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Hazard recognition and regulation : an asbestos chronology

Diane A. Colson
New Jersey Institute of Technology

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ABSTRACT

HAZARD RECOGNITION AND REGULATION:
AN ASBESTOS CHRONOLOGY

by
Diane A. Colson

One hundred years after it was first mined, several uses of asbestos were banned in the United States. The latency period that typifies asbestos-related diseases was but one factor in the delayed recognition of asbestos as a health hazard. Many other factors delayed the initiation of practical measures to alleviate the hazards.

Controversies surrounding exposure to asbestos continue. Epidemiological studies have answered many questions, but many others are, and may remain, unanswered. Some of the controversies surrounding exposure to asbestos are embedded in the development of the United States of America as a country. An understanding of the historical basis for these controversies helps to explain the present relationships between business, labor, and government, and how these groups view issues of health and safety. Asbestos is an integral part of that history. It is used as the focus of this thesis, to examine the interplay of factors and forces which influenced the development of federal regulation of health and safety in the United States.

HAZARD RECOGNITION AND REGULATION: AN ASBESTOS CHRONOLOGY

by
Diane A. Colson

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APPROVAL PAGE

HAZARD RECOGNITION AND REGULATION: AN ASBESTOS CHRONOLOGY

Diane A. Colson

Dr. Howard Gage, Thesis Advisor — Date
Professor of Industrial Engineering, NJIT

Dr. Joseph Bozzelli — Date
Distinguished Professor of Chemistry, NJIT

Mr. Arjit Sengupta — Date
Special Lecturer, Industrial Engineering, NJIT

BIOGRAPHICAL SKETCH

Author: Diane A. Colson

Degree: Master of Science in Occupational Safety and Health Engineering

Date: May 1995

Undergraduate and Graduate Education:

- Master of Science in Occupational Safety and Health Engineering,
  New Jersey Institute of Technology,
  Newark, NJ, 1995
- Bachelor of Science in Civil Engineering
  Newark College of Engineering,
  Newark, NJ, 1974

Major: Civil Engineering

DEDICATION

The thesis is dedicated to the many people who contributed to its completion. They are too numerous to mention individually, but the consequences of naming some and not others are about to be suffered. Forgiveness is requested in advance for limiting an endless list to the following:

My family, especially my mother, Clara S. Colson, a wise woman, who, in 1987, saved a newspaper clipping about another new program starting at NJIT; and brother, Kenneth, head of the "Glitch Team";

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PREFACE

Although it develops in, and is mined from rock formations, asbestos breaks into fibers that have the characteristics of silk or cotton, but will not burn. Not all fibrous minerals are asbestos minerals. The term asbestos refers, variously, to the group, or to a type in the group, or more specifically to the fibers, of six commercial minerals.

Asbestos is a worldwide commodity that was first mined in the 1870s. Commercial production of asbestos insulating materials was recorded by 1874. Worldwide production and use of asbestos increased from fifty tons in 1877, to four million tons per year in 1967. From 1900 to 1980, some thirty-six million metric tons were used worldwide.[1] Asbestos has been used in more than thirty-six hundred different commercial, consumer, and industrial products.[2]

The unique properties of asbestos fibers, which make them so useful commercially, also cause specific diseases. The risk of developing an asbestos-related disease depends on exposure to airborne asbestos fibers.[3] An apparent connection between exposure to asbestos dust and resulting sickness was noted as early as the first century A. D.

The scarring, or fibrosis, of the lungs caused from inhaling asbestos fibers, was identified at the turn of the nineteenth century, and given the name asbestosis in 1927. Even though there was some awareness that inhalation of asbestos dust was hazardous, exposure does not cause any

short term effects, and there are no warning properties. The ill effects are long term. Twenty years may elapse between exposure and the manifestation of an asbestos-related disease.

In 1963, the only major asbestos-producing mine operating in the United States was GAF's Lowell mine in Vermont. During that year, four other mining operations began in California.[4] In 1978, there were six mine and mill operations, run by as many companies. Three of those were located in California, and one each was in Arizona, Vermont, and North Carolina. Total employment for the six firms was four hundred.[5]

In his 1978 book, Death On The Job, Berman estimated that ninety thousand people worked directly with asbestos, and that another five million worked with asbestos-containing products every day, commenting that "profits and jobs linked to asbestos have made it politically difficult to cut back its use."[6]

In 1964, a seminal study on asbestos exposure was presented at the New York Academy of Sciences International Conference on Asbestos Disease. The study, by Selikoff et al., found high rates of asbestosis, lung cancer, and mesothelioma, among the cohort when compared with the general population. It was not the first study to show these results, but it was the first to use data gathered independent of the asbestos industry. The study was deemed, by asbestos manufacturers, to be the first definitive study on the ill health effects from exposure to asbestos.

The discussions at, and published proceedings of, the 1964 conference initiated a discourse that helped to create a favorable atmosphere for enactment of the Occupational Safety and Health Act of 1970.

Under its power to regulate commerce, Congress passed the Occupational Safety and Health Act in 1970. Asbestos was the first substance to undergo the formal rulemaking procedure established by the OSHAct. It thus became the subject of the first federal, enforceable, comprehensive standard for "protecting the health and safety of workers," by regulating workplace conditions.

In 1990, the Collegium Ramazzini sponsored a conference on The Third Wave of Asbestos Disease which categorized three phases of asbestos exposure. The first included small populations involved in mining the raw material, and manufacturing asbestos-containing products. The second phase involved shipyard and insulation workers who installed those products. The third phase is exposure to asbestos-containing materials (ACM) that were put in place between 1930 and 1980. More than thirty million tons of asbestos was used in the United States from 1900 to 1980.[7]

Researchers at Mt. Sinai's Occupational Health Clinic in New York City estimate that twenty-seven million people worked with asbestos between 1940 and 1980. Of that population, more than one million are expected to develop an asbestos-related disease by the year 2000. Their research also estimated that, within the next thirty years, exposure

to asbestos in the workplace will cause ten thousand cancer deaths above the normal cancer rate.[8]

OSHA considers occupational exposures which cause more than one death per one thousand workers over a working lifetime to be significant. Its 1994 Final Rule for Occupational Exposure to Asbestos estimates that the population at risk from exposure during new construction, renovation, abatement, maintenance work and custodial activities ranges from 1,758,006 to 5,751,586.[9]

The other sphere of asbestos exposure is regulated by EPA under AHERA, which applies to primary and secondary school buildings. Benarde, in Our Precarious Habitat: Fifteen Years Later, estimates the average level of exposure in schools ranges from 0.001 to 0.004 fibers per cubic centimeter. He notes a study by Hughes and Weil of Tulane University which predicts the lifetime risk for school children exposed for five years, starting from age ten, at from 0.02 to 0.37 deaths per million children per year.[10] An article by Mossman, in the January 19, 1990 issue of Science, advocates the "amphibole theory." [11] This supports the view that other types of asbestos are more dangerous than chrysotile, which comprised more than ninety-five percent of all asbestos used in buildings in the United States. A review of the article in Time notes that Mossman estimates the risk to the general public to be no more one percent of the level deemed safe for workers, even in buildings where asbestos is damaged and flaking. The

Time review states that "the risk of dying from smoking, drowning, airplane crashes or even playing high school football is 100 to 1,000 times as great as the risk of dying from asbestos exposure in buildings."[12]

All or none of these estimates may or may not be confirmed, but no "safe" level of exposure to asbestos has been determined. Public health policy advocates reducing exposures to the lowest level possible. OSHA's 1994 rule reduced the permissible exposure limit to 0.1 fibers per cubic centimeter (0.1 f/cc), while acknowledging the risk of developing an asbestos-related disease that still exists at that level.

The thread of asbestos woven through the development of the United States of America was slow to unwind, and hard to break. Events which got the spool rolling are presented herein, as are some of the medical studies which helped distinguish and establish the hazards of exposure to airborne asbestos fibers. The final chapter presents a sampling of the many factors which continue to prompt controversy concerning asbestos-related diseases. The many aspects and controversies concerning sampling and analytical procedures for estimating exposure levels are only discussed in the context of the impact of technology in establishing limit levels of exposure.

It is hoped that the general reader will gain a better appreciation of how and why the fairly recent clamor about asbestos developed. Presenting events in chronological

order is not always possible due to the long span of time over which the history unfolds. Recounting the one hundred year boom-to-bust sojourn of asbestos in the United States speaks to the power of labor, of industry, and of consumerism. Each is a driving force of the country's economy, but in the final analysis, no one should have to sacrifice their life to boost profits or, without warning, jeopardize their health to make or use the products that are produced.

CHAPTER 1

THE MAGIC MINERAL

1.1 Historical Names and Sightings

Asbestos is the common name used in North American and Northern European languages to identify a group of fibrous minerals. The use of the Greek word 'sabestos' was recorded before the first century AD, to describe the asbestos wicks which kept their temples lamps and candles burning, but were not themselves consumed by the flames. The word meant inextinguishable or unquenchable, derived from the Greek verb sbennumi, meaning to quench, die down, or extinguish.[13] This contradicts an essential characteristic of the mineral, which is that it does not burn. Its mode of formation was unknown, but the remarkable physical and chemical properties of asbestos were obvious, and did not go unnoticed in the annals of ancient history.

Pliny the Elder, the Roman naturalist, thought that it grew in the deserts of India, and reported that asbestos cloth 'came out of the flames whiter and cleaner than it could possibly have been rendered by the aid of water'. The incombustible cloth shown to Marco Polo in Siberia was called salamander, said to be made from the skin of the salamander which lived in fire.[14] It is said that Charlemange had a tablecloth made of asbestos to impress his warrior guests. He would throw it into the fire after dinner and withdraw it later, clean and intact.[15]

In England, the mineral was originally known as amiantus or amianthus, derived from the Greek amiantos, meaning the material which is undefilable. It was known as amiante in French, and amianto in Italian, and Spanish. In Germany it was known as stone flax (Steinflachs). French Canadian miners referred to it as cotton-stone (pierre-a-coton).[16]

1.2 Properties of Asbestos

The chemical, and morphologic composition of asbestos differs according to where it is mined, and results in corresponding changes in physical properties such as range of the fiber diameter, degree of flexibility, tensile strength, and surface properties. In addition to elemental substitutions, the asbestos varieties are influenced to some extent by the presence of impurities. Impurities may be a part of the crystal structure, or they may be introduced by associated minerals.

In general, chrysotile deviates less in composition, and is less variable than the amphibole types.[17] The properties may vary considerably with the different types of, and to a lesser extent, between specimens of, the same mineral. Various properties of the commercial varieties of asbestos are included in Appendix D.

1.2.1 Unique Properties

Properties vary among asbestos types, but commercial value depends largely on the two common physical characteristics of noncombustibility, and unique fibrous structure.

1.2.1.1 Water Content, Dehydration, and Mineral Type Chrysotile contains approximately fourteen percent water. The water content of the amphibole varieties is only one or two percent, and they will withstand somewhat higher temperatures than chrysotile.

X-ray studies in the 1920s confirmed earlier work by W.T. Schaller, in concluding that all amphiboles contain water of crystallization. Myril C. Shaw's work, published in 1950, indicated that chrysotile contains both (OH), the hydroxyl radical or water of constitution, and H_2O, the water of crystallization.

Unlike vegetable and animal fibers, asbestos will not burn, but it will decompose and lose its essential physical properties at moderately high temperatures. Dehydration is accompanied by a pronounced change in physical properties. For example, olivine, also called chrysotile or peridot, is an anhydrous magnesium-iron silicate that does not exhibit the "asbestiform habit." Chrysotile, the fibrous form of serpentine, is a hydrous magnesium silicate. The adsorbed water of chrysotile is driven off at about 300 degrees centigrade. Between 550 and 600 degrees centigrade, all water of crystallization is driven off, and the mineral gradually alters to olivine.[18]

1.2.1.2 Fibrous Structure The outstanding physical characteristic of asbestos is its unique fibrous structure. When asbestos minerals are crushed, they have the property

of splitting lengthwise into bundles of fibers of varying strength and flexibility, exhibiting the "asbestiform habit".[19] Unlike hard crystal fragments typical of most minerals, asbestos minerals yield fibers that can be spun. The fiberization is a cleavage process, defined as a tendency in some minerals to split in a certain direction along planes related to the molecular structure of the mineral, and parallel to possible crystal faces. Cleavage is described in five steps from poor (bornite) to fair, good, perfect, and eminent (mica).[20] The asbestos varieties exhibit good to perfect cleavage, crystallizing in bundles that look like organic fibers. Each fiber is composed of smaller fibers, or fibrils. A strand of human hair is more than twelve hundred times larger than a typical asbestos fiber. Asbestos fibers can stay airborne for weeks. The aerodynamic behavior is a function mainly of diameter, but also of size, shape, and density.[21]

1.2.2 Morphology of Asbestos FIbers

Perhaps equal in importance to the fibrous nature is the difference in morphology or fiber structure. The most striking difference between chrysotile and amphibole fibers is in their shape.

1.2.2.1 Chrysotile No other mineral is as fibrous as chrysotile, and its fibers are the most flexible of any asbestos fibers. In its natural state, chrysotile is

slippery and soapy to the touch. The fibrous surface is so hard that it will dull a sharp knife, yet it can be scraped with the fingernails into a soft, fluffy, fine, fibrous mass, ranging from a lustrous white to shades of gray. The fine fibers have a delicate texture, and a perfectly smooth surface.[22] Chrysotile fibers can be separated into fibrils, with diameters in the range of 200 to 400 angstroms (one angstrom equals one ten-billionth of a meter). A chrysotile fiber can readily yield one thousand fibrils of the same length. Fibers mined from serpentine rock deposits in Quebec and Vermont varied from one-half to three inches in length.[23] The fibers in a piece of chrysotile as big as the tip of a finger would stretch around the world if placed end to end.[24]

The typical tubular structure of chrysotile is formed by the tendency to roll up onto itself as the fiber cleaves, giving the appearance of long, curved, hollow cylinders. Some fibers which are solid show an unusual growth pattern. Filling the cylinder with foreign material may also result in a solid appearance, and a change in density. The density of Arizona and African chrysotile is compatible with tubular structure, but Canadian chrysotile with a high density has at least fifty percent filled tubes.[25]

1.2.2.2 Amphiboles Originally, the term asbestos was applied to fibrous minerals closely related to amphiboles. Amphibole fibers are usually straight, and exhibit good

cleavage. The cleavage planes of amphibole minerals are at about 55 degrees and 125 degrees, forming wedge-shaped fragments. The crystals are often long or needle-like, and when short, they are six-sided.[26] The amphibole fibers are acicular, with minimal diameters of 600 to 2500 angstroms. After fiberizing processes, the amphiboles produce a great flocculent volume, composed mainly of air held in minute cells by the framework of fibers.[27]

1.2.3 Specific Properties by Type

Heat resistance and ease or difficulty of fiberization were important properties for the commercialization of asbestos. Other important properties include flexibility, durability, high tensile strength, high strength-to-weight ratio, chemical, corrosion and moisture resistance, and resistance to searing temperatures. The varying chemical compositions and physical properties of the asbestos varieties made them more or less suitable to certain commercial applications.

1.2.3.1 Properties of Chrysotile Chrysotile, also called white asbestos, is the only variety with a positive electric charge. It is very elastic, and the most flexible of the asbestos varieties. It also has the highest resistance to heat, becoming brittle around 650 degrees centigrade, and fusing at well over 1,000 degrees centigrade. Chrysotile has virtually no resistance to acids, and is readily decomposed by sea water and moist air. The fibers are long, and strong, easily separable, and have a silky luster.

1.2.3.2 Properties of the Amphiboles Amphiboles are straighter and harsher than chrysotile, but will withstand somewhat higher temperatures. Amosite and crocidolite were the most widely used amphiboles.

Amosite, or brown asbestos, is a ferrous silicate in which a proportion of the iron is replaced by magnesium. It has good tensile strength, heat resistance and flexibility. It is less resistant to acids than crocidolite, but fuses at a higher temperature. Its fibers are fairly strong, and unusually long, but coarse.

Crocidolite, or blue asbestos is a complex silicate of iron and sodium. It is the strongest of all natural fibers, with a basic tensile strength 1-1/2 times that of steel piano-wire.[28] It is noted for resistance to acids, alkalis, neutral salts, and organic solvents, but will discolor and loose strength at temperatures above 360 degrees centigrade, as a result of oxidation.

Tremolite is a calcium-magnesium silicate. It is characterized by the lack of iron, and resistance to acids. The fibers are long and silky, but brittle, and low in tensile strength.

Anthophyllite is a silicate of magnesium and iron, usually with small amounts of aluminum. Its fibers are usually brittle, and lack tensile strength. Both tremolite and anthophyllite are superior to chrysotile in resistance to chemical reaction.

Actin is similar to tremolite, except that iron replaces some of the magnesium. It has good resistance to acids, and the lowest tensile strength of the asbestos varieties. Its fibers are weak and brittle.[29]

1.3 Historical Early Uses

Sometimes called the "Funeral Dress of Kings," asbestos cloth was used in ancient Egypt as a burial shroud to keep the ashes of the royally deceased separate and protected from those fueling the funeral pyre.[30] Clay pots from Finland in this same era, cira 2500 BC, contained asbestos as a binder to enhance the material strength.[31]

The first recorded use of the material for acoustic purposes was in 28 BC, when Anaxilaus, a Greek doctor, described how a tree could be felled noiselessly if it was surrounded by asbestos linen. Pliny the Elder recorded that it would not burn in the first century BC, and also noted its apparent debilitating effect on slaves weaving asbestos cloth.

Until the end of the 17th century, asbestos was more an object of superstition and curiosity than a commercial commodity.[32] As late as the 1880s, its properties were so singular, and its occurrence in nature so unusual, that it almost defied classification.[33]

1.4 Mining

Asbestos is mined like other minerals, in huge open-pit or underground workings. "Crude" asbestos was separated from dirt and rock, and sorted into groups for milling. "Milled" asbestos consists of all grades produced by mechanical treatment such as crushing, screening and air separation. The end product is graded by fiber length.[34]

All uses of asbestos are as processed fiber. Long fibers were used in the textile industry. The shorter fibers were used in a variety of products, according to their individual properties. Because the various asbestos minerals differ in their chemical composition, crystalline structure, fiber dimensions, and chemical properties, they share certain properties to varying degrees, but each has distinctive properties.

The asbestos miner had virtually no control over the percentage of the various grades produced, and the properties of a particular asbestos type were often associated with the mine or location from which it was extracted.

Commercial mining of asbestos began in the 1870s, in a remote region of the Italian Alps. The Italian chrysotile asbestos fibers were straight, smooth, and slippery. Cotton fibers were mixed in to enable spinning. The large chrsotile deposits in Canada were first mined in 1876. Although not as long as the Italian variety, the Canadian fiber had a slight hook on it that enabled easy spinning. Within a few

years there were four mines operating in the vicinity of Thetford, Quebec.[35]

1.5 Deposits

Development of the extensive chrysotile deposits in eastern Quebec in the 1880s was followed by further exploitation of already known and extensive deposits. Major deposits of chrysotile occur in the southern Ural Mountains of Russia, eastern and southeastern Quebec, and the Italian Alps. The Italian Alps deposits are in Susa, Lanzo, and Val Malenco. Limited deposits were located in Italy and Cyprus. Significant deposits in the United States were mined in Arizona, California and Vermont.[36]

South Africa is the major source for both amosite and crocidolite. Deposits of the famous Cape blue variety of crocidolite near Prieska were first mined in 1891.[37] Limited deposits of crocidolite were also mined in Australia, Brazil and Canada. Amosite was first discovered in the Transvaal, South Africa, in 1907, where commercial mining began in 1908.[38]

1.6 Summary

In antiquity, asbestos was known for its resistance to fire. The wide spread modern use of asbestos is intimately connected with industrialization, and the use of asbestos for the conservation of heat, to make the steam-powered machinery

which propelled that era more efficient. The variety of favorable and adaptable properties probably accounts for the diverse applications and multiple uses of asbestos.

Crude asbestos fibers could be separated by standard industrial procedures into useful sizes, and were easily incorporated into the manufacturing operations of already established industries such as commercial mining, and textiles. The relatively limited use of asbestos was greatly expanded during the Industrial Revolution. A vast industry evolved, using asbestos as the raw material of choice, to manufacture a great variety of valuable products.[39]

facing page 12

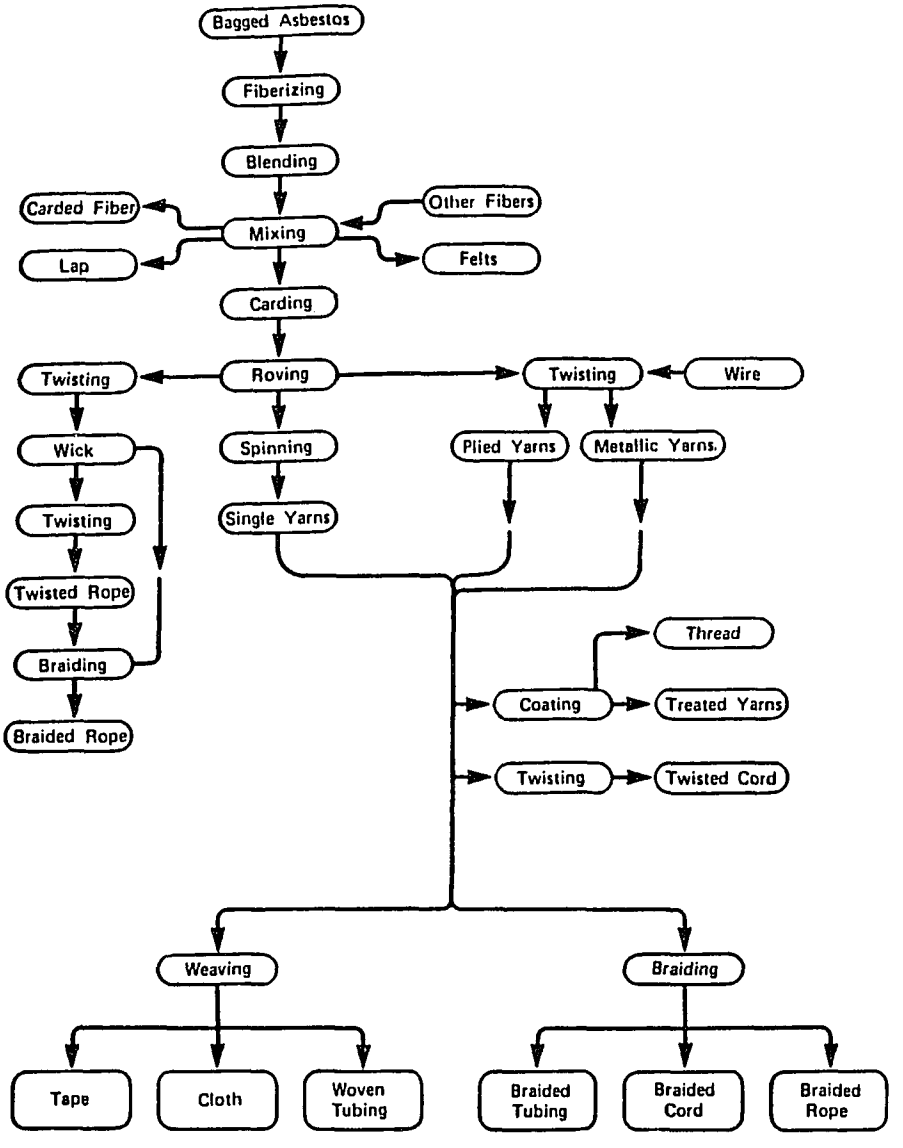

Figure 2.1 Flow diagram for asbestos textile manufacture.

Source: Burgess William A. Recognition of Health Hazards in Industry: A Review of Materials and Processes. (New York: John Wiley and Sons, 1981): 155.

CHAPTER 2

DEVELOPMENT AND GROWTH OF COMMERCE

2.1 Textiles

The Industrial Revolution began in England around 1760, and later spread to other countries, including the American colonies. The most important developments in England's Industrial Revolution came in the field of textiles.[40]

2.1.1 Textile Operations

Asbestos fibers pass through the same steps as cotton, an operation which was well established by the time the first Italian asbestos was used as a raw material in England's textile industry. The principal textile operations are carding, spinning, and weaving.

After leaving the mines, crude asbestos was shipped to England for further processing. At first, workers picked out the longer fibers by hand. Eventually, the raw material was separated by mechanical processes. The asbestos fiber was then grouped and graded according to length. Those of suitable length were used to make yarn, rope, and assorted fabrics.[41] The operation starts with breakers and crushers to separate and clean the fiber, while guiding it over slats, screens, and rollers. The fibers are formed into a large, round, soft roll called a picker "lap." In this form it is ready for carding.[42]

Carding is the specific operation for opening up the fibers, further eliminating dirt and foreign substances. It also straightens the fibers somewhat and removes some of the very short fibers. The fibers leave the carding machine with no twist, in a long, soft, round rope called a "card sliver."

The slivers are drawn out, to straighten the fibers and lay them as parallel as possible and at the same time to reduce the strand in diameter. Roving continues the drawing-out process and gives a twist to the sliver. Leaving the roving machine, the strand is called a "roving" and is ready for spinning into yarn. Drawing and roving are the necessary final operations prior to spinning.[43]

Spinning completes the drawing operation. The strand is drawn out to the final size required; it is then given the desired amount of twist, and is wound on bobbins, spools, or other suitable packages. In this form it is called "yarn," and can be woven, knitted, twisted, or plaited into fabrics. A small percentage of cotton was usually blended with the straight, smooth Italian fibers
to facilitate the spinning operation. This addition was not necessary for the Canadian fiber, which already had a slight hook. The characteristic hook and more accessible location, made Canadian fiber more desirable than the Italian fiber as a raw material.

2.1.2 Textile Industry, England

The number of textile mills grew as various inventions speeded production. Spinning and weaving were slow, hand-crafted arts practiced with little change for about seven thousand years until 1733, when John Kay invented the "flying shuttle." This device allowed a single weaver to sit at the center of the loom and pull a handle that sent the shuttle twice the distance previously covered by hand. It enabled weavers to weave fabric faster than spinners could make thread.

That same year, John Wyatt and his partner Lewis Paul introduced the use of mechanical rollers to form a tough, compressed thread that could be stretched out in spinning to any desired fineness. In 1769 Richard Arkwright, a barber who became Britain's first great industrialist-capitalist, gave England "the power of cotton." Arkwright made and patented the first spinning machine powered by waterwheel. Called a "water frame", it had a series of rollers that could spin a much stronger cotton thread faster, and made it possible for cloth to be woven entirely of cotton. Arkwright was also the first to develop the factory system by taking textile workers out of their homes.[44]

In 1770, James Hargreaves invented the "spinning jenny," which could operate a number of spindles simultaneously.[45] It produced more thread in a day than six or eight human spinners and their handwheels. In 1779, Samuel Crompton

combined the water frame's rollers and the jenny's movable carriage into a "mule," able to produce stronger thread at higher speed.[46] Mule spinning is an intermittent process, but drawing out and twisting operations are performed at the same time.[47] Crompton's mule became the standard for the textile industry. One worker with a Crompton mule could spin as much thread in a day as three hundred workers could spin in 1760 with handwheels.[48]

The conversion to steam powered machinery started in 1785, when Edmund Cartwright, a clergyman, built a power loom. It used horse power at first, and then steam.[49] By 1785 steam power was applied to drive machines for spinning and weaving. Spinning mules driven by water or steam were soon producing literally millions of miles of yarn and thread. The textile mills made Britain the world's richest industrial power, boosting exports from insignificance in 1780, to nearly eight million pounds sterling two decades later. By 1824, the textile industry, regarded as "the sacred staple and foundation" of British wealth, was well on the way to complete conversion to steam [50] as the main power source for the various machines used in the textile industry.

2.1.3 Textile Industry, United States

Factories using jennies were established in 1787, in Philadelphia, in Beverly, Massachusetts, and elsewhere in the

country, but none proved successful, partly because of the inferior quality of the product. To protect its cotton trade, England forbade export of textile machinery, or its design. The textile industry was smuggled to the United States by Samuel Slater, an apprentice working in Belper, England for one of Richard Arkwright's partners.

Slater left England in 1789 to pursue his fortune in America. Early in 1790, he sought out Moses Brown, who ran a spinning mill in Providence, Rhode Island. They set up a business agreement, and Slater set to work building textile machinery from memory. Four days before Christmas, 1790, the machines started to spin, powered by the arm of an elderly Negro man named Samuel Brunius Jenks. Later, water power was supplied from the falls of the Blackstone River. Within four years of leaving England, Slater built the first successful cotton-spinning mill in the United States, on a site in Pawtucket, Rhode Island.

By 1804, a Pittsburgh factory was selling textile machinery, and by 1807, there were fifteen or twenty mills, with about 8,000 spindles in operation. By 1810 there were some ninety mills operating perhaps 80,000 spindles. Ten years later the number of spindles in use had climbed to 191,000.

In 1812, upon his return from two years in England studying the industry, Francis Cabot Lowell designed, from memory, a textile operation at a plant in Waltham, Massachu-

setts. This may have been the first time anywhere that both the spinning and weaving processes came together under one roof.[51]

2.2 Transportation

It is said that the United States of America could not have been without the steam engine. The steam engine consumed and wasted considerable fuel, but it also fueled the development of America and the transportation network needed to support it. The American Revolution officially ended with the signing of the Peace Treaty of Paris, on September 3, 1783. What emerged from the Revolutionary War were some four million people living in thirteen states, joined in a loose federation, strung out along the Atlantic seaboard. Wagons and roads were an expensive way to carry merchandise, especially bulk freight. The network of waterways available made the steamboat a natural alternative to overland transport.[52]

2.2.1 Steamboats

The first commercially successful steamboat run in America was developed by Robert Fulton of Pennsylvania.[53] In 1806, Fulton returned from twenty years in Europe with a Boulton & Watt engine that he had managed to take out of England. In August, 1907, he launched the flat-bottomed, 100-ton North River Steamboat on the Hudson River. Within two months, it was carrying sixty to ninety passengers on each trip between

New York and Albany, covering the 150 miles in thirty-six hours or less. In time, Fulton renamed his flagship the Cleremont.[54]

The prosperity of the shipping industry flourished throughout America from 1792 to 1807. Output from the shipyards rose from 200,000 tons in 1789 to 1,400,000 in 1810. By this time, American ship's were hauling ninety per cent or more of both the nation's exports and imports, the value of which had more than tripled since the 1760s.

Improved engines and the use of screw propellers produced more powerful boats, cutting travel times substantially. An upriver trip from New Orleans to Louisville, which once required twenty-five days, was reduced to less than five days. By 1820, sixty steamboats were operating on the western rivers. Exports had climbed to $70 million, and imports to $74 million. Steamboats were largely responsible for the growth of Mississippi Valley commerce, estimated at more than $650 million by 1852. Navigation of the waterways was a natural development, especially on the eastern rivers where passenger traffic was more important, but the railroad was recognized as a key factor in developing the transportation network and the push to go west. The railroads got a much later start than steamboats, partly because of the large capital expense required and partly due to the lag in technology.[55]

2.2.2 Railroads

Like the steamboat, the railroad, got its start in England. The early steam engines were bulky, extremely hot, and could not be used where fire was a hazard. They also consumed huge amounts of fuel. The possibility of using steam locomotives instead of horses to haul coal from the mine pits of England inspired experiments using faster, more compact engines. This produced a variety of coaches and configurations intended for rail transport, some of which had trouble staying on the rails.

In 1829, the Rainhill contest was held to determine which locomotive to use on the thirty-five mile track built for the new Liverpool & Manchester Railway. Stephenson's Rocket won by hauling a thirteen-ton train an average of fifteen miles per hour. With this event, problems of technology were considered solved.

The first successful railroad in America, the Baltimore & Ohio, was chartered in 1827. The B & O had a 300-mile (480 km) right of way from Baltimore, Maryland, across the Allegheny wilderness to Wheeling, Ohio. The first thirteen mile (21 km) stretch of B & O track was designed for horse-drawn cars traveling between Baltimore and Ellicott's Mills, Maryland. By 1833, 137 miles (220 km) of track to Harper's Ferry, Virginia had been completed. It was the longest stretch of railroad track in the world.[56]

The Mohawk & Hudson, chartered in 1826, was the earliest forbear of the New York Central. The Mohawk & Hudson railroad opened in 1830, as did the Charleston & Hamburg,

followed by the New York and Erie in New York, the Western in Massachusetts, and many more small lines.[57] By 1830, some 230 steamers were churning America's rivers and the American railroad industry was developing full steam ahead. Within ten years, the fame of American locomotives had spread abroad and their manufacturers began filling orders from Russia, Germany, Austria, Cuba, even England itself.[58]

One bottleneck to railroad expansion was overcome in 1839, when the Burden railroad spike machine was developed. It formed spikes in one operation, and produced them at a rate of fifty per minute, much more than fifty blacksmiths and their helpers could forge.

By 1840 more than 400 companies were operating almost 3,000 miles of track, and heavy duty steam-driven machinery was invented or adapted as needed to continue the push west. Between 1820 and 1850 the population of the United States grew from 7.9 million to 19.6 million, with the population living west of the Alleghenies rising from 27 to 45 per cent in that period.[59]

Between 1850 and 1860 rail mileage tripled to thirty thousand (40,000 km). Most of the new construction occurred in the Midwest, connecting cities of the east with those on the Great Lakes and along the Mississippi, and the Ohio Rivers.[60] In 1868, George Westinghouse invented the air brake, making the use of more powerful locomotives possible. The first transcontinental railroad was established on May 10, 1869, when the Golden Spike driven at Promontory Point,

Utah, joined the Union Pacific and Central Pacific railroads. A cross-country trip that had taken 118 days twenty years ago was now completed in a little over six days.

Between 1868 and 1873, thirty-three thousand miles of rails were laid, and many lines were double-tracked, with steel rails beginning to replace iron.[61] In addition to greater passenger comfort and speed, railroads cut inland freight rates still further and brought a mobility that was vital to the nation's commerce.[62]

2.2.3 Automobiles

The search for an internal combustion engine was advanced in 1860 when French inventor Etienne Lenoir built a small, single-cylinder, internal-combustion gas engine, but its running costs were excessive compared with steam engines.

Ford built his first successful automobile in Dearborn, Michigan, in 1896. It had a two-cylinder, four horsepower (3 kW) gasoline engine, and could travel sixty miles (100 km) on its three gallon maximum capacity tank. Three years later, Ford quit his engineering job with the Detroit Edison Company to concentrate on building the car he envisioned. In 1908, he introduced the Model T, a sturdy, practically indestructible car that cost $850.

The long sought concept of cost effectiveness through assembly-line production was finally achieved at Ford's Highland Park, Michigan plant. In 1913, assembly of the Model T 's five thousand parts were consolidated into

defined, successive, linear operations. The car drove out onto John R. Street on operation number forty-five.

As production climbed from 78,440 cars in 1911-1912 to 785,432 in 1916-1917, the Model T's price plummeted from $690 to $360. Before Ford discontinued them in 1927, some fifteen million Model Ts had rolled off the assembly line. Ford's Model T made automobile ownership possible for the average working person, and demonstrated the efficiency of assembly line production. Since then, both the number and variety of products and assembly lines have multiplied beyond reckoning.[63]

The overwhelming success of the automobile brought a new asbestos friction product into the market. In 1907, a woven asbestos fabric and wire composition product was introduced. It replaced the use of leather in brake shoes and clutch facings.[64] The best braking materials convert kinetic energy into heat very rapidly, and then dissipate the heat as quickly as possible.[65] This ability, along with other properties, including durability, traction, resistance to thermal and chemical breakdown, and incombustibility, made asbestos ideal for brakes.[66]

2.3 New Products and Uses Developed for Asbestos

The Industrial Revolution that had started in 1760 in England reached America about fifty years later, and was embraced with a vengeance in those industries which had been imported to the American colonies. Growth of the nation's industries

and transportation network was made possible by adopting, adapting, and improving steam technology.

2.3.1 The Power of Steam

The steam engine was the preeminent source of power in the nineteenth century. Because it was virtually impossible to machine cylinders and pistons to fit together exactly, various kinds of seals or packings had to be used. Most common in the mid-nineteenth century were leather packings or hemp soaked in grease. These and other organic materials deteriorated rapidly under the extreme conditions in the interior of steam engines.[67]

A major improvement in precision parts machining in 1776 led to the development of mass production.[68] By the 1850s, the standardized part was a symbol of American industrial practice. By the 1860s, asbestos was being spun, woven, or fashioned into a type of millboard or corrugated cardboard for use as a packing and sealing material. Asbestos was uniquely suited to this use "owing to its power of resisting moisture, friction, high temperatures, and even flame itself."[69]

Advances in steam engineering led to higher steam pressures and temperatures. Wasted fuel due to heat loss was still a major problem. Eighty to ninety percent of the heat loss could be prevented by providing insulated coverings.[70] Asbestos was a component in a large number of the new products developed for insulating hot engines, boilers,

and piping. One of these new products was called eighty-five percent (85%) magnesia.

2.3.2 Eighty-five Percent Magnesia Insulation

In 1885 Hiram M. Hanmore, a pipe coverer, began to mix magnesia with other pipe-covering materials and patented his idea. The addition of asbestos fiber acted as a binder to give strength and cohesion to the magnesia sludge, which could then be cast or molded into standard shapes and dried. The optimum mix was eighty-five percent magnesia and fifteen percent finely divided, long fiber chrysotile asbestos, the only type available when eighty-five percent magnesia was developed.

The use of eighty-five magnesia insulation spread rapidly, especially in the United States. By 1888 the U.S. Navy used coverings of eighty-five magnesia almost exclusively in its capital ships "because by maximum conservation of heat these coverings enabled ships to steam farther on a given coal capacity." In the 1880s and early 1890s, such products could be sold on the basis of their efficiency as well as their cost effectiveness.[71]

As the variety of materials enhanced by the thermal, condensation, and noise insulating properties of asbestos became more popular, they were used for a variety of purposes in shipbuilding. A characteristic difference among the types of asbestos was also utilized by the Navy to reduce the tonnage of naval vessels.

2.3.3 Use of Asbestos in Naval Vessels

During World War I, the allied powers agreed to a proposal by Charles Evans Hughes, Secretary of State under President Harding.[72] The 1921 Washington Treaty of Limitations in Tonnage called for a drastic reduction in the gross weight of naval vessels. The use of amosite reduced the density of eighty-five magnesia insulation from 16-26 to 14-18 pounds per cubic foot. Long fiber amosite was also less costly than the Canadian chrysotile. By switching to eighty-five magnesia insulation made with amosite, heavier weapons could be utilized without increasing the total weight of the vessels.

Amosite felt was developed in 1934. It was lighter, more resistant to higher temperatures (up to 900 degrees Farenheight), and more flexible than any previous insulating material. Loosely compacted amosite felt did not pack under vibration. The fabric retained its insulating qualities after it got wet and dried out again. Amosite felt was used as a protective covering for eighty-five magnesia insulation and other forms of insulation, and for turbines, valves, and fittings on virtually all U.S. combat vessels built just before World War II.[73]

Other weight saving materials which incorporated amosite into naval vessels included a lightweight, fireproof wallboard, called Marinite, used for partitions, and amosite insulated mattresses. The amosite mattresses weighed 9-1/2 pounds per cubic foot. Those made of chrysotile weighed up

to fifty percent more, and would not conform with Navy specifications. Asbestos was also used in other personal items, such as safety clothing, curtains and blankets on board the ships.[74]

Asbestos was utilized extensively by the Navy during building, conversion, and repair to insulate and fire-proof hulls, boilers, engines, electrical lines, and piping. The work was often carried out in confined spaces under cramped conditions without any ventilation or other respiratory protection.

CHAPTER 3

THE ASBESTOS INDUSTRY

3.1 Development

Speculation in asbestos contributed to one of the severest depressions the country had ever experienced during the Panic of 1893. In one six-month period, eight thousand businesses failed. One company that managed to survive was the H.W. Johns Manufacturing Company of New York.[75] The company's name would be as indelibly linked to asbestos as the Industrial Revolution's was to the power of steam.

3.1.1 The H. W. Johns Manufacturing Company

Henry Ward Johns had moved to Brooklyn in 1858 at the age of twenty-one, and began a small business handling roofing materials. Upon learning about the properties of asbestos, Johns experimented with various ways to incorporate it into his products. He patented roofing material made with asbestos in 1868 and also developed a prefabricated pipe covering. This new insulation product was lined with asbestos paper and precut to standard sizes. It could be wrapped or wired around pipes. The company's sales increased immediately.

Asbestos fibers added strength to the insulation and roofing materials. Shorter, inferior fibers were suited for these products and much less expensive than importing the

long fiber Italian asbestos. The raw material needed by Johns' products could be provided from local sources, such as the minor deposit of asbestiform material discovered on Staten Island in 1874. By 1880, the H.W. Johns Manufacturing Company had a second factory in Brooklyn manufacturing paints made with asbestos, marketed as a fireproofing product.[76]

3.1.2 The Birth of an Industry

In 1880 several European mining and manufacturing companies combined to form the United Asbestos Company, Inc., and subsequently monopolized the Italian chrysotile deposits.[77] The demand for noncombustible fabrics produced by the cotton industry in England and France provided incentive for commercial development of the mines. Incorporating asbestos gave the fabrics increased durability and better sealing properties.[78]

The chrysotile deposits discovered in Canada in 1876 opened a new supply source, and the slight hook on the Canadian fibers saved steps in processing for textile applications. An asbestos industry that had been virtually non-existent in the United States was about to evolve. Johns and his American competitors soon became the major consumers of Canadian asbestos. By 1889 the United Asbestos Company had acknowledged the superiority of the Canadian fiber and obtained mines in Quebec.[79]

The cost of asbestos had risen steadily during the 1880s. In 1889, first-quality fiber ranged from $80 to $120 per ton. By 1891, first-quality asbestos sold for $250 per ton or more. The typical cost of production and preparation for market of a ton of asbestos was about $25. As demand for asbestos increased, additional efforts were made to use the cruder fibers and to extract even more fibers from what had previously been considered waste material. Removal of impurities became important when it was discovered that they, and not the asbestos fibers in the packing material used in steam engines, caused scoring of the piston rods. The introduction of more capital equipment and machinery to accomplish the needed operations decreased production costs to about $15 dollars a ton. Mining was a profitable operation for companies which could finance the needed acquisitions, and asbestos mining was recommended as an ideal investment.

3.1.3 The Johns-Manville Corporation

In 1891 the H. W. Johns Manufacturing Company of New York retained its name and joined with several other firms and competitors to become the largest asbestos manufacturer and dealer in the world. Now the company had factories in New York, Chicago, and Philadelphia, which more than quadrupled Johns's former production capacity. By the early 1890s the new company had obtained its own mine in Canada, as did other

asbestos manufacturing companies in both the United States and Europe.[80] The first asbestos textile mill in the United States opened in 1896.[81]

In 1898, Johns died in Yonkers of scarring of the lungs.[82] His death certificate listed the cause as "dust phthisis pneumonitis," which was probably a medical euphemism for asbestosis.[83] His son took over the company. One vendor of Johns' products was a company started in 1886 by Charles B. Manville and his three sons. The Manville Coverings Company of Milwaukee also manufactured pipe and boiler coverings, but none contained asbestos. Manville sold Johns' asbestos insulation products in the midwest for high temperature applications. By the end of the century, the Johns Company was deeply in debt. The paint company was sold first, and a buy-out of the entire company to the Manville Covering Company of Milwaukee was arranged in 1901.[84]

Thomas F. Manville, a son of the Manville Company's founder, became president of the new Johns-Manville Company. Rather than focus on research and development as Johns had, Manville further diversified the company's product line by adding hundreds of non-asbestos products.[85] He built a big hotel in the small town of Manville, which had developed around the complex of asbestos factories started there in 1912.[86] He also established an extensive network of sales offices throughout the country staffed by salaried agents. By the fall of 1925, when T. F. Manville suddenly dropped dead in his hotel room, the company had sales of almost forty

million dollars a year and a catalogue of two thousand different products.[87]

3.2 Products and Competition

Growing recognition of the adaptability and utility of asbestos for modern industrial use stimulated the rapid exploitation of the Canadian deposits, and an expansion of asbestos manufacturing.[88]

3.2.1 Fiber Sizes Used in Products

The longer fibers (greater than 6 mm) were spun and woven into rope, tape or cloth.[89] Fabrics made of asbestos were soft, flexible, and very strong. They were widely used commercially where fire protection was essential. Common uses were curtains in theatres, or protective clothing in certain industries, including aprons, helmets, arm protectors, gloves, leggings, shoes, coats, and overalls.[90] As early as 1853, the uniform worn by the Parisian Fire Brigade consisted of a full hooded body jacket of asbestos-containing material (ACM). Fifty years later, fire fighters uniforms made of ACM encapsulated the whole body.[91]

Intermediate (2 to 6 mm) and the smallest size fibers (less than 2 mm) were more abundant than the longer fibers. They were used in products such as packing for steam engines; building materials such as roofing, shingles, and caulking; millboard; paper products; paint; brake linings;

clutch facings; electrical insulation; floor tiles; and filters.[92]

3.2.2 Friction Products

American companies competing with Johns-Manville included Keasbey and Mattison of Ambler, Pennsylvania, and Philip Carey Company of Cincinnati. Each had their own mines, and manufactured roofing and insulation products. The Asbestos Corporation, a combination of Canadian mining companies, was also active in this market. Other companies concentrated on newer specialty asbestos products, one of which was the manufacture of "friction products." These were primarily brake shoes and clutch facings, in demand due to the continuing rise in automobile sales.[93]

At the end of World War I, there were 5.5 million motor cars in America - one for every nineteen Americans.[94] In 1920 there were 8.25 million registered automobiles. The high numbers of people killed and injured each year in automobile accidents from driving without experience prompted a rapid conversion in the mid-1920s from two-wheel to four-wheel brakes as a safety measure.[95] By 1927 the number of automobiles had more than doubled the 1920 figure. By the end of the 1920s, there were more than 23 million cars on the roads - about one for every five Americans.[96] All of those vehicles had brakes and clutches that required asbestos friction products.[97]

Companies competing with Johns-Manville in the friction products market included the Raybestos Company, formed in 1916 in Connecticut; the General Asbestos and Rubber Company of South Carolina, started in 1895; and the Manhattan Rubber Manufacturing Company, which began in New Jersey in 1893. Raybestos bought out the General Asbestos and Rubber Company in 1925. Four years later, it consolidated with the Manhattan Rubber Company to become Raybestos-Manhattan Company, the country's largest manufacturer of friction products.[98]

Johns-Manville continued to dominate the building products and insulation sector, listing over fifty sales offices in the company's 1927 annual report. After T. F. Manville died, his younger brother had become president. Hiram Edward Manville consolidated, then promptly sold most of his holdings, retained a large block of Manville stock, and went into semi-retirement as chairman of the executive committee.[99] By 1927, Johns-Manville was no longer a family-owned business.[100] That was the same year in which Cooke coined the term asbestosis to describe an industrial lung disease caused by inhaling asbestos dust.

3.3 Summary

The asbestos industry was poised to take off, and their main concern was the ability to sustain a very profitable business. Industry, medical, and government interests and interactions, in combination with a number of other factors,

served to delay the consideration of practical measures to prevent exposures to asbestos.

As the industry grew, its executives collaborated to preclude damaging information about asbestos, while influencing legislation favorable to their business interests. Industry manipulated the dissemination of medical data as a condition of sponsorship, and controlled the workplace as a function of ownership. Industry's influence on the medical community compromised the research that was being conducted, and what was known about the hazards.

CHAPTER 4

MEDICAL AWARENESS in ENGLAND

4.1 Introduction

The ability to control hazards depends on the knowledge and assessment of the cause, as derived from some measure of exposure. The most accurate measures of occupational exposures are obtained from workers and workplace conditions.[102] During the first quarter of the nineteenth century, there were few regulatory restrictions and almost nothing was done to control dust levels in asbestos factories. Little medical information was gathered on asbestosis and few dust samples were taken.[103]

The long latency period of asbestos-related disease, ignorance about the cause, confusion with other pulmonary disease-causing agents, and evaluation of exposure were factors in establishing the hazards of working with asbestos. Awareness of the dangers posed by inhalation of asbestos fibers existed long before medical evidence established the nature of the hazard.[104] Some of the studies and reports from England which established the nature of the hazard are presented briefly in this section.

4.2 Early Reports of Disease

4.2.1 British Home Office Report, 1898

Until the 1870s, there had been minimal importation of asbestos into England. The Industrial Revolution and the inventions it spawned occurred there first, as did the first documented case of asbestos-related disease.[105] It was reported approximately thirty years after chrysotile was first mined, but serious health effects were noted before the turn of the century.

The earliest British government document to cite the hazards of asbestos fiber was The Annual Report of Her Majesty's Women Inspectors, in 1898.[106] The report mentioned asbestos textile work specifically, and identified common symptoms exhibited from the effects of the dust on the respiratory system.[107] Eight years later, the Lady Inspector of Factories reported that, of all the "dusty trades" for which complaints were received, none surpassed the injuriousness of asbestos processes."[108]

4.2.2 Report of Mortality, 1906

The results of the first study of mortality among asbestos workers appeared in 1906, in the Bulletin de l'Inspection du Travail et de l'Hygiene Industrielle.[109] The article was an account by an inspector in the Department of Labor at Caen named Auribault. He was reporting on conditions at an asbestos-weaving mill that had been established in 1890. Fifty workers died in the first five years of the mill's

operation, including all but one of seventeen workers recruited by the factory's director, from the staff of a cotton mill he had previously owned.

4.2.3 Murray, 1900

The first fatal case of pulmonary asbestosis was observed in 1900 by Dr. H. Montague Murray, Senior Physician at Charing Cross Hospital in London. It involved a thirty-three year old man who had worked for fourteen years in an asbestos textile factory. He claimed to be the last survivor of ten men who had been working in the carding room of the factory when he started in 1886. The patient had been suffering from severe pulmonary fibrosis. At autopsy, Murray found spicules of asbestos in the lung tissue.[110] Although not the first such case reported in detail, Murray's was the first with a post-mortem description of the victim's lungs as extremely tough and fibrous, especially in the lower parts.[111] As the first documented case of a death resulting specifically from asbestos, it established a presumptive connection between occupation and disease.[112]

In testimony before a British Departmental Committee on Compensation for Industrial Diseases in 1906, Murray said, "that considerable trouble is now taken to prevent the inhalation of dust, and so the disease is not so likely to occur as heretofore."[113] His correlation between asbestos dust and disease prevailed for the next two decades.

Attention focused on reduction of the high dust levels and dusty conditions commonly found in industry.

4.2.4 Cooke, 1927

In 1927, the year in which Johns-Manville ceased to be a family-owned business, Dr. W. E. Cooke, an English physician, coined the term asbestosis. Cooke used the term to describe[114] the unusual fibrosis of the lungs he had observed and previously reported, regarding the case of a thirty-three year old female patient. At the age of thirteen, she had started working in an English asbestos-textile factory that had no system to remove dust. By 1917, after thirteen years of exposure, she was coughing and in bad health.[115]

The case had been detailed in Cooke's article "Fibrosis of the Lungs Due to the Inhalation of Asbestos Dust," published in the July 26, 1924, edition of the British Medical Journal.[116] The autopsy showed extensive pulmonary fibrosis and dense strands of abnormal fibrous tissue connecting the lungs and the pleural membranes surrounding them.[117] It was the first clear case of death due to asbestos exposure, and only the second death from asbestosis that had been so identified.[118]

Cooke's discovery sparked intensive study in England over the next seven years. The most important work was conducted by Dr. E.R.A. Merewether, Medical Inspector of Factories for the British Home Office.[119]

4.3 Medical Studies in England

4.3.1 Merewether, 1930 - Asbestosis and Silicosis

In 1928 the British Medical Journal published Seiler's South African case of "pure" asbestosis in an asbestos textile worker. This was only the third such case reported. It prompted the Factories Department of the Home Office to initiate a study of asbestos textile workers in England's factories.[120]

Between 1928 and 1929, a comprehensive study was conducted by Merewether and Price. Their investigation was confined to workers exposed to pure asbestos fibers.[121] After excluding other possible causes, they concluded that over one-quarter of the workers had contracted the disease because of their occupation.[122]

Merewether and Price determined that, over a period of years, inhalation of asbestos resulted in serious fibrosis in those air-cells of the lungs where the asbestos came to rest. The normal reserve capacity of the lungs masked the effect for some years. They found that the disease could be fatal, with no detectable difference evident, when considering the different types of asbestos fibers used in British industry. They also discovered that the disease was dose-related. Workers exposed to higher concentrations of dust had a greater probability of becoming ill.

Of the 160 factories employing 2,200 workers in manufacturing asbestos products in England in 1930, Merewether and Price found 18 factories involved in weaving textiles

from fibers. MuCulloch outlines how the subjects were selected and gives a description of conditions in the most dusty plants, where mattresses were made using asbestos as a filler and covering material. Brouder also gives an account of the study selection and results.[123]

Merewether and Price's study was entitled "Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry: Part 1 - Occurrence of Pulmonary Fibrosis and Other Pulmonary Afflictions in Asbestos Workers; Part 2 - Processes Giving Rise to Dust and Methods for its Suppression". Published in 1930, it was the first major study of the effects of asbestos on occupational health published in Britain. The authors' reasoning - that reducing the dust levels found in industry would reduce the dose, and therefore lengthen the time before the appearance of disease - emphasized elimination of the hazard by controlling dust levels in industry.[124]

Based on the results of the study, the Home Office issued a notice to all manufacturers about the pathological and clinical distinctions between silicosis and asbestosis. Each disease was described in detail. Dust suppression, such as wet versus dry methods of processing, and other engineering controls were recommended. Periodic medical examinations for all employees was also encouraged.[125] By 1930, the hazard had grown to such proportions that measures were adopted to suppress dust in asbestos factories.[126] Parliament passed legislation in 1931 that instituted

periodic medical examinations for workers engaged in particularly dusty processes in the asbestos-textile industry, made asbestosis a compensable disease, and required improved methods of ventilation and dust suppression in asbestos-textile factories.[127]

4.3.2 Merewether, 1947 - Lung Cancer

As dust levels decreased, more workers lived long enough to develop lung cancer. In this regard, another study conducted by Merewether is topical, because it provided the first evidence of a causal link between asbestos and lung cancer. An association between lung cancer and asbestosis was first suggested by clinicians in the 1930s.[128] Some articles appeared in the British medical journals, but lung cancer was not a frequent diagnosis at the time and no firm conclusions were drawn.[129]

In 1934, Wood and Gloyne described lung cancer in asbestos workers.[130] The following year, the first published reports began to suggest that asbestos workers with asbestosis also appeared be at unusual risk of developing cancer of the lung. Two of those reports were by Gloyne, and one was by Lynch and Smith from Charleston, South Carolina.[131]

In 1936, Gloyne reported a case of oat cell carcinoma in an asbestos factory worker, and Middleton reported three lung cancers among fifty-four cases of asbestosis. Another

American case by Egbert and Geiger, from New Haven, Connecticut, brought to six the total number of published reports by 1936.

Three more cases were reported in 1938. These came from Germany,[132] whose physicians by that time were calling lung cancer an occupational disease of asbestos workers. The following year, asbestosis became a compensable disease in Germany.[133] An editorial in the British Medical Journal reviewed the general question of the relationship between lung cancer and dust diseases, and urgently called for further study.

Six more articles appeared between 1938 and 1942 that mentioned a suspected relationship between asbestos exposure and cancer of the lung. Seven pages were devoted to asbestosis in Wilhelm Hueper's 1942 textbook, Occupational Tumors and Allied Diseases, in which he reviewed the evidence and concluded that "there is an incidence of lung cancer in asbestosis of the lung which is definitely excessive."[134]

In 1947, Merewether investigated the link between lung cancer and asbestosis. He considered 235 autopsy reports in the period from 1924 until 1946, in which the acknowledged cause of death was asbestosis, and found 31 with cancer of the lung or pleura. In addition to the causal link, Merewether found a much higher incidence of cancer when compared with silicosis sufferers.

Merewether's report was published in 1949. It was substantiated by other studies which culminated in Richard Doll's work.[135] Doll studied post-mortems dating back to 1935, and provided epidemiological evidence of a causal link between cancer and asbestos exposure. Doll concluded that cancer of the lung was a specific hazard for asbestos workers.[136]

4.4 State of Medical Awareness

That exposure to asbestos may lead to diffuse pulmonary fibrosis has been known since early in this century.[137] Because of ignorance about the cause of the disease, and because it manifests itself slowly and insidiously over a period of ten to twenty years or more, asbestosis was probably, more often than not, misdiagnosed as pulmonary tuberculosis, fibrosing pneumonia, or silicosis.[138]

In referring to the uncertain nature of the hazard, the 1898 Annual Report of Her Majesty's Women Inspectors stated that, "the general symptoms produced by dust on the various respiratory organs are to the lay mind so similar to those produced by other causes that it is not always easy to trace the connection."[139] This difficulty in diagnosis was evident in earlier works of Murray, Cooke, Merewether and others.

Cooke's 1924 case, and others previously reported, was complicated by co-existing tuberculosis or pneumonia, making

an exact specification of the effects of asbestos dust less certain.[140] Over the next five years, a number of articles on the subject of pulmonary disorders would indicate that asbestos-related disease of the lungs was quite distinct from that in patients with tuberculosis.[141]

Industrial lung diseases such as silicosis and asbestosis were well defined in the medical literature by 1930. A specific chronic disease of the lungs attributed to breathing asbestos was well described in dozens of different medical articles, involving hundreds of cases. In addtion to confirming the link between lung cancer and asbestosis, Merwether's 1949 report showed a much higher incidence among those suffering from asbestosis than for those with silicosis.[142] An awareness of the individuality of diseases caused by asbestos dust was reflected by the title of the Third International Conference of Experts on Pneumoconiosis held in Sydney, Australia in February and March, 1950. Previous conference titles had only used the term "silicosis."[143] The 1950 conference title acknowledged the broader spectrum and individuality of dust diseases.

CHAPTER 5

DEVELOPMENT OF AWARENESS in the UNITED STATES

5.1 Introduction

The English studies documented the very real hazards of occupational exposure to asbestos dust, and differentiated asbestosis from other forms of pneumoconiosis. As early as 1907, more than 280 American entries were listed in a preliminary bibliography on occupational diseases and industrial hygiene.[144] Despite this information, asbestosis would be confused and overshadowed in the United States by the contemporary pulmonary diseases of tuberculosis and silicosis.[145]

5.2 Early Reports of Disease

5.2.1 Pancoast, 1917

The first published case of asbestos-related disease in the United States was reported by Dr. Henry K. Pancoast.[146] Like Murray of England, Pancoast was a pioneer in the use of X-rays for medical diagnosis, and was considered to be the foremost roentgenological authority in the United States.[147]

In 1915, Pancoast and his colleagues at the University of Pennsylvania began an investigation of chest X-rays in the diagnosis of workers exposed to dust in various occupations. These included potters, metal grinders, cement workers, coal miners, marine firemen, and asbestos workers.

Pancoast concluded that the "effects are the same in general but may vary in degree." The report was published in 1917. Its reception in the medical community focused on the economic impact of the undue prevalence of tuberculosis in industrial workers. The following year, Pancoast et al. reported findings of lung scarring and fine fibrosis in X-rays of fifteen asbestos factory workers.[148]

5.2.2 Hoffman, 1918

In 1918, the United States Bureau of Labor Statistics published a special study titled "Mortality from Respiratory Diseases in Dusty Trades (Inorganic Dusts)."[149] The study was conducted by statistician Frederick L. Hoffman, chief actuary for the Prudential Insurance Company of America, Newark, New Jersey.[150] The report's conclusion noted "that in the practice of American and Canadian life insurance companies, asbestos workers are generally declined on account of the assumed health injurious conditions of the industry." Hoffman concluded that the industry involved "considerable dust hazard," and cited the fact that American asbestos workers were experiencing unusually early deaths.[151] Hoffman called urgently for "more qualified and extensive investigation of the health aspects of asbestos manufacture."

Hoffman's findings and his urgent call for further investigation were repeated in future years by others researching asbestos-related disease. Their calls went

unanswered for various reasons. Among these were the laissez-faire relationship between government and industry, events of national scope that displaced attention, and lack of any adequate response to the research results sponsored by industry. There was also the ongoing problem of recognition of asbestos as a disease causing agent.

5.3 "The New Public Health"

The earliest American reports of asbestos related disease were published amidst changing ideas about, and approaches to, disease identification, causation and control. The belief that disease resulted from miasmas emitted by decaying organic matter held sway in the United States until the 1890s.[152] This was superseded by the germ theory of disease, which focused on the individual as the key factor in disease causation. H. W. Hill, of the Minnesota State Board of Health, expounded the belief that disease producing germs resided chiefly in relatively few people. In a widely read and influential book for the general public, called The New Public Health, Hill announced the New Order: "The old public health was concerned with environment; the new is concerned with the individual. The old sought the sources of infectious disease in the surroundings of man; the new finds them in man himself."[153]

Unlike the Inspector of Factories in England, there was no centralized data collection, assessment, or federal regulation of workplace hazards in the United States.

Responsibility for public health rested with territorial, state, or local governmental units which were spread out across the frontier society.[154] Most of the state and local agencies were thinly staffed, minimally funded and often politically controlled. These agencies had little if any regulatory power and lacked laboratory and other technical resources so vital to the surveillance of hazards in industry.[155] Even federal government agencies, such as the Public Health Service, required the cooperation of industry to gain access to their factories.[156]

Until the 1920s, the asbestos industry was so small that no individual physician would have come across a sufficient number of cases of asbestosis, or lung cancer among asbestos workers, to be alerted to the connection. The American Medical Association (AMA), in representing the interests of individual physicians, and in promoting free market competition, ostracized contract practice, as economically and professionally detrimental to the medical profession. From 1901 to 1920, during the period in which mergers created giant corporations in industry, the AMA waged a negative campaign against contract doctors while reorganizing to become one of the most effective and powerful lobbying organizations in the United States.[157]

Public health reformers did not perceive the environment of the worker as their main concern even though industrial settings provided the "great human laboratory" envisioned by pioneer industrial physician Henry Mock.[158]

5.4 Government Initiatives

Physicians had always been employed to treat injured workers, but when the rate of industrial accidents became a national scandal early in the twentieth century, state and federal agencies were forced to respond to conditions in industry. Disasters, especially epidemics, created the political pressures necessary for initiating action.[159] It was during this period that the new field of industrial hygiene emerged.

5.4.1 *Regulating Working Conditions*

The first federal law to regulate working conditions was the Hours of Service Act passed in 1907. A coal mine explosion in West Virginia that same year led to creation of the Bureau of Mines in 1910, and in 1913 the Department of Labor was formed to "foster, promote and develop" the lot of wage workers. One of the first reports on asbestosis in the United States appeared in a 1918 Department of Labor publication.[160]

Between 1911 and 1920 most states passed worker compensation laws requiring employer's to carry liability insurance to ensure payment of legitimate claims. Worker's compensation tion was intended to be an incentive to reduce accidents and injuries by improving working conditions. Due to industry's influence on the lawmakers, the legislation proved to be a boon for employers. They were allowed to compensate for loss of life or limb at bargain prices during a time when injury

and death rates had soared in coal mines, steel mills and textile factories.[161] In the 1930s, the public outcry, over another disaster in West Virginia, prompted a change in some of these laws.[162]

5.4.2 U. S. Public Health Service

In 1914, the United States Public Health Service formed a Division of Occupational Health. It was headed by Dr. Joseph Schereschewsky, first president of the American Association of Industrial Physicians and Surgeons (AAIPS). Mock was one of the 125 physicians and surgeons, in industry or government service, who met in Detroit to form the AAIPS in 1916. Like members of similar groups, such as the Conference Board of Physicians in Industry, members of the AAIPS identified more strongly with the large corporations for which they worked, than did the average contract physician.[163]

Schereschewsky also headed the Public Health Service's Division of Industrial Hygiene until 1918. On staff with this Division were Anthony Lanza and William McConnell, who both participated in industry sponsored asbestos research in later years. Under Dr. Lewis Thompson, the Division of Industrial Hygiene carried out a number of extensive dust studies using equipment developed jointly by Dr. Leonard Greenburg and George W. Smith, of the Public Health Service and the Bureau of Mines, respectively. Soon after taking over in 1923, Thompson, hired a young sanitary engineer named Jack Bloomfield. Bloomfield pioneered the use of the

Greenburg-Smith impinger over a ten year period. It became the standard instrument for dust measurement in the United States.[164]

5.5 Harvard's Program in Industrial Hygiene

One of the first comprehensive programs in industrial hygiene was developed by Philip Drinker, a professor at Harvard University. The new program began as a joint venture between the Sanitary Engineering Department of the Engineering School, and the Department of Ventilation and Illumination at the new School of Public Health.

When the School of Public Health was formally founded in 1918, retired Jackson Professor Frederick Shattuck raised $125,000 from manufacturing firms in New England to support the new program. The program was managed by Philip's older brother, Cecil Drinker, who succeeded Shattuck as Jackson Professor in 1916, and eventually became the second dean of the School of Public Health.

The objective of the program was to train company doctors, and teach students how to investigate industrial health problems. Industrial hygiene was regarded principally as a medical problem. A certificate of public health was issued after completion of a five month course. As the program expanded, it became apparent that industry was more interested in engineering problems, some of which were beyond the scope of the industrial physician's ability to solve.

In 1922, the School of Public health entered a new phase, paying more attention to the measurement and control of hazards. Physicians in the program were soon outnumbered by engineers and scientists, who were taught to design less dangerous workplaces. In this capacity, the university's facilities were made available for research and training.

The Schools of Public Health and Engineering launched Harvard's new program in industrial sanitation in 1927. The title is significant. Until 1925, sanitary engineering was the only discipline that addressed the impact of the environment on public health. This impact was exemplified by the great improvement in public health following reforms such as water purification and waste disposal earlier in the century.

Eventually, the program became the Industrial Hygiene Department of the School of Public Health.[165] The first engineering oriented graduates from Harvard's industrial sanitation program would again connect public health with the environment, this time in industrial settings. They graduated just in time to address the growing silicosis epidemic.

5.6 Silicosis

The medical literature of the 1930s pointed to dust disease as the prevalent threat in occupational settings, and an avalanche of litigation by workers suffering from silicosis got the most attention. Silicosis is a disease associated

with those who drill, crush, or pulverize rocks, such as granite and sandstone, where silica is released into the air as respirable dust. Breathing becomes difficult as the lung tissue becomes scarred, and shortness of breath is a common result. Those who are thus affected are more susceptible to tuberculosis, a complicating factor in diagnosis.

Measures to prevent the disease were suggested as early as 1700, by Bernardino Ramazzini, a professor of medicine at the University of Padua. The first pathological description of silicosis was given in 1762 by Ijsbrand van Diemerbroeck, a professor of medicine at the University of Utrecht. In the United States in 1887, silicosis was found at autopsy in the lungs of a stove-foundry worker in Poughkeepsie, New York.

Though well established in the medical literature, and despite protective practices standardized by the Bureau of Mines, more than one million workers in the United States had been exposed to harmful amounts of silica dust by 1930. At least one in four of them could have been expected to develop silicosis. In 1933, damage suits totaling more than $100 million were pending, and a year later the figure had risen to $300 million.[166] One source of the swelling number of lawsuits was the Hawks Nest tunnel disaster, near the town of Gauley Bridge in Fayette County, West Virginia.

5.6.1 Gauley Bridge - Hawk's Nest Tunnel Disaster

Excavation on the tunnel began in late March, 1930, with a crew of about two thousand laborers. Three-fourths of them

were Black, many had migrated to the area, and all of them were paid depression era wages. The contractor, Rinehart and Dennis of Charlottesville, Virginia, permitted dry drilling of the 3-3/4 mile tunnel through Gauley Mountain, which was, in parts, 99.44% pure silica. Construction was completed in December, 1931.[167] It was reported that almost five hundred workers died, and fifteen hundred were disabled from severe silicosis within a few years.[168]

At the time, worker's compensation only covered those occupational injuries which occurred at a definite time and place. Silicosis was a compensable disease in only six states, and West Virginia was not one of them.[169] Eighty claims had been filed with the West Virginia State Compensation Commission. The contractor's appeal to have the claims settled through the Commission was denied by the state supreme court in February of 1933. The only other redress for injured tunnel workers was through common-law negligence suits, which were eventually filed by more than 500 of the 2,500 tunnel workers.[170]

The first lawsuit came to trial in the Fayette County District Court on March 16, 1933. It lasted six weeks, and ended in a hung jury. In an out-of-court settlement, the contractor paid $130,000 to 157 plaintiffs. Half of that amount was in exchange for the plaintiff's seventeen attorneys' agreement not to prosecute.[171] By the time the trial concluded, more than 330 suits were pending, and the courts were clogged. More than two hundred

additional new lawsuits were thrown out by the courts, citing the trial agreement to forgo further prosecution.

In March, 1935, the West Virginia legislature enacted new provisions to cover silicosis, including a one year statute of limitation for filing claims, and a two year employment requirement. No action was ever taken on the original eighty claims filed with the Commission, and the new time restraints jeopardized the viability of the pending lawsuits.[172] As a result, Rinehart and Dennis was able to settle the remaining cases for some $70,000, with the stipulation that all evidence and data be relinquished.[173]

The story of Gauley Bridge might have remained a local tragedy. By chance, early in 1934, a playwright named Albert Maltz learned about it, and passed the story on. Late in 1935, the People's Press, a weekly labor tabloid, broke the story under a banner headline that read, "476 DEAD, 1500 DOOMED, IN W. VA. TUNNEL CATASTROPHE."[174]

The resulting publicity prompted hearings before a subcommittee of the House Committee on Labor in January, 1936, but Congress made no further effort to investigate the charges. However, the public outcry resulting from the hearings did promote a change in legislation, with more attention paid to occupational disease.[175]

5.6.2 Industry Response

In business circles, silicosis was known as the "Depression disease." One remedy for industry to deal with the rising

number of claims had been recommended in 1932, by Andrew Farrell, a Chicago insurance-company lawyer. In an article in Industrial Medicine, concerning the onslaught of silicosis claims, Farrell recommended that silicosis and other pulmonary dust diseases be taken out of the courts and covered by worker's compensation acts wherever possible, using the statute to limit the amount that could be recovered. Worker's compensation statutes for pulmonary dust diseases were enacted in some twenty states over the next decade, and business interests were once again placed before the health and welfare of the afflicted workers.[176]

Research and training opportunities were other responses to the mounting silicosis claims. The Air Hygiene Foundation was created in 1935, at industry's request and with backing from the Mellon Institute. It was established specifically to address technical issues concerning silicosis that industry felt were beyond its own capability. Among its twenty members were representatives from the Aluminum Company of America, Dupont, Owens-Illinois Glass, the Public Health Service, and the Bureau of Mines. Anthony Lanza chaired the Medical Committee. The Preventive Engineering Committee was chaired by Philip Drinker.

In 1941, the name of the organization was changed to the Industrial Hygiene Foundation. At that time, John D. Harper, Vice-President of the Aluminum Company of America, wrote that "It is encouraging to me that in an era when the extensive

grasp of government is reaching out to regulate and control more and more phases of economic life, there is a voluntary and nongovernmental research organization to turn to on problems of environmental engineering."[177]

5.7 The State of Industrial Medicine

The employer's consideration for workers' health was not so much with installing costly preventive or protective measures. Their concern was that escalating disability payments would negatively effect production and profits. Their insurer's were frightened about the prospect of having to make massive compensation payments.

The primary social role assigned to contract doctors was to keep workers' compensation costs down, and their primary tool was the physical examination. For industrial use, the examination was reduced to a ten minute assessment of a worker's fitness for work. Knowledge regarding a worker's medical condition was, by law, the exclusive property of the employer, not of the employee.[178]

The ambiguity between an industry that is economically healthy and one with healthy workers was a blurred distinction that lay at the heart of early industrial medicine. A 1930 editorial in the Journal of the American Medical Association set the boundaries: "Industrial medicine...must deal with the worker as a producing unit not as a social unit." The editorial warned that to do otherwise would

result in loss of status as specialists and a reversion to their former reputation as "poorly educated, low salaried 'hacks'."[179]

The restrictions placed on physicians in the workplace is exemplified in Brouder's account of the Drs. Wise. David Wise began as a contract physician for Johns-Manville's Pittsburgh, California plant in 1926. His son, Kent, took over in 1962, and was specifically instructed to ignore medical evidence of disease among the workers. Both doctors were named in a lawsuit, filed by former workers against the company in the 1970s, at which time Johns-Manville refused to defend either one.[180] Even though distinguished as a specialty and separated from the discredited tradition of contract practice, industrial medicine in 1930 was still not a part of mainstream organized medicine.[181]

As described in The Health and Safety of Workers, the alignments and divisions that resulted from the reorganization of medicine in the United States had unintended side effects on the discipline of public health and the nascent specialty of industrial medicine. For the latter, Ozonoff notes that "the result would be a shift in focus from the individual employee to the interests of the employing firm."[182] Access to, and conditions in the workplace, and knowledge of the hazards of asbestos exposure were still controlled by industry.

5.8 The State of Industrial Hygiene

Against this backdrop, industrial hygiene was emerging as a specialty in its own right. In 1930, only five states and one city (New York City) had official industrial hygiene programs. In an effort to increase those numbers, the Public Health Service initiated a series of short training seminars in industrial hygiene, with funding provided from the 1936 Social Security Act. The seminars were held in conjunction with the territorial and state health agencies. A group that formed to help coordinate actives for the fledgling state industrial hygiene units became the American Conference of Governmental Industrial Hygienists (ACGIH).

The American Industrial Hygiene Association (AIHA) was the major support organization for those employed full time by private companies, many of which held large government contracts.[183] Viewing themselves as worker rather than company oriented, "the ACGIH split from the AIHA in 1938 expressly to keep out corporate influence."[184] Ironically, the ACGIH would later recommend an inadequate threshold level for exposure to asbestos dust that was adopted as the standard by industry.

5.9 Concern Over Mounting Litigation

The Gauley Bridge tunnel tragedy was a symbolic turning point. It focused all attention on silicosis, even though tuberculosis was still the prevalent concern. Concern with

asbestos-related disease was overshadowed, but it was not unnoticed. The first disability claim for asbestosis was filed in 1927, by a foreman in the weaving department of an asbestos-textile mill in Massachusetts. The claim was upheld by the Massachusetts Industrial Accident Board. In 1930, the first case of asbestosis found at autopsy in this country was reported in a journal called Minnesota Medicine. The subject of the case was a worker who had been employed at an asbestos mine in South America.[185] In 1932, there was a report of an asbestos-related disability of a hospital maintenance worker in Wisconsin.[186]

Construction of the Gauley Bridge tunnel began as the depression was starting. By the time the incident was finally publicized in late 1935, the depression was in full swing. By then, the public's attention was focused on jobs, not on job health.[187]

5.9.1 Insurance Companies

During this period, industry and their insurers assessed the possibility of a similar situation regarding their products' manufacture and liability. The position they took gave asbestos-related diseases more time to incubate. The insurers position appeared in the Philadelphia Record in August 1935, in an article of response, by John L. Spivak, about the insurance companies' awareness of the asbestos hazard. Spivak wrote of an intercompany occupational rating

conference that had been held in May, 1928, at the home office of the John Hancock Mutual Life Insurance Company in Boston, Massachusetts. At the conference, officials of the Penn Mutual Life Insurance Company of Philadelphia delivered a paper advising their colleagues, that:

> until we have the benefit of our experience with this class of workers, we should continue to look upon those who may be exposed to large quantities of dust...as risks to be selected with great care and only at an extra premium that will provide for an estimated extra mortality of fifty percent, disability not to be granted."[188]

The following year, the nation's largest life-insurance carrier, Metropolitan Life Insurance Company, began a survey of health conditions in asbestos-textile mills. The report was completed in 1931, but was not published until 1935. By that time, the perception of the hazard had changed.[189]

5.9.2 Industry

The concern with litigation was also recorded in Johns-Manville's Board of Directors meeting minutes dated April 23, 1933. Negligence suits filed by former employees who had developed asbestosis while working at the Manville, New Jersey plant were pending in federal court. The company's attorneys had been approached by the attorney for the plaintiffs "with an offer to settle all the cases upon a much

lower basis than had ever been previously discussed." The recommended settlement offer was "approximately $30,000 provided written assurance were obtained from the attorney for the various plaintiffs that he would not directly or indirectly participate in the bringing of new actions against the Corporation." The minutes of this meeting came to light during court proceedings which took place more than forty-five years later.[190]

Various points of view emerged as disability and compensation claims became big items on the business agenda, and issues of occupational health crept into public debate.

CHAPTER 6

A DECADE OF MEDICAL STUDIES

6.1 Introduction

The precise data required to permit appropriate medical and engineering controls was only available from impartial scientists in industry or in the public sector.[191] Research studies were sponsored by the industry, and conducted by the Public Health Service. The dichotomy of views about the nature and extent of the asbestos hazard was reflected in the research results.

Industry executives decided to fund medical research that would discredit reports of asbestos hazards, and "keep...a check on workers' health while telling them as little as possible."[192] To this end, the asbestos industry funded a substantial amount of research, some in conjunction with the insurance companies. In 1928, industry began a long-term relationship with the Saranac Laboratories.

The body of work sponsored by the public sector was conducted by the US Public Health Service in conjunction with various state units. Much of the work involved devising detection methods, equipment, and techniques with which to make dust measurements during field. One of the first of these studies became the basis for the ACGIH's recommendation for an allowable level of exposure to asbestos in industry.

6.2 Lanza, 1929

Silicosis and Asbestosis was a popular medical text written by Dr. Anthony Lanza. In a chapter on the health and economic aspects of pulmonary dust disease, Lanza wrote that "Silicosis and asbestosis burst upon the amazed consciousness of American industry during the period 1929-1930," citing "Among the first claims for damages were those for asbestosis, a hitherto unheard of disease in the United States."

At the time Merewether began his study of workers in England's asbestos textile mills, Lanza began similar work on a smaller scale in the United States. "In 1929, the Metropolitan Life Insurance Company was approached by firms representing the asbestos industry in the United States with the request that a hygienic study be made of that industry, to ascertain "... whether asbestos dust was an occupational hazard in their establishment and if so, what was the nature of this hazard and what should be done to prevent or control it."[193] So began the introduction to the first epidemiological study of asbestos workers in the United States.

The study was conducted by Lanza and two of his colleagues for their employer, the Metropolitan Life Insurance Company. Lanza was assistant medical director for Metropolitan Life. Working with him were William J. McConnell, formerly of the Public Health Service's Industrial Hygiene Division, and William Fehnel, a Harvard trained industrial hygienist. After attending Harvard, Fehnel

returned to the New Jersey Zinc Company. Previously, he had worked there as a chemist, and was assigned to assist the Drinkers in measurements of dust levels during their investigation in the early 1920s, of manganese poisoning at the plant. Fehnel returned to the company to monitor the program, and may be the first person hired by industry specifically to perform industrial hygiene work. He left New Jersey Zinc shortly thereafter to work at Metropolitan Life.[194]

From October 1929 through January 1931, Dr. Lanza and his colleagues examined and took X-rays of workers [195] in five asbestos plants and mines in the United States and Canada.[196] Results showed an unusual prevalence of enlarged hearts. Signs of fibrosis appeared in the X-rays of forty-three percent of those engaged less than five years in asbestos factory work, and rose dramatically to eighty-seven percent for those with over fifteen years.[197] Fifty-three percent of the workers studied were classified as asbestotics. Only seventeen percent were asymptomatic.[198]

The written report, entitled "Effects on the Inhalation of Asbestos Dust Upon the Lungs of Asbestos Workers," was submitted to industry sponsors Johns-Manville and Raybestos-Manhattan for their review just as a New Jersey legislative commission was trying to decide if silicosis should be made a compensable disease under worker's compensation. With the increased attention on dust borne diseases, the sponsors were

concerned that the report, if published as written, might prompt inclusion of asbestosis in the legislation.[199]

The sponsors complicity in editing the report was revealed in correspondence contained in files belonging to Sumner Simpson, president of Raybestos-Manhattan. The files were recovered in 1977 during deposition for a product liability suit filed in May 1975 on behalf of former workers at a Raybestos-Manhattan plant in Passaic, New Jersey. They had been saved by his son, William, who was then CEO of the company. The recovered files included correspondence between Simpson and Vandiver Brown, head of Johns-Manville's legal department. Sumner Simpson had died in 1953, and the plaintiff's attorneys were told that Vandiver Brown was also dead. In fact, he was in Glascow, Scotland, very much alive, but mentally debilitated.[200]

Upon reviewing the 1931 report, Brown had consulted with attorney George S. Hobart, of the law firm of Hobart & Minard, Newark, NJ, who had handled some damage claims for Johns-Manville. In stressing the importance of a medical report which drew a distinction between asbestosis and silicosis, Hobart's reply to Brown outlined a "state-of-the-art" defense argument that the industry would use for the next forty years:

> ...one of our principal defenses in actions against the company on the common law theory of negligence has been that the scientific and medical knowledge

> have been insufficient until a very recent period to place upon the owners of plants or factories the burden or duty of taking special precautions against the possible onset of the disease in their employees.[201]

Variations of the "state-of-the-art" defense were used successfully by the industry, until Borel vs. Fibreboard Paper Products Corp. et al. That suit was filed by Ward Stevenson, in federal district court in Beaumont, Texas, on October 29, 1969, against eleven asbestos insulation manufacturing companies. In the first case of its kind in the nation, Stevenson argued successfully that the doctrine of strict liability applied to asbestos insulating materials. By failing to provide adequate warnings of foreseeable dangers associated with their products, the manufacturers had breached warranties, and could be held strictly liable in the death of the plaintiff, Clarence Borel. In Borel vs. Fibreboard, the courts established that exposure to asbestos was cumulative. Each exposure represented a separate, additional injury.[202]

In November 1933, more than two years after receiving Lanza's report, Simpson invited discussion on his idea to standardize methods of dust control in asbestos factories. A month later, on December 29, 1933, Simpson and three Johns-Manville executives (W.R. Seigle, Chairman of the Board; E.M. Voorhees, Secretary; and Brown) agreed to allow Metropolitan Life to bring its survey up to date.[203]

During the 1931, study Lanza had observed that the severity of symptoms exhibited was less than might be expected from the X-rays, and concluded that asbestosis was a clinically milder disease than silicosis. By late 1934, he was aware that other scientists, including Merewether, were finding asbestotics dying at a younger age, and contracting their disease under dust conditions that appeared to be safe in other industries. Lanza revised the 1931 report accordingly, and submitted the proofs.

Vandiver Brown requested a compromise from Lanza. "All we ask is that all of the favorable aspects of the survey be included and that none of the unfavorable be unintentionally pictured in darker tones than th circumstances justify."[204] Among the changes proposed was a reinstatement of the 1931 report's conclusion, complete with the suggested wording that "Clinically, from this study, it appears to be of a type milder than silicosis." Presenting asbestosis as a less dangerous disease than silicosis obscured the serious health hazards of asbestos as demonstrated by the study. Yet, the changes were accepted as editorial comments by Lanza, sanctioned by Metropolitan Life, and incorporated in the final report.[205]

The final Metropolitan Life report emphasized the differences between asbestosis and silicosis. The finding that fifty-three percent of the workers were classified as asbestotics was absent. The study was finally published on

January 4, 1935 by the Public Health Service. By then, Lanza had become a leading figure in the field of occupational medicine.[206]

Lanza's Silicosis and Asbestosis was published in 1938. In it, he identified dyspnoea, or shortness of breath, as the most striking symptom of asbestosis. He explained the detectable changes in X-rays preceding the reduced lung function that caused shortness of breath. He also noted that misdiagnosis of asbestosis was frequent because other cardiac ailments were thought to cause enlargement of the heart, a common symptom of asbestosis sufferers. In the latter stages, clubbing of fingers and toes, and anorexia are outward signs of disease progression.

Prior to the books' publication, Lanza knew of only seventy-eight cases in England which documented asbestosis as the cause of death. These occurred from 1930 to 1936. In his book, Lanza suggested a link between asbestos exposure and lung cancer, but drew no firm conclusions due to the lack of statistical evidence. Like his peers, Lanza believed that the chemical composition of asbestos was most likely responsible for its harmful effects. Although it was not accurate, this view was predominant for many years.

Questions about the mechanics of disease causation remain, but modern research has identified the morphology of asbestos fibers as the enabling factor, rather than chemical composition. The indestructibility and physical structure of the fibers wreck havoc on the natural defense systems of body tissue.[207]

Silicosis and Asbestosis remained in print for twenty years. Lanza left Metropolitan Life after World War II, and finished his career as a Professor of Industrial Hygiene at New York University Medical Center. Before he died at the age of eighty in 1964, the university renamed its laboratories at Sterling Forest the Anthony J. Lanza Research Laboratories for Environmental Medicine.[208]

6.3 Saranac Laboratories

Many of the industry sponsored studies were conducted at the Saranac Laboratories in Saranac Lake, NY. Originally named the Trudeau Foundation's Saranac Laboratory for the Study of Tuberculosis, the institute was founded by Edward Livingston Trudeau, a pioneer of the anti-tuberculosis movement of the 1890s and early twentieth century.

6.3.1 Gardner

In 1927, Dr. Leroy Upson Gardner became director of the Saranac Labs. Gardner had been diagnosed with tuberculosis in 1917, and sent to the Trudeau Sanatorium at Saranac Lake to recover. By 1919, he was the pathologist for the sanatorium's laboratory. During this period, Gardner learned of the high tuberculosis mortality rate among granite cutters in Barre, Vermont compared to marble cutters in nearby Proctor, who had fewer cases than expected. As a result of Gardner's investigations, the laboratory's research turned to the relationship between tuberculosis and mineral dusts.[209]

Loss of support from private philanthropies in the 1920s caused the Laboratory to seek other sources of funding. An alliance with industry was formed, and in 1928, Gardner was retained by Johns-Manville to conduct animal studies.[210] During these experiments, Gardner was able to produce asbestosis in the test animals by allowing them to inhale pure asbestos. Industry discounted the results claiming that they would only apply to factory workers exposed to one hundred percent asbestos fiber.[211] In 1930, Gardner began conducting experiments with asbestos dust. By the early 1930s, he was in charge of one of the few groups with experience and information on dust borne diseases.[212]

On November 20, 1936, Gardner received an offer to finance his animal experiments on asbestosis for a three-year period, at a cost of five thousand dollars per year. The offer came from Johns-Manville counsel Vandiver Brown, representing eight or ten asbestos-products manufacturers. Communications and finances were handled by Brown and Sumner Simpson, President of Raybestos-Manhattan.[213] Brown explained the conditions for support in his letter to Gardner:

> It is our further understanding that the results obtained will be considered the property of those who are advancing the required funds, who will determine whether, to what extent and in what manner they shall

> be made public. In the event it is deemed desirable they shall be made public, the manuscript of your study will be submitted to us for approval prior to publication."[214]

Three days later, Gardner agreed to the terms and began his experiments late in 1937. An article by Gardner entitled the "Etiology of Pneumoconiosis" appeared in the November 1938 issue of the Journal of the American Medical Society. It was included in the 1938 Saranac annual report along with other addresses by Saranac staff about their work on asbestosis. In May 1939, Brown wrote to Simpson concerning Gardner's article. "The information covered by these references has presumably been derived from the experiments which Dr. Gardner is conducting for, and with funds provided by, the group members of the Asbestos Textile Industry." Simpson agreed that Gardner was "certainly not living up to his agreement of November 1936."[215]

Even though Gardner had breached the agreement, it must have extended beyond three years, because by 1943, he had received almost thirty thousand dollars. By then, nineteen cases of lung cancer in asbestos workers had appeared in the medical literature. In his studies, Gardner had noted an excessive incidence of pulmonary cancer among a small group of white mice inhaling asbestos dust for a period of from fifteen to twenty-four months.[216] Rather than approach the industry for additional funding, he applied to the National

Advisory Cancer Council to finance a new series of experiments on the cancer-causing potential of asbestos. Gardner was turned down, and so was not able to conduct the new series of tests he had planned.

Many of the test animals in Gardner's studies had developed pulmonary disease from exposure to asbestos dust.[217] Shortly before he died suddenly, in October, 1946, Gardner was said to be very distressed because the 1936 agreement would not allow him to publish his findings.[218]

6.3.2 Kaylo - 1943 to 1952

In 1943, Saranac Labs was retained by another asbestos company to study a new insulation product that used asbestos as a reinforcing agent.[219] After receiving samples of the insulation material, and determining its components, Gardner wrote to the company on March 12, stating "the fact that you are starting with a mixture of quartz and asbestos would clearly suggest that you have all the ingredients for a first class hazard."[220]

The product was called Kaylo, a name derived from the "K-factor" rating used in heat transmission. The lower the heat loss or K-factor, the better the insulation. Kaylo was developed during the 1930s by the Owens-Illinois Glass Company of Toledo, Ohio. The product was made from calcium hydroxide and silica, and had a chrysotile asbestos content of approximately fifteen percent. The study began in 1944.

By May, Gardner had produced asbestosis-like symptoms in test animals whose lungs had been injected with the product. He was directed to proceed with inhalation experiments. Interim reports were sent to the company by Saranac Lab until the study was completed in 1952.[221]

6.3.2.1 Vorwald Gardner was succeeded by Dr. Arthur Vorwald, who was a pathologist at the Saranac Laboratory from 1934 to 1942. A year after Gardner died, Vorwald sent an interim report which "tentatively concluded that Kaylo alone fails to produce significant pulmonary damage when inhaled into the lung." The following year Vorwald reversed that conclusion, informing the company that "...Kaylo on inhalation is capable of producing asbestosis and must be regarded as potentially-hazardous material" that "might pose a grave danger to the company's employees."[222]

Despite these warnings, Owens-Illinois prepared to manufacture Kaylo, and started production in the early 1940s at factories in Berlin, and in Sayerville, New Jersey. Pre-employment and annual X-rays were instituted to monitor the 505 people who worked in these plants.

The final report of the study, entitled "Investigation Concerning the Capacity of Inhaled Kaylo Dust to Injure the Lung," was marked "Confidential," and sent to Owens-Illinois on February 7, 1952, along with Vorwald's comments on its publication. He promised that neither the company or the

product would be mentioned by name, and assured review of the final manuscript by the company prior to its release for publication. The study on Kaylo was finally published in September 1955. By that time, Kaylo, and other new asbestos-containing products, had been installed in schools and other public buildings that had been constructed following World War II.[223]

6.3.2.2 **Schepers** After the study on Kaylo was finally published, the company requested an evaluation by the new director of the Saranac Laboratories, Dr. G. W. H. Schepers. Schepers had been sent to America in 1949 by the South African government to apprentice under Lanza. He also spent three months at the Saranac Laboratories with its director, Dr. Vorwald. During this time, Schepers had the opportunity to meet Vandiver Brown, who asked him to delete statements that embarrassed Johns-Manville from his reports. In 1954, Schepers returned to the United States to succeed Vorwald as director of Saranac Laboratories.[224]

Like his predecessor, Schepers did not mention the company or the product by name in his article. He noted that lesions and fibrosis on the test animals' lungs, as a result of inhaling the product, were similar to experimental asbestosis, and concluded that "this harmful effect was probably caused by the chrysotile asbestos it contained."[225] The company's delight with Schepers's

conclusion was reflected in an intercompany memo dated October 5, 1955: Kaylo was "no more harmful than the universally used heat insulation - asbestos - in fact less so, since Kaylo contains a very small percentage of asbestos." [226]

6.3.2.3 **The Kaylo Files** Files of the Saranac experiments were transferred to new owners when Owens-Illinois sold their Kaylo line to Owens-Corning in 1958. Upon purchase of the plant in Berlin, New Jersey, Owens-Corning became the sole manufacturer of Kaylo. Two years earlier, insulators in Owens-Corning's employee contract units had begun to file worker's compensation claims for disability caused by asbestosis. A decade would pass before Owens-Corning began issuing warnings in December, 1966 that Kaylo was dangerous to inhale.

The Kaylo documents first came to light in 1979 as a result of litigation. That spring, the director of the Saranac Laboratory was subpoenaed in connection with two asbestos cases in Albany, New York. This led to Vorwald's medical papers at the Armed Forces Institute of Pathology, in Washington, D.C., deeded there by his widow following his death in 1974. The remaining documents were produced by Owens-Corning from the summer of 1979 through the winter of 1980. Litigation in that case involved workers at the Berlin, New Jersey plant.[227]

The reference to chrysotile as the specific disease causing agent, and the "very small percentage of asbestos" contained in a product, would be used by industry to plead ignorance about the harmful effects of asbestos in their "state-of-the-art" defense. This defense worked as long as the industry was able to manipulate the published literature, or suppress publication altogether.

6.3.3 Seventh Symposium

The total amount of information suppressed by the industry is unknown, but an indication of the extent is evident in the case of the Saranac Laboratories. Documents detailing the industry's meddling were uncovered during litigation in the late 1970s and early 1980s. During that time, it was discovered that thousands of documents had disappeared. Among these were some thirteen hundred experimental studies of asbestos and other hazardous substances that had been conducted at Saranac over a thirty year period.[228]

In addition to the research it conducted, this pre-eminent institution held a yearly symposium. Vorwald organized the Seventh Saranac Symposium. The week long conference on pulmonary dust disease was held in September, 1952. Merewether, Lynch, Lanza, and Philip Drinker were among more than two hundred attendees, who heard from some of the leaders in the field of asbestos research. Johns-Manville, Owens-Illinois, the U.S. Public Health Service, the

American Cancer Society, the Industrial Hygiene Foundation and major insurance companies were also represented. Unlike the proceedings of six previous meetings, those of the Seventh Symposium were never published, supposedly due to lack of funding. Despite the groundbreaking data which was presented, none of the participants publicized any of the conference's discussions. It would be another twelve years before the scope and definitive nature of the Seventh Saranac Symposium were duplicated. That event was the 1964 New York Academy of Sciences Conference on the Biological Effects of Asbestos, organized by Dr. Irving J. Selikoff. Its proceedings were both published, and well publicized. In the interim, the number of exposures to asbestos continued to increase.

6.4 Dreessen, 1938

The acceptable level of exposure to asbestos dust in industry was a guideline adopted by the American Conference of Governmental Industrial Hygienists (ACGIH) in 1946. The guideline set the level of exposure at five million particles per cubic foot (5 mppcf) of air. The guideline was based on a 1938 study sponsored by the Public Health Service.

In 1937, the Public Health Service began a series of field investigations in conjunction with various state and territorial boards of health. One of the first of these investigations was done at the request of North Carolina's

State Board of Health and its Industrial Commission, which administered the state worker's compensation act.[229]

The study team was headed by Dr. Waldemar Dreessen of the Public Health Service. The team inspected three asbestos textile plants, only one of which existed before 1920. Fourteen occupational groups containing a total of 541 workers were examined after their occupational histories were obtained. Two hundred of them had previously worked with other fibers in cotton and wool textile factories.

Asbestos workers were the youngest group of workers examined. Ninety-four percent were under fifty years old, with an average age of approximately thirty-two. Eighty percent were employed less than ten years, and more than half less than five years. Only twenty three had previously worked in a dusty trade before working in the asbestos textile factories.[230]

The 126 page report of the study was published in 1938, as a Public Health Service bulletin. Its findings were typical for people working in dusty trades. Almost one-quarter of all workers examined showed signs of asbestosis. Evidence of the disease increased rapidly as dust exposure increased. This duplicated the conclusion reached by Merewether and Price in their 1930 study in England; i.e., workers exposed to higher concentrations of dust had a greater chance of becoming ill.

A total of 242 dust counts were made using the Greenburg-Smith impinger, the standard sample collection instrument for obtaining dust measurements.[231] The impinger was a dust trapping device. The particles which settled at the bottom of the impinger's collecting cell could only be viewed at a magnification of one hundred times (100X).[232] The impinger did not differentiate between asbestos and any other airborne fibers. Dust concentration was measured in millions of particles per cubic foot (mppcf) of air.

Dreessen's team noted that concentration and duration were dual factors responsible for causing a higher prevalence of asbestosis. The team combined these factors to describe exposures in "millions-of-particle-years." This combined system diluted the importance of the duration factor. An exposure of five mppcf for ten years was comparable to ten mppcf for five years. Each was equivalent to fifty million particle-years.[233]

Using this measurement system, half of the workers with more than one hundred million particle-years had asbestosis. This figure was extremely low compared to the dust levels needed to cause a similar degree of silicosis. Later, it was discovered that fifteen months prior to the study, 150 workers had been discharged and replaced with others who had little or no previous exposure to asbestos. Sixty-nine of the 150 displaced workers were located and examined. More

than sixty percent of them showed evidence of asbestosis, more than double the rate for those still on the job.

The continuing problem of what to consider as a "safe" threshold level of exposure was addressed in the study report:

> Ideally, a threshold concentration of dust should be the highest dust concentration that would not produce pneumoconiosis in originally healthy workmen during their entire working life. The chief difficulty in this study, as in most of the earlier studies of the Public Health Service, is that very few workmen are exposed for a long period of time to low concentrations of asbestos dust.[234]

Study results showed that three-quarters of the workers in the 50-99 million particle-year category, equivalent to a ten to twenty year exposure at five mppcf, had developed asbestosis. An earlier study in Pennsylvania also indicated that exposures below five mppcf would lead to asbestosis.[235] Even though decidedly inadequate for disease prevention, five mppcf was tentatively proposed by the Dreessen team "as the threshold level for asbestos-dust exposure...for the guidance of factory managers and engineers until more complete data are available." Below that level symptoms were developing, but above that level definite cases of asbestosis had already developed.[236]

The rationale for the proposed level was that new technology was available and capable of reducing dust exposures below five mppcf for the majority of the workers. Reducing levels to five mppcf would have been a major accomplishment because most levels without dust control exceeded fifty mppcf. The team also assumed that the explicitly tentative nature of their recommendation would prompt further and more extensive investigation on a wider scale.[237]

6.5 Drinker, 1946

In 1938, Philip Drinker was asked to establish a health and safety program for the Navy's shipyards. By 1941, Harvard's School of Public Health began intensive, four-month courses to train Naval personnel in occupational medicine and industrial hygiene. By this time, Drinker chaired the Schools' Industrial Hygiene Department. He had also maintained a friendship with a former school mate, Jim Forrestal, who was then Undersecretary of the Navy.

The friendship came in handy when the U.S. Maritime Commission and the Navy requested that Drinker conduct a survey of health conditions at shipyards. For his staff, Drinker selected eight Naval officers who had taken the Harvard course, but could not get the Navy to assign them. His request was denied until he spoke directly to Forrestal, and Chester Nimitz, Commander-in-Chief of the Pacific Fleet.

The study began in 1942 with an inspection of the shipyard at the Bath Irons Works in Maine. Two years later, the team returned because of "concern among the pipe covering crews that the amosite [asbestos] was causing some respiratory troubles." Of the thirty-eight workers X-rayed, twelve displayed changes significant enough to prompt further investigations.[238]

Four more shipyards on the East Coast were selected. All of them had high labor turnovers, and dust levels that greatly exceeded five mppcf. In the dustiest yard, forty-eight workers were examined, representing less than ten percent of the workforce. Three of the forty-eight had ten or more years of exposure. No asbestosis was found. In the least dusty yard, all of the workers were examined. Nine of the 168 workers had ten or more years of exposure. Two cases of asbestosis were found among the nine. Of the total of 1,074 men examined, three cases of asbestosis were found among the group of fifty-one workers who had ten or more years of exposure.

Though well aware of the hazards from his work at Harvard and previously documented studies, Drinker emphasized the more positive aspects of the survey. The results were published in 1946, in The Journal of Industrial Hygiene and Toxicology, a periodical edited by Drinker.[239]

6.6 Summary

Like others before them, Dreessen and his team assumed that additional research would be conducted. Exposures, although high in dosage, were still largely confined to factory settings. Dusty environments were common in the workplace, but people working with asbestos were obviously at greater risk. Unfortunately, in addition to the subterfuge practiced by the industry and their insurers, one national crisis after another served to forestall sustained, impartial, or comprehensive efforts to answer the questions being raised about asbestos-related diseases.

Despite sporadic medical reports, asbestos-related diseases had not yet occurred in any great numbers. The definitive medical evidence that did exist was overshadowed, or ignored altogether. Failure to comprehend the latency period characteristic of asbestos-related diseases served to increase the numbers of people who were eventually exposed.

In the public domain, the reality of the hazard lay dormant. McCulloch notes that "the lapse in time between Merewether and Doll's publications was the period during which the industry expanded most rapidly in Britain and in North America."[240] The industry, and the number of asbestos-containing products it produced, continued to grow without restraint during the twenty year period in which diseases were developing.

CHAPTER 7

THE WAR YEARS

7.1 Between Two Wars

The asbestos industry began a long-term expansion after World War I.[241] From 1925 on, thousands of new uses were developed, and annual sales swelled from millions to billions of dollars over the next half century.[242] Asbestos made the leap from factories and workplaces to the public domain in the mid-1930s, when widespread use began in homes, schools, office buildings, and other public facilities.[243] A new process for spraying asbestos-containing material widened the uses of, and exposures to, asbestos-containing materials.[244]

There were no enforceable regulations regarding worker safety or workplace conditions, but the first official federal dust standard was published by the government in the "Basic Safety and Health Requirements for Establishments Subject to Walsh-Healy Public Contracts Act". The Walsh-Healy Act of June 30, 1936, applied only to government contracts. It required companies contracting with the government to insure that no hazardous health conditions existed in the plants in which their goods were produced. There was no authority to compel compliance. The only way to enforce the Act was to deny or withhold a contract from the offending company, which proved to be impractical at

the time. The country was gearing up for World War II. Large scale naval construction was already underway, and total focus was on the war effort.[245]

By 1942, the first asbestos litigation crisis had disappeared.[246] Compensation laws in many states had already been amended to prevent workers with asbestosis from suing their employers under common law. Asbestos manufacturers and their insurers continued to settle dozens of worker's compensation claims for several thousands dollars apiece.

The upward trend in asbestos use, which began in the 1930s, expanded after World War II, and continued to the 1980s. During this fifty year period, asbestos came to be known as the "miracle" fiber or the "magic" mineral. The many cases of asbestos-related disease that began to appear in the 1960s actually got their start during World War II. It was at that time that hundreds of thousands of people were employed to carry out the massive build-up for the war effort, especially in the nation's shipyards.[247]

7.2 World War II

The Navy and Maritime Commission issued minimum safety and industrial health requirements based on Drinker's work, and on surveys conducted by other health and safety consultants [248] but there were no changes in work practices.[249] Twenty years passed before further studies of asbestos

exposure in shipyards were conducted. "Neither the Navy nor the Maritime Commission was aware of asbestos-related illness in shipyard employees until December of 1944," was the government's response to a lawsuit filed against them almost fifty years later by Johns-Manville.[250]

7.2.1 Restrictions on Asbestos

The United States was the largest consumer of the world's raw asbestos supply, but produced less than eight percent of all grades, and only two percent of strategic grades, domestically. In June 1940, government officials met with asbestos importers and manufacturers to help formulate the National Defense Commission's strategy to "insure production and importation of adequate supplies..." of certain grades and types of asbestos. These were strategic materials considered vital to the success of the war effort.[251]

The Office of Production Management issued the first in a series of restrictive orders on January 20, 1942. Conservation Order M-79 "was issued to effect conservation of the asbestos supply and restrict the usage of African fibers." Amosite was only available from South Africa and Southern Rhodesia. There is no record of its being imported before 1930.[252] Imports of amosite increased from 500 tons in 1935, to 4,500 tons in 1945. Order M-79 was revoked on August 20, 1945. By the end of that month, all restrictive orders on the asbestos industry were lifted.

7.2.2 Repercussions

In 1983, Manville filed suit against the federal government for harming the health of shipyard workers by exposing them to asbestos products during the war. Manville claimed that the government took control over every aspect of asbestos fiber, including expanding mining and manufacturing facilities, purchasing, stockpiling, allocation, processing, exporting, and importing. The suit also cited the change to a less dense variety of asbestos which broke up (fiberized) more easily.[253] This referred to the particular characteristic which makes some types of asbestos more suitable than others for certain uses. The lighter weight of amosite, and its superior heat and acid resistance, had been utilized to meet the requirements for lighter naval vessels.

The aerodynamic quality typical of all asbestos fibers is more pronounced with amosite. Resistance to wetting makes its fibers more readily airborne for longer periods of time, with greater potential for contaminating adjacent spaces.[254] Becklake notes that differences in the physical properties of the various types of asbestos fibers may account for differences in the health effects of exposure.[255]

7.3 Beyond the Shipyards

Those who worked in factories supplying the naval yards were also at risk. The Union Asbestos & Rubber Company of Chicago (UNARCO) established a plant in Paterson, New Jersey

to supply amosite insulation for the pipes, boilers, and turbines on navy and cargo vessels.

Between 1941 and 1954, a total of 1664 workers were employed at the facility. Twenty years later, workers from this plant were being referred to a medical clinic in Paterson, founded in 1953 by Dr. Irving J. Selikoff,[256] a native of Paterson. By 1990, two-thirds of the workers had died, 554 of them from cancer.[257]

7.3.1 The Trail to Tyler, Texas

UNARCO closed the Paterson plant in 1954, and established a similar operation in Tyler, Texas. In 1962, UNARCO sold the Tyler operation to Pittsburgh Corning, and got out of the asbestos business completely. Twenty years later, UNARCO went bankrupt due to the mounting asbestos lawsuits filed against the company.[258]

Pittsburgh Corning was a joint venture between Corning Glass Works and Pittsburgh Plate Glass (PPG Industries). Over a period of six and a half years, Owens-Corning conducted five surveys on health conditions at the Tyler plant. On February 3, 1972, two months before a compliance deadline imposed after an OSHA inspection in October, 1971, the board of directors chose to shut down the Tyler plant.

7.3.2 Repercussions

Yandle vs. PPG Industries was the class action suit filed on behalf of former workers at the Tyler plant. It was the

first case to involve the federal government directly in a major asbestos health claim. The suit was filed on January 2, 1974, in first district court in Tyler, by Frederick Baron. Baron used the strategy developed by Ward Stevenson in Borel vs. Fibreboard. Yandle vs. PPG Industries was settled in secret in September, 1977, when the defendants agreed to pay twenty million dollars (Oil, Chemical and Atomic Workers International Union - $1 million; PPG and Pittsburgh Corning - $8.05 million; Cape Industries - $5.2 million; and the US government - $5.75 milion) to 445 former employees represented in the case. No money was disbursed until the following spring because of the government's reluctance to pay its $5,750,000 share of the settlement.

7.4 A Period of Peak Exposures

Exposures to asbestos in the shipyards reached their peak during the war years. "The use of asbestos insulation in 'Liberty Ships' during World War II has proven to be the most fertile source of asbestos-related cancer."[259] The number of shipyard workers increased from 177,300 in 1940, to 1.89 million in 1944.[260] Selikoff estimated that 4,325,000 workers were employed in the nation's shipyards between 1940 and 1945.[261] Some thirty years later, that period of naval construction would represent half of the asbestos-related health claims in litigation.[262]

CHAPTER 8

EXPANDED USE OF ASBESTOS

8.1 Post-World War II Construction Boom

On Tuesday, August 14, 1945, President Truman announced the unconditional surrender of the Japanese government. World War II was suddenly over, along with the contracts that had been anticipated from a lengthier war. Inflation and union strikes took over in the post-war period. When the dust settled, many Americans had a higher material standard of living. Pent up demand from the war years produced an unprecedented boom in building construction.

From 1940 to 1950, the demand for asbestos increased by two hundred percent.[263] Half of that increase occurred in the five years following the end of World War II. The use of asbestos insulation for fireproofing accounted for a major portion of that increase.

8.2 New Products and Uses for Asbestos

The four major uses of asbestos in insulation products were fireproofing, thermal insulation, acoustical and decorative uses, and condensation control. The products, manufactured for a variety of applications, came in many forms. The proportion of asbestos fibers, other fibers, binders, adhesives, and agents contained in insulation products varies according to end use. Thermal insulation for turbines contains almost

all amosite or crocidolite asbestos, or a combination of both. Materials used for decorative or acoustical purposes may contain little or no asbestos. Material used for fire-proofing may contain from five to thirty percent asbestos fibers.

The focus for marketing asbestos products was switching from insulation to fireproofing. New asbestos-containing products developed during this period included theatre and welding curtains, high temperature corrugated paper, cold-applied adhesives, joint compound, spackles, air tight paints and coatings, and cementitious products such as siding panels, roof tiles, and pipe. Vinyl asbestos floor tile and sheeting was introduced in 1950.[264]

Products containing amosite or chrysotile asbestos, or a combination, were marketed aggressively, and used extensively in the construction industry.[266] Following World War II, all state building codes specified asbestos insulation as the material of choice for fire retardation.[266] A method for applying asbestos insulation in a spray-on form made it the premier fire-proofing material. "By the late 1950s, the major American producers of asbestos-containing construction products were positioned to market their products as school construction took off as a result of the "baby boom."[267]

8.3 Spray-On Asbestos

8.3.1 The Process

Dry materials are prepared for spray application by either the wet or the dry method. In the dry method, the dry material is dumped into a hopper, mixed, and blown into a hose, which conveys it to the mixing nozzle. The operator can control the mix of air, material, and water by adjusting valves located at the mixing nozzle. In the wet method, the dry material is premixed with water in a hopper and is then pumped through a hose. By either method, a slurry results which can be sprayed or troweled onto the surface to be coated. After being applied, the material can be shaped. After it dries, the material is inherently friable.

8.3.2 Development in England

Soon after the British government adopted measures for dust suppression based on Merewether's 1930 study, a method for applying insulation by spraying it onto surfaces was developed in England. Speights and Runyan credit the Turner & Newall Company with developing 'Limpet', the first spray-applied asbestos-containing product, in 1931.[268] Limpit was apparently both a process and a product name. Reitze credits N.L.Dolbey, Director of Research for the J. W. Roberts Company, with developing a sprayed inorganic fiber insula- tion with the Limpet process, which the company introduced in 1932.[269] The daily press carried

illustrations of an experiment to reduce noise in the London subway system by coating the tunnel walls with asbestos. The Lancet warned about the foreseeable consequences of such an experiment in its February 20, 1932, issue:

> In view of the fact that the dust particle producing the lesion in the lung has a diameter of only 2u to 10u, it is obvious that the dust must be suppressed at its source; the use of respirators is futile; ...
> Experience in asbestos factories would indicate that, should the experiment of the London tube railways lead to an extensive use of this method for deadening noise in all the underground railways of London, then a large increase in industrial pulmonary asbestosis may be expected in years to come.[270]

8.3.3 Use in the United States

The spray process was first used in the United States in 1935 to apply decorative finishes to ceilings and walls in nightclubs, restaurants, and hotels. In 1950, the National Gypsum Company had its brand of spray insulation approved as a fireproofing material by Underwriters Laboratories. Underwriters Laboratories also approved an inorganic fibre blend developed by the Asbestospray Company in 1951.[271]

8.3.3.1 High Rise Building Construction Spray-on asbestos insulation was particularly useful in the construction of steel framed buildings. Previously, structural steel was encased in concrete to prevent buckling and subsequent failure resulting from the high temperatures during a fire, but steel roof decks were left exposed. Spray-on asbestos insulation provided adequate fire protection, reduced the weight load on steel building components considerably, and lowered installation costs. Where condensation was a problem, the wicking action of asbestos fibers drew moisture away from inner insulation materials, preventing extensive corrosion of the structural steel.[272]

The first large high-rise project to use spray-on fireproofing was the Chase Manhattan Bank headquarters building, constructed in New York City, in 1958. This sixty story building used 1,500,000 square feet of sprayed fire-proofing product. By the early 1960s, spray-on fireproofing cost just twenty-five cents per square foot. A thirty story building might use two hundred tons. An estimated forty thousand tons was used in 1968 by the construction industry for fireproofing alone, exclusive of thermal, acoustical, or condensation control uses.[273] In 1970, seventy percent of the world's asbestos supply was reportedly used for products used by the construction industry.[274]

By 1970, more than half of all large, multi-story buildings being constructed around the country were being sprayed with fireproofing products. In New York City, six

to eight buildings over thirty stories tall were being sprayed on any given day. Five thousand tons was used for fireproofing in the 110 story twin towers of the World Trade Center. Only non-asbestos-containing spray materials were used above the fortieth floor, due to a change in city regulations.[275]

8.3.3.2 New York Responds In April 1970, New York City became the first governmental agency to address the occupational hazards associated with sprayed asbestos insulation. The city issued its Sprayed Insulation Regulations, seeking to control or minimize the hazards by engineering methods and industry procedures, as an alternative to an outright ban on the practice. The city's Department of Air Resources was in charge of the program. Less than two years later, citing an inability to ensure compliance or control of environmental hazards, the New York City Council banned all spraying of asbestos as of February 15, 1972.

In 1991, the New York State Supreme Court ruled to allow tax abatements for liabilities incurred due to ACM in buildings. Chase Manhattan received a $24 million refund for fiscal years 1984-1990, for its headquarters building at One New York Plaza.[276] In 1994, adding a new wrinkle to the health-related damage suits that were piling up, Chase sued Turner & Newell, claiming losses of $185 million in property damage caused by the use of their product in the construction of the bank building.[277]

CHAPTER 9

THE ROAD TO REGULATION

9.1 Introduction

Selikoff's work at the Paterson, New Jersey clinic, which he opened in 1953, sparked his interest in asbestos-related diseases. At the time, he was an epidemiologist at the Mount Sinai Hospital in New York, specializing in the detection by X-ray of lung dysfunction. Among his first patients were seventeen men from the UNARCO plant who had been referred by a local attorney. All of these men were still working, and appeared to be in good health, but fifteen showed signs of pulmonary defects from the inhalation of asbestos fibers. By 1961, four of them had died.[278]

By 1961, nearly half of all insulation workers were dying from occupational cancer and asbestosis, even though their average exposure was comparable to the exposure level recommended by the ACGIH in 1946.[279]

9.2 Selikoff, 1964

Selikoff sought support for a study of the Paterson plant from UNARCO, and from the US Public Health Service, but was denied. In 1962, he established a working relationship with two locals of the International Association of Heat and Frost Insulators and Asbestos Workers (IAHFIAW).

9.2.1 The IAHFIAW

The International Association of Heat and Frost Insulators and Asbestos Workers (IAHFIAW) represents insulation workers in the construction trade, and also employed in refineries, industrial plants, shipyards, and powerhouse construction and repair. The IAHFIAW was charted by the American Federation of Labor in 1910 to represent independent locals throughout the country, and the Salamanders Association of New York.

The Salamanders was the first union of insulation workers in the United States. It started in New York City in 1884 as the Salamander Association of Boiler and Pipe Felters. Local 12 in New York City is directly descended from the Salamanders, and Local 32 is an affiliate located in Newark, New Jersey.[280] Detailed records kept by the union dated from 1914.

9.2.2 The 1964 Studies

With the cooperation of the locals, and his associates at the Mount Sinai Hospital, Selikoff, along with Dr. Jacob Churg, Chief Pathologist at Barnert Memorial Hospital, in Paterson, New Jersey, and Dr. E. Cuyler Hammond, Vice-President for Epidemiology and Statistics of the American Cancer Society, began the first comprehensive studies of the effects of asbestos exposure among insulation workers, using data independent of the asbestos industry.[281]

Preliminary research confirmed that the asbestos content of the products used by the insulation workers had remained fairly consistent over the years. Magnesia block, the most commonly used product, contained approximately fifteen per cent asbestos. Cement products contained from fifteen to twenty percent asbestos. The appearance and increased use of amosite in insulation for shipbuilding, beginning in 1934, was also noted.

Several stages of disease manifestation were investigated using the extensive database compiled in cooperation with the IAHFIAW and its members. Insulation workers not included in the study were those working in other trades or other unions, spray-insulation workers, nonunion workers, and some maintenance workers.

In 1963, the IAHFIAW rolls contained 14,803 members. Of this number, 1,258 were counted in the New York-New Jersey locals. In addition, 264 men had died from January 1, 1943 to December 31, 1962. The cohort consisted of the latter two groups, a total of 1522 cases. The studies began on January 1, 1963.

9.2.2.1 The Twenty Year Latency Period Previous reports mentioned the appearance of neoplasia associated with asbestosis that seemed only to occur after twenty years from onset of exposure. This question was addressed by a separate study on the mortality of asbestos workers with twenty or more

years of exposure. The cohort was a sub-group of 632 workers who were members of either of the locals as of Dec 31, 1942. 339 of these men had been exposed to asbestos dust prior to 1924, and had twenty years of exposure as of 1943. The others were counted in the study as they reached the twenty year exposure mark, which was before 1962. Those who died before the twenty year exposure mark were not counted. A total of 8,737.5 man-years of exposure histories of twenty years or longer were compiled.[282]

Using five year time periods, the death rate among the asbestos workers was compared to the average age-specific death rate of the US white male population. In the first period, the rate among asbestos workers was substantially lower, but was slightly higher in the second period. In the third period, from 1953 to 1957, 85 deaths occurred compared to 56.6 expected. From 1958 to 1962, 88 deaths occurred compared to 54.4 expected.[283] Excluding seven men who died before the twenty-year-exposure mark, 255 of the 632 asbestos workers died before Jan. 1, 1963. This was twenty-six percent in excess of the 201.5 deaths that would have been expected.[284]

Death from cancer of the lung or pleura was far higher than expected in each of the five year periods. Forty-five of the 632 workers died from cancer of these sites, when only 6.6 such deaths were expected. The three deaths listed as neoplasms of the pleura were all mesotheliomas. This one

percent incidence was considered extremely high for such a rare tumor. The other 42 deaths were from bronchogenic carcinomas. Cancers of the stomach, colon, and rectum were three times more frequent than expected. Cancer from all other sites was consistent with the expected rates.[285]

The question of environmental exposure was briefly discussed, with particular concern for other tradespeople on job sites where asbestos was used. Because the "floating fibers do not respect job classifications," these workers were subject to passive, or second hand, rather than direct exposure. The mortality study on asbestos exposure and neoplasia was published in the Journal of the American Medical Association in April, 1964.

9.2.2.2 Asbestosis The study of asbestosis among insulation workers included all 1522 members on the roster of the New York-New Jersey locals from January 1, 1943 through December 31, 1962. 264 members had died. Of 1258 living members, 1086 were still working in the trade, 63 had retired, and 34 were not working due to illness. The remaining 75 had left the union and were no longer employed as insulation workers. Union records, which were compiled on a weekly basis, detailed the work history of each member. These were used to fill in information on workers who were not examined, and supplement the occupational histories obtained from those who were. Death certificates were used to obtain and analyze hospital and autopsy records for the 264 members who had died.[286]

From the examinations and records, such details as: onset and lapses of employment, protection measures used, materials handled, occupational settings, and smoking habits were established. Reasons for inactivity or leaving the trade were ascertained. All physical examinations included X-rays, measures of vital lung capacity, and fluid samples. Many of the examinations were even more detailed.

Radiological change was the sole criteria used to establish evidence of pulmonary asbestosis in those who were examined. Examination was voluntary and 1117 members participated. This included 984 of the 1806 active working members, 50 retired members, 28 who were ill, and 55 who had left for other employment.[287] Evidence of pulmonary asbestosis was present in half of this group. The extent of fibrosis varied directly with the duration of exposure.[288]

Time from onset of exposure to time of examination of the 1117 workers was divided into ten year periods. Only 36 (10 percent) of the 346 workers with less than ten years showed some sign of radiological abnormality. For workers with exposures from ten to nineteen years, 167 of 379 (44 percent) showed some abnormality, nine of which were greater than minimal. Pleural fibrosis was present in 35, and 5 showed evidence of calcification.[289]

The majority of the 392 workers with more than twenty years showed evidence of pulmonary asbestosis. This group included the majority of older members who had retired, or were ill. Of this group, 56 of the 77 (72 percent) with

twenty to twenty-nine years had abnormal films. This increased to almost ninety percent for 194 workers with thirty to thirty-nine years, and to ninety-five percent for those with over forty years. The extent and severity of asbestosis increased with the time from onset of exposure.[290]

9.2.3 Study Results

Dyspnea, or shortness of breath, is the physical complaint most often associated with asbestosis. The study found that dyspnea on exertion rarely correlated with X-ray evidence of fibrosis.[291] Only twenty-five percent of ninety-nine cases with twenty or more years of exposure, in which moderate or severe dyspnea was present, had X-rays showing moderate or extensive parenchymal fibrosis. "Thus significant disability may be present with relatively little to be seen on X-ray and, conversely, X-ray changes may be extensive with little functional difficulty."[292]

Particular attention was paid to the known increased risk of lung cancer among asbestos workers. Seven cases (0.7%) were found among 984 of the 1086 members who were still active in the trade. Among the 172 who were no longer active, 133 were examined and four cases (3%) of lung cancer were found. These cancers occurred in the 392 members with more than twenty years of exposure. None was found in those with less than twenty years exposure prior to examination. In 307 consecutive deaths examined in the cohort, lung cancer

was found to be seven times greater than expected, and cancer of the gastrointestinal tract was three times as common. Ten cases of mesothelioma of the pleura or peritoneum were found.

The difficulty of comparison with earlier studies was cited, noting the conditions under which some of these took place, the skewed populations involved, and the lack of prolonged exposures. Of particular interest is mention of a report by Fleischer et al. in 1945. It is cited as "the only large scale survey of asbestos insulation workers undertaken ...in the US." The men were examined while at work, and only 51 of the 1074 examined had more than ten years of work experience. Only three cases of asbestosis were found, and the study concluded that 'asbestos pipe covering of naval vessels is a relatively safe operation.'

The study by Selikoff et al. concluded that pulmonary asbestosis, and the complications of lung cancer and mesothelioma associated with it, were significant hazards among insulation workers exposed to asbestos, particularly for those with more than twenty years from onset of exposure. Results of the study were presented at the New York Academy of Science's International Conference on the Biological Effects of Asbestos, held at the Waldorf-Astoria Hotel in October 1964 in New York City.[293]

9.3 The State of Awareness

9.3.1 The Medical Literature

From 1930 to 1960, industry executives had conspired to suppress damaging information about the health effects of asbestos exposure, by either editing the results to appear more favorable, or not allowing publication, of at least nine statistical health studies performed on humans or animals. Independent studies in the 1930s and 1940s linked asbestos exposure to pulmonary disease, lung cancer, and mesothelioma tumors.[294] These were mainly occupational diseases, but the possibility of environmental or community exposures had been suggested. By the 1950s and 1960s, public health officials and scientists were paying more attention to the ill health effects attributed to asbestos exposure.

By 1960, sixty-three papers on the problems of asbestos exposure had been published in the United States, England, and Canada. The papers sponsored by industry downplayed asbestosis and dismissed any connection between lung cancer and asbestos exposure. The other fifty-two papers, published independently of the industry, concluded the opposite - that asbestos exposure was a dangerous source of asbestosis and lung cancer.[295] After 1960, the industry sponsored the publication of three times as many studies as it had in the preceding thirty years, and spent more than thirty times as much on research as any government agency.[296] By the end of 1964, more than seven hundred medical articles worldwide detailed the hazards of asbestos exposure.[297]

9.3.2 New York Academy of Science Conference, 1964

"The Occurrence of Asbestosis Among Insulation Workers in the United States" appeared in the Annals of the New York Academy of Science on December 31, 1965, which published the proceedings of the 1964 conference. The study was used by attorney Ward Stevenson, whose precedent setting strategy of suing asbestos manufacturers under product liability law successfully breached the industry's "state-of-the-art defense".[298] The decision rendered by the court in the case of Borel vs. Fibreboard Paper Products "triggered the greatest avalanche of toxic-tort litigation in the history of American jurisprudence."[299]

The extensive scope, objectivity, and thoroughness of the database and methodology of the 1964 study presented conclusive evidence of definite hazards resulting from asbestos exposure.[300] At the time, less than half of the state governments had enacted regulations pertaining to asbestos exposure. Most of these only addressed asbestosis, and all of them were based on less than reliable data.[301] Unions led the drive to channel funding to Mount Sinai for further research and education efforts. By the end of 1964, unions were pressing for federal government intervention.

The 1964 New York Academy of Science conference, and the publication of its proceeding had opened the passage to public debate on the use of asbestos, asbestos exposure and its ramifications. The reality of decades of asbestos exposure was well known, but the twenty year latency period,

and long term hazards of exposure had not been so apparent. The 1964 studies confirmed the possibility that millions of workers exposed during the war years were currently at risk. It was also apparent that the five mppcf exposure limit value adopted by the ACGIH did not provide an adequate level of protection. The TLV for asbestos and how it was set were major topics of discussion at the 1964 conference.

9.4 The ACGIH

9.4.1 Formation

TLVs were the brainchild of the American Conference of Governmental Industrial Hygienists. Despite its name, the ACGIH is a voluntary organization with no formal ties to the federal government. It was an outgrowth of training seminars funded by the 1936 Social Security Act. For the first time, states received money for public health activities, including education and training. The focus was on industrial hygiene, and the Public Health Service worked with the Conference of State and Provincial Health Authorities of North America to establish active industrial hygiene units in state and local health departments.[302]

The American Industrial Hygiene Association was the dominant group representing industrial hygienists. Most of its membership worked in private industries which held large contracts from the federal government. The AIHA had industry support and was viewed as "industry-friendly," whereas, the ACGIH saw itself as "worker-friendly". The

groundwork for its formation was laid after the second series of training seminars in the summer of 1937. By 1938, the Public Health Service had organized industrial hygiene units in twenty-eight states. The ACGIH held its first convention apart from the AIHA in June, 1938, and continued to do so until 1943.[303]

The reason for forming the ACGIH was that a non-governmental organization would have more flexibility in responding to issues when the Public Health Service could not. The model for the ACGIH was the Conference of State Sanitary Engineers. This private group met annually with the Public Health Service's Division of Sanitary Engineering to discuss policy and training matters.[304] The purpose of the ACGIH was to:

> promote industrial hygiene in all its aspects and phases; to coordinate industrial hygiene activities...by official federal, State, local and territorial industrial hygiene agencies; to encourage the interchange of experience among industrial hygiene personnel in such official organizations; to collect and make accessible to all governmental industrial hygienists such information and data as may be of assistance to them in the proper fulfillment of their duties.[305]

9.4.2 The 1946 Guideline

The ACGIH adopted a strategy to maximize the impact of the new group's national standing and impact. To that end, standing committees were formed to recommend guidelines for levels of hazardous agents in the workplace.[306] These guidelines were called Maximum Allowable Concentrations (MACs).

The purpose of the MAC list was to define acceptable limits for exposures to ambient concentrations of substances used in the workplace, as a guide for industrial hygienists. A MAC value was based on an average concentration over an eight hour day, called a time-weighted average (TWA). The MAC value was not a maximum concentration. During an eight hour period, the value could be exceeded, as long as lower values occurred to balance the exposure to an average concentration that did not exceed the MAC.[307] The MAC list included some materials that were known or suspected human carcinogens. This information was not specified, and did not appear to be the basis for including such materials on the list.[308]

The ACGIH established a formal Committee on Threshold Limit Values (TLVs) in 1941. Its members included William G. Fredrick, Bureau of Industrial Hygiene for Detroit, as chair; Leonard Greenburg, co-inventor of the Greenburg-Smith impinger; and Philip Drinker of Harvard.[309] The TLV Committee began publishing an annual report in 1946, the same year in which asbestos was approved as an addition to their

MAC list. The guideline adopted for asbestos was based on the tentative recommendation of the 1938 Public Health Service study conducted by Dreessen, et al. It was not based on engineering solutions or modified work practices, but on a numerical limit. The limit applied only to asbestosis, based on a total average dust count of five mppcf, over an eight hour period. The 1938 study had already documented that workers exposed at the five mppcf level were developing asbestosis. The limitations of the study were thus incorporated, when the ACGIH adopted five mppcf as the allowable exposure limit for asbestos in 1946.

9.4.2.1 The 1947 Hemeon Report New Jersey adopted the ACGIH guideline as a workplace standard in 1946, and also considered restricting its industrial hygiene code to apply only to long fibers. Gardner, in his industry funded research at Saranac Labs, had already produced asbestosis in guinea pigs breathing ball milled asbestos. The total concentration of asbestos dust was 138 mppcf, but only 0.8 mppcf of the fibers were longer than ten microns. Shorter asbestos fibers had produced lung damage in rabbits in a study by King in England, which also found interstitial fibrosis produced by asbestos fibers as short as 2.5 microns. Despite the insistence of Johns-Manville's counsel Vandiver Brown, Gardner refused to endorse the New Jersey proposal by testifying that short fibers were harmless.[310]

The strongest criticism of the five mppcf guideline came in a 1947 report by the Industrial Hygiene Foundation of America (IHF). The IHF evolved from the Air Hygiene Foundation (AHF), a research laboratory begun in 1935 by the Mellon Institute. The AHF had been formed, at the request of industry, in response to the silicosis problem prompted by the Gauley Bridge Tunnel disaster.[311]

The year it was formed, Brown wrote to the industry trade magazine Asbestos, praising the new foundation for "approaching various problems relating to air hygiene from an unbiased viewpoint". He also noted that it was "nevertheless the creature of industry and is the one institution upon which employers can rely completely for a sympathetic appreciation of their viewpoint." Brown urged the co-operation and "unqualified support of all members of industries faced with a dust disease hazard."[312] Asbestos companies supporting the AHF formed a group named the Asbestos Textile Institute (ATI). Their purpose was to exchange information on, and promote the use of successful methods of dust control, among themselves. In 1941, the Air Hygiene Foundation became the Industrial Hygiene Foundation.

In 1947, the ATI requested an evaluation by the IHF to define the nature and magnitude of the asbestosis problem. A preliminary investigation was conducted that spring by Dr. W. C. L. Hemeon, and Dr. Richard Walmer, medical director and chief engineer, respectively, for the foundation. They took dust surveys, and evaluated medical studies in ten asbestos

textile plants owned by ATI member companies. Only five of the ten plants had programs for taking chest X-rays. In two of those plants, the Raybestos-Manhattan factory in Manheim, Pennsylvania, and the Johns-Manville plant in Manville, New Jersey, about twenty percent of the workers had developed asbestosis. The average total dust level for the Raybestos-Manhattan plant was only two mppcf.[313]

Hemeon concluded that "the information available does not permit complete assurance that five million [ppcf] is thoroughly safe nor has information been developed permitting a better estimate of safe dustiness." He urged the ATI to find a method for "accurately measuring any remaining hazards in the dust zone below five million [ppcf] for the elimination of future asbestosis depends on the degree of control effected now." He also recommended that further studies be done at ATI companies in North Carolina, where annual X-rays of textile workers were required by state law, especially "in one or two plants with a long history of high order of dust control...".[314]

The 1947 report was never published, and none of the further studies that Hemeon suggested were done.[315] The 1947 report was uncovered by plaintiff attorneys thirty years later, and subsequently publicized during the trial of Bob Alan Speake vs. Johns-Manville Corp. et al.[316]

As a tribute to the ACGIH's initial strategy to gain national impact and influence for its work, the five mppcf guideline stood firm as the only available industry standard

for asbestos, despite continued criticism of its inadequacies. Hemeon felt that the five mppcf limit was too high. Brown questioned whether it should exist at all. At the Sixth Saranac Symposium on Dust Diseases, organized by Gardner's successor Vorwald, Brown presented a speech on "Management's Viewpoint" in which he addressed the ACGIH guideline that New Jersey had recently adopted. Brown used Hemeon's report to support his own argument that "no one can state with certainty what is the maximum allowable limit for asbestos dust." He stated with assurance that "no study has been made specifically directed toward ascertaining this figure and I question there exists sufficient data correlating the disease to the degree of exposure to warrant any determination that will even approximate accuracy."[317]

9.4.2.2 Modifications Proposed Vorwald, a member of the TLV Committee in 1952, tried to incorporate some aspects of Gardner's earlier Saranac study into the standard. Vorwald proposed a companion limit of one mppcf for fibers longer than ten microns in length. The proposal was not adopted by the ACGIH, but it was considered by Johns-Manville.

In 1954, Johns-Manville's safety executive directed a change in the company's internal reporting of MAC values. The five mppcf standard would still be used for total airborne dust, but fibers longer than ten microns would be reported in fibers, rather than particles, per cubic foot. The change was an attempt to measure long asbestos fibers

more accurately because they were thought to be the most hazardous airborne contaminants. Johns-Manville decided on the change despite the difficulty of reconciling and then applying to humans, the contradictory conclusions from two studies that involved different animals (guinea pigs and rabbits).

Another attempt to modify the ACGIH standard was made by toxicologist Herbert Stokinger of the Public Health Service. In 1955, after Doll's study confirming the link between cancer and asbestos exposure, Stokinger spoke at a "Symposium of Threshold Limits" held jointly by the ACGIH and the American Industrial Hygiene Association. Even though he was skeptical about the carcinogenicity of asbestos, Stokinger proposed a safety factor of from one hundred to five hundred for industrial carcinogens. Stokinger was a member of the TLV Committee for the next twenty years, but his proposal was never implemented.[318] The addition of asbestos to the MAC list in 1946 had been followed by arsenic in 1947 and chromates in 1950. Nickel carbonyl was added in 1955, at the time of Stokinger's presentation at the symposium.[319] The substances were finally identified as carcinogens in the ACGIH's "Documentation of the TLV Values" for 1966, but no limits were recommended for them.[320]

Dr. Paul Gross became a member of the TLV Committee in 1965, primarily to address the setting of limit values for mineral dusts including asbestos. Gross was a pathologist with the Mellon Institute. He started there in 1954 and over

a six year period, had examined various lung samples sent from Johns-Manville. Cancer was present with asbestosis in most of these lung samples, leading Gross to the belief that asbestos was carcinogenic. He was the principal handler of the asbestos standard for the TLV Committee until 1983.[321]

In 1968, the ACGIH issued a "Notice of Intent" inviting comment from "industry-connected individuals principally, but others also," on proposed changes to the 1967 TLV list.[322] The proposal for asbestos included special consideration for crocidolite:

> A limit of 5 MPPCF, based on impinger samples counted by light-field technics (sic), is satisfactory to control exposures to most forms of asbestos. Crocidolite, however, has been shown to produce, in addition to the asbestotic inflammation, also mesothelioma. Since no safe limit can be established for this form of asbestos, it is recommended that workers exposed to crocidolite be equipped with air-supplied helmets.[323]

Official recognition of crocidolite as a cancer causing agent, even at low levels of exposure, would create a problem for the industry. Most of the crocidolite imported to the United States was used to manufacture large-diameter, asbestos-reinforced cement pipe for drinking water supply

systems. A blend of chrysotile and fifteen percent by weight of crocidolite produced the best product. Selikoff and other public health officials raised doubts about the wisdom of this practice, and questioned whether crocidolite was actually more hazardous than chrysotile or amosite asbestos. Stokinger recommended banning further use of asbestos-cement pipe for water supply systems.[324]

9.4.3 Setting the Guidelines

The TLV Committee faced inherent difficulties. The ACGIH was formed "expressly to keep out corporate influence," but was virtually dependent on industry to supply data the TLV Committee needed.[325] Speaking as chair of the committee in 1969, Stokinger summarized his view of this dilemma:

> The TLVs are industry's values...industry has the sole responsibility to develop data on its own products; government is not in a position to develop the facilities to handle the problem in total, nor should it, when reliable toxicological consultants are now available."[326]

Stokinger went on to acknowledge the inability to obtain "appropriate industrial hygiene data as the greatest problem facing the TLV Committee" in setting standards.[327] The industry was not forthcoming, and the government was not

able to exert influence, forcing the TLV Committee to rely on other sources.

The ACGIH opted not to open membership to those in private industry when it split with the American Industrial Hygiene Association in 1938. As a result, the TLV Committee was an insular body of toxicologists and industrial hygienists, most from government health agencies. "Outsiders" such as Vorwald and Gross had indirect connections with industry, but their professions were similar to those represented on the TLV Committee. The persistent lack of physicians on the committee increased the ire of the more established industrial doctors, especially as the ACGIH guidelines became more widely recognized.[328]

9.4.3.1 Emphasis on Prevention The TLV Committee concentrated on prevention by seeking some minimum level of exposure which would not lead to sickness. The data on which to base a lower limit were not available to them, so other criteria were used. These criteria are amply described by McCulloch's comment about the coal dust standard for New South Wales: "The standard was determined through a trade-off between technical efficiency, acceptable cost to the producer and the existing dust levels found in the industry." McCulloch's comment referred to discussions of preventive measures and setting standards, which took place at the Third International Conference of Experts on Pneumoconiosis, in Sydney, Australia in 1950. The conference unanimously

adopted a proposal favoring prevention because "...no reliance should be placed on an appliance which depended on the individuals using it correctly, unless it was quite impossible in the circumstances to use any other." Prevention, a higher level of protection, took precedence over safety equipment in setting standards.[329] Although the ACGIH achieved its goal of gaining national impact, its standards were not enforceable unless adopted by state law. Even when enforceable, the five mppcf standard for asbestos was not an adequate level of protection to prevent asbestosis, the only asbestos-produced disease it addressed. The standards could be no more stringent than what the technology at the time was capable of measuring.

9.4.3.2 Changes in the TLV The ACGIH had approved the addition of asbestos to the first MAC list published in 1946. In 1963, the ACGIH changed the name for its list, from maximum allowable concentrations (MACs) to threshold limit values (TLVs).[330] The need for a more stringent standard was somewhat acknowledged by the "Notice of Intended Changes" for 1968. Although the proposal for special handling of crocidolite was dropped, the TLV for asbestos was lowered to twelve fibers per cubic centimeter of air (12 f/cc), or two mppcf for all particles. One particle per cubic centimeter (ppcc) equals 35.3 times one mppcf. A conversion factor, of one mppcf equal to six fibers per cubic centimeter, was used to facilitate a more accurate system of measurement which, like the old system, could only count microscopic dusts.[331]

In 1970, the ACGIH established a new subcommittee on carcinogenic substances. Over the next six years, representatives from major corporations in private industry became so prevalent that they made up half of the committee members. In some cases, primary responsibility for reviewing new products was done by representatives from the companies which made those same products. In 1974, the TLV for asbestos was listed at 5 f/cc, two years after OSHA had adopted a standard at that level through the formal rulemaking process.[332]

In 1980, the ACGIH revisited its earlier proposal for special consideration for crocidolite, and lowered the TLV for crocidolite to 0.2 f/cc. The TLV for amosite was lowered to 0.5 f/cc. The TLV for chrysotile and other forms of asbestos was lowered to 2 f/cc. By 1980, the 2 f/cc level of exposure had been in effect for four years in the United States, and for eleven years in England.[333]

CHAPTER 10

FEDERAL REGULATION OF ASBESTOS

10.1 Introduction

In 1969, a federal standard of twelve fibers per cubic centimeter (12 f/cc) of air was adopted under provisions of the Walsh-Healy Public Contracts Act, which applied to government contract jobs costing $10,000 or more.[334] After several years of consideration, a watershed of federal legislation concerning environmental issues was pending in the Congress.

10.2 The OSHAct of 1970

"Through the exercise of its power to regulate commerce among the several States and with foreign nations," the 91st Congress passed the Occupational Safety and Health Act of 1970. The OSHAct of 1970 was signed into law by President Nixon, on December 29, 1970. It became effective on April 28, 1971.

10.2.1 Provisions of the OSHAct

The purpose of the Act was to "assure safe and healthful working conditions for working men and women by authorizing enforcement of the standards developed under the Act; by assisting and encouraging the States in their efforts to assure safe and healthful working conditions; by providing

for research, information, education, and training in the field of occupational safety and health; and for other purposes."[335]

10.2.1.1 Administration The Department of Health, Education and Welfare (HEW), and the Department of Labor (DOL) were the agencies designated to carry out the provisions of the OSHAct. The term "Secretary" in the OSHAct, refers to the Secretary of Labor. The Occupational Safety and Health Administration (OSHA) was the enforcement agency created in the Department of Labor. OSHA was the first centralized, federal depository for information on workplace conditions in the United States.

The Department of Health, Education and Welfare was established in 1953 to combine the government's various health related activities under the administration of one federal agency.

10.2.1.2 NIOSH Section twenty-two of the OSHAct created the National Institute for Occupational Safety and Health (NIOSH) in the Department of Health, Education and Welfare (HEW), which later became the Department of Health and Human Services (HHS). NIOSH is administered by the Centers for Disease Control. Its functions include research, training, employee education, and related activities. NIOSH is responsible for developing and recommending standards.

10.2.1.3 Setting Standards The primary responsibility for setting occupational safety or health standards is assigned to the Secretary of Labor. Section six of the OSHAct established the procedure for setting a "national consensus standard." In the event of "conflict among any such standards, the Secretary shall promulgate the standard which assures the greatest protection of the safety or health of the affected employees."[336] Standards for toxic or harmful physical agents were to assure, "to the extent feasible, on the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life."[337] Promulgation of an OSHA standard would supersede those safety or health standards which predated the OSHAct.

10.2.2 The Rulemaking Process

The first test of OSHA's rulemaking procedure for setting standards in the public forum was set in motion within one year of the act's passage.

10.2.2.1 The First Consensus Standard The first consensus standard adopted under the OSHAct limited asbestos exposure to 12 f/cc of air, averaged over an eight hour day. It was adopted by OSHA on May 29, 1971. In October, 1971, NIOSH investigators surveyed the Owens-Corning plant in Tyler,

Texas, at the request of its owners. More than seventy-five percent of the fifty-four air samples taken, failed to meet the existing standard of 12 f/cc, but this was not mentioned in the inspection report. Instead, an "extremely serious occupational-health situation" was said to exist. Extensive improvements to the ventilation and dust control systems were to be completed by March 31, 1972, and a $210 fine was imposed for other "non-serious" violations.[338] The situation at the Tyler plant prompted action from the AFL-CIO. On November 4, 1971, the Industrial Union Division of the AFL-CIO petitioned Secretary of Labor James D. Hodgson to declare an emergency temporary standard (ETS) for asbestos exposure of 2 f/cc.

10.2.2.2 The First Comprehensive Standard On December 7, 1971, Secretary Hodgson issued an ETS of 5 f/cc, and a peak exposure of 10 f/cc as a "scientific compromise".[339] The Secretary called for hearings on a permanent standard to begin in March, 1972, and assigned a five person advisory committee representing government, labor, industry and science. OSHA retained the Arthur D. Little consulting firm to conduct a survey of the health effects, at various levels of exposure (0.2, 2, 12, and 30 f/cc), over a working lifetime. Labor unions, research scientists, and the advisory committee recommended a standard of 2 f/cc. Industry supported the ETS of 5 f/cc that was in effect.

The consensus standard of 12 f/cc was no longer considered feasible by any group. NIOSH prepared a Criteria Document recommending a standard of 2 f/cc, to be effective in two years, along with requirements for recordkeeping, monitoring, medical surveillance of workers, and labeling. The report was submitted to the Secretary on February 1, 1972.[340]

OSHA promulgated its first comprehensive health standard for asbestos on June 7, 1972. The emergency standard was retained until July 1, 1976, when a reduction to 2 f/cc became effective. A decision was made on May 24, 1983 to apply any new standard adopted for general industry to construction, as well.

10.2.2.3 The 1984 Hearings An ETS of 0.5 f/cc, published on November 4, 1983, was invalidated in March, 1994, by the US Circuit Court of Appeals. On April 10, 1984, OSHA began the formal rulemaking process again, by announcing two possible exposure levels for asbestos (0.5 and 0.2 f/cc), and permitting the use of respirators.[341]

At OSHA's public hearing on June 21, 1984, a NIOSH representative testified that there was "no safe airborne fiber concentration for any of the asbestos minerals. NIOSH stated that not even the lowest fiber exposure limit could assure all workers of absolute protection from exposure-related cancer," and reaffirmed the agency's earlier conclu-

sion, that "there is no scientific basis for differentiating health risks between types of asbestos fibers for regulatory purposes."[342]

10.2.2.4 Risk Analysis OSHA considers as significant those occupational exposures which cause more than one death per one thousand, over a working lifetime. Through its risk assessment analysis, OSHA determined that a reduction of the PEL from 2 f/cc to 0.2 f/cc would reduce the lifetime risk of death from asbestos-related cancer from 64 to 7 deaths per 1000 workers. Excess cancer risk for a twenty year period of exposure was estimated at 4.5 per 1000 workers. For asbestosis, the incidence over a working lifetime was estimated at 5 per 1000, with an incidence for twenty years of exposure of 2 per 1,000 workers.[343]

10.2.3 A Ten-fold Reduction Over Ten Years

On June 17, 1986, OSHA issued two revised standards, one for occupational exposures in general industry, and the other for construction. The construction standard included specific requirements for asbestos abatement and demolition work. The time-weighted average permissible exposure limit (TWA-PEL) for asbestos was the same for both standards. It was reduced from 2 f/cc to 0.2 f/cc, and an action level of 0.1 f/cc was established.

Both standards included occupational exposure to the non-asbestos varieties of tremolite, anthrophyllite and actinolite asbestos. This provision was subsequently deleted in June, 1992. The 1986 standards became effective on July 21, 1986. In the ten year period from 1976 to 1986, OSHA had reduced the PEL for asbestos tenfold, from 2 f/cc to 0.2 f/cc.[344]

10.2.4 OSHA Regulations

Congress created OSHA under its power to regulate commerce, and the OSHAct of 1970 focused on occupational settings, mostly in private industry. The regulations are codified in the Code of Federal Regulations (CFR) for the construction, and maritime industries, as well as for general industry. In general, OSHA regulations cover employees and employers in occupational settings, through federal jurisdiction, or through OSHA-approved state programs.[345] OSHA has never had the resources to inspect all of the nation's workplaces, which means that compliance is largely voluntary. Despite OSHA's extensive recordkeeping and reporting requirements, lack of data on the causes of injury are still one of the agency's greatest problems.

10.3 Mounting Litigation

The landslide of claims in the 1970s and the 1980s from asbestos-related exposures helps to explain why the federal government was forced to intervene. As a matter of

commerce, it was becoming difficult to obtain financing and insurance for buildings which contained asbestos.[346] The numbers of product liability claims, and the amounts being awarded, were piling up.

10.3.1 Increasing Damages

The average settlement cost had tripled in the ten year period from 1970 to 1979, then doubled in the next four years. In mid-1982, the courts began awarding punitive damages as well. By the end of 1982, manufacturers and their insurance companies had paid approximately six hundred million dollars in compensation, to settle some four thousand product liability cases.[347] A study completed in March, 1984, surveyed compensation paid for asbestos-related disease claims in trials that began between January 1, 1980 and August 26, 1982. The average total compensation, for the fifty-three percent of the plaintiffs who won their cases, was $388,000.[348]

10.3.2 Johns-Manville

By 1982, claims against Johns-Manville were coming in at a rate of six thousand per year. Seventeen thousand cases were still pending. Compensation payments with no end in sight prompted the company to change its name, and file a petition in Federal Bankruptcy Court.

10.3.2.1 Bankruptcy On August 26, 1982, the Manville Corporation filed for reorganization under Chapter 11, seeking protection from the continuing onslaught of personal injury lawsuits. The next day, Manville Chairman and CEO John A. McKinney's full page ad appeared in the Washington Post, the New York Times, and the Wall Street Journal. McKinney chastised Congress for failing to pass a statutory compensation program, and held the government responsible for conditions at naval shipyards during World War II. Selikoff, who was then Director of the Environmental Sciences Laboratory at the Mount Sinai School of Medicine in New York City, called the filing "simply the latest episode in a fifty year history of corporate malfeasance and inhumanity to man unparalleled in the annuls of the private enterprise system."

At the time of filing, the company had over two billion dollars in assets, and was ranked 181st on the Fortune 500 list of domestic industrial concerns. Bankruptcy was seen as the only viable solution for keeping the company alive to face the onslaught of product and strict liability lawsuits. [349] Manville voluntarily declared bankruptcy to force government intervention on the industry's behalf[350] so that, as CEO McKinney stated, "the thousands of citizens and voters caught up in this problem will be spared the expensive, inefficient, and haphazard litigation system we have been saddled with."[351]

10.3.2.2 Manville Sues the Government The suit filed by Manville in May 1983 was the first of its kind against the federal government. The company sought reimbursement of compensation it had already paid to former shipyard workers. Half of the claims were from shipyard workers. Most of them had never even worked for the company, but had used its products.[352] Manville argued that Navy specifications and government actions during World War II made the federal government liable for damages. The suit was Manville's way of forcing the government to do something about the prospect of never ending claims.

10.3.3 Organizing to Settle Claims

On June 19, 1985, the Wellington Agreement was signed in the United States by thirty-four asbestos producers and sixteen insurers.[353] Manville did not participate, as it had already established a trust for settling claims during its reorganization. The agreement established the Asbestos Claims Facility to settle bodily injury claims, and to provide a joint defense against claims brought by asbestos victims. Sixty-five thousand lawsuits were settled under the Asbestos Claims Facility, but it could not handle the number of cases being brought, or the increasing number of cases from new and different sources of exposure. The Claims Facility was dissolved in 1988. It was succeeded by the Center for Claims Resolution, formed by some of the producers and insurers who had signed the original agreement.[354] Disputes continue regarding settlement of claims.

10.4 EPA

Overall workplace conditions have probably improved because of OSHA, but the agency responsible for bringing the subject of asbestos fully into the public arena was the Environmental Protection Agency (EPA) through its administration of the asbestos-in-schools programs.

10.4.1 Provisions for Regulating Asbestos

OSHA regulates matters of health and safety in occupational settings. EPA enforces public health and environmental safety laws.[355] The EPA Worker Protection Rule of 1987 extended OSHA's asbestos regulations for the construction industry to cover workers in the public sector who are engaged in asbestos abatement activities.

In 1979, the agency established the Technical Assistance Program (TAP) to provide guidance and technical assistance for the identification and control of ACM in buildings.[356] EPA regulates asbestos under several provisions, and each of its ten regional offices has a Regional Asbestos Coordinator.

Emissions to ambient air are regulated under the National Emission Standards for Hazardous Air Pollutants (NESHAP), which EPA administers under authority of the Clean Air Act of 1971.[357] NESHAP revisions in 1973 specified work practices and procedures to be followed in order to prevent "visible emissions" of dust. Removal of asbestos

prior to renovation and demolition projects, and prior notification to EPA of same, are requirements of NESHAP.

Under NESHAP, asbestos was designated as a hazardous air pollutant in 1971. The use of asbestos in spray-on insulation or fireproofing applications was banned in 1973, followed by a ban on its use for decorative purposes. The disposal of asbestos waste in landfills is regulated under the Resource Conservation and Recovery Act (RCRA) of 1976, which pertains to solid, but not hazardous, wastes.

10.4.2 The Asbestos-in-Schools Programs

EPA regulates the asbestos-in-schools programs to monitor asbestos abatement in public and private, elementary and secondary school buildings. The most prominent of these programs is regulated under the Asbestos Hazard Emergency Response Act (AHERA) of 1986.

AHERA was originally created as Subchapter II under the Toxic Substances Control Act (TSCA). TSCA was enacted in 1976 to fill the gaps left in other federal laws regulating toxic substances.[358] Under TSCA, EPA issued the Friable Asbestos-Containing Materials in Schools Identification and Notification Rule (Asbestos-In-Schools rule), effective in June, 1982.

10.4.2.1 Identification and Notification Rule The Identification and Notification rule required private schools and school districts to inspect for friable asbestos by June 28,

1983, and notify parents and employees, whether or not it was present.[359] Abatement was not required, and there were no guidelines for the inspections, or for establishing when abatement would be an appropriate response.

No funding was allocated to carry out the inspection and notification program. Schools that had complied with the Asbestos-In-Schools rule could apply for funding under the Asbestos School Hazard Abatement Act of 1984, which provided loans and grants to correct serious exposure situations in financially needy schools.[360]

An EPA survey of 2600 schools, completed in January, 1984, showed that ninety-three percent of them had been inspected, but only thirty-four percent were in full compliance with the rule. One year later, EPA had issued 147 civil complaints nationwide, for failure to comply. Fines levied on the school districts totaled more than one million dollars.[361]

10.4.2.2 AHERA of 1986 To correct deficiencies in the rule, Congress directed the EPA to regulate response actions to friable ACM found in the schools. Friable asbestos is defined as any material, containing more than one percent asbestos by weight, that can be easily crumbled by hand pressure. Representative Jim Florio, of Gloucester Township, New Jersey, sponsored the federal legislation which became known as the Asbestos Hazard Emergency Response Act (AHERA)

of 1986.[362] The act was signed into law by President Reagan on October 22, 1986.

AHERA required local education agencies (LEAs) to establish management plans with inspection, monitoring and notification procedures for the control of asbestos in the schools. Approximately 107,000 schools in the nation were affected by AHERA,[363] and abatement work was anticipated for 45,000 of them.[364]

Control options included repair, encapsulation, enclosure, and removal. Removal of non-friable asbestos-containing materials was rarely required by AHERA. A poorly done removal job could increase, rather than eliminate, the risk of exposure. Abatement work in the schools, including inspections and the design of management plans, was to be done only by EPA accredited personnel. A readily available work force of such personnel was not yet established.

The implementation of AHERA required a vast education and training effort. The EPA published numerous guidebooks on controlling asbestos-containing materials in buildings, and in January, 1985, established the first supervisory level training centers at the University of Kansas, Tufts University, and Georgia Institute of Technology.[365] Maryland, New Jersey, and Alaska were the first states to establish training and certification programs for asbestos abatement personnel.

The original deadline for completing the inspections and submitting the asbestos management plans was October

12, 1988. Most school districts experienced difficulties, and many of the plans that were submitted were technically inadequate. In New Jersey, less than twenty percent of the school districts had filed by the deadline. President Reagan signed a bill in mid-July allowing the LEAs to request delays until May 9, 1989, but the original July 9, 1989 deadline for implementation of the plans remained unchanged. By the end of August, EPA had approved training and certification programs for asbestos abatement in only eight states (New Jersey, Alaska, Arkansas, Illinois, Iowa, Kansas, Massachusetts, and Michigan).[366] A general lack of understanding of the regulations, and the temperament of the times, resulted in a plethora of removal projects, undertaken during the summer of 1989.

10.4.3 The 1988 Study

One provision of AHERA required EPA to determine whether public and commercial buildings should be subject to the asbestos-in-schools regulations. In February 1988, the EPA Study of Asbestos-Containing Materials in Public Buildings: A Report to Congress was issued. The study detailed the results of extensive inspections conducted in a statistically representative sampling of 231 buildings located at ten sites around the country. The sample represented 3.5 million buildings in the United States, divided into three classes - federal government, private non-residential or commercial, and residential apartments.[367] The study found

that almost sixty percent of the buildings contained ACM. The worst levels of airborne asbestos fibers in a sample of forty-three federal buildings were no higher than those found in ambient outdoor air.[368] The study found that asbestos was commonly found in large, residential apartment buildings and most commonly used in heating systems to insulate boilers and piping.

From the sample, EPA estimated that 733,000 federal and commercial buildings contained friable asbestos-containing material (ACM), and 317,000 of those buildings contained some areas with significantly damaged ACM that was likely to become airborne.[369] It was estimated that 190,000 buildings contained 1.2 billion square feet of sprayed-on or troweled-on materials, with an average asbestos content of fourteen percent.[370] Estimates for removal of ACM in these buildings ranged from $100 to $150 billion. EPA estimated that 22.5 million office workers, and 369,200 custodial workers worked in the buildings, and predicted 3,300 deaths from asbestos-related disease by the year 2118, among the population using these buildings.[371] An extension of EPA regulations to encompass these additional buildings was estimated to cost $53 billion over a thirty year period. Instead of widening the scope of its oversight, EPA recommended a $6.2 million increase in its annual budget over the next three years: to increase the number of trained asbestos-control personnel, to develop safe abatement methods, and to supplement educational, technical assistance

and enforcement programs. No new regulations were proposed because of limited resources available in terms of funding and trained personnel. The schools were given top priority, "both because of the minerals' greater prevalence in schools and because asbestos exposure poses a greater cancer risk to children than adults."[372]

10.4.4 Asbestos Ban and Phase Out Rule

The EPA had already banned several uses of asbestos in the 1970s and began investigating other asbestos containing products after French researchers reported that asbestos-impregnated floor tiles could release fibers through normal wear.[373] On June 20, 1986, the agency published a proposal to ban all uses of asbestos over the next ten years. The EPA estimated that the proposal would prevent 1900 deaths per year from asbestos-related cancers. On July 12, 1989, the EPA promulgated the Asbestos Ban and Phaseout Rule under TSCA. The Asbestos Ban and Phaseout Rule was designed to phase out the importation, manufacture and processing of 94% of all remaining asbestos-containing products in the United States, in three stages, over a seven year period.

The schedule for the Ban and Phase Out rule began in 1990, when the manufacture, importation, or processing of asbestos in roof and floor felt, vinyl-asbestos tile, asbestos clothing and asbestos-cement products was prohibited. Commercial distribution was prohibited in 1992. After 1994, the same applied to gaskets and some friction products,

followed in 1996 by paper products, roofing and other coating products, additional friction products, and asbestos-cement pipes and shingles. Under the rule, all commercial distribution of these products in the United States would be prohibited after 1997. Exemptions to the ban could be granted on a case-by-case basis. The rule did not affect existing asbestos-containing materials in buildings.[374]

The Asbestos Information Association of North America (AIA/NA) filed a petition in the US Court of Appeals in Richmond, Virginia.[375] The AIA was an industry supported lobbying group and public relations agency that was formed in 1970. The group challenged the authority under which the Asbestos Ban and Phase Out rule was established, but specified that the legal action did not pertain to friable asbestos products being abated from schools and other structures. The Asbestos Cement Pipe Producers Association joined the suit.

A decision by the US Fifth Circuit Court of Appeals, in October, 1991, vacated enforcement of the Asbestos Ban and Phase Out Rule. The health effects of exposure to asbestos were not the basis for the suit, and they were not considered in the court's decision. The EPA has interpreted the court's decision to apply only to products that were still found in commerce after the rule was enacted, and in 1993, published a list of product categories still subject to the rule. It is not clear which products will remain in commerce, but the business of asbestos has been sharply

curtailed in the United States.[376] Ten years after the OSHAct became effective, the apparent annual consumption of asbestos had decreased by half.[377]

CHAPTER 11

DISCUSSION
SCIENCE, TECHNOLOGY, and PUBLIC POLICY

11.1 Introduction

Asbestos was the miracle fiber that became the curse of the twentieth century. An understanding of its role in the development of the United States helps to explain how the hazards of exposure were perceived, interpreted and addressed.

Asbestos was remarkably marketable because of its versatility. Between 1900 and 1980, 36 million metric tons were used in over 3,000 products. Millions of buildings benefited from its unique fireproofing and insulating properties.[378]

In the 1950s, marketing non-regulated asbestos-containing products to the schools was a profitable venture for industry. Thirty years later, regulations for the asbestos-in-schools programs created a new market for asbestos abatement, forcing technology and the practitioners needed to service that market to catch up.

11.2 Regulation of Hazardous Materials

Laws promulgated in the 1980s to protect workers exposed to hazardous materials included, for the first time, specific mandates that required more precise assessment, monitoring and recordkeeping procedures.

11.2.1 Origin of Federal Laws

The origin of these laws involved OSHA's experience in responding to oil spills in the chemical industry.[379] OSHA's worker protection laws applied to the chemical industry, but laws for hazardous wastes and accidental releases were monitored by the EPA. OSHA had no jurisdiction over workers responding to spills inside the plants, and the EPA had no mandate to regulate worker safety.

The first regulations to address hazardous materials and emergency response training were issued in 1980 under the Resource Conservation and Recovery Act (RCRA) to cover workers in treatment, storage, and disposal facilities. This was followed by the Hazardous Communications Act of 1984 (29 CFR 1910.1200), which applies to employees in companies using or manufacturing hazardous materials. In 1986, OSHA was directed to regulate workers at hazardous sites or responding to emergencies at those sites. These regulations were promulgated in AHERA, which included standards for training asbestos abatement workers handling AHERA contracts.[380]

11.2.2 Creating A New Industry

Federal regulations created the asbestos abatement industry to address the health concerns of asbestos in the schools. The market for asbestos abatement grew from about $600 million in 1986[381] to over $1.5 billion by the following year, in contractor services alone.[382] New and improved

technologies were developed to improve efficiency, and to meet the flurry of increasingly stringent regulations.

By 1989, asbestos abatement contractors and equipment suppliers were also engaged in hazardous materials management and emergency response remediation. Eliminating economic losses faced by owners whose buildings contained asbestos provided another market for the growing abatement industry.[383]

11.3 Public Policy

Like the TLV, the PEL was not meant to imply a safe level of exposure, but one that could be assessed using technology that was widely available. Limitations in both technology and available data were reflected in the standards. Advances in both of these areas provided some basis for setting limits, but many other factors were considered. Unlike the TLV, the PEL is codified in regulations and procedures that are enforceable under federal law. Court cases brought by both industry and labor challenging, the validity of OSHA's standards and how they were developed, are ongoing, as of 1994.

11.3.1 Changing Conditions

As a matter of public policy, establishing an exposure limit involves elements of risk and burdens of uncertainty that must be evaluated in the decisionmaking process. Changing

conditions produce different situations that influence how those risks and burdens are allocated. OSHA's first regulations addressed unsafe conditions in manufacturing plants, mines and steel mills.[384] Changes in the economy, and advances in technology have produced new industries, with occupations and processes with different conditions. Regulations addressing health and safety issues will always lag behind changes in the economy because hazardous conditions are not always readily discernible.

11.3.2 Defining the Problem

A problem has to be recognized before it can be defined or addressed. Klaidman comments on the high level of uncertainty typical of health-risk issues, which makes them especially manipulable.[385]

Peters notes that government regulations may be responsible for some common misconceptions about the hazards of asbestos. Asbestos is regulated as a mineral dust. The required warnings and labels caution against exposure to asbestos dust. In his 1930 report, Merewether emphasized control of dust levels. As Peters points out, most people believe dust is *something* that can be seen, but the most harmful asbestos fibers are too small to be seen by the naked eye, and they can stay airborne for long periods of time. A situation in which dust can be seen is one of gross contamination.[386]

11.3.3 Addressing the Problem

How a problem is addressed reflects the perspectives and values of the decision makers. Industry executives knew that exposure to asbestos dust was a health hazard, but they were not compelled to recognize it as such, or institute preventive measures. Their primary concern was the possibility of escalating compensation costs from the adverse health effects from exposure to asbestos dust.[387] The industry's problem was originally defined in terms of lost production time and cost containment. Efforts to determine the nature and extent of the problem were undertaken from that perspective. The problem was addressed by suppressing information, influencing unfavorable legislation, and disclaiming positions contrary to their interests. Before the hazards of asbestos exposure became a matter of public debate, there were no effective rebuttals to the industry's position.

Industry was able to carry on business as usual as long as it did, because it retained control over conditions in the workplace. The OSHAct of 1970 finally established federal regulation of workplace conditions, six years after pressure to produce such legislation was initiated in 1964.

11.4 Summary

The 1964 studies established evidence of the hazards from occupational exposure to asbestos, which helped initiate the movement toward environmental legislation. Remediation of

asbestos in the schools was addressed as a national priority because of fears that the situation placed an entire generation in jeopardy of developing debilitating asbestos-related diseases. Environmental exposures exploded on the scene in New York City in the summer and fall of 1989, when ruptures of asbestos-cement steam and water pipes seemed to occur on a weekly basis.[388]

One of the first of these ruptures occurred in Gramercy Park. The fine mist spewed from the steam pipe released two hundred pounds of asbestos, in a geyser that reached the rooftops of a row of affected apartment buildings. In that case, Con Edison was fined late in 1994, for failure to give proper notification and follow other required procedures. The utility was able to settle OSHA's proposed sixty thousand dollar fine for seven thousand dollars.[389]

The Occupational Safety and Health Act was passed under the authority of Congress to regulate commerce. Issues of health and safety, even when recognized as hazards, are weighed against economic interests. Science, research, and technology can provide information, but the results can only be explained through interpretation. The industry fought against stricter regulation of asbestos to the bitter end, citing lack of scientific proof as an argument for delay. Various aspects of exposure to asbestos will continue to prompt debates that could apply as well to: cigarette smoke, materials substituted for asbestos, hazardous waste disposal sites, and electromagnetic fields (EMFs).[390]

Perception of risk and what is considered to be safe, are subject to opinion in any number of situations when health, safety, and the cost of peoples' lives are weighed against economic interests, by entities with diverse agendas and the power to impact policy decisions. As Peters notes, government mandated requirements are minimum guidelines "representing a real-life political compromise between influential interest groups" which, if used in a vacuum, "can induce unsafe behavior, promote harmful misconceptions, and create detrimental attitudes."[391]

Standards for health and safety will continue to be based on incomplete data until better information is available. That may involve the amount of time it takes for the consequences of a hazard to outweigh the failure to recognize and address it.

There is some scientific consensus regarding the causation and progression of asbestos-related diseases. Unanswered questions about these processes are a primary source of the longstanding controversies regarding exposure to asbestos. Uncertainties include which fiber characteristics induce disease mechanisms; the synergy of smoking and asbestos exposure that increases the risk of lung cancer; extrapolating the results of animal studies to humans; transferring occupational experience to non-occupational settings; using past exposure and disease experience (heavy in textiles) to predict present circumstances (light in schools); a threshold level below which disease will not

occur; and the role of differences in human susceptibility. Research and technology may eventually provide better information about some of these processes.

CHAPTER 12

CONTROVERSIES

Some of the current knowns, unknowns, and continuing controversies regarding the regulatory aspects of asbestos, are outlined briefly in this final chapter, including OSHA's 1994 rule for occupational exposure to asbestos, the latest since the 1986 standard.

12.1 Regulatory Definitions Limited by Technology

For regulatory and analytical purposes, asbestos is defined as a fiber in terms of length and aspect ratio. The aspect ratio is a fiber's length compared to its width. OSHA defines a fiber as greater than 5 microns in length, with an aspect ratio of three to one or greater. EPA defines a fiber as greater than or equal to 0.5 microns in diameter, with an aspect ratio of five to one, with substantially parallel sides.

Exposure to airborne asbestos is determined by analyzing fiber counts of samples taken to be representative of conditions at the site. Collecting the samples, preparing them, and counting the fibers, are all subject to human and other errors. The instrumentation and procedures used are explained in Asbestos: The Hazardous Fiber, pages 102-142, and are detailed in OSHA's regulations.

NIOSH Method #7400 prescribes the method for analyzing fiber counts using phase contrast microscopy (PCM) at a magnification of four hundred times (400X). There are two sets of rules: The A rules apply to fibers with aspect ratios of three to one or greater; the B rules apply to aspect ratios of five to one or greater, where the diameter is less than three microns. The units of measurement are miniscule. There are one million microns in one meter. One meter equals 39.37 inches, or 3.28 feet, or 100 centimeters. One fiber per cubic centimeter (1 f/cc) of air is equivalent to one million fibers in each cubic meter of air which, by definition, includes both asbestos and nonasbestos fibers.

PCM is not capable of detecting fibers with diameters of less than 0.25 microns, and less than 0.5 microns in length. It cannot distinguish between asbestos and other fibers.[392] The skill and accuracy of the microscopist counting the fibers is critical, but it is difficult to count what cannot be seen. Ultimately, the limits of readily available technology determined the lower value for an exposure limit.

The regulatory definition of a fiber reflects technological limitations in the detection, measurement, and counting of asbestos fibers. A 1985 EPA guidebook entitled, "Measuring Airborne Asbestos Following Abatement Action," stated that transmission electron microscopy (TEM) was "the best method for measuring airborne asbestos." TEM has a magnification of 10,000X, and is capable of distinguishing

asbestos fibers from other fibers. As of December 14, 1989, AHERA required the use of TEM analysis for determining the final clearance levels in asbestos abatement projects.

12.2 Fiber Characteristics and Disease Effects

Asbestosis, lung cancer, mesothelioma, and non-malignant asbestos-related diseases are detailed in Sourcebook on Asbestos Diseases by Peters, and Work-Related Lung Disorders by Cotes[393]. One of the long standing controversies concerns the causal, versus the collaborative or incidental, effect of exposure to asbestos fibers. The degree to which various types of asbestos promote specific diseases is still being debated, but all commercial forms of asbestos have now been shown to produce these diseases.

12.2.1 Long vs Short Fibers

Regulatory emphasis on longer fibers, and the difficulty of detecting and counting shorter fibers, led some to conclude that only longer fibers were potentially harmful, and shorter fibers relatively harmless.[394] The longer, thinner fibers are thought to be more carcinogenic, especially for mesothelioma, whereas, shorter fibers migrate deeper into body tissue.[395] The most harmful fibers are only one tenth the size of an object that can be seen by the naked eye.[396] Studies indicate that the fibers most likely to cause cancer are too thin to be observed by a light microscope.

12.2.2 Asbestos Fibers in Body Tissue

Inhalation and ingestion are the primary routes by which asbestos fibers enter the body. Orally, asbestos does not appear to present a significant health hazard. Inhalation is the route which leads to ill health effects.

Asbestos fibers can be widely disseminated in the human body. Their transmission through body tissue has not been fully explained,[397] and the amount of fibers required to cause adverse reactions in living organisms has not yet been determined. The large surface area and specific surface reaction of the fibers are suspected to have some effect on cells.[398]

A latency period of twenty to forty years is typical before physical ailments become apparent. The length of the latency period varies with the type of disease. The likelihood of developing disease follows a dose/response relationship,[399] but the mechanism that initiates the malignant transformation remains unknown.

12.3 Latency and Liability

Latency was a key issue in the class action lawsuits. The question was whether insurers were liable for claims from the time of first exposure, or from the time that disease became apparent. The U.S. Court of Appeals for the District of Columbia in 1981 answered the question of insurer liability in Keene vs. INA. In that case, the court ruled that "all insurance policies in effect from the day of exposure

until the time the illness manifests itself are responsible for the entire amount of the loss...".

Fibreboard had been insured by the Chubb Corporation's Pacific Indemnity Company under a twelve month general liability policy that began in 1956. That was followed by a three year policy, issued by a subsidiary of CNA Financial Corporation, which began in 1957, but was cancelled after twenty-two months. The terms of the policies were argued during litigation of a personal injury class action lawsuit against Fibreboard that began in 1979 in California. In 1990, the California court ruled that the two insurers involved in the case faced unlimited liability. A three billion dollar settlement was proposed in 1993. At the time, Chubb's general counsel stated that there were 148,000 past or pending claims, and 186,000 new claims were anticipated against Fibreboard over the next twenty or thirty years. Chubb's share of the settlement proposal was $358 million, bringing their total reserves for injury claims arising from the one year policy to $1.25 billion.[400]

12.4 Changes in Classifications

Dust levels were the highest during a period when few measurements of dust levels were taken. More precise measurement techniques were adopted, but comparisons with earlier data, gathered using impingers, were compromised. Decades later, when disease became apparent, there was no reliable basis for assessing past exposures. Categories

for classifying causes of death were also changed over the years[401] as the various pulmonary diseases were more precisely differentiated and defined.

In 1972, the Bureau of Mines began revising the format it used for collecting data on domestic asbestos consumption. Adjustments were made to reflect one hundred percent of apparent consumption for eleven major uses of asbestos. The revisions negated the possibility of comparisons with earlier years. The difficulty of establishing a true or complete picture of past situations based on historical data, is aggravated when changes negate the basis for comparison.

12.5 Chrysotile

There is still considerable controversy over the ill health effects of chrysotile, in part because exposure to chrysotile does not produce mesotheliomas at the same rate as the amphiboles.[402] Most of the Canadian chrysotile that was used in the United States is naturally contaminated with approximately one percent tremolite, an amphibole, which was blamed for producing mesotheliomas found among Canadian miners. Tremolite is found in talc, and in children's play sand. It is controversial in its own right.[403]

12.5.1 Mesothelioma

Mesothelioma tumors, first linked to asbestos exposure in 1943, were very rare. In 1960, Wagner reported thirty-three deaths from mesothelioma over a four year period, all from

the North Western Cape Province of South Africa, where crocidolite was mined.[404] For this reason, mesothelioma was associated with crocidolite asbestos for a long time. Meso thelioma progresses rapidly, and is usually fatal within two years of diagnosis. It does not appear to be dose related, and smoking does not appear to increase the risk.[405]

12.5.2 The "amphibole hypothesis"

The argument that chrysotile is less carcinogenic than other forms of asbestos is outlined in an article on the "amphibole theory" by Mossman et al., in the January, 19, 1990 issue of Science.[406] In rebuttal, Nicholson, notes that chrysotile can partially dissolve in body fluids, and does not tend to remain in the lungs. This makes an estimation of dose difficult to ascertain. Amphiboles fibers do accumulate in body tissue, and are visible upon autopsy. Nicholson also notes that lung cancer is three times more prevalent than mesothelioma among groups of workers exposed to asbestos.[407]

12.6 Lung Cancer

Cancer is a collection of diseases that affect various sites of the body in different ways. The progression of asbestos-related cancer is indistinguishable from cancer caused by some other agent.[408] Cancer is the second leading cause of death in the United States, after heart disease, and lung cancer is the number one cause of cancer related deaths.

It is estimated that as many as sixteen thousand lung cancer deaths per year may be due to occupational exposure.[409] Whether or not asbestosis progresses to bronchial carcinoma, or lung cancer, is another controversial topic.[410]

12.6.1 Asbestosis

Asbestosis is a diffuse pulmonary fibrosis of the lungs, initiated by the inhalation of asbestos fibers.[411] The aerodynamic behavior and deposition of asbestos fibers as they travel through the respiratory tract are described in Bernarde's Our Precarious Habitat, and in The Normal Lung: The Basis for Diagnosis and Treatment of Pulmonary Disease by John F. Murray. The body's natural defense mechanism attempts to destroy the foreign fibers by surrounding them with scar tissue. The build-up of scar tissue causes a thickening or fibrosis of the alveoli walls, and of the pleural tissue of the lung (pleural) cavity. The gas exchange function of the lungs is reduced as more air sacs are affected, causing a reduction in functional lung capacity. Straining to breathe produces an enlarged heart, typical of asbestosis sufferers. When this process is initiated by asbestos fibers, it is called asbestosis.[412] Eventually, the sufferer suffocates.

Fifty percent of asbestosis sufferers may develop lung cancer, the predominant form of respiratory cancer. Moderate exposure to asbestos increases the risk of developing lung cancer by a factor of five to ten.[413]

12.6.2 Cigarette Smoke and Smoking

For those who smoke, and are exposed to asbestos, the risk factor for developing lung cancer increases by more than ninety. The risk appears to be dose related, and suggests some synergistic relationship between asbestos fibers and tobacco smoke. Asbestos is considered to be the second most important cause of lung cancer after tobacco smoke.[414] Bernarde notes the numbing effect of cigarette smoking on the cilia, which reduces the output of mucus and permits more asbestos fibers to slip deeper into the respiratory tract to the alveoli.[415]

Klaidman, in his book on media coverage and public health issues, notes that lung cancer was rare in the first third of the twentieth century, before smoking became popular. Between 1922 and 1952, cigarette sales rose by 639 percent while the population was growing by only 54 percent. As smoking became more popular, yet another use was found for asbestos - in the filter tips of cigarettes. The rate of cancer among women increased almost four-fold between 1930 and 1967, the same period in which the proportion of adult women smokers rose, from ten percent to an estimated thirty-five percent.

Smoking was linked to lung cancer in 1936 by Dr. Alton Ochsner who, in the 1950s, predicted that lung cancer would account for 18 percent of all deaths from cancer in 1970. In fact, lung cancer accounted for 19.7 percent of deaths from cancer in 1970.[416] The cancer rate in 1994 was eighteen

percent higher than it was in 1971. At that rate, one third of the population can expect to develop some type of cancer during the course of a lifetime.[417]

12.7 Unregulated Chemicals

Benjamin Goldman, in The Truth About Where You Live, details the geographic disparities in mortality rates among counties in the United States, and notes that they are declining for all causes of death except cancer, especially lung cancer. Goldman also discusses the large number of unregulated substances in every day use for which there are no data. The National Toxicology Program lists 117 chemicals as potential human carcinogens. The International Agency for Research in Cancer (IARC) lists 30 chemicals, mixtures, or occupational exposures as carcinogenic to humans, 61 as probable human carcinogens, and 64 as carcinogenic to animals.[418] "Less than 2 percent of the 70,000 chemicals in commerce have been fully tested for human health effects, and there are no health data whatsoever for over 70 percent."[419] Goldman attributes the dearth of data to the high cost of performing thorough human tests and industry's reluctance to conduct them. He also notes evidence that "animal tests themselves cause the cancer, because massive doses of almost anything seem to make cells divide, increasing the risk of mutations associated with cancer."[420]

12.8 Experimental Animal Studies

Klaidman also addresses the question of bias in the interpretation of experimental animal studies for risk assessment purposes, citing EPA's conservative guidelines requiring that animal experiments select the most sensitive strain of the species. He gives a specific example of using rats in testing formaldehyde:

> ". . . although rats get nasal cancers from formaldehyde and appear to be the species that is most sensitive to it, rats are unusual because they can breathe only through their noses. In contrast, other animals, notably humans, can breathe through the mouth and this would reduce significantly the estimate of cancer risk from formaldehyde in humans."[421]

A person engaged in heavy labor is more likely to breathe through the mouth, inhaling more of whatever is in the air. In an eight hour day, a worker breathes in at least five cubic meters of air. Heavy labor can increase the volume of inhaled air by a factor of five.[422] The example emphasizes how the effects of a decision that is based on an interpretation can be multiplied throughout the experimental, analytical, and policy-making stages of risk assessment and risk management.

12.9 The One Fiber Theory

The question of low level exposures encompasses debates surrounding environmental exposures, the "one fiber theory," and the reasonableness of enacting legislation such as AHERA. At issue is the reliability of extrapolating past occupational exposures to predict expectations of future disease produced under present day conditions, and the variability of individual human susceptibility to disease.

The one fiber theory postulates that one fiber can initiate the progression of disease. Opponents, such as Mossman, declare that "if one fiber of asbestos could kill, we'd all be dead, as the general population all contain asbestos fibers in their lungs." In supporting this position, Bernarde cites a study conducted in the 1970s, of the population of Paterson, New Jersey. The study was of interest because the former UNARCO asbestos textile mill was located in a residential area. The study concluded that contact with asbestos did not appear to have an adverse effect on the residents of Paterson.[423]

12.10 Substitutes

Glass wool, rock wool, steel wool, iron wire, synthetic rubber, organic plastics, cellulose, and treated paper are some of the materials that have been tested as possible substitutes for asbestos, but none of them can duplicate the variety and combination of advantageous properties found in asbestos.[424]

During World War II, a fabric made from glass and asbestos yarns was developed to extend the limited supply of asbestos fibers. The United States Rubber Company manufactured a similar fabric named "Asbeston," which was said to be well-adapted for theatre curtains as well as fireproof draperies in ships, schools, hospitals, libraries, and hotels. Glass fibers have proven to be unsuitable as a replacement for asbestos in asbestos-cement and in friction products.[425]

In 1990, the EPA added man-made minerals, including glass fibers, rock wool, and slag wool fibers, to the list of hazardous air pollutants.[426] On July 1, 1994, the Department of Health and Human Services classified fiber-glass insulation as a suspected cancer-causing agent, and listed it, along with radon, as one of seven additions to the Annual Report on Carcinogens. Fiberglass insulation, common in most homes, was said to be "perfectly safe when handled properly."[427] The same could be said about asbestos. Currently, there is no defined or universally accepted threshold level below which exposure to airborne asbestos fibers can be considered safe.

12.11 OSHA Final Rule, 1994

12.11.1 Challenges to the 1986 OSHA Standard

Various provisions in OSHA's 1986 standard were challenged by the Building and Trades Department of the AFL-CIO, and the Asbestos Information Association (AIA). The Court of

Appeals upheld most of the provisions, but found that the ban on all spraying of asbestos containing products was unsubstantiated. The court ruled that the use of asbestos in encapsulating sprays would have to be allowed. In that process, asbestos is simultaneously sprayed and coated with resin, binding the fibers onto a surface. OSHA was ordered to take action on the ban, and other issues, by December 14, 1988.

A second set of issues included smoking controls, the effective use of respirators, and the use of bilingual warnings and labels. OSHA responded to these issues on January 28, 1990. With the court's consent, an extension of time was granted to coordinate a proposal for a third set of three issues, with EPA and other regulatory agencies. These revisions were published in a Notice of Proposed Rulemaking on July 20. The time scheduled for receiving comments on these issues was extended, and an informal hearing was held from January 23 to February 8, 1991. The post-hearing briefing period was extended to July 24, 1991.

On June 8, 1992, the definition of asbestos in the 1986 standard was amended to delete the non-asbestos forms of tremolite, actin, and anthrophyllite. In November, the comment period was re-opened, to allow additional public commentary on options to protect workers from inadvertent exposure to asbestos in buildings. Although not part of the court's remand order, the issue had been raised by OSHA and others.

In 1988, the Service Employees International Union (SEIU) petitioned, and subsequently sued EPA, seeking regulation of asbestos usage in public and commercial buildings. As a result, EPA convened a series of joint meetings between May 1989 and May 1990. Representation at these meetings included OSHA, unions, states, asbestos manufacturers, consultants and contractors, public interest groups, and other interested parties from the realty, lending, and insurance industries. "The major area of disagreement in the group dealt with the characterization of risk to general building occupants and office workers."[428] The need for specific federal inspection requirements was also disputed, but the group agreed that building service workers should be made aware of the presence of asbestos. As a result of these discussions, OSHA and EPA collaborated to enhance the compatibility of their respective regulations. The comment period closed on January 4, 1993.

12.11.2 OSHA 1994 Final Rule

On August 10, 1994, OSHA published its final rule for exposure to asbestos, which became effective on October 11. The evolution of the current PEL for asbestos, and details of the standards are included in the Codes of Federal Regulation (CFR). Occupational exposure to asbestos in general industry is codified in 29 CFR 1910.1001, in the construction industry in 29 CFR 1926.1101 (previously 1926.58), and in the shipyard industry in 29 CFR 1915.1001. The standards

include four classes of construction activity. Class I, for removal of known or presumed ACM, is the most hazardous, high risk activity. Class II involves removal activities that are not high risk. Class III involves repair and maintenance work where ACM or presumed ACM is disturbed. Class IV includes maintenance and custodial activities where employees may contact ACM. Clean up of ACM waste and debris is also included in Class IV. As the hazard of an activity increases, the prescribed controls become more stringent. Mandatory methods of control for brake and clutch repair are included in the standard for general industry. This group of workers remains the most highly exposed to episodic releases, although the exposures are sporadic.

The TWA-PEL was reduced to 0.1 f/cc for all industries, because "this limit is feasible for most industry sectors to reach most of the time." A lower PEL "would be particularly unsuitable as compliance criteria because it is difficult to reliably measure." Such measurements, when taken, would not insure that employers had complied with the standard. In contrast, it would be easier to determine whether or not specified work practices were being followed.

OSHA's 1984 risk assessment had shown that significant risk would be reduced, but not eliminated, at the 0.1 f/cc level of exposure. At that level, the risk of cancer would be reduced to 3.4 per 1,000 workers, with a twenty year exposure risk of 2.3 per 1,000 workers. OSHA acknowledged that significant health risk remained at the 0.1 f/cc level,

and mandated "operation-specific work practices" in the 1994 standard, as the "most cost-effective means of assuring that significant risk is eliminated to the extent feasible."[429]

12.12 Conclusion

The use of asbestos has been banned in several countries, but it is still a marketable commodity in segments of the global economy. The possibility of financial ruin from disability claims, the potential of future lawsuits, the massive education and training campaign conducted to launch AHERA, the educated consumer's disfavor, and the specter of regulatory enforcement, were factors which led to a drastic reduction in domestic production, and use of asbestos-containing products in the United States.

The public rulemaking process, formalized by the OSHAct, cut its teeth on asbestos. The regulatory policy adopted for friable asbestos, to reduce the exposure level to the lowest feasible limit, was realized in the 1994 OSHA standard. The limits of detection of the most widely and readily available technology now matches the regulatory definition of an asbestos fiber at the 0.1 f/cc PEL, a level recommended by NIOSH in 1976.[430]

OSHA standards will continue to come under attack by various industries and interests which continue to downplay the adverse health effects of their products. However imperfect the regulatory mechanism may be, the reality of enforceable federal regulations with comprehensive standards

and practices to protect workers, and prevent injury and death as a consequence of performing one's duties in the workplace, has changed the laissez-faire relationship that previously existed between government and industry.

Having executed the policy adopted for exposure to asbestos, regulatory agencies have turned their attention to other pressing matters. The prodigious amount of asbestos that was used in the United States has provided reason, at various times, for both profit and panic. The amount of ACM still in place, and still in commerce, will continue to provide sources of potential exposure.

Thirty years after the 1964 studies by Selikoff et al., and twenty-five years after the OSHAct, a much wider aware ness of the hazards and how to prevent or protect against them exists. Regarding the scientific literature, Peters notes the importance of distinguishing between causation of cancer and causation of asbestosis, because much, if not most, of the attention, experimental studies, and general conclusions have been based on the latter, as were the first TLV and the latest PEL limits levels for exposure to asbestos. Information and literature on every aspect of asbestos is more abundant than ever. These are tools to be utilized, because the bottom line is still caveat emptor.

APPENDIX A

GLOSSARY

ACM	Asbestos-Containing Material. EPA defines ACM as any material containing more than one percent asbestos by weight.
ACGIH	American Conference of Governmental Industrial Hygienists recommends TLVs.
AFL-CIO	American Federation of Labor and Congress of Industrial Organizations labor union.
AHF	Air Hygiene Foundation. Begun by the Mellon Institute in 1935, and renamed the Industrial Hygiene Foundation (IHF) in 1947.
AIA	Asbestos Information Association.
AIHA	American Industrial Hygiene Association.
AMA	American Medical Association.
Abatement	Procedures to control fiber release from ACM, which may include repair, encapsulation, enclosure, or removal.
Air Sampling	Samples taken under ambient conditions, then analyzed to determine the concentration of airborne contaminants, as a measure of the level of exposure.
Asbestos	The common and commercial name for a group of fibrous minerals.
CAG	Carcinogins Assessment Group of the EPA.
CPSC	Consumer Product Safety Commission.
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act.
DOL	U. S. Department of Labor
EPA	U. S. Environmental Protection Acency.
Encapsulation	Using a paint or sealant product to adhere asbestos fibers to a surface.
Enclosure	Using barriers to surround, enclose, or seal off ACM.

f/cc	Fibers per cubic centimeter of air.
FDA	Food and Drug Administration.
Friable	Material which can be crumbled, pulverized, or reduced to powder by hand pressure.
IAHFIAW	International Association of Heat and Frost Insulators and Asbestos Workers.
IARC	International Agency for Research in Cancer classifies chemicals according to their cancer-causing potential.
IHF	Industrial Hygiene Foundation of America, evolved from the AHF.
ILO	International Labor Organization.
impinger	A dust trapping device that was used to obtain samples, which were analyzed by microscope at a magnification of 100X.
Industrial Hygienist	A professional qualified by education, training, and experience to anticipate, recognize, evaluate and develop controls for occupational health hazards.
Latency	The time period between exposure and manifestation of disease.
mppcf	Million parts per cubic foot.
MSHA	Mine and Safety Health Administration.
NESHAP	National Emission Standards for Hazardous Air Pollutants.
NIOSH	National Institute for Occupational Health and Safety.
NTP	National Toxicology Program tests chemicals and reviews evidence for cancer.
O & M	Operations and Maintenance plans and programs, initiated under AHERA, to control and prevent exposure to friable asbestos in buildings.
OSHA	The Occupational Safety and Health Administration within the Department of Labor administers the OSHAct.

PEL	Permissible Exposure Limit. Levels of occupational exposure set by OSHA.
PCM	Phase contrast microscopy. The most readily available, lowest cost method for analyzing air samples. NIOSH Method #7400 describes the procedure.
PLM	Polarized Light Microscopy, used to analyze bulk, and surface or wipe samples.
RCRA	Resource Conservation and Recovery Act.
TAP	Technical Assistance Program, started in 1979 by EPA, established a Regional Asbestos Coordinator in each of agencies ten regional offices.
TEM	Transmission electron microscopy. Required by AHERA for analyzing air samples for final clearance in abatement projects. Magnifications range from 1000x to over 250,000X. Can distinguish asbestos fibers from others. NIOSH Method #7402 prescribes the procedure.
TLV	Threshold Limit Values. Levels of exposure recommended by the ACGIH.
TLV-C	TLV-Ceiling. The concentration which should not be exceeded.
TWA	Time-weighted average. Refers to the average air concentration of contaminants during a particular sampling period.
UNARCO	The Union Asbestos & Rubber Company.

APPENDIX B

Numerical limit levels adopted for exposure to airborne asbestos, 1946-1994

		OSHA PEL	ACGIH TLV
1946			5 mppcf
1963	ACGIH changed MACs to TLVs		
1968			12 f/cc, or 2 mppcf *
1970	OSHAct signed Dec. 29		
1971	OSHAct eff. Apr. 29		
	First consensus, May 29	12 f/cc	
	ETS issued, Dec. 7	5 f/cc	
1972	First comprehensive, June 7	5 f/cc	
1974			5 f/cc
1976	Effective July 1	2 f/cc	
1978			
1980			0.2 f/cc crocidolite 0.5 amosite 2.0 others
1986		0.2 f/cc	
1994	Issued August 10 Eff. Oct. 11, 1994	0.1 f/cc	

* 12 f/cc for asbestos or 2 mppcf for all fibers. The conversion factor used was 6 f/cc = 1 mppcf.

APPENDIX C

MEASURING AIRBORNE ASBESTOS CONCENTRATIONS

C.1 Common Units for Measuring Fiber Concentrations

mppcf = millions of particles per cubic foot
f/cc = fibers per cubic centimeter
ppcc = particles per cubic centimeter
= 35.3 x mppcf

Length

1 meter (m) = 39.37 inches, or 3.28 ft
= 100 centimeters (cm)
= 1,000,000 microns (um)
= 10,000,000,000 Angstroms (A)

Volume

1 cubic meter (m^3) = 35.3 cf
= 1,000,000 cubic centimers (cc)
1 liter (l) = 1,000 milliliters (ml)
= 61.02 cubic inches

Weight

1 pound = 454 grams (g)
1 gram = 1,000,000,000 nanograms (ng)

Source: EPA. "Guidance for Controlling Asbestos-Containing Materials in Buildings." (Purple Book)(Washington, D.C.: GPO, June 1986): B-1.

C.2 Regulatory Definitions of Asbestos Fiber

	Length	Aspect Ratio (Length to Width)	
OSHA Industry Standards	Greater than or equal to 5 microns	3:1 or greater	
EPA AHERA	Greater than or equal to 5 microns	5:1 or greater	
New Jersey NJAC 5:23-8 (Subchapter 8)	Greater than 5 microns	3:1 or greater	A Rules.
NIOSH #7400 A and B Rules		5:1 or greater	B Rules. Diameter less than 3 microns.

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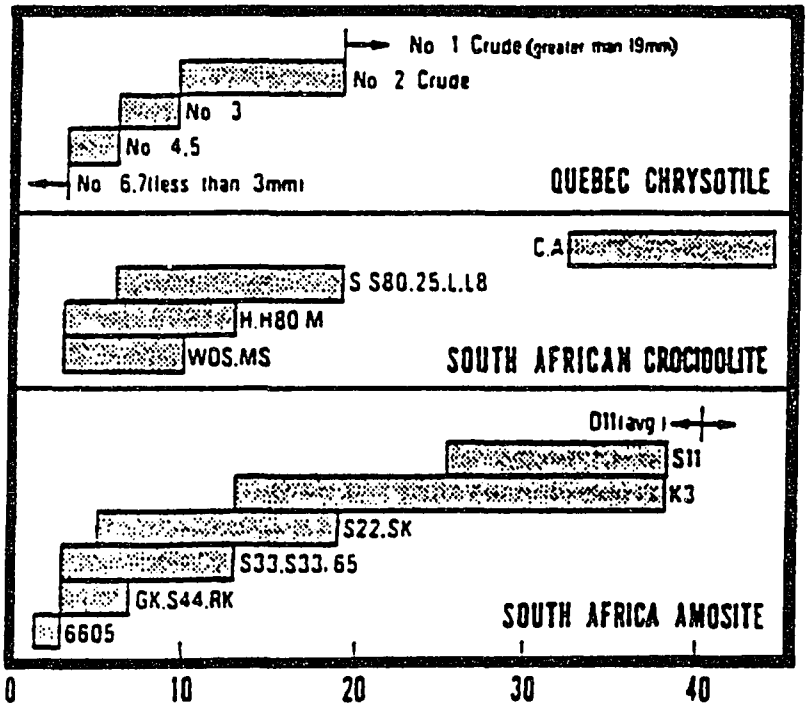

Figure D.1 Asbestos fiber lengths in millimeters with grade designations.

Source: Bureau of Mines. Asbestos. (Department of Commerce (Washington, D. C.: GPO, 1979): 4.

APPENDIX D

PROPERTIES OF ASBESTOS MINERALS[a]

	Chemistry[b]
Chrysotile	$Mg_3Si_2O_5(OH)_4$
Amosite	$(Fe^{2+}, Mg)_7Si_8O_{22}(OH)_2$
Crocidolite	$Na_2(Fe^{2+}, Mg)_3Fe^{3+}_2Si_8O_{22}(OH)_2$
Anthophyllite	$(Mg, Fe^{2+})_7Si_8O_{22}(OH)_2$
Tremolite	$Ca_2Mg_5Si_8O_{22}(OH)_2$
Actinolite	$Ca_2(Fe^{2+}Mg)_5Si_8O_{22}(OH)_2$

	Mineralogy[c]	Crystal Structure	Crystal System[c]
Chrysotile	Sheet silicate	Fibrous and asbestiform	Monoclinic and orthohombic
Amosite	Double chain	Prismatic, lamellar to fibrous	Monoclinic
Crocidolite	Double chain	Fibrous	Monoclinic
Anthophyllite	Double chain	Prismatic, lamellar to fibrous	Orthohombic
Tremolite/ Actinolite	Double chain	Long and thin columnar to fibrous	Monoclinic

	Morphology[b]	Cleavage[c]	Specific Gravity	Filtration
Chrysotile	Wavy, curly fibers	Perfect	2.4-2.6	Slow
Amosite	Straight, rigid fibers	Good	3.1-3.25	Fast
Crocidolite	Straight, rigid fibers	Good	3.2-3.3	Fast
Anthophyllite	Straight, single fibers	Perfect	2.85-3.1	Medium
Tremolite/ Actinolite	Straight, single, or composite fibers	Good	2.9-3.2/ 3.0-3.2	Medium

	Fusion Point, °F	Specific Heat, Btu/lb/°F	Electric Charge	Resistance to Destruction from Heat
Chrysotile	2770	0.266	Positive	Good. Brittle at high temp.
Amosite	2550	0.193	Negative	Good. Brittle at high temp.
Crocidolite	2180	0.201	Negative	Poor, fuses
Anthophyllite	2675	0.210	Negative	Very good
Tremolite/ Actinolite	2400/ 2540	0.212/ 0.217	Negative	Fair to good/ ---

	Color	Luster	Texture
Chrysotile	Green, gray amber to white	Silky	Soft to harsh
Amosite	Gray, yellow to dark brown	Vitreous	Coarse but pliable
Crocidolite	Blue	Silky to dull	Soft to harsh
Anthophyllite	Yellowish, brown, gray-ish white	Vitreous to pearly	Harsh
Tremolite/ Actinolite	Gray-white, greenish, yellowish, bluish	Silky	Harsh

	Flexibility	Spinnability	Tensile Strength, psi
Chrysotile	High	Very Good	824,000 max.
Amosite	Good	Fair	16,000 to 90,000
Crocidolite	Good	Fair	876,000 max.
Anthophyllite	Poor	Poor	4,000 and less
Tremolite/ Actinolite	Poor	Poor	1,000 to 8,000/ 1,000 and less

[a]Source unless noted: Handbook of Asbestos Textiles, 2nd ed., (Philadelphia, Pa.: Asbestos Textile Institute, 1961) 8-9.

[b]Melvin A. Benarde, ed., Asbestos: The Hazardous Fiber, (Boca Raton, Florida: CRC Press, 1990) 14.

[c]Asbestos in the Great Lakes Basin with emphasis on Lake Superior: A Report to The International Joint Commission from The Great Lakes Research Advisory Board (Feb. 1975) 9.

APPENDIX E

ASBESTOS COMPARED with OTHER SUBSTANCES

Table E.1 Airborne Concentrations of Dust, mppcf

Substance	Approximate concentration (millions of particles per cf of air)
Pollen	0.05 - 0.2
Dust, rural air	0.2 - 2.0
Dust, city air	0.5 - 5.0
Dust, industrial district	1.0 - 20.0
Dust, dust storm	2,000 - 3,000

Source: Gross, William F. Applications Manual for Paint and Protective Coatings. (New York: McGraw Hill, 1970): 64

Table E.2 Diameter of Various Fibers

	Fiber Diameter, inches	Fibrils in one linear inch
Human hair	0.00158	630
Ramie	0.000985	1015
Wool	0.008 to 0.0011	910 to 1250
Cotton	0.0004	2500
Rayon	0.0003	3300
Nylon	0.0003	3300
Rock wool	0.000142 to 0.000284	3520 to 7040
Glass	0.00026	3840
Chrysotile	0.000000706 to 0.00000118	850,000 to 1,400,000

Table E.3 Average Tensile Strength of Various Materials

	Tensile Strength, lbs. per sq. in.
Ingot iron	45,000
Wrought iron	48,000
Carbon steel	155,000
"Nichrome" steel	243,000
Piano steel wire	300,000
Cotton fiber	73,000 to 89,000
Rock wool	60,000
Glass fiber	100,000 to 200,000
Chrysotile	80,000 to 200,000
Crocidolite	100,000 to 300,000
Amosite	16,000 to 90,000
Tremolite	1,000 to 8,000

Table E.4 Surface Area of Various Fibers

	Surface Area by N_2 Adsorption, square centimeters per gram
Nylon	3100
Acetate rayon	3800
Cotton	7200
Silk	7600
Wool	9600
Viscose rayon	9800
Chrysotile	130,000 to 220,000

Source for Tables E.2,E.3, E.4: Rosato, D. V. Asbestos: Its Industrial Applications (New York: Reinhold Publishing, 1959) 42-43, 50.

The density of chrysotile is 2600 g/m^3. The density of the amphiboles is 3000 g/m^3.

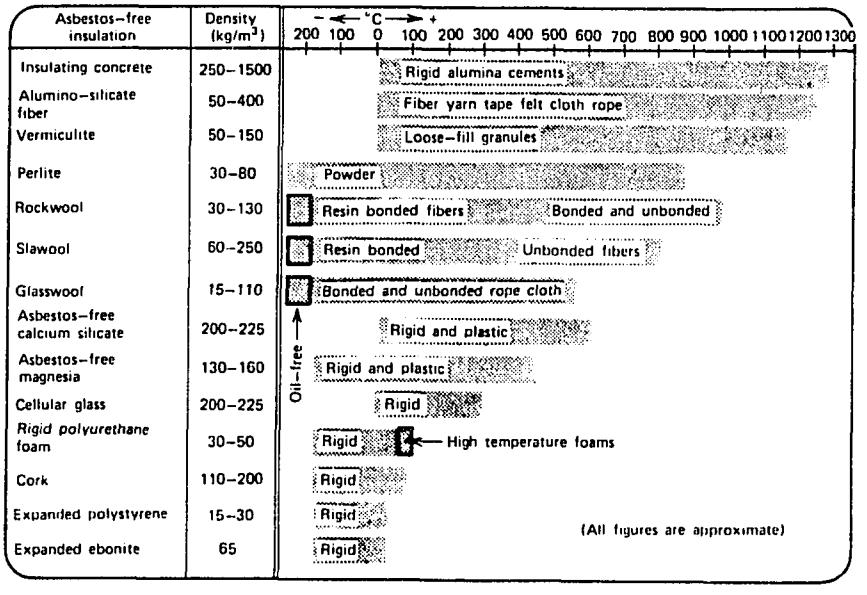

Figure E.1 Density and temperature of non-asbestos insulation materials

Source: Burgess, William A. Recognition of Health Hazards in Industry: A Review of Materials and Processes. New York: John Wiley & Sons, 1981): 158.

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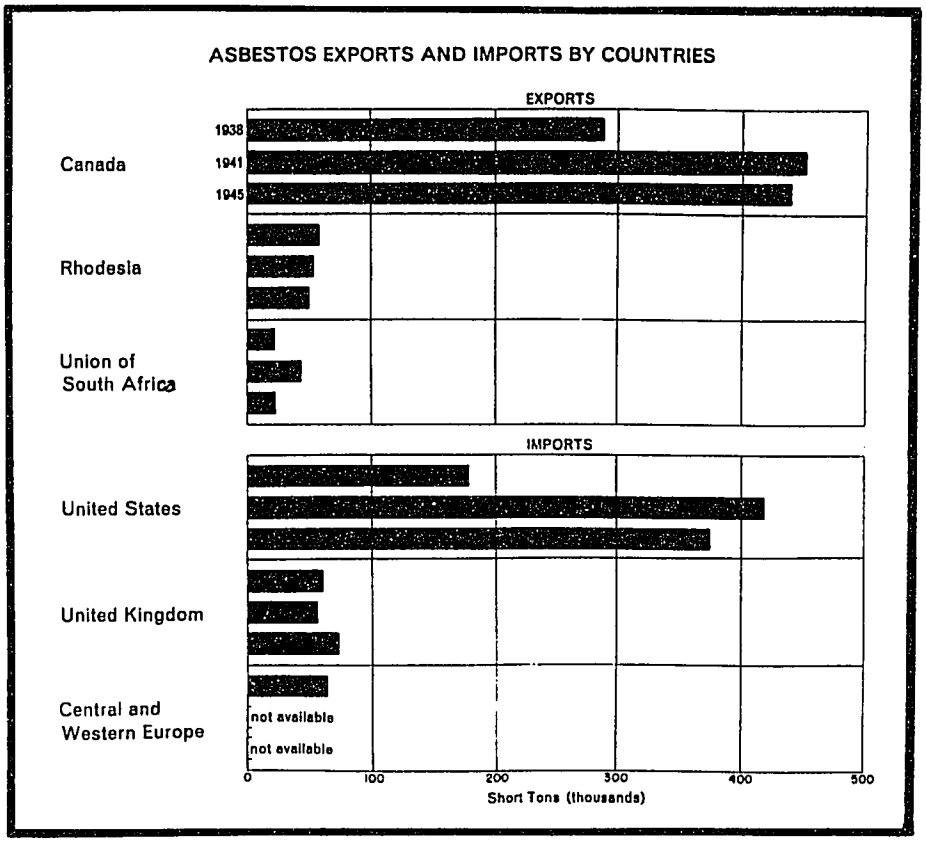

Figure F.1 Asbestos exports and imports by country in 1938, 1941, and 1945.

Source: Bureau of Mines. 1950 Materials Survey on Asbestos. (Washington, D. C.: GPO, 1952): IX-1

APPENDIX F

U.S. ASBESTOS CONSUMPTION

Table F.1 Allocation of Strategic Grades of Asbestos by End Use, in 1944

Canadian Spinning Fibers (Chrysotile)	Percent
Woven brake linings and clutch facings	30
Mechanical packings and gaskets	19
Navy cable filler	18
Navy lagging cloth	13
Maintenance and repair of other than electrical	4
Asbestos safety clothing	4
Aircraft	3
Asbestos yarn in flexible metal tubing	1
Miscellaneous textile uses	8
	100
Rhodesian C. & G. Nos. 1 and 2 (Crocidolite)	
Navy cable insulation	65
Electrical equipment new and maintenance	25
Laminated plastics	10
	100
African Amosite	
Navy felt insulation (lightweight blankets)	39
Molded amosite insulation	25
85% magnesia and other high temp. insulation	22
Marine insulating board	11
Sprayed insulation	3
	100

Source: U.S. Bureau of Mines, 1950 Materials Survey on Asbestos, National Security Resources Board (Washington, D. C.: GPO, 1952) XII-9.

Table F.2 Annual Imports to the U.S., 1935-1950

Short Tons			
1935	166,585	1943	440,255
1936	243,602	1944	383,049
1937	307,188	1945	374,354
1938	179,490	1946	456,688
1939	242,561	1947	594,839
1940	246,613	1948	647,881
1941	419,196	1949	509,366
1942	419,242	1950	705,253

Table F.3 Asbestos Production, 1943-1950

	Metric Tons				
	U.S.	World		U.S.	World
1943	5,456	633,800	1947	21,804	872,000
1944	6,048	602,000	1948	33,649	995,000
1945	11,091	632,000	1949	39,360	895,000
1946	12,769	724,000	1950	38,495	1,206,000

Source: U.S. Bureau of Mines, 1950 Materials Survey on Asbestos (Washington, D. C.: GPO, 1952) IX-6.

Table F.4 U.S. Asbestos Consumption, 1971-1981

	Metric Tons		
	Domestic Production	Imports	Apparent Consumption
1971	119,000	620,000	699,000
1972	12,000	670,000	748,000
1973	137,000	721,000	804,000
1974	103,000	706,000	779,000
1975	91,000	523,000	572,000
1976	104,000	596,000	659,000
1977	92,256	550,693	609,157
1978	93,097	570,000	618,706
1979	93,354	513,084	560,600
1980	80,079	327,296	358,700
1981	75,618	337,618	348,800

Source: Melvin A. Benarde, ed. Asbestos: The Hazardous Fiber, (Boca Raton, Florida: CRC Press, 1990) 31. Benarde notes that figures may not total due to rounding and deposits/withdrawals from stock piles.

Table F.5 Asbestos reprocessing plants in New Jersey in 1968

Company	Location	
Friction Materials		
Johns-Manville Corp.	Manville	
Raybestos-Manhattan	Newark	
Reddaway Manufacturing	Passaic	
H. K. Porter Co., Inc.	Trenton	
Asbestos Cement Products		
Johns-Manville Corp.	Manville	
National Gypsum Co.	Millington	
Philip Carey Manufacturing Co.	Perth Amboy	
GAF Corp.	So. Bound Brook	
U.S. Plywood-Champion Papers, Inc.	South River	
Floor Tile		
Congoleum-Nairn, Inc.	Kearny	
Johns-Manville Corp.	Manville	
American Builtrote Rubber Co.,, Inc.	Trenton	
Other A=Textiles, B=Paper, C=Miscellaneous		
Howard Industries	Berkley Hgts	
Asbestos Corp.	Bloomfield	A
Kavon Filter Products Co.	Cranford	C
Janos Asbestos Co.	E. Rutherford	ABC
GAF Corp.	Gloucester City	B
Columbia Filter Co.	Hawthorne	C
Cellulo Co.	Hoboken	C
Imperial Products Co.	Hoboken	
Ladden Asbestos Corp. of New Jersey	Irvington	AB
Smyth Rubber & Packing Co.	Jersey City	C
Smith & Kanzler Corp.	Linden	BC
Johns-Manville Corp.	Manville	BC
Flaherty-Kennedy Filter Fabrics	Maplewood	C
Asbestos Products Manufacturing Corp.	Newark	C
Asbestospray Corp.	Newark	C
Johns-Manville Corp.	New Brunswick	C
Electrical Insulation Sales Co.	North Bergen	C
Brassbestos Manufacturing Corp.	Paterson	
La Favorite Rubber Manufacturing Co.	Paterson	C
J. T. Baker Chemical Co.	Phillipsburg	
Minerals & Insulation Co., Inc.	Rochelle Park	C
Baldwin-Ehret, Inc.	Trenton	C

Source: Rajhans, Gyan S., and Gordon M. Bragg. Engineering Aspects of Asbestos Dust Control. (Ann Arbor, Michigan: Ann Arbor Science Publishers, Inc., 1978): 176-182.

APPENDIX G

REGULATION OF ASBESTOS

G.1 Principal Federal Agencies

G.1.1 OSHA

The Occupational Safety and Health Administration in the Department of Labor is responsible for administering the OSHAct. The agency regulates matters of health and safety in occupational settings, and sets standards and permissible exposure limits for worker exposure.

G.1.2 EPA

The Environmental Protection Agency enforces public health and environmental safety laws. EPA regulates the handling, hauling, and disposal of toxic substances in air, water, and land, under a number of provisions, including the Toxic Substances Control Act (TSCA), RCRA, and CERCLA.

Under the Asbestos Hazard Emergency Control Act, EPA regulates the asbestos-in-schools programs, to control and eliminate exposure to friable asbestos in the nation's elementary and secondary school buildings.

G.1.3 CPSC

The Consumer Product Safety Commission regulates asbestos in consumer products, under the Consumer Product Safety Act, and the Federal Hazardous Substance Act.

G.1.4 MSHA

The Mine and Safety Health Administration regulates mining and milling of asbestos, and approves respirators.

G.1.5 FDA

The Food and Drug Administration is responsible for preventing asbestos contamination in food, drugs, and cosmetics, which might occur during the manufacturing process.

Sources: Environmental Protection Agency, Asbestos Fact Book (Washington, D. C.: GPO, Feb., 1985) 3.

Asbestos Exposed: The Inside Story (Philadelphia, Pa.: Asbestos Victims Special Trust Fund, 1992) 20.

G.2 Regulations Governing Asbestos

G.2.1 OSHA

Construction Industry Standard for Asbestos
29 CFR 1926.1101 (Previously 29 CFR 1926.58).

General Industry Standard for Asbestos
29 CFR 1910.1001

Maritime Industry Standard for Asbestos
29 CFR 1915.1001

Respiratory Protection Standard
29 CFR 1910.134

G.2.2 EPA

Worker Protection Rule
40 CFR 763 Subpart G

Asbestos Hazard Emergency Response Act (AHERA)
40 CFR 763 Subpart E

National Emission Standard for
Hazardous Air Pollutants (NESHAP)
40 CFR 61 Subpart M

G.2 **Regulations Governing Asbestos** (continued)

NESHAP The National Emission Standard for Hazardous Air Pollutants prohibits visible emissions to the outside air. NESHAP applies to demolition and renovation projects that disturb more than 260 ln.ft. or 160 sq.ft. of ACM over a one year period.

RCRA The Resource Conservation and Recovery Act regulates the handling, manifesting, and disposal of solid waste in landfills. Some states classify ACM waste as hazardous waste under RCRA.

CERCLA The Comprehensive Environmental Response, Compensation, and Liability Act Recovery Act, or "Superfund" Laws, classifies friable asbestos as a hazardous substance.

Source: EPA, "Managing Asbestos in Place: A Building Owner's Guide to Operations and Maintenance Programs for Asbestos-Containing Materials (Washington, D. C.: GPO, July 1990): 26-30.

APPENDIX H

SELECTED EPA PUBLICATIONS for ACM

1979 March. Orange Book.
"Asbestos-Containing Materials in School Buildings: A Guidance Document."

Part 1 outlines steps schools can take to conduct an asbestos control program.

Part 2 addresses school personnel, contractors and others, with emphasis on inspection and control work.

1983 March. Blue Book
"Guidance for Controlling Friable Asbestos-Containing Materials in Buildings"

1985 June. Purple Book
"Guidance for Controlling Asbestos-Containing Materials in Buildings"

1985 October. Pink Book.
"Asbestos in Buildings: Simplified Sampling Scheme for Friable Surfacing Materials."

1985 Silver Book
"Measuring airborne asbestos following an abatement action."

1986 June. "Guidance for Preventing Asbestos Disease Among Auto Mechanics."

1990 July.
"Managing Asbestos in Place: A Building Owner's Guide to Operations and Maintenance Programs for Asbestos-Containing Materials."

B.2 CONSUMER PRODUCT LISTING

This list is a selection of consumer products suspected of containing asbestos by the Consumer Product Safety Commission. Not all brands or models of these products necessarily contain asbestos.

ASBESTOS PAPER PRODUCTS

Acoustical ceiling tile

Lamp sockets

Burner mats for gas stoves

Roofing felt (outer layer)

Pipe and boiler covering

Vinyl sheet flooring backing

Radiator top insulation

Appliance heating shielding (paper)
- Slow cookers
- Hair dryers

Paper sheets for heat insulation

Millboard
- TV and other electronic switch plates
- Electric switch boxes
- Metal reinforced gaskets (for air-cooked engines)
- Electrical washers
- Linings for ovens, kilns, safes, safety boxes, incinerators
- Millboard sheet
  - Wall protection behind heat-generating products
  - Floor protection under wood and coal stoves

Soldering and welding blocks

Iron rests

Appliance heat shielding (millboard)
- Toasters
- Rotisserie broilers

Fireproof wallboard

Metal-clad fire doors and partitions

Tent garments

Stove pipe rings

CLOTH AND WOVEN PRODUCTS

Appliance wiring
- Barbecue fire starters
- Broilers
- Curling irons
- Electric blankets
- Hair dryers
- Heating pads
- Ranges
- Slow cookers
- Toasters
- Irons
- Deep fat fryers
- Electric fry pans

Awnings

Candlesticks

Catalytic heater mantles

Cigarette lighter wicks

Cord

Seals for high temperature gaskets

Valve steam packings

Insulation for glass handling tools

Reinforcing for braided wall stem hose

Theater curtains

Felt
- Reinforcements in plastics
- Gaskets
- Reinforcements in asbestos tapes
- Secondary insulation in high-temperature wire and cable

Asphalt impregnated

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- Secondary insulation in high-temperature wire and cable
- Asphalt impregnated roofing felts
- Piano and organ felts

Flexible air conductors for heating, cooking and ventilation equipment

Heating pads (element insulation)

Ironing board pads and covers

Lamp and lantern mantles

Pipe and boiler covering

Pot holders and oven mitts

Flame resistant garments
- Gloves
- Hats
- Helmets
- Hoods
- Mittens
- Overgaiters
- Sleeves
- Suits
- Umbrellas
- Aprons
- Arm protection
- Flame-resistant blankets
- Boots
- Caps
- Smokers' bibs

Stoves-Coal and wood burning

Tape for pipe insulation

Braid and rope for packing

Motion picture screens

Tent grommets

ASBESTOS CEMENT PRODUCTS

Water, sewer and septic drain field pipe

Airduct pipe

Sheet products
- Roofing clapboard
- Siding
- Shingles
- Interior walls
- Boiler and furnace baffles
- Bulk sheeting
- Welding shields
- Baking sheets
- Blackboards
- Laboratory table tops
- Linings for vaults, safes, humidifiers and filing cabinets

VARIOUS MATRIX PRODUCTS

Adhesives (glues and epoxies)

Air duct cement for asbestos-cement air duct

Buffing and polishing compounds

Caulks and putties

Floor tile cement & mastic

Auto body filler

Flashing cement

Furnace cement

Glazing compound for ceramics

Pipe & boiler coverings

Roof & driveway coatings

Stains & varnishes

Automotive undercoating

Refrigerant cements

Automotive muffler repair compounds

PRODUCTS SUBJECT TO INADVERTENT ASBESTOS CONTAMINATION

Driveway gravel

Fertilizer & lawn care products

Potting materials (vermiculite)

Playsand

Talcs for noncosmetic or food use applications

MISCELLANEOUS PRODUCTS

Acoustical & thermal insulation material, sprayed

Ammunition shell wadding

Automotive mufflers

Barbecue firebed materials in gas barbecue grills

Boat hull repair kits

Flower pots

Friction materials
- Clutch plates
- Brake linings

Potter's kilns

Reinforcement in molded plastic & rubber

Automotive radiator sealant

Pottery clay

Powder (asbestos)

Bulk Fiber

Vinyl asbestos floor tiles

Abrasive wheels

Aerial distress flares

Molded plastics & phenolic laminates

Paints

Textured paint

Cement, drywall & plaster patching compounds

Artificial gas fireplace emberizing material

Phonograph records

CONSUMER PRODUCTS POSSIBLY CONTAINING ASBESTOS

APPLIANCES
- Air conditioners
- Dishwashers
- Hand-held mixers
- Portable electric heaters
- Popcorn poppers
- Refrigerators
- Vacuum cleaners
- Waffle makers

MISCELLANEOUS PRODUCTS
- Carpet padding
- Fireplaces
- Instant papier mache
- Light fixtures on railroad passenger cars
- Welding masks
- File cabinets

Source: "Asbestos Exposed: The Inside Story." (Philadelphia, Pa.: Asbestos Victims Special Trust Fund, 1992): 37-39.

APPENDIX J

Lifetime Risk Values for Selected Situations

Selected risk situations, mainly U.S. data[269,272]	Lifetime risk per 100,000
Extra high risk	
Smoking (all causes of death)	21,900
Smoking (cancer only)	8,800
High risk	
Motor vehicle, U.S., 1975 (deaths)	1,600
Elevated risk	
Frequent airline passenger (deaths)	730
Cirrhosis of liver, moderate drinker (deaths)	290
Motor accidents, pedestrians, U.S., 1975 (deaths)	290
Skiing, 40 hours per year (deaths)	220
Moderate risk	
Light drinker, one beer per day (cancer)	150
Drowning deaths, all recreational causes	140
Air pollution, U.S., benzo(a)pyrene (cancer)	110
Natural background radiation, sea level (cancer)	110
Frequent airline passenger, cosmic rays (cancer)	110
Low risk	
Home accidents, U.S., 1975 (deaths)	88
Cycling (deaths)	75
Person sharing room with smoker (cancer)	75
Diagnostic x-rays, U.S. (cancer)	75
(Risk level where few would commit their own resources to reduce risk; Royal Society, London, 1983), (270)	70
Very Low risk	
Person living in brick building, additional natural radiation (cancer)	35
Vaccination for small pox, per occasion (death)	22
One transcontinental air flight per year (death)	22
Saccharin, average U.S. consumption (cancer)	15
Consuming Miami or New Orleans drinking water (cancer)	7
(Risk level where very few would consider action necessary, unless clear causal links with consumer products, Royal Society, London, 1983), (270)	7
Extremely low "rare-event" risk	
One transcontinental air flight per year, natural radiation (cancer)	4
Lightning (deaths)	3
Hurricane (deaths)	3
Charcoal broiled steak, one per week (cancer)	3
ENVIRONMENTAL ASBESTOS RISK," 1985, (cancer) ("around one per 100,000 or lower"; this Report)	1

Source: Benarde, Melvin A., Ed. Asbestos: The Hazardous Fiber. (Boca Raton, Florida: CRC Press, 1990): 69.

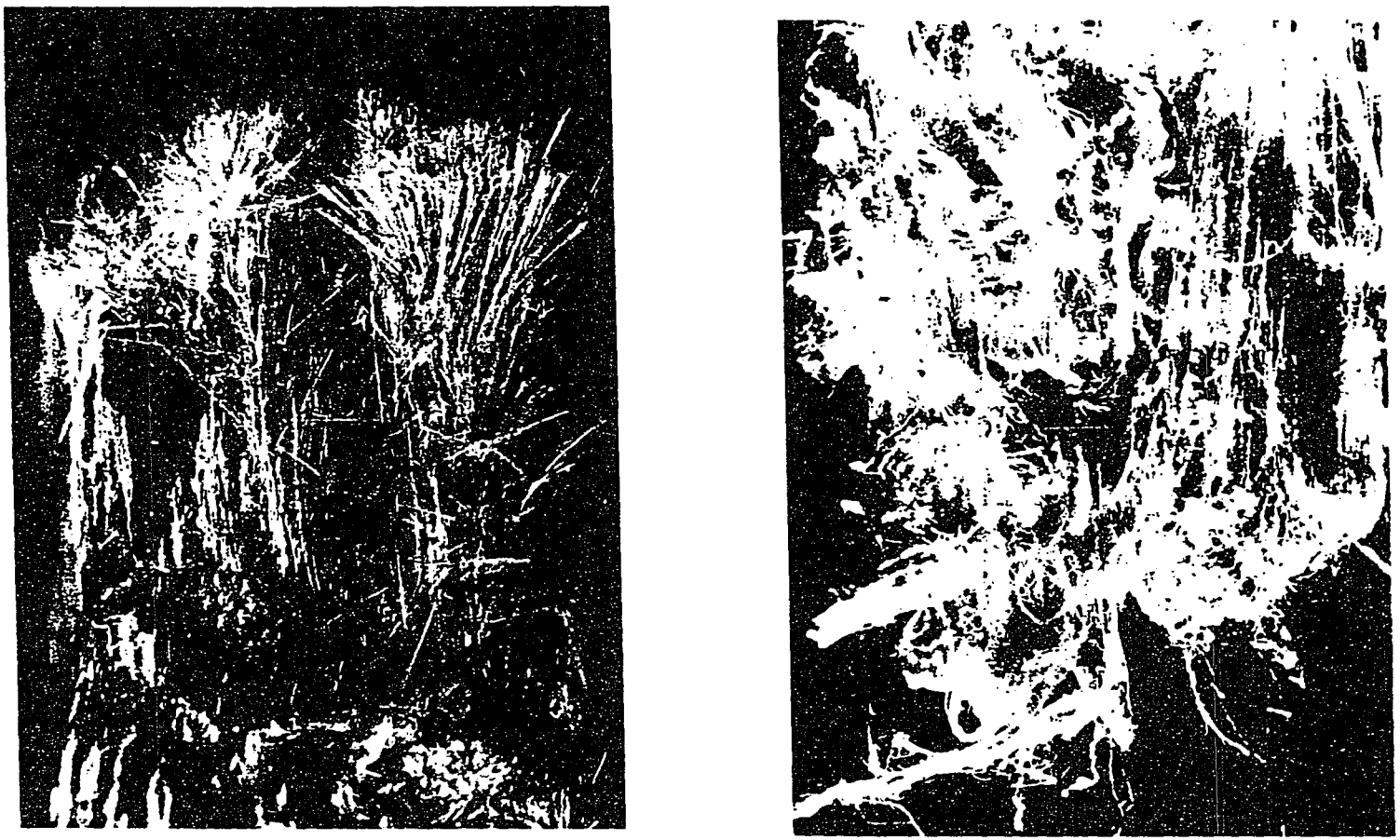


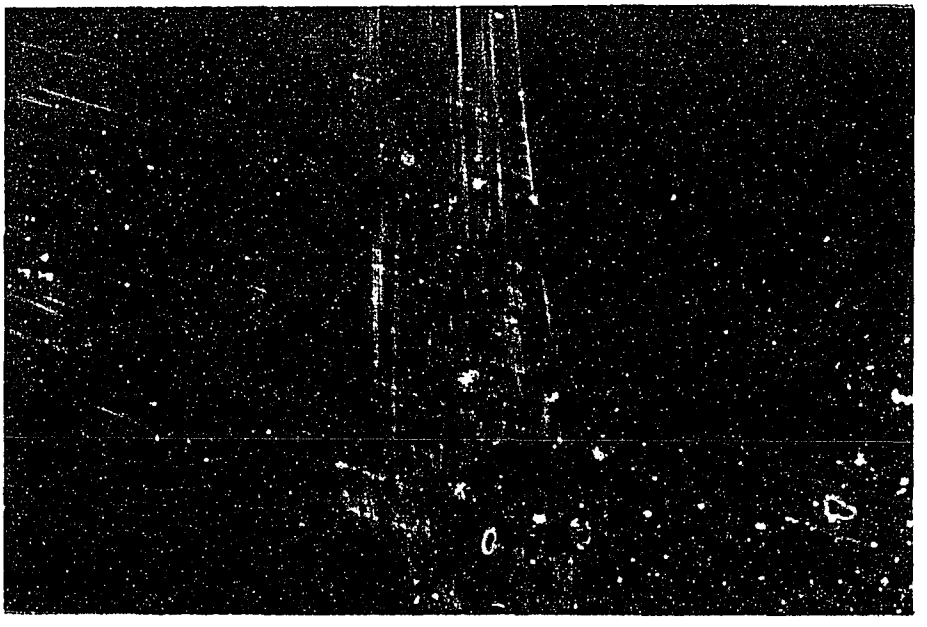
Figure K.1 In their natural, raw state, fibers typical of amosite (left) are straight and rigid; fibers typical of chrysotile are curly and silky.

Source: Bernarde, Melvin A., Ed. Asbestos: The Hazardous Fiber. (Boca Raton, Florida: CRC Press, 1990): 46-47.

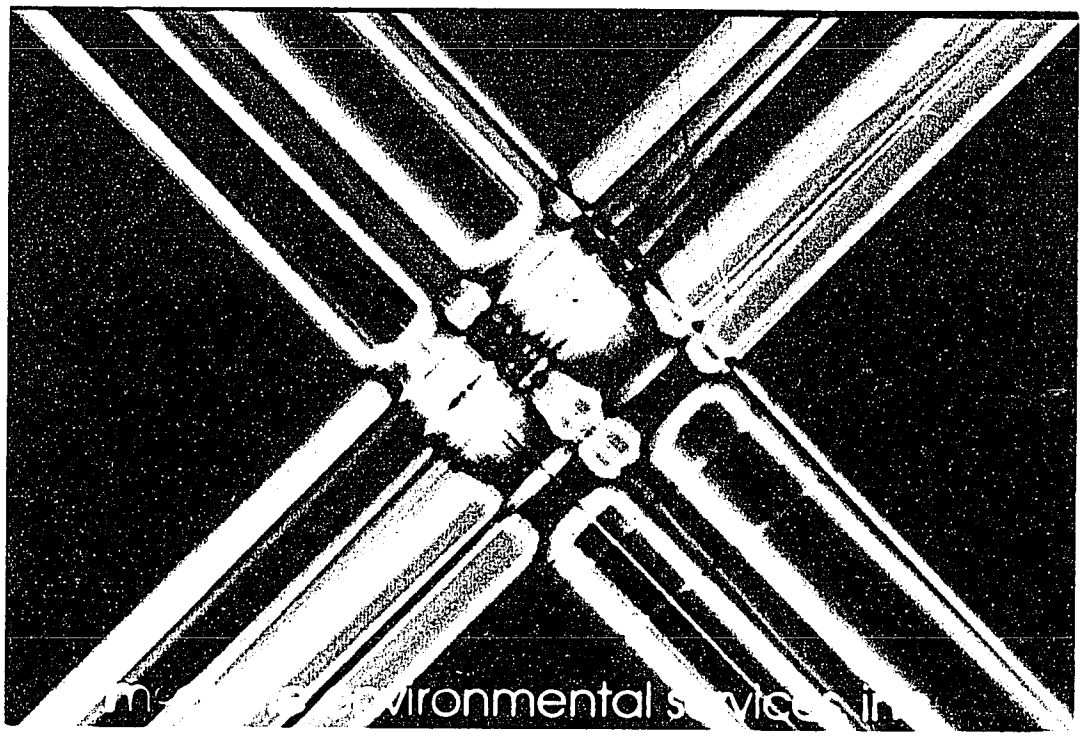
APPENDIX K

PHOTOGRAPHS

Microscopic Identification of Chrysotile and Amosite Fibers

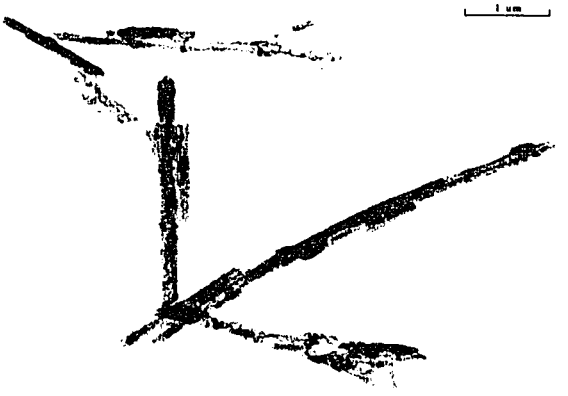


Chrysotile
Dispersion Staining
Refractive Index Liquid-1.550
Total Magnification-148X

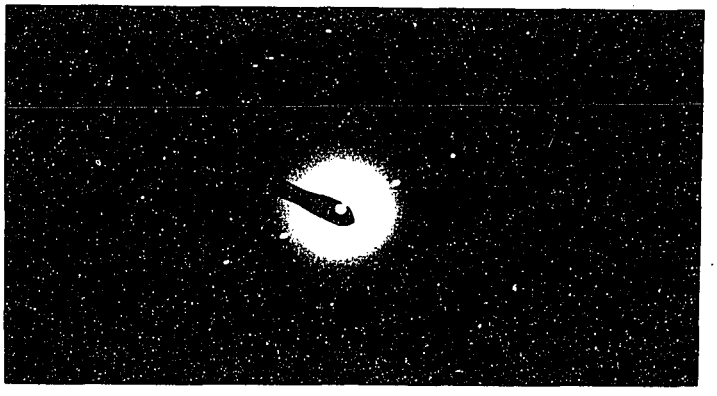


Chrysotile
Crossed Polars with
First Order Red Plate
Total Magnification-155X

Photo by Thomas J. Hopen

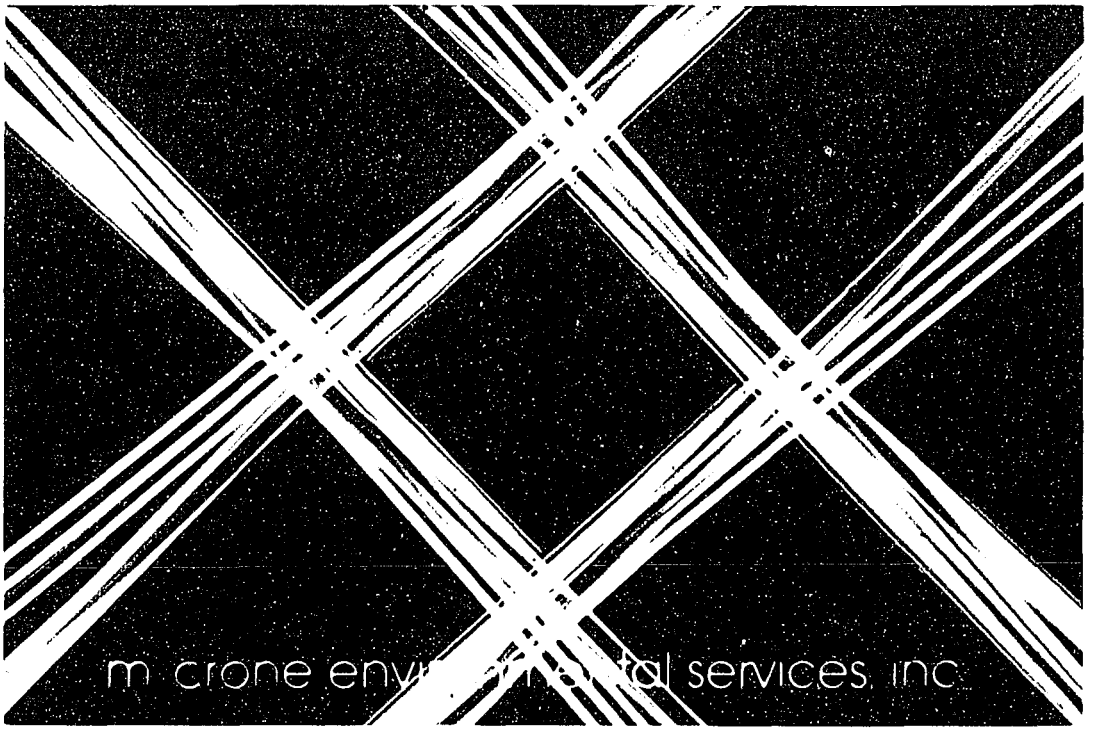


mccrone environmental services, inc.



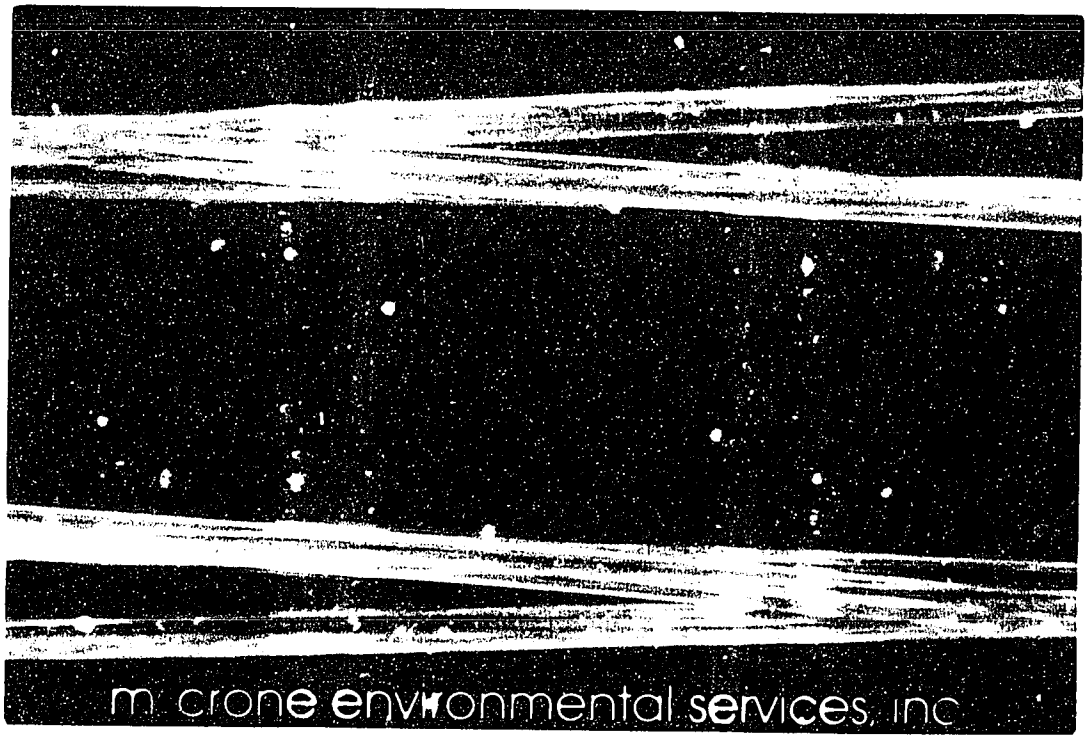
Chrysotile Asbestos
AHERA Cluster with Selected Area Electron Diffraction Pattern
(Camera Constant = 64.76 mmA)

Photo by Steve Burris



Amosite
Crossed Polars
Total Magnification-155X

Photo by Thomas J. Hopen



Amosite
Central Stop
Dispersion Staining
Refractive Index Liquid-1.680
Total Magnification-155X

Photo by Thomas J. Hopen

Color Photographs Courtesy of McCrone Environmental Services, Inc., 850 Pasquinelli Drive, Westmont, Ill. 60559.

APPENDIX L

AN ASBESTOS CHRONOLOGY

1760	The Industrial Revolution begins in England.
1776	The signing of the Declaration of Independence establishes the United States of America.
1791	The New England textile industry begins with a cotton spinning plant in Pawtucket, Rhode Island.
1858	Henry Ward Johns moves to Brooklyn, and starts a small roofing materials business.
1861	The Civil War begins.
1866	Asbestos fiber mixed with sodium silicate was first used as an insulation material.
1869	May 10. The first transcontinental railroad is established.
1870s	Mining of chrysotile begins in the Italian Alps.
	Asbestos coverings for insulating boilers, and asbestos-cement products were introduced.
1871	The first asbestos textile factory opens in England.
1876	Mining of chrysotile begins in Canada.
1883	The Salamander Association of Boiler and Pipe Fitters, the first union of absestos workers, was formed in New York City, N.Y.
1885	Eighty-five percent magnesia insulation using asbestos fiber as a binder was introduced.
1890	The H. W. Johns Manufacturing Company obtains its own mine in Canada.
1891	Mining of the first deposits of Cape blue crocidolite, discovered by a German geologist named Lichtenstein, near Prieska, South Africa.
1891	The H. W. Johns Manufacturing Company becomes the largest asbestos manufacturer and dealer in the world.
1896	The first asbestos textile mill opens in the United States.

1898 Johns dies from scarring of the lungs.

The Annual Report of Her Majesty's Women Inspectors cites asbestos textile work, and describes the ill effects of the dust.

Corrugated asbestos paper for air-cell coverings is introduced.

1900 Murray's post-mortem description of the lungs of a thirty-three year old asbestos textile worker documents the first case of death from asbestosis.

1901 Manville buys out the H. W. Johns Manufacturing Company to form the new Johns-Manville Company.

1906 The first study of mortality among asbestos workers appears in an article in the Bulletin de l'Inspection du Travail et de l'Hygiene Industrielle.

1907 Amosite discovered in the Transvaal, South Africa. Large scale extraction begins in 1908.

Murray reports the first asbestos-related death in England.

Brake shoes made with asbestos introduced.

The Hours of Service Act regulates working conditions nationally for the first time.

1912 Johns-Manville begins a complex of asbestos factories, establishing the town that bears half its name - Manville, N. J.

1913 The Department of Labor is formed to "foster, promote and develop" the lot of wage workers.

1914 The Public Health Service's Division of Occupational Health is formed.

1917 The United States enters World War I.

The first published report of asbestos-related disease in the United States, by Pancoast.

1918 The U. S. Bureau of Labor Statistics publishes a special study conducted by Frederick Hoffman, chief actuary for the Prudential Insurance Company of America.

1918 Formal founding of Harvard University's School of Public Health. The Drinker brothers develop one of the first comprehensive programs in industrial hygiene.

1919 Wisconsin enacts worker's compensation legislation covering asbestosis.

1921 Lighter weight amosite is used in insulation instead of chrysotile to reduce the weight of naval vessels, as called for in the Washington Treaty of Limitations in Tonnage.

1924 July 24. The first clear case of death due to asbestos exposure is described by W. E. Cooke in the British Medical Journal.

1927 W. E. Cooke coins the name asbestosis to describe scarring of the lungs caused by inhalation of asbestos fibers.

The first disability claim for asbestosis is upheld by the Massachusetts Industrial Accident Board.

Gardner becomes Director of the Saranac Laboratories.

1928 Seiler's reports a case of "pure" asbestosis from South Africa, which prompts a study of hundreds of asbestos textile workers in England, conducted by Merewether, Chief Inspector of Factories.

1929 The Raybestos Company, and the Manhattan Rubber Company consolidate to become Raybestos-Manhattan, the country's largest manufacturer of friction products.

1929 Lanza begins a study for the Metropolitan Life Insurance Company. Completed in 1931, the study is published four years later, by the Public Health Service.

1930 April. Construction begins on the Gauley Bridge tunnel.

Minnesota Medicine reports the first case of asbestosis found at autopsy in the United States.

Merewether and Price's report on asbestos and occupational health is published in England. Legislation on workplace conditions is enacted the following year, when asbestosis becomes a compensable disease.

1930 Industrial medicine has emerged as a distinct specialty, but industrial hygiene is not yet recognized as a specialized discipline.

1931-32 Limpet, the first spray applied asbestos containing product, is developed in England.

1933 Major asbestos producers settle compensation claims filed by contract insulation workers.

Pancoast testifies as an expert witness for the defense concerning Gauley Bridge in the case of Raymond Johnson v. Rinehart and Dennis and E. J. Perkins, which came to trial in March.

1934 Amosite felt is developed.

1935 January 4. Lanza's study is published by the Public Health Service.

The Mellon Institute starts the Air Hygiene Foundation, at industry's request, in response to the "silicosis problem." The name was changed to the Industrial Hygiene Foundation (IHF) in 1941.

The spray-on application of asbestos for decorative finishes is introduced in the United States.

1936 Summer. Courses in industrial hygiene, sponsored by the Public Health Service, lead to formation of the American Conference of Governmental Hygienists.

1938 Lanza's textbook, Silicosis and Asbestosis, is published.

1938 The Public Health Service has organized industrial hygiene units in twenty-eight states.

The ACGIH splits from the American Industrial Hygiene Association (AIHA).

A study by Dreessen is published as a Public Health Service bulletin. It becomes the basis for the exposure level recommended by the ACGIH.

1938 German physicians are calling lung cancer an occupational disease of asbestos workers.

1941 The United States enters World War II.

The AGCIH's Committee on Threshold Limits is formally established.

1941 UNARCO begins operation of its plant in Paterson, New Jersey, to supply insulation for naval vessels.

1942 January 20. Government restrictions are enacted to insure an adequate supply of strategic types and grades of asbestos for the war effort.

1943-45 Eight states enact worker's compensation legislation to cover asbestosis.

1944 Gardner begins the study of Kaylo for the Owens-Illinois Glass Company.

1945 August. All restrictive orders imposed on asbestos during World War II are lifted by the end of the month.

1946 The TLV Committee approves the addition of asbestos to the MAC list, and begins publishing an annual report.

Drinker's study of naval shipyards is published.

1947 Hemeon's study of ten asbestos textile plants is completed, but never published.

1949 Merewether's study on asbestos and lung cancer is published in England.

1950 February. The Third International Conference of Experts on Pneumoconiosis is held in Sydney, Australia.

From 1940-1950, the demand for asbestos increased by two hundred percent.

1951 The Lancet publishes Gloyne's study on lung cancer and asbestosis.

1952 September. The Seventh Saranac Symposium is held but, unlike the preceding six, its proceedings are never published.

1953 Selikoff establishes a medical clinic in Paterson, New Jersey.

1954 UNARCO closes its Paterson plant and begins a similar operation in Tyler, Texas.

1955 Richard Doll's study in England shows high rates of lung cancer among asbestos workers.

September. The study on Kaylo is published.

1958 The Chase Manhattan Bank building in New York City is the first modern skyscraper to use spray-on fireproofing containing asbestos.

1960 Wagner's report of thirty-three cases of mesothelioma in South Africa.

1962 UNARCO sells its Tyler, Texas plant to Pittsburgh Corning, and leaves the asbestos business.

1963 ACGIH maximum allowable concentrations (MACs) are renamed threshold limit values (TLVs)

1964 Selikoff's seminal study of insulation workers.

The New York Academy of Science's International Conference on the "Biological Effects of Asbestos" is held at the Waldorf-Astoria in New York City.

1967 Asbestos production increased to four million tons per year, from fifty tons in 1877.

1968 ACGIH issues a Notice of Intended Change which includes special consideration for crocidolite. The proposal is dropped, but is reconsidered at a later date.

1969 Under provisions of the Walsh-Healy Public Contracts Act, a federal standard of 12 f/cc is enacted.

1970 The Asbestos Information Association is formed.

President Nixon signs the Occupational Safety and Health Act on December 29.

Seventy percent of the world's asbestos supply is used in construction products.

1971 March. EPA lists asbestos as a toxic air pollutant under NESHAP.

1971 April 28. The OSHAct becomes effective, and items on the TLV list become enforceable under federal law. The TLV for asbestos, remains at 5 mppcf.

May 29. The first consensus standard adopted under the OSHAct sets an exposure level of 12 f/cc.

October. NIOSH inspects the Tyler, Texas plant.

The AFL-CIO petitions OSHA to reduce the exposure level to 2 f/cc.

1971 December 7. OSHA issues an emergency temporary standard (ETS) of 5 f/cc, and a peak exposure of 10 f/cc.

1972 February 3. Pittsburgh Corning closes the Tyler, Texas plant.

February 15. The New York City Council bans all spraying of asbestos.

June 7. OSHA's first comprehensive health standard for asbestos sets the permissible exposure limit (PEL) at 5 f/cc, with a ceiling limit of 10 f/cc.

1973 April. NESHAP is revised to include the "no visible emissions" standard. The use of spray-on asbestos material for thermal, insulating, or fireproofing purposes is banned.

Asbestos consumption in the United States reaches a high point, exceeding 800,000 tons.

1974 ACGIH lists the TLV for asbestos at 5 f/cc.

1975 October 9. A proposal by OSHA to reduce the PEL to 0.5 f/cc is withdrawn because of the Supreme Court's decision on benzene.

Collection and disposal of ACM are included under the NESHAP "no visible emissions" standard.

Three thousand known commercial uses have been developed for asbestos.

1976 July 1. The 2 f/cc PEL becomes effective, as called for in the 1972 standard.

1977 Feb. The CPSC is petitioned to ban consumer products containing tremolite talc. Dry patching compounds are banned. The ban requested on other products, including play sand, is denied in 1981.

1978 The EPA bans the use of spray-on asbestos for decorative purposes. The "no visible emissions" standard is extended to require removal of ACM prior to demolition.

April 26. Health, Education, and Welfare Secretary Joseph A. Califano, Jr. orders the Surgeon General to send an advisory letter describing the health risks of exposure to the nation's 400,000 physicians, and urges those who are or were exposed to asbestos to stop smoking cigarettes.

1978 June. The New York Academy of Science's second major conference, the "Health Hazards of Asbestos Exposure," is held at the New York Hilton.

Total employment at six mine and mill operations in the United States is four hundred.

Total world production of all grades and varieties of asbestos is estimated at 5.6 million metric tons.

1979 March. The Technical Assistance Program is established, which includes a Regional Asbestos Coordinator for each of the EPA's ten regions.

EPA issues the two-part "Orange Book," a guidance document for handling ACM in the schools.

CPSC negotiates with manufacturers to voluntarily remove asbestos materials from hair dryers.

1980 September. EPA issues a proposed rule on the identification and notification of friable ACM in schools.

From 1900 to 1980, thirty-six million metric tons of asbestos were used, and some thirty million tons was put in place.

1982 June. EPA's final Identification and Notification Rule affects public and private, elementary and secondary schools nationwide.

UNARCO (name changed to UNR) files for bankruptcy.

1982 August 26. The Manville Corporation files for reorganization under Chapter 11, after changing the company's name and moving its headquarters to Denver, Colorado. Johns-Manville became a subsidiary against which more than eleven thousand lawsuits were filed between 1980 and 1982.

Asbestos consumption drops to 300,000 tons.

1983 Feb. EPA reports to Congress on ACM in buildings.

Manville sues the government for reimbursement of health claims paid to former shipyard workers, whose claims comprise half of those against the company.

1983 June 28. The deadline for schools to meet EPA's Friable Asbestos-Containing Materials in Schools: Identification and Notification Rule.

1983 November 4. OSHA publishes an ETS of 0.5 f/cc, which is challenged by the industry and subsequently overturned in Federal District Court, in March, 1984.

1984 April 10. OSHA publishes a proposed rule covering all industries governed by the OSHAct (maritime, construction, and general industry).

August 11. President Reagan signs the Asbestos School Hazard Abatement Act (ASHAA), a loan and grant program to help schools eliminate asbestos hazards.

November 4. OSHA issues an ETS of 0.5 f/cc and permits the use of respirators. A court decision, answering a challenge by the AIA, reverts the PEL to 2 f/cc.

December. The Asbestos Action Program is established in the EPA's Office of Pesticides and Toxic Substances.

1985 January. EPA establishes the first supervisory level training centers for training and certification of asbestos workers.

June. EPA issues the "Purple Book," a guidance document for ACM in buildings.

Approximately two thousand tons of asbestos per year is mined and processed in the United States.

1986 January. A proposed Ban and Phase Down Rule is issued by EPA.

June 20. OSHA publishes new regulations for general industry and for the construction industry. The PEL is reduced to 0.2 f/cc and an action level of 0.1 f/cc is established. The effective date is July 21.

1986 October 22. President Reagan signs Public Law 99-519, the Asbestos Hazard Emergency Response Act, which includes standards for training asbestos abatement workers.

The Johns-Manville complex in Manville, N. J. is closed. At peak production, almost five thousand workers were employed to manufacture fire-proof products made with asbestos.

1988 February. EPA reports to Congress on ACM in public buildings.

1988 October 12. This original deadline for schools to complete inspections and submit management plans for AHERA is extended until May 9, 1989.

Manville emerges from bankruptcy.

1989 July 9. Deadline for implementation of AHERA.

July 12. EPA's Asbestos Ban and Phaseout Rule would ban ACM products by 1997. The rule is challenged by the AIA, and overturned by the Fifth Circuit Court of Appeals in October, 1991.

Dec. 14. AHERA requires TEM analysis to establish final clearance levels for all abatement projects.

1990 Sept. 10. Fire destroys Building A at the empty Johns-Manville plant in Manville, N. J.

1991 March 6. EPA issues "An Advisory on Asbestos in Buildings."

1994 February 2. The AFL-CIO challenges the 0.2 f/cc PEL, which is upheld by the court.

August 10. OSHA issues a final rule for occupational exposure to asbestos, setting the PEL at 0.1 f/cc. The effective date is October 11, 1994.

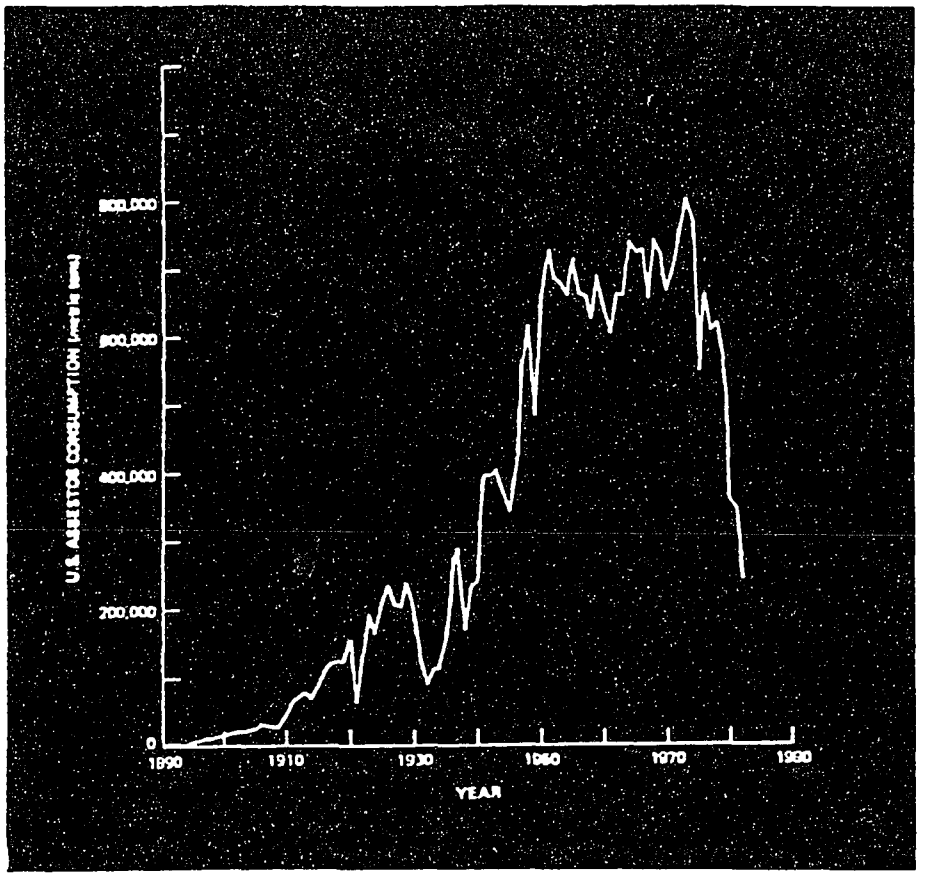


Figure L.1 Trend of Asbestos Use, 1890 to 1980, in metric tons.

Source: Benarde, Melvin A., Ed. Asbestos: The Hazardous Fiber. (Boca Raton, Florida: CRC Press, 1990): 10.

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