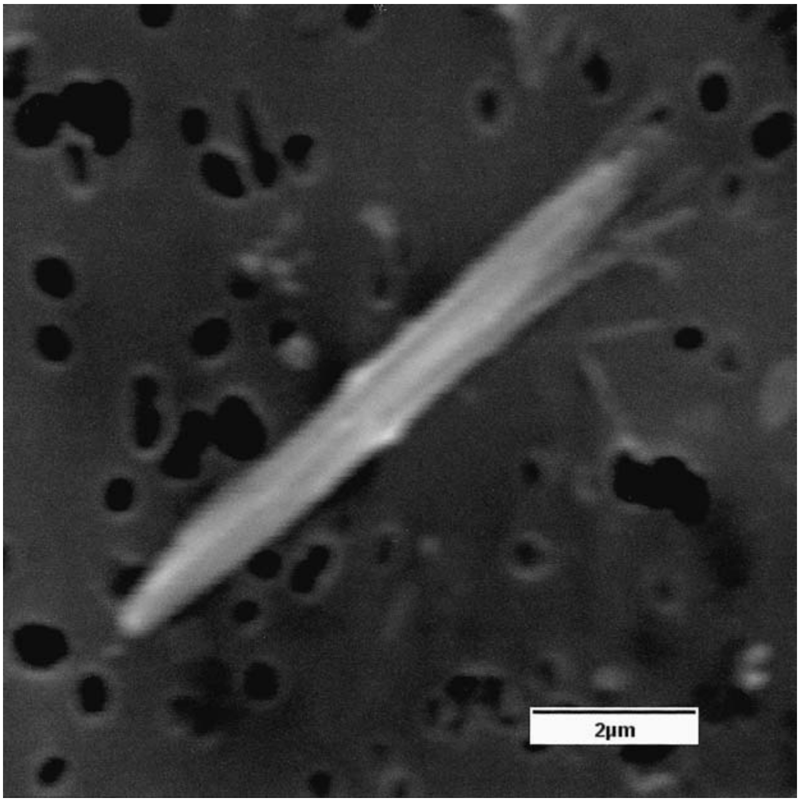

Figure 2-1. Scanning electron micrograph of chrysotile bundle isolated from bronchoalveolar lavage fluid from a New York City firefighter working on site for two weeks after the World Trade Center towers collapsed on September 11, 2001. Nuclepore filter preparation, magnified ×14,000.

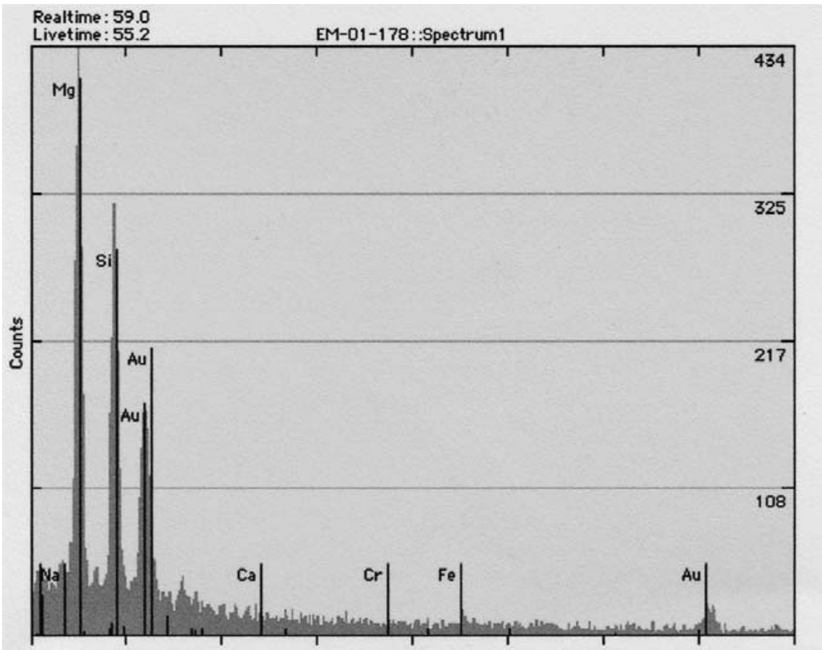

Figure 2-2. Energy dispersive spectrum from fiber shown in Figure 2-1. Note the large peaks for magnesium and silicon, characteristic for chrysotile asbestos.

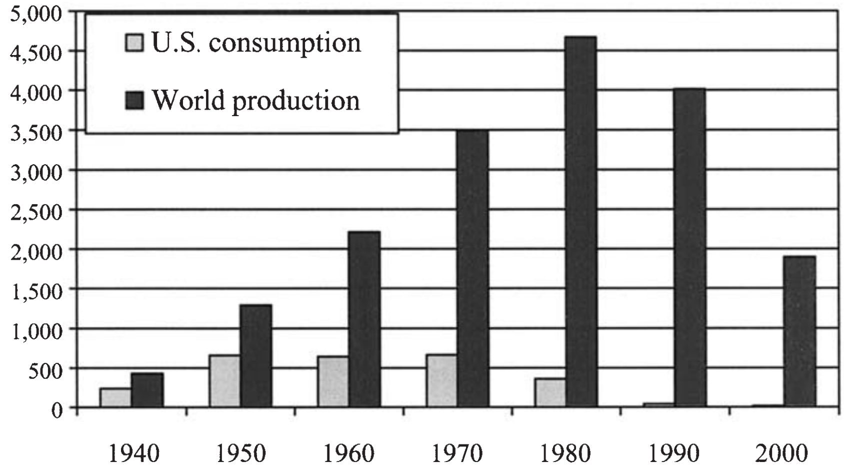

Figure 2-3. Asbestos consumption in the United States and world production of asbestos, which is used as a guide to world consumption. Peak U.S. consumption of asbestos was 801,000 metric tons in 1973. Peak world production was 5.09 million metric tons in 1975. Data from Minerals Yearbook, v. 1 (published by the U.S. Bureau of Mines until 1995 and by the U.S. Geological Survey after 1995, with permission).

into individual fibers) generates worker exposure levels of 6.0 to 12.1 fibers/ml.[1]

Asbestos is shipped in bags, historically made of porous cloth but recently of paper and plastic. The handling of this material in secondary industries routinely begins with cutting open these bags and manually emptying them into hoppers, for example, for mixture with concrete. Because this material is both dry and nonaggregated, the likelihood of dispersal is then at its maximum. The waste packaging material constitutes a source of exposure separate from the intended construction or industrial application.

Manufacture of Asbestos-Containing Products

The exposures that occur during the manufacture of asbestos products are extremely variable. Production of asbestos textiles involved higher exposure than other products. Carding and conventional spinning produced extreme air concentrations, resistant to environmental controls. Methods of manufacture utilizing liquid dispersion rather than dry asbestos are more successful at controlling potential exposure.

Work with material where the asbestos fibers were already entrapped (e.g., in roofing materials, floor tile, or cement pipe) presented considerable exposure opportunities, but only when such products are broken, thereby releasing respirable fibers. The job title sometimes provides information that offers some basis for assessing actual exposure, but it is often incomplete or misleading in estimating the degree of exposure. Certain jobs are more variable than others; for example, exposures for "inspectors" in manufacturing depend on the amount of loose asbestos dust remaining on the finished product.

Asbestos Insulation Materials

During the 1940s, 1950s and 1960s, covering boilers and furnaces with asbestos was universal. Before the health effects of asbestos exposure were well recognized, the use of asbestos insulation material was considered an effective safety practice, preventing burns, heat release, and fire. Boilermakers and pipe coverers constitute the most important and widely evaluated cohort of exposed workers. Selikoff's 1964 study of New York insulation workers unionists was one of the earliest U.S. reports of the health consequences of this work. Among the 255 deaths evaluated in this mortality study, 18% were the result of lung cancer, 11% of direct pulmonary damage from the dust, and 1.2% of mesothelioma. This staggering impact was an early demonstration of asbestos exposure risk.[8]

The construction industry application of asbestos coating to structural steel beams increased the societal scope of this exposure. The spraying of asbestos-cement mixtures was initiated in 1935, and from 1958 through 1978 was widely employed for railway carriages, naval ships, and newly constructed buildings. By one estimate, 1.2 billion square feet of asbestos-containing insulation (averaging 14% in concentration) is present in 190,000 American buildings.[9] The process was actually employed more—rather than less—frequently in the final years of this period, until the practice was halted when health issues became widely known.

Friction Materials

The use of asbestos for vehicular brakes takes advantage of its heat resistance and material strength. Asbestos concentrations in these materials are sizable, ranging from 30–80%. Because manufacture and repair of automotive wheels is geographically widely distributed, this application exposes individuals in a wide variety of trades and geography. The number of workers exposed as a consequence of asbestos brake and clutch work is estimated at 900,000. The practices of "blowing out" brake surfaces and beveling or grinding brake shoes produce modest airborne fiber concentrations for considerable periods of time and at distances extending many feet from the actual operation. The dispersion of asbestos dust particles from brake surfaces (even in situations of automotive traffic) remains a distinct possibility.[10]

Construction Materials

In floor tile and in roof shingles and coatings, asbestos mixtures utilize the flexibility and strength of the mineral additive as an important stabilizing feature. Because these materials are popular for home improvement activities, this application provides additional opportunity for exposure to nonprofessional workers, who lack specific occupational monitoring or training. Ordinarily, exposures are quite low and require considerable disruption of the product's integrity to release respirable particles with asbestos content.

Shipyards

Shipbuilding makes unusually intense use of insulation materials because of the nature of the construction. Ships have greater vulnerability to fire because of their isolation and the confined spaces. Noise and heat from the immediate proximity of a shipboard power plant create an important need for effective thermal and acoustic insulation, which must also be fireproof. In addition, preparing ships brings workers not necessarily directly involved with asbestos work (e.g., electricians, metal workers) into an asbestos-containing closed environment for the entire duration of the project. This closed-space exposure is, by its nature, difficult to control by the usual industrial measures, such as ventilation, wetting of the fiber sources, and containment.

Because workplace safety efforts were relaxed during the establishment of the wartime economy of the 1940s, the massive shipbuilding effort of that period put the largest segment of workers at risk for subsequent asbestos-related disease. The conditions of enclosed, poorly ventilated, and unmonitored assignments produced prolonged and heavy exposure to all interior ship workers.

Asbestos Removal

As a result of the regulatory recommendation that asbestos must be removed from schools, industrial work sites, and residences, the most significant and identifiable current exposure to asbestos occurs during asbestos abatement.[11] In the removal of pure asbestos lagging, for example, potential exposures of 62 to 159 fibers/ml have been reported.[12] This process often takes place in considerable disorder, because the surfaces are no longer easily accessible, and the work site is either in demand or in current use. Geographic isolation, soaking of the asbestos source, and personal containment represent the most important strategies for reduction of exposure.

The safety advantage in this process is that workers are required to be trained and to become aware of the nature of the task and its hazards. Current regulations for asbestos exposure provide detailed rules for these workers that contrast dramatically with the historically careless handling of the same material.

The administrative demands of asbestos worker protection are extensive. Currently, workers involved in asbestos abatement are required to undergo a pre-employment evaluation of their ability to work wearing a HEPA (high-efficiency-particulate air) filter respirator and impermeable (thus hot and humid) disposable clothing.[13] Baseline and periodic chest radiographs are taken, and measurement of pulmonary function. Before initiation of asbestos work, these individuals receive mandatory instruction regarding the health effects of asbestos-related disease and the means of dust and exposure control. Educational opportunities regarding the multiplicative effect of tobacco smoking on the risks from asbestos exposure are now a required component of asbestos worker training.

The area for asbestos removal is enclosed with a plastic barrier of specified 6-mil-thick polyethylene sheeting and by toxic-hazard

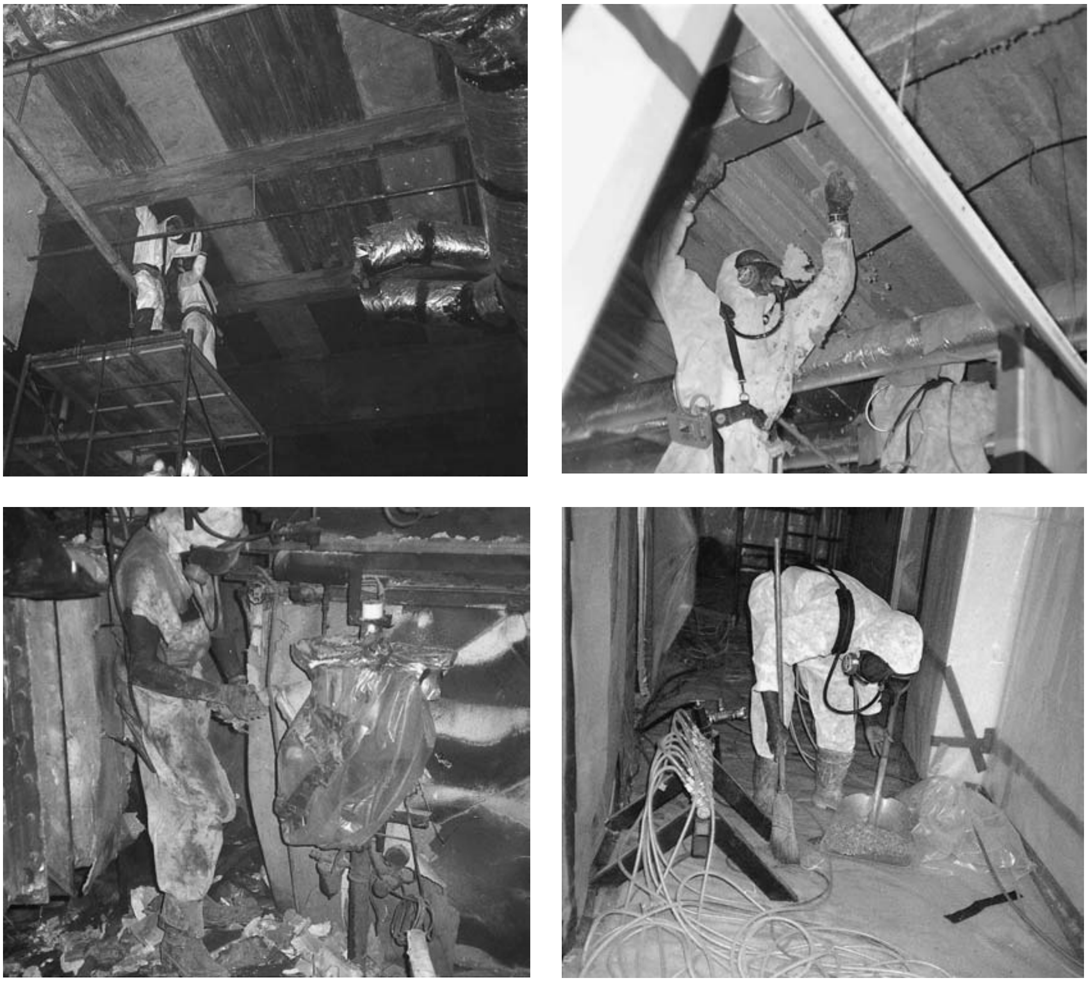

Figure 2-4. Workers in the North Carolina Asbestos Abatement Program. Asbestos removal occurs within confined spaces. Note the respiratory equipment and special protective clothing.

warning signs. The site is kept at negative barometric pressure (relative to the surrounding area) by having fans blow air outward through HEPA filters. If possible, asbestos-containing material is covered in plastic bags to encase escaping fragments. Additionally, workers wear personal protective gear (mask, gown, and gloves, as in Figure 2-4). Throughout removal, every effort is made to keep the material soaked so that respirable dust is minimized. Waste products are labeled and are handled with special care. Monitoring for airborne asbestos concentration is performed outside the confined asbestos-abatement area. Following each work period, workers are required to discard all outer clothing and shower, to prevent secondary contamination from work clothes. Periodic medical monitoring is also required, although the decades-long latency of asbestos-related disease makes these sessions more appropriately an opportunity for discussions of health risk and for counseling on smoking cessation.

Nonoccupational Exposure to Asbestos

Exposure to asbestos in the ambient indoor and outdoor environments results from many sources, both natural and manufactured. Chrysotile asbestos, which accounts for more than 90% of the asbestos used in the United States, has become a ubiquitous contaminant of ambient air. It has been noted that asbestos fiber can be found in the lungs of almost everyone in the American population.[14] Natural sources of asbestos fiber release include weathering and erosion of asbestos-containing rock and of road surfaces composed of asbestos ores. If the primary areas of source rock are compared 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.

Manufactured sources of exposure in the past have included off-site releases from mining, milling, and manufacture of asbestos products, exposing residents in nearby communities. Before occupational work practices improved in the 1970s, secondary contamination of homes occurred when employees brought home asbestos-laden work clothes. Weathering of asbestos cement wall and roofing materials is a relatively minor environmental source of exposure from man-made construction materials. However, off-site release from construction sites (primarily from sprayed-on asbestos fireproofing) have resulted in ambient asbestos levels 100 times background.[15]

Asbestos brake and clutch pads in automobiles contribute to the environmental load of asbestos. However, it is uncertain how much respirable fiber is released, because thermal degradation occurs at the high temperatures generated during braking. Waste disposal has become a growing source of potential exposure to asbestos fibers, and promises to continue as removal, abatement, and renovation occur in the existing building stock. Consumer products, water supplies, and food sources have been contaminated with asbestos-containing materials in the past. These manufactured sources of exposure have been significantly reduced by regulatory activity over the past 30 years and will continue to decline.

Currently, the most important source of nonoccupational exposure is the release of fibers from existing asbestos-containing surface materials in schools, residences, and public buildings or from sprayed asbestos-containing fireproofing in high-rise office buildings. The greatest potential for future exposure will be determined by the asbestos released during the maintenance, repair, and removal of these structures. The implementation of the Asbestos Hazard Emergency Response Act (AHERA), requiring inspection of the nation's public and private schools for asbestos, has resulted in an explosive commercial growth of the industry involved in asbestos identification and removal.

Some have argued that removal itself presents more of an exposure hazard than leaving the materials undisturbed or encapsulated.[16]

Measuring Exposure

Different techniques have been developed for measuring the concentration of asbestos in ambient air and in the workplace. The phase-contrast light microscope for counting fibers in the workplace has been less useful in the ambient environment, where fiber identity and character are usually unknown, fibers are too small to be seen by light microscopy, and concentrations expressed as mass are usually hundreds or thousands of times lower than those in the workplace.

Fiber concentrations in the workplace have generally been measured as the number of fibers longer than 5 microns. Ambient concentrations are now determined by transmission electron microscopy and usually are expressed as mass per unit volume (nanogram per cubic meter). Because of intrinsic variability in the unit weight of individual fibers, the conversion factors relating mass concentration to optical fiber concentration range from 5 to $150\,\mu g/m^3/f/ml$. These conversion factors have been adopted by the EPA[15] and other scientific bodies.

Measurements acquired through transmission electron microscopy have established background concentrations of asbestos in urban ambient air at generally less than 1 nanogram per cubic meter (0.00003 fibers per ml) and rarely more than 10 nanograms per cubic meter (0.00033 fibers per ml).[17] Table 2-1 summarizes fiber concentration data from a variety of studies in both urban and rural areas.

Asbestos concentrations in buildings, on the other hand, are more variable, revealing threefold variability among arithmetic mean concentrations.[17] Earlier studies often focused on buildings in which asbestos surface materials were visibly damaged and friable, which were not representative. In buildings with evidence of severe damage or deterioration, the probability of detecting excessive asbestos levels

Table 2-1. Summary of asbestos exposure samples in different environments

		Measured concentration (ng/m^3)		Equivalent concentration (fibers/cc)*	
Sample set	**Sample No.**	**Median**	**90^{th} %ile**	**Median**	**90^{th} %ile**
Air of 48 U.S. cities	**187**	**1.6**	**6.8**	**0.00005**	**0.00023**
Air in U.S. school rooms without asbestos	**31**	**16.3**	**72.7**	**0.00054**	**0.00242**
Air in Paris bldgs with asbestos surfaces	**135**	**1.8**	**32.2**	**0.00006**	**0.00107**
Air in U.S. bldgs with cementitious asbestos	**28**	**7.9**	**19.1**	**0.00026**	**0.00064**
Air in U.S. bldgs with friable asbestos	**54**	**19.2**	**96.2**	**0.00064**	**0.00321**

Source: Modified from Ref.17. *Based on conversion factor of $30\,\mu g/m^3$ = 1 fiber/cc.

Table 2-2. Summary statistics for average airborne fiber concentrations in U.S. schools and buildings

Statistic	Schools (71)	Outdoor air (48)	Public buildings Category 1 *(6)*	Category 2 *(6)*	Category 3 *(37)*
Median		0.00000	0.00010	0.00040	0.00058
Mean	0.00024*	0.00039	0.00099	0.00059	0.00073
Standard Deviation	0.00053	0.00096	0.00198	0.00052	0.00072

Source: From Ref. 18, with permission.

*80th percentile = 0.00045; 90th percentile = 0.00083.

The data used in the calculation of each statistic are the average concentrations (expressed as number of fibers greater than 5 μm in length per cubic centimeter of air) in a building (for indoor samples) or the concentration outside each building (for outdoor samples). By visual inspection, category 1 buildings contained no asbestos-containing material (ACM), category 2 buildings contained ACM in primarily good condition, and buildings in category 3 showed at least one area of significantly damaged ACM. In the study on public buildings, 387 indoor and 48 outdoor air samples were evaluated. No asbestos fibers were detected in 83% of the 387 samples. The sample size is given in parentheses below each heading.

over background was high. If the asbestos-containing surface materials or thermal insulation was undamaged or encapsulated, lower air concentrations were observed.

Table 2-2 shows summary statistics for average airborne fiber concentrations near schools and buildings Levels are comparable to outdoor air and are several orders of magnitude lower than current workplace standards (OSHA permissible exposure level (PEL) of 0.1 fibers per ml).

Asbestos abatement work is a significant potential source of asbestos exposure, particularly in schools and public buildings. While abatement procedures already specified by the EPA should minimize building contamination following renovation, removal, enclosure, or encapsulation of asbestos materials, these procedures may be violated.

The EPA has monitored the efficacy of the specified controls and cleanup procedures. Table 2-3 presents the results of one study of five schools where removal and encapsulation of asbestos-containing surfaces followed EPA procedures.[17] Although escape of asbestos fibers did occur during encapsulation and removal, there appeared to be a net reduction in fiber levels after encapsulation. Little improvement occurred in asbestos fiber levels following physical removal, with pre- and postabatement fiber levels being virtually the same. These results have brought into question both the health risk/benefit and the cost-benefit considerations of removal versus encapsulation. Currently, widespread removal of asbestos is not frequently recommended, and encapsulation is preferred in many situations.

Table 2-3. Geometric mean of chrysotile fiber and mass concentrations before, during, and after asbestos abatement

	Concentration							
	Before abatement		During abatement*		Immediately after abatement		After school resumed	
Sampling location	(f/l)[†]	(ng/m^3)	(f/l)	(ng/m^3)	(f/l)	(ng/m^3)	(f/l)	(ng/m^3)
Encapsulation								
Rooms with unpainted asbestos	1423.6	6.7	117.2	0.6	13.7	0.1	248.1	1.2
Rooms with painted asbestos	622.9	2.7	—	—	0.8	0.0	187.2	0.8
Asbestos-free rooms	250.6	1.2	0.5	0.0	9.3	0.0	30.7	0.2
Outdoors	3.5	0.0	0.0	0.0	6.5	0.0	2.8	0.0
Removal								
Rooms with asbestos	31.2	0.2	1736.0	14.4	5.6	0.1	23.9	0.2
Asbestos-free rooms	6.1	0.1	12.0	0.1	1.6	0.0	18.1	0.1
Outdoors	12.6	0.1	1.3	0.0	20.0	0.1	7.9	0.0

Source: Reprinted from Ref. 17, with permission.
*Measured outside work containment areas.
†Fibers of all lengths.

Regulatory Activity

Public health concern over the occupational and nonoccupational sources of asbestos exposure has created a vast array of governmental regulatory activity and the phasing out of asbestos production and its use in consumer products. This marked reduction in use is the result of regulatory activities in the 1970s and 1980s, during which time five government agencies invoked statutory authority to regulate asbestos.

The Occupational Safety and Health Administration (OSHA) regulates workplace exposure to asbestos and has set a PEL (an 8-hour time-weighted average for a 40-hour-per-week work shift) for occupational exposures. The PEL has been steadily lowered, as concern over health hazards and better monitoring methods have become established (Table 2-4). The first permanent standard, set in 1972, was 5 fibers/ml. This was lowered in 1976 to 2 fibers/ml and in 1986 to the lowest agreed to be technologically feasible at that time, 0.2 fibers/ml. The National Institute for Occupational Safety and Health (NIOSH) recommended a PEL of 0.1 fibers/ml, and this was also proposed as a regulatory standard by OSHA in 1990 and adopted in 1993.

The Mine Safety and Health Administration regulates the mining and milling of asbestos ore. The Food & Drug Administration (FDA) is responsible for regulating asbestos in food, drugs, and cosmetics. Consumer product bans on the use of asbestos in garments, dry-wall patching compounds, and fireplace-emberizing materials have been implemented by the Consumer Product Safety Commission. Despite these selected events, most of the regulatory activity has emanated from the Environmental Protection Agency.

Table 2-4. Regulatory exposure levels in the workplace

Date	U.K. Advisory Committee on Asbestos	British Occupational Hygiene Soc.	National Institute of Occupational Safety and Health	OSHA (proposed)	OSHA (regulation)	EPA
1969		2 fibers/ml. TWA chrysotile				
March 1971						Asbestos listed as hazardous air pollutant
1972					5 fibers/ml STEL	
April 1973						No visible emissions, milling and manufacturing Ban: Spray application of friable materials containing more than 1% asbestos
Oct. 1975				0.5 fibers/ml TWA		
July 1976					2 fibers/ml TWA 10 fibers/ml STEL	
Dec 1976			0.1 fibers/ml TWA			
1979	0.5 fibers/ml TWA chrysotile 0.5 fibers/ml TWA amosite 0.2 fibers/ml crocidolite					

Table 2-4. *Continued*

Date	U.K. Advisory Committee on Asbestos	British Occupational Hygiene Soc.	National Institute of Occupational Safety and Health	OSHA (proposed)	OSHA (regulation)	EPA
1983		0.5 fibers/ml TWA chrysotile				
June 1986					0.2 fibers/ml TWA	
Sept 1988					1.0 fibers/ml STEL	
July 1989						Ban: cloth, felt, tile, gaskets, brakes, after-market brakes, air conditioning, pipe, shingles, roof materials to be phased out over several years†
July 1990				0.1 fibers/ml TWA		
July 1993					0.1 fibers/ml TWA	
Nov 1993						Revision of ban on asbestos, vacating and remanding most of 1989 rule
Nov 2000						Asbestos Worker Protection Rule, cross reference to OSHA standards to protect state and local government employees‡

† Federal Register, Vol. 54, No. 132, July 12, 1989
‡ Federal Register, Vol. 65, No. 221, November 15, 2000

Through the National Emissions Standards for Hazardous Air Pollutants (NESHAP) program, the EPA regulates external emissions from asbestos mills and from manufacturing and fabrication operations. The EPA also regulates the use of asbestos in roadway surfacing and in insulation materials, and has banned most uses of sprayed-on asbestos materials and pipe wrapping. These standards also require specific work practices during demolition and renovation involving asbestos materials, and regulate the removal, transport, and disposal of asbestos-containing materials. The EPA has also established programs to evaluate and certify asbestos-removal contractors and established work rules to protect workers during asbestos-abatement activities.

Since 1982, when the EPA issued the Asbestos and Schools Identification and Notification Rule, the agency has required all local education agencies to inspect for friable asbestos materials; to notify parents and teachers if such materials are found; to place warning signs in schools where asbestos is found; and to keep accurate records of their actions eliminating this problem. With Congressional approval of the Asbestos School Hazards Abatement Act of 1984, the EPA was given responsibility for providing both financial and technical assistance to local education agencies.

Assessing Nonoccupational Risk

Asbestos-related disease resulting from nonoccupational exposure to asbestos has been recognized in published reports of mesothelioma among household contacts of asbestos workers and in residents living near asbestos mines and factories. An increase in the prevalence of malignant mesothelioma and asbestos-related disease has been reported in nonoccupationally exposed populations in Turkey, Cyprus, the Metsovo region of Greece, and Northeast Corsica. The causal factor for at least some of the excess mesothelioma in Turkey may be due to the geologic presence of a nonasbestos mineral fiber, erionite (see Chapter 5).

A metaanalysis of eight published studies conducted in populations with relatively high household and neighborhood exposure to asbestos showed significantly elevated relative risks for developing pleural mesothelioma. In the neighborhood exposure groups, the risk ranged between 5.1 and 9.3. In the household exposure groups the risk ranged between 4.0 and 23.7.[19]

However, these study populations were exposed to ambient concentrations much higher than those observed in U.S. homes and public buildings, and these data are insufficient to estimate the magnitude of the excess risk for pleural mesothelioma at levels of environmental exposure commonly encountered by the general population in industrialized countries.

In an effort to assess the health risk of nonoccupational exposure to asbestos in buildings and schools, numerous international panels have been convened. In the absence of undisputed evidence, several mathematical models have been proposed to assess the lifetime risk of lung cancer and mesothelioma. Underlying these varying risk assessment models are assumptions and uncertainties that make the interpretation of these risk estimates inherently difficult.

The estimation of risk is based upon extrapolation from high-dose workplace exposures in the past to low doses found in buildings and the ambient environment. Modern ambient exposures are orders of magnitude less than even today's OSHA permissible exposure level of 0.1 fibers per ml. Estimates of exposure assigned to these retrospective worker cohorts cannot be fully characterized, due in part to poor sampling and analytical methodology and the use of surrogate exposure categories based on job title. Mass-to-fiber conversions utilized in these models add substantial uncertainty. Models that include an assumption of a linear dose-response assume that exposure to one fiber of asbestos carries an inherent and finite risk for lung cancer and mesothelioma, and that the risk is cumulative for each fiber to which an individual is exposed. There appears to be no evidence of a threshold level below which there is no risk of mesothelioma.[20] This hypothesis is still debated.

In a review of the potential health risk associated with working in buildings constructed of asbestos-containing materials, the lifetime risk for premature cancer death was estimated to be 4 per million for those exposed for 20 years working in office buildings (estimated exposure levels ranging from 0.0002–0.002 fibers per ml). For those exposed for 15 years in schools the risk was estimated to be 1 per million (estimated exposure levels ranging from 0.0005–0.005 fibers per ml).[21] In comparison, the risk associated with the OSHA permissible exposure level of 0.1 fibers per ml for 20 years was estimated at 2000 per million exposed. The risk estimates associated with building exposure to asbestos are orders of magnitude lower than some commonplace risks from drowning, motor vehicle accidents, and household accidents. They are also far less than the background estimate of mesothelioma of 1 to 2 cases per million population per year.

Calculations of unit risk for asbestos at the low concentrations measured in the environment must also be viewed with great caution. The World Health Organization proposed a range of cancer risks based on an exposure of 0.0005 fibers/ml. The predicted lifetime cancer risk per 100,000 (smokers and nonsmokers) for the general population is 1–10 for mesothelioma and 0.1–1 for lung cancer.[22] The EPA's best estimate of the risk to the U.S. general population for a lifetime of continuous exposure to 0.0001 fibers/ml is 2.8 mesothelioma deaths and 0.5 excess lung cancer deaths per 100,000 females; and for males, 1.9 mesothelioma and 1.7 excess lung cancer deaths per 100,000. The risk assessment model used by the EPA was recently called into question in a study by Camus et al. of nonoccupational exposure to chrysotile asbestos.[23]

Some studies of asbestos workers have observed an increased risk of cancer at other sites, including the gastrointestinal tract, larynx, esophagus, and kidney (see Chapter 8). However, these findings have not been consistent and there is still considerable controversy as to whether these cancers are associated with asbestos exposure. All reviews regarding asbestos risk considered asbestosis to be of little importance at levels now typical in the ambient environment and buildings.[15]

References

1. Gilson JC: Asbestos. In: *Encyclopedia of Occupational Health and Safety* (Parmeggiani L, ed.), Geneva: International Labour Office, 1983, pp. 185–187.
2. Federal Register, Vol. 54, No. 132, July 12, 1989.
3. EPA Asbestos Material Bans, 1999, http://www.epagov/opptintr/asbestos/asb-bans2.txt.
4. Collegium Ramazzini: Call for an international ban on asbestos. *J Occup Environ Med* 41:830–832, 1999.
5. Camus M: A ban on asbestos must be based on a comparative risk assessment. *Can Med Assoc J* 164:491–494, 2001.
6. Nicholson WJ, Selikoff IJ, Seidman H, Lilis R, Formby P: Long-term mortality of chrysotile miners and millers in the Thetford Mines, Quebec. *Ann NY Acad Sci* 330:11–21, 1979.
7. U.S. Department of Health Education and Welfare, Public Health Service: Asbestos: An information resource (Levine RJ, ed.). 1980, DHEW Publication No. 78–1681, pp. 41–60.
8. Selikoff IJ, Churg, J, Hammond EC: Asbestos exposure and neoplasia. *JAMA* 188:142–146, 1964.
9. U.S. Environmental Protection Agency. Asbestos Fact Book, USEPA Office of Public Affairs (A-107), 1985, p. 5.
10. Kohyama N: Airborne asbestos levels in non-occupational environments in Japan, IARC Scientific Publications No 30: Lyon, 1989, pp. 262–276.
11. Report to the Congress, Study of Asbestos-Containing Materials in Public Buildings, U.S. Environmental Protection Agency, February 1988, p. 5.
12. Dement JM: Asbestos. In: *Occupational Respiratory Diseases, National Institute for Occupational Safety and Health, U.S. Department of Health and Human Services* (Merchant JA, ed.), 1986, p. 288.
13. Code of Federal Regulations. Title 29 Chapter XVII, Subpart 1926.58, 1986.
14. Churg A: Current issues in the pathologic and mineralogic diagnosis of asbestos-induced disease. *Chest* 84:275–280, 1983.
15. Airborne Asbestos Health Assessment Update. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, EPA/600/8-84/003F, June 1986.
16. Whysner J, et. al.: Asbestos in the air of public buildings: a public health risk? *Prev Med* 1:119–125, 1994.
17. Nicholson WJ: Airborne mineral fiber levels in the non-occupational environment, In: *Non-Occupational Exposure to Mineral Fibers* (Bignon J, Peto J, Saraccci J, eds.), IARC Scientific Publication No. 90: Lyon, 1989, pp. 239–261.
18. Mossman BT, et al: Asbestos: Scientific developments and implications for public policy. *Science* 247: 294–301, 1990.
19. Bourdes V, Bofetta P, Pisani P: Environmental exposure to asbestos and risk of pleural mesothelioma: Review and meta-analysis. *Eur J Epidemiol* 16:411–417, 2000.
20. Hillerdal G: Mesothelioma: Cases associated with non-occupational and low dose exposures. *Occup Environ Med* 56:505–513, 1999.
21. Health Effects Institute—Asbestos Research. 1991. *Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge.* Cambridge, MA: Health Effects Institute.
22. World Health Organization, Air Quality Guidelines, Copenhagen, 1987.
23. Camus M, Siemiatycki J, Meek B: Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. *N Engl J Med* 338:1565–1571, 1998.

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