As the Asbestos Crumbles: A Look at New Evidentiary Issues in Asbestos-Related Property Damage Litigations

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NOTE

AS THE ASBESTOS CRUMBLES: A LOOK AT NEW EVIDENTIARY ISSUES IN ASBESTOS-RELATED PROPERTY DAMAGE RELATED LITIGATIONS

I. INTRODUCTION

It may be helpful to think of asbestos removal as akin to slicing a pie. As a pie sits in its tin it may look or smell perfect, but attempts to remove a slice are far more difficult than would first appear. The results are often oozing fruit and crumbling crust; the pie is no longer in its undisturbed pristine state. Quite similarly, removing asbestos from its resting place is also far more difficult than it appears. The abatement of asbestos, however, poses severe health risks from potential cancer causing fibers that can be released into the atmosphere during removal or disturbance.

The scientific community agrees that asbestos is a health hazard that must be severely restricted in its use and heavily regulated.


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where use is permitted. Asbestos was shown at the turn of the century to cause asbestosis, and its association with the causation of lung and pleural tumors was demonstrated in the 1950s. Although concerns about the health risks posed by asbestos emerged in the 1930s, it was not until the creation of the Occupational Safety and Health Administration ("OSHA") in 1970 that American asbestos exposure and use limits were set. Since 1970, OSHA and the Environmental Protection Agency ("EPA") have worked to lower asbestos exposure levels to industrial workers and the public at large.

Asbestos has been an important fire retardant since the time of Caesar, and has been employed extensively in the United States. Health Act of 1970, 29 U.S.C. §§ 651-678 (1988). Examples of state regulation include a New York law prohibiting the use of asbestos in cement piping used to convey water, N.Y. EXEC. LAW § 378 (McKinney 1992). But see J. Bignon, Mineral Fibres in the Non-occupational Environment, in NON-OCUPATIONAL EXPOSURE TO MINERAL FIBRES 3-25 (J. Bignon et al. eds., 1989) (calling the EPA ban of asbestos-containing products unnecessary and scientifically unjustifiable).

3. Asbestosis is a form of lung disease, known medically as pneumoconiosis, which is caused by the inhalation of asbestos fibers. Asbestosis is an irreversible non-malignant scarring of lung tissue, which can ultimately prevent the necessary exchange of oxygen and carbon dioxide. RICHARD A. SLOANE, THE SLOANE-DORLAND ANNOTATED MEDICAL-LEGAL DICTIONARY 66-67 (Richard A. Sloane ed., 1987). See generally GEORGE A. PETERS & BARBARA J. PETERS, SOURCEBOOK ON ASBESTOS DISEASES: MEDICAL, LEGAL, AND ENGINEERING ASPECTS B1-B24 (1980) (discussing the medical aspects of asbestos-related diseases).


5. A recommendation for limiting exposure to asbestos in the workplace was made in 1938 by the U.S. Public Health Service. PUBLIC HEALTH SERVICE, supra note 1, at 5. Prior to that time the United Kingdom had already moved to regulate asbestos exposure with the passage of the Asbestos Industry Regulations of 1931, pursuant to the Factory and Workshop Act of 1901. Also, documents as early as 1918 reveal that the Prudential Insurance Company of New York considered refusing to issue life insurance to asbestos workers. SELIKOFF & LEE, supra note 1, at 21.


7. See Joseph Hooper, The Asbestos Mess, N.Y. TIMES, Nov. 25, 1990, § 6 (Magazine) at 38, 39. The earliest known use of asbestos dates to the year 2500 B.C.E., when the mineral was found in Finnish pottery. See SELIKOFF & LEE, supra note 1, at 3. It is also known that the Athenians employed asbestos in the wicks of lamps in the 5th century B.C.E. Id. (citing GEORGIUS AGRICOLA: DE RE METALLICA (H.C. Hoover & L.H. Hoover trans., 1950)). The Roman, Pliny the Younger, near the year 100 C.E., is credited as the first author to employ the word asbestos, using it to comment on the sickliness of asbestos mine workers. See F.D. Pooley, Asbestos Mineralogy, in ASBESTOS-RELATED MALIGNANCY, supra note 6, at 3; see also SELIKOFF & LEE, supra note 1, at 4.

8. Approximately a maximum figure of 800,000 short tons of asbestos were consumed...
As a result of its strength and durability, asbestos found its way into thousands of manufactured products,9 and is still currently used in many diverse products.10 Despite attempts to ban it,11 asbestos remains an important element internationally in construction, and as a friction material.12

The broad use of asbestos in structures since the 1940s and its presence in brake linings and other products, combined with its natural presence in the environment, has resulted in the release of asbestos fibers into urban air.13 Asbestos is not a man-made product, but rather is a naturally occurring fibrous material mined from the earth. Since asbestos is a material that occurs naturally in rock formations, it is virtually everywhere—in the air, in the water, and in the food chain.14 In fact, asbestos fibers are released into the atmosphere virtually every time an automobile or train applies its brakes. These fibers become airborne and are inhaled by urban and suburban dwellers on a daily basis.15 It is not surprising, therefore, that studies have per year in the United States throughout the 1960s and 1970s. PUBLIC HEALTH SERVICE, supra note 1, at 9-13.

9. A partial list of those products include: yarn; thread; felt; rope packing; plain and corrugated paper; rollboard; millboard; insulating wire; 85% magnesia pipe covering; blocks; high temperature insulation; compressed sheet packing; molded composition for electrical and other purposes; molded brake linings; brake blocks; filler in plastics; flooring; pottery; asbestos cement; shingles; siding and tile; asbestos wall tile; flat sheets; corrugated roofing; roof sheathing; panels; insulating board; floor tile backing; pipes; boiler insulation; roofing cement; furnace cement; plaster; stucco; paints; varnishes; acoustical sprayed asbestos; insulation of walls, floors, and mattresses; filter fibers; filter pads; sewer pipe; asphalt floor tile; automobile body coverings; and even gas mask filters. See SELIKOFF & LEE, supra note 1, at 19.

10. For example, asbestos fibers are still used in automobile brake linings and clutch pads, jointing and gaskets, asphalt coats and sealants, paper, plastics, and other similar products. Furthermore, asbestos continues to be employed, both nationally and internationally, as an insulator and fire retardant in construction materials such as cement piping, roofing, and shingles. See Mossman et al., supra note 4, at 295.

11. See Bignon, supra note 2, at 10.
12. See Mossman & Gee, supra note 4, at 1721.
13. Andrew Churg & Martha L. Warnock, Asbestos Fibers in the General Population, 122 AM. REV. RESPIRATORY DISEASE 669 (1980); see also Bignon, supra note 2, at 9; Mossman & Gee, supra note 4, at 1721. Asbestos fibers have been identified in the urban air of Canada, France, South Africa, the Netherlands, the United Kingdom, the United States, Germany, Italy, Switzerland, and Japan. B.W. Case & P. Sebastien, Fibre Levels in Lung and Correlation With Air Samples, in NON-OCCUPATIONAL EXPOSURE TO MINERAL FIBRES, supra note 2, at 207 (citations omitted).

14. PUBLIC HEALTH SERVICE, supra note 1; J. Corbett McDonald, Health Implications of Environmental Exposure to Asbestos, 62 ENVTL. HEALTH PERSP. 319 (1985); B.T. Commins, Estimations of Risk from Environmental Asbestos in Perspective, in NON-OCCUPATIONAL EXPOSURE TO MINERAL FIBRES, supra note 2, at 476-85; see infra note 62 and accompanying text.

15. NATIONAL RESEARCH COUNCIL, COMMITTEE ON NONOCCUPATIONAL HEALTH RISKS
found asbestos levels in natural dustfalls along roadways to occur in concentrations in excess of OSHA regulations for the workplace. However, "[t]he emission of mineral fibres from fibrous materials used for building construction is the main source of indoor fibre pollution at the present time." It is estimated that significant amounts of asbestos are present in roughly 20% of all U.S. public and commercial buildings—a total of about 733,000 structures. The revelation of these numbers, combined with reports of asbestos fibers in water supplies and in some food products, has created widespread concern "about the possible risks of exposure to asbestos outside the working environment."

The response to the public's concern about asbestos in buildings has been to remove asbestos from buildings, a process of ripping and scraping asbestos fibers from a building's superstructure. Total nationwide abatement, or asbestos removal, costs are estimated at anywhere from $50 billion to $150 billion, and may in fact amount to much more. As society's concern about asbestos spreads from occupational exposure to non-occupational exposure, the question of what to do with the millions of tons of "in place" asbestos persists.

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16. Johnine J. Brown, Asbestos Removal: A Really Major Boondoggle, ILL. LEGAL TIMES, May 1990, at 14, 15; see also N. Kohyama, Airborne Asbestos Levels in Non-occupational Environments in Japan, in NON-OCCUPATIONAL EXPOSURE TO MINERAL FIBRES, supra note 2, at 262 (finding that air samples taken from major roadways in Japan showed "extremely high" levels of asbestos concentrations).

17. Bignon, supra note 2, at 10.

18. Hooper, supra note 7, at 39; Mossman et al., supra note 4, at 294. A study of the 800,000 buildings within New York City, conducted by the City of New York, found that 544,000 of the buildings studied contained significant amounts of asbestos-containing materials. The study further revealed that approximately 87% of the 544,000 buildings had asbestos in fair to poor condition. Daniel J. Sitomer & Susan G. Rosmarin, NYC May Mandate Inspection, Sampling of Asbestos-Containing Materials, N.Y.L.J., Oct. 9, 1990, at 1 (citing David E. Pitt, Costly Proposals to Curb Asbestos Ignite New York City Debate, N.Y. TIMES, July 14, 1990, at 23 (Metropolitan)).

19. Churg & Warnock, supra note 13 (citing SELIKOFF & LEE, supra note 1, at 101-31).

20. Mossman and Gee, supra note 4, at 1721.


22. Mossman et al., supra note 4, at 294 (citing a presentation by Morton Corn at the 22nd International Congress on Occupational Health, held in Sydney, Australia, Sept., 1986).

23. The term "in place asbestos," for the purpose of this Note, and generally, refers to
scientific community is torn over this question and no answer appears to be forthcoming.\textsuperscript{24} Presently, two schools of thought dominate the arena.\textsuperscript{25} In what I have deemed the “Mount Sinai” camp, the philosophy, which adheres to traditional asbestos thought, is that all types of asbestos are equally dangerous and carcinogenic, and thus the problem of asbestos in buildings should be approached similarly to asbestos in the workplace. However, the “Mossman” camp has challenged this traditional view, arguing that one type of asbestos, which accounts for approximately 95\% of all in place asbestos,\textsuperscript{26} poses little or even no health risk when properly managed.

While the scientific community continues to debate the merits of a blanket removal policy, a “second wave” of asbestos litigation is beginning to crest.\textsuperscript{27} This wave consists of numerous litigations by building owners seeking abatement, and other related expenses, from manufacturers, installers, and miners of asbestos-containing materials. These litigations could number in the tens of thousands, and damages could escalate to billions of dollars.\textsuperscript{28}

The following pages present some of the new scientific thinking in the evaluation of the health risks of asbestos to non-occupational building occupants, and how this new research may effect an asbestos-related property damage litigation. At the core of a defense is the need to persuade the jury to abandon its traditional “one fiber” phobia. To do this, a defendant must utilize new scientific research that indicates that in most instances abatement produces negligible results at best, and at worst, greatly increases the health risks to building occupants.

This Note primarily presents an overview of the evolving asbestos debate and then delves into the background information necessary

\textsuperscript{24} See generally Hooper, supra note 7, at 38 (documenting the dispute between rival scientific thought as to the health risks posed by in place asbestos).


\textsuperscript{26} Mossman & Gee, supra note 4, at 1722; see also PUBLIC HEALTH SERVICE, supra note 1, at 9-10; Abelson, supra note 21; Pooley, supra note 7.

\textsuperscript{27} Stanley J. Levy & Ivan B. Rubin, Asbestos Cost Containment Litigation, in THE FOURTH ANNUAL SEMINAR ON ASBESTOS IN BUILDINGS 23, 27-28 (1991) [collection of works hereinafter SEMINAR ON ASBESTOS].

\textsuperscript{28} Cf. Suzanne L. Oliver & Leslie Spencer, Who Will the Monster Devour Next, FORBES, Feb. 18, 1991, at 75 (discussing the glut of personal injury cases pending against the asbestos industry, with potential damages in the billions of dollars).
to understand how the debate has arisen. After exploring the basic physiological characteristics of asbestos and how researchers measure the presence of asbestos in the air, this Note will show how researchers and scientists arrived at the "Amphibole Hypothesis," which argues that the most prevalent type of asbestos found in U.S. buildings does not pose a significant health risk to building occupants. Finally, this Note presents comparative risk evidence that indicates that playing high school football is a greater danger than exposure to asbestos in buildings.

II. ASBESTOS: THE EVOLVING DEBATE

Dr. Irving J. Selikoff, the prominent researcher in the field of asbestos disease and leader of the "Mt. Sinai" camp, told the New York Times that "there is no issue, no debate about asbestos."29 Selikoff, the former director of the Mount Sinai Medical School’s Environmental and Occupational Health Division, was the first to conclusively document the relationship between asbestos and lung ailments.30 His group's work was the primary catalyst for government regulation of asbestos in the 1970s and 1980s.31 "For most of the last decade, the work of Selikoff and his colleagues pointed to a seemingly unassailable conclusion: Asbestos, a tragedy for industrial workers, posed a grave threat to the general public as well. Countless articles and television programs hammered this simple message home."32 The work of the Mount Sinai camp, however, has been under attack and traditional opinions about asbestos are being questioned and changed.33

29. Hooper, supra note 7, at 38.
31. See Hooper, supra note 7, at 38.
32. Id.
33. In a recent column in the New York Law Journal, the authors summarized the current public perception of the threat from asbestos:

The issue of the health risk to building occupants from the presence of asbestos, and the related issue of the responsibility of the building owners for asbestos abatement, has been the subject of widespread debate for several years. During this period, there has been a change in the public attitude regarding the presence of asbestos—from initial demands that all asbestos-containing materials, regardless of condition, be removed from buildings immediately, to a recognition that most asbestos can be managed safely in place.
Dr. Brooke T. Mossman of the University of Vermont and her co-authors are at the center of the current asbestos controversy. Mossman's two recent articles have been the focal point of the "to-abate-or-not-to-abate" war among asbestos scientists. Mossman's first article appeared in the New England Journal of Medicine and while ruffling scientific feathers, it raised few eyebrows. Her second article appeared some seven months later in the more mainstream journal Science, where her positions received greater attention.

In essence, Mossman challenges the view that chrysotile asbestos, which accounts for more than 90% of the world's production of asbestos, is a carcinogen at low levels. The natural implication of the Mossman camp's findings is that widespread abatement is unnecessary. According to Mossman and her followers, there is no evidence that there is any significant health risk posed by well-managed, in place asbestos. "Low level exposure is not a threat to human health. The scare [i.e. one fiber phobia] is unprecedented, and the amount spent on removal is ridiculous." In fact, she and her colleagues find that unnecessary removals are a much greater health threat than containment of damaged asbestos.

Mossman's followers are not outside the mainstream. The Moss-
man position was adopted by a Harvard University Symposium in 1989, and most recently the American Medical Association ("AMA") took a position similar to Mossman's. Recent revisions in EPA policy follow the Mossman position, which calls for the management of in place asbestos and rejects abatement except where necessary.

The unwarranted amount of asbestos removal in the United States is attributable to "one fiber" thinking. The fear of a single fiber of asbestos has been the boon to a $3 billion per year abatement industry. Dr. Mossman blames the asbestos abatement "craze" on one fiber phobia. "Panic has been fueled by unsupported concepts such as the 'one fiber theory,' which maintains that one fiber of inhaled asbestos will cause cancer." There is no merit to the one fiber theory, and the myth is slowly being dispelled.

43. Block & Kessler, supra note 33.
44. William K. Stevens, Doctors Reassess Risk of Asbestos, N.Y. TIMES, Aug. 7, 1991, at A15 (reporting that an AMA position paper concluded that in place asbestos poses far less risk to building occupants than smoking, alcohol or drug abuse, or improper diet, and that Americans should learn to live with asbestos safely).
45. National Emission Standards for Hazardous Air Pollutants; Asbestos NESHAP Revision, 55 Fed. Reg. 48,406 (1990). "[The] EPA's best advice on asbestos is neither to rip it all out in a panic nor to ignore the problem . . . [r]ather, we recommend a practical approach that protects [the] public health by emphasizing that asbestos material in building should be . . . appropriately managed." EPA, AN ADVISORY TO THE PUBLIC ON ASBESTOS IN BUILDINGS (1991). The EPA concluded, in "fact five" that the "EPA does recommend in-place management whenever asbestos is discovered." Id; see also EPA, MANAGING ASBESTOS IN PLACE: A BUILDING OWNER'S GUIDE TO OPERATIONS AND MAINTENANCE PROGRAMS FOR ASBESTOS-CONTAINING MATERIALS (1990).
46. Mossman et al., supra note 4, at 299; see also Brown, supra note 16, at 14 (categorizing the public perception concerning asbestos in buildings as "hysteria"); Corn, supra note 4, at 522 (calling for the scientific community to "lend perspective to [one fiber phobia thinking], to not permit understandably emotional responses to documented past severe health effects . . . carry over into conditions of very low exposures in the public domain").
48. Mossman et al., supra note 4, at 299.
Lung cancer and other lung ailment models used by the EPA and other agencies in assessing the risk from asbestos assume a linear dose response, and therefore, presume greater exposure to be consistent with greater risk. Linear models, however, presuppose that any exposure, no matter how small, poses some risk. Id. at 156, 162-63. Additionally, it is now believed that asbestosis only results from intensive exposure to asbestos fibers and particles. Richard Siegler, Developments Concerning Asbestos, N.Y.L.J., July 3, 1991, at 3 (Cooperatives and Condominiums). See infra section VIII for more on in-place asbestos as a health hazard.
50. See supra note 33 and accompanying text.
Another type of fear, related to one fiber phobia, is motivating owners, and more importantly, banks: the fear of liability. According to Richard Jones, senior partner at Pepe & Hazard, a Hartford asbestos specialty firm, no building owner has ever been found liable for asbestos-related medical damage to tenants. Abatement, however, remains an attractive avenue for building owners because of questions and fear of liability.\textsuperscript{51} Jones says that he expects to continue to see banks requiring abatement, in spite of the solid evidence against that course of action.\textsuperscript{52}

Abatement, however, is certainly not an absurd option, and when properly done, has the endorsement of the Mount Sinai camp. At an international asbestos conference, held by the Mount Sinai School of Medicine in 1990, the theme was that chrysotile asbestos is as toxic as all other types of asbestos. The data shows "beyond any shadow of a doubt, that chrysotile asbestos is every bit as hazardous as other forms of asbestos," reported Dr. Philip J. Landrigan, co-chairman of the conference.\textsuperscript{53} Sponsors of the conference further reported that the fifty-six scientific papers presented were all "unanimous in expressing data that all types of asbestos fibers created some type of health risk."\textsuperscript{54} And in early 1991, the National Institute for Occupational Safety and Health ("NIOSH"), rejected the Mossman position, stating that it believed that the results of some studies indicated that chrysotile fibers are at least as toxic as other asbestos fibers.\textsuperscript{55}

Dr. Mossman and members of her camp criticized the conference papers and the NIOSH report.\textsuperscript{56} The Mossman response was that first, the studies have not been reported in scientific journals so that they can be duplicated, and that second, the conference relied mostly on studies concerning occupational exposure, and only non-occupational exposure is at issue.\textsuperscript{57}

However, in spite of the professional bickering, members of the Mount Sinai camp recognize that there have been unnecessary asbestos removals. Dr. Landrigan conceded that building owners have too

\textsuperscript{51} Slutsker, \textit{supra} note 47, at 303.
\textsuperscript{52} Id.
\textsuperscript{56} See Stevens, \textit{supra} note 53, at B7.
\textsuperscript{57} Id.
often unnecessarily abated asbestos, increasing the risks to building occupants.\textsuperscript{58} He said that this is often done to improve a building's value, and that he agreed with the EPA, AMA, and the Mossman camp, that undamaged asbestos should be left alone.\textsuperscript{59} Dr. William J. Nicholson, a member of the Mount Sinai camp, also agreed that the removal of intact asbestos was not necessary.\textsuperscript{60}

Still, despite the apparent consensus among the scientific community that the abatement of undamaged asbestos-containing material is unwarranted,\textsuperscript{61} and the less general consensus that most damaged asbestos-containing material should be contained, building owners continue to abate and seek damages.

\textbf{III. ASBESTOS TYPES AND CHARACTERISTICS}

Asbestos is a naturally-occurring fibrous mineral mined from the earth. Since asbestos is a material that occurs naturally in rock formations, it is found in the air, and often in water supplies and in food.\textsuperscript{62} In fact, we live on a planet on which there is an abundance of asbestos-containing rock.\textsuperscript{63} The natural processes of erosion have been releasing fibers throughout Earth's history, so that today the typical American breathes in about one million fibers a year via natural and man-made sources.\textsuperscript{64}

Asbestos is defined as a group of naturally-occurring fibrous materials.\textsuperscript{65} More precisely, "[a]bestos is a broad commercial term

\begin{footnotesize}
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\item 58. \textit{Id.}
\item 59. \textit{Id.}
\item 60. Stevens, \textit{supra} note 25, at D24.
\item 61. \textit{See id.} (reporting that most scientists support the view that asbestos should be managed in place rather than removed arbitrarily).
\item 62. \textit{See supra} notes 13-16 and accompanying text.
\item 63. \textit{See generally} Pooley, \textit{supra} note 7, at 3-25 (discussing geological and mineralogical aspects of asbestos and asbestos-containing rock).
\item 64. Abelson, \textit{supra} note 21. Man-made industrial sources of asbestos fiber inhalation include the shredding of brake linings, the decay of asbestos-containing water pipes, and the release of asbestos fibers during building renovations, among other sources. \textit{See supra} notes 9-13 and accompanying text.
\item 65. \textit{See Pooley, supra} note 7; \textit{see also} Andrew Churg, \textit{Nonneoplastic Diseases Caused by Asbestos}, in \textit{Pathology of Occupational Lung Disease} 213-24 (Andrew Churg & Francis H.Y. Green eds., 1988).
\end{itemize}
\end{footnotesize}
for a group of naturally occurring hydrated silicates that crystallize in a fibrous habit.\textsuperscript{66} For EPA purposes, the legal definition of an asbestos fiber is one that possesses an aspect ratio (i.e., a ratio of length to diameter) of equal to or greater than three to one.\textsuperscript{67}

The commercial name asbestos is actually six distinct minerals: actinolite, amosite, anthophylite, chrysotile, crocidolite, and tremolite. The chemical makeup of each fiber type is complex, and fibers often consist of a variety of trace metals and organic compounds acquired organically or during processing. For example, iron oxides and quartz may be found with crocidolite and amosite fibers, whereas the more versatile chrysotile is often contaminated with forsterite, magnetite, brucite, quartz, and feldspar.\textsuperscript{68}

Asbestos ore forms as a crystalline rock, and is found on or just below the surface, and is easily mined.\textsuperscript{69} The minerals fall into two classes: serpentine and amphibole fibers.\textsuperscript{70} Chrysotile asbestos is the most abundant type of asbestos and is a serpentine fiber, with the other five minerals falling into the amphibole family.\textsuperscript{71} Abundant deposits of amphibole asbestos are found in South Africa and Australia, while large amounts of chrysotile asbestos are mined in Canada.\textsuperscript{72} Chrysotile asbestos accounts for more than 90\% of the world's asbestos production,\textsuperscript{73} and 95\% of all in place asbestos in the United States.\textsuperscript{74}

Chrysotile fibers consist of pliable, curly fibrils which resemble scrolled tubes.\textsuperscript{75} Amphibole fibers, on the other hand, are longer rod or needle-shaped spikes.\textsuperscript{76} Studies of the fibers show that the

\textsuperscript{66} Mossman et al., supra note 4, at 294.
\textsuperscript{68} Mossman & Gee, supra note 4, at 1722 (citing MINERAL RESOURCES OF THE UNION OF SOUTH AFRICA 357-67 (4th ed. 1959)); see also SELIKOFF & LEE, supra note 1, at 36.
\textsuperscript{69} Asbestos ore is usually mined from open pits by bulldozers and power shovels; however, where the ore is deeper, conventional mining practices are employed—including blasting. SELIKOFF & LEE, supra note 1, at 51.
\textsuperscript{70} Id. at 33-36; Mossman et al., supra note 4, at 294; Pooley, supra note 7, at 3-25.
\textsuperscript{71} Mossman et al., supra note 4, at 294.
\textsuperscript{72} Slutsker, supra note 47, at 303. Asbestos has also been mined in Italy, the former Soviet Union, the United States, China, Bolivia, and India, as well as other nations.
\textsuperscript{73} See supra note 39 and accompanying text.
\textsuperscript{74} See supra note 26.
\textsuperscript{75} Mossman and Gee, supra note 4, at 1722. "In simplistic terms, the macromolecular structure of chrysotile consists of parallel sheets of silica and brucite that are stacked with varying degrees of overlap and curvature." Id.; see also Pooley, supra note 7, at 14-18, for a detailed, but easy to understand, discussion of the structure of chrysotile fibers.
\textsuperscript{76} Mossman & Gee, supra note 4, at 1722. The amphibole group is composed of parallel chains of silica tetrahedra, separated by a band of cautions that vary in type, number,
chrysotile fibers tend to occur in bundles, or groups of fibers, as a result of its tubular structure, and can become quite large. They are "readily intercepted at airway bifurcations because of their curliness." Those chrysotile fibers that do enter the lungs generally, as a result of their shape, do not become imbedded in the lung wall, and are "digested" by the lung. This process of "digestion" is not understood; however, a potential explanation for the increased solubility of chrysotile fibers may be found in the longitudinal fragmentation and leaching of magnesium from chrysotile fibers. In contrast, the longer amphibole fibers do not group, and therefore, have little difficulty penetrating the lungs. Upon entrance, the fibers bury themselves deep in the lung wall and are inextricable by the body. Once in the lung wall, the asbestos fibers begin the long process of impairing the lungs' ability to function properly.

and capacity for substitution. Id.; see also Pooley, supra note 7, for a detailed, but easy to understand, discussion of the structure of amphibole fibers.

77. See L. Le Bouffant, Physics and Chemistry of Asbestos Dust, in Biological Effects of Mineral Fibres 15, 26-27 (J.C. Wagner ed., 1980); Mossman & Gee, supra note 4, at 1722; Mossman et al., supra note 4, at 295.


79. J.M.G. Davis, Mineral Fibre Carcinogenesis: Experimental Data Relating to the Importance of Fibre Type, Size, Deposition, Dissolution and Migration, in Non-Occupational Exposure to Mineral Fibres, supra note 2, at 33-40; Mossman and Gee, supra note 4, at 1722-23; Mossman et al., supra note 4, at 296; see also M.C. Jaurand et al., Leaching of Chrysotile Asbestos in Human Lungs: Correlation with In Vitro Studies Using Rabbit Alveolar Macrophages, 14 Envtl. Res. 245 (1977). See infra section V, the Amphibole Hypothesis, for a further discussion of the increased solubility of chrysotile fibers, and the correlation between asbestos exposure and lung burdens.

80. Davis, supra note 79, at 33-40; Mossman and Gee, supra note 4, at 1722-23; Mossman et al., supra note 4, at 296.

81. When fibers enter the lung, they can cause disease by piercing the cells in the lung walls. Once the cells are pierced, the asbestos fibers are not cleared from the respiratory system, and thus begin to oxidize, or rust, and tumors begin to appear around them. As the fibers accumulate they can scar the lung tissue (asbestosis, which is non-malignant), or cause lung cancer or mesothelioma (malignant tumors in the lining of the lung and digestive tract), as well as other lung ailments.

Asbestosis, the most common asbestos-related disease, develops as follows. The initial lesions occur upon the fiber's lodgement in the alveoli. Soon thereafter, a network of reticulin fibers form around the lesion. As the cellular degeneration continues, the reticulin fibers are replaced with collagen, resulting in the obliteration of the alveoli. The collagen fibers are then carried into the alveolar ducts, infecting remaining healthy lung regions. The effect is known as scarring, and it reduces both lung expansion and lung surface area, as well as narrowing airways. The result is a diminished ability to mix gases, which ultimately leads to
Undisturbed, both amphibole and chrysotile asbestos types are not respirable, or capable of being inhaled. Asbestos presents a health risk only when inhaled. Asbestos fibers, however, are extremely friable and when disturbed become airborne and are easily inhaled. Asbestos fibers in ores are usually not respirable until released during mining and processing, or sometimes through wind and water erosion. Likewise, asbestos in buildings does not become respirable unless disturbed, often during renovation or demolition, and always during abatement.

Usually a visual inspection of known asbestos-containing material can reveal a friable or damaged condition; however, only air sampling can measure asbestos air concentrations.

IV. MEASUREMENT TECHNOLOGY

The scientific evidence indicates that there are important differences between asbestos fiber types in their chemical composition, durability, solubility, and morphology. As indicated above, asbestos fiber type may be a significant factor in the risk posed by exposure to asbestos. As it appears that different fiber types may affect the body differently, enhanced technology, used to examine asbestos fibers and air samples with greater detail and clarity, becomes extremely important.

A. Historical Perspective

In the early 1970s when NIOSH established the P&CAM 239 optical microscopy method to measure asbestos concentrations, there was no reason to be overly concerned with measurement specificity. "In factories, asbestos was used in thousands of products. The
material was shipped in bags that were ripped open and routinely dumped into hoppers. It was certainly reasonable to assume that any fiber taken from a worker's breathing zone was asbestos." Obviously, industrial hygienists and researchers did not have a problem obtaining measurable deposits on their air filters. NIOSH commonly reported workplace exposure levels up to 75 fibers per cubic centimeter of air ("f/cc") throughout the early 1970s. The OSHA permissible exposure level ("PEL") in 1970 was, however, 12 f/cc in the workplace. According to workplace studies of that time, the greatest concern of technicians was to avoid overloading filters with fibers.

Since the birth of OSHA there has been a continually decreasing airborne asbestos concentration level enforced by regulation. The original OSHA PEL of 12 f/cc in 1970 was soon reduced to 5 f/cc in 1972. In 1976, the PEL was reduced again, and today the PEL is set at 0.2 f/cc, with an action level of 0.01 f/cc. This represents a 120-fold decrease from the original 12 f/cc level. Also, it is not uncommon for abatement specifications to require removal at 0.005 f/cc—2,400 times lower than the original OSHA PEL. At these lower levels measurement specificity takes on greater importance, and places "quite a demand on the sensitivity of any analytical method." Measurement specificity has become more important as air samples are no longer replete with long asbestos fibers and contain many non-asbestos fibers.

Air samples are collected today in much the same process as in the 1970s, by pulling air across a filter. A proportionate number of particles in the air will gather on the filter. By counting the number of fibers deposited on the filter, technicians can determine the asbestos air concentration for the tested site. An asbestos fiber, statutorily,

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88. Id. at 157.
89. Id.
90. Id.
91. See generally Nowinski, supra note 6, at 99-134 (outlining the history of asbestos legislation and regulation).
92. Lippy & Boggs, supra note 87.
93. Id.
94. Id. at 157.
95. See infra notes 103-09 and accompanying text.
is one that possesses a ratio of length to diameter of equal to or greater than three to one, and a length greater than five microns.  

B. Phase Contrast Microscopy

Asbestos levels in buildings are generally measured by phase-contrast microscopy ("PCM") or transmission electron microscopy ("TEM"). PCM, or optical or light microscopy, was the technology employed during the 1970s, and continues to be the most widely used asbestos measuring technology. Despite its wide use, PCM has several limitations, which leave Mossman camp members questioning its continued use. PCM measures fibers only longer than 5 microns in length, hence the statutory definition. Furthermore, PCM achieves a magnification of only about 600, which renders fibers thinner than between 0.25 and 0.5 microns in diameter invisible to PCM technology. Individual fibrils, however, have been measured as thin as 0.01 microns in diameter, using transmission electron microscopy. As a result of these limitations, PCM cannot distinguish between asbestos and other types of fibers, nor can it differentiate among asbestos fiber types. As a consequence of PCM's inability to distinguish between asbestos and non-asbestos fibers, it tends to overestimate ambient asbestos concentrations. Moreover, the limit for detection of fibers by PCM is about 0.01 f/cm, a concentration higher than that reported in many schools, as measured by TEM.

98. Lippy & Boggs, supra note 87; Mossman & Gee, supra note 4, at 1722; Mossman et al., supra note 4, at 299. See H. Well, Asbestos—A Summing Up, in BIOLOGICAL EFFECTS OF MINERAL FIBRES, supra note 77, at 867, 873 (noting that PCM, as of 1980, was the most common method of analyzing air samples for asbestos concentrations).
99. Crump, supra note 49, at 163; Lippy & Boggs, supra note 87; Mossman & Gee, supra note 4, at 1722; Mossman et al., supra note 4, at 299 (among other sources documenting the limitations of PCM technology).
100. See supra note 96 and accompanying text.
101. Crump, supra note 49, at 163; Mossman et al., supra note 4, at 299. According to Le Bouffant, most asbestos fibers found in lung tissue are not seen with PCM. Le Bouffant, supra note 77, at 26.
103. Id.; see also Lippy & Boggs, supra note 87, at 158; Mossman et al., supra note 4, at 299.
104. Crump, OSHA Testimony, supra note 102 at 248; Lippy & Boggs, supra note 87, at 158.
105. Bignon, supra note 2, at 11; Mossman et al., supra note 4, at 299.
As a result of PCM's shortcomings, PCM is of limited value in building settings because the majority of fibers measured by PCM in buildings usually are not asbestos. Additionally, in a building, the erosion process releases extremely small amounts of asbestos fibers into the air. While airborne, fiber bundles and individual fibers are further reduced in diameter and length through the process of comminution.

The problem is that in buildings the fibers are different [from in the workplace]. Asbestos fibers appear to be smaller (fewer bundles) and make up a smaller fraction of all airborne fibers. The slow degradation of asbestos-containing materials in ventilation airstreams . . . produces much thinner and shorter fibers than the physical removal of asbestos from ceilings and mechanical systems during abatement.

In a West German study, researchers established that less than 10% of all fibers, 5 microns in length or longer, found in air samples were asbestos fibers. Samples were taken from 231 sites; 219 indoor ambient air samples taken during asbestos abatement, and 21 from workplaces with asbestos-containing materials. For all samples combined, there was an average of 1,000 fibers (all types) per cubic meter found. Of that number, however, only 100 fibers, on average, were deemed to be asbestos, as counted by scanning electron microscopy. In another study, comparing phase-contrast microscopy with transmission electron microscopy, the conclusion was that only 6% to
10% of the fibers identified by PCM as asbestos actually are asbestos fibers, as viewed under the electron microscope.\(^{112}\)

PCM technology is not well suited for the changed focus from the workplace to the non-occupational setting. Asbestos fibers found in non-occupational air are usually thin fibrils, shorter than 5 microns, and not detectable by phase-contrast microscopy.\(^ {113}\) "To the extent that one is concerned with the concentration of fibers of smaller dimensions . . . [PCM] is clearly inadequate."\(^ {114}\)

The demands of measuring in place asbestos surpass the technology provided by phase-contrast microscopy. PCM, however, is still considered useful for measuring asbestos levels during abatement operations, because a larger percentage of airborne fibers in the air during removal is likely to be asbestos.\(^ {115}\)

\section*{C. Transmission Electron Microscopy}

Transmission electron microscopy ("TEM"), on the other hand, is well suited for the task of measuring and identifying asbestos fibers. Whereas PCM could not measure fibers less than 5 microns in length and 0.25 microns in width, TEM achieves a resolution of 100,000, and therefore is "capable of identifying the thinnest asbestos structure."\(^ {116}\) By employing TEM to study air samples,\(^ {117}\) researchers are able not only to count more fibers, but also to identify and differentiate between asbestos and non-asbestos fibers and among asbestos fibers. "Transmission electron microscopy of air samples is essential for the identification and quantitation [sic] of finer asbestos fibers."\(^ {118}\) It is undisputed that TEM is "far superior to the other methods" of microscopy.\(^ {119}\) TEM, however, is vastly more expen-

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\(^{112}\) Crump, \textit{OSHA Testimony}, supra note 102, at 258.

\(^{113}\) Bignon, supra note 2, at 11; see also Churg & Warnock, supra note 13, at 672 (finding that in a study of diseased human lung tissue, the majority of asbestos fibers were less than 5 microns in length; additionally, 90% of chrysotile fibers were less than 5 microns, while only 25% of commercial amphibole fibers were less than 5 microns).

\(^{114}\) SELIKOFF & LEE, supra note 1, at 78.

\(^{115}\) Crump, supra note 49, at 163.

\(^{116}\) Crump, \textit{OSHA Testimony}, supra note 102, at 249.


\(^{118}\) Mossman et al., supra note 4, at 299.

\(^{119}\) Lippy & Boggs, supra note 87, at 158. "These instruments [transmission electron microscopes] have now been shown to be the most useful for both detecting and identifying fibrous particles of all sizes . . . ." A. Gaudichet et al., \textit{Identification and Quantification of
sive than PCM, and government standards are still set with and according to the ability of PCM technology.120

D. Microscopy and Public Policy

The continued use of phase-contrast microscopy, therefore, presents two problems. First, numerous smaller fibers and thinner fibers, which tend to be amphibole fibers, will not be detected. Second, many non-asbestos fibers will continue to be classified as asbestos, thus overestimating the risk from non-occupational asbestos exposure.121 Yet, by employing the new measurement technology provided by the electron microscope, researchers are able to distinguish chrysotile fibers from amphibole fibers, and establish hyper-accurate fiber counts. Although this enhanced ability is costly, especially when considered in the aggregate, the use of TEM technology provides more reliable data. This leads to realistic risk assessment models for non-occupational asbestos exposure, and therefore, may obviate costly building abatements.

E. Transmission Electron Microscopy and the Amphibole Hypothesis

The utilization of transmission electron microscopy has not been limited to the evaluation of air samples. Researchers now use TEM to view the effects and concentrations of different fiber types in animal tissue, including human tissue. Application of this enhanced microscopy technology has allowed researchers to learn more information about the differences among asbestos fiber types in their chemical composition, durability, solubility, and morphology.122

The Mossman camp, utilizing this heightened knowledge of

Asbestos Fibres in Human Tissues, in Biological Effects of Mineral Fibres, supra note 77, at 61; see also Cherrie et al., supra note 107, at 304 (comparing PCM, TEM, and scanning electron microscopy ("SEM"), and finding that TEM and SEM are far superior to PCM at all levels, and that TEM achieves more accurate results than SEM measuring fibers less than 5 microns in length).

A new technique for measuring asbestos concentrations, however, has been advanced. Use of the infrared spectrophotometry ("IR") technique was described by Valerio and Balducci, in a study of asbestos-cement products and insulating materials. F. Valerio & D. Balducci, Qualitative and Quantitative Evaluation of Chrysotile and Crocidolite Fibres with Infrared Spectrophotometry: Application to Asbestos-Cement Products, in Non-Occupational Exposure to Mineral Fibres, supra note 2, at 197. The authors describe IR as a simple and rapid qualitative and quantitative method of evaluation, as well as being low cost. Id.

120. Lippy & Boggs, supra note 87, at 158.
121. Id. at 158-59.
122. See supra notes 58-84 and accompanying text.
asbestos characteristics, together with detailed information of asbestos levels in buildings and in animal tissue, have concluded that exposure to chrysotile asbestos in buildings does not pose a significant health risk to building occupants. This highly controversial theory is known as the Amphibole Hypothesis.

V. THE AMPHIBOLE HYPOTHESIS

In essence, the Amphibole Hypothesis suggests that the amphibole group of asbestos fibers is the principal cause of asbestos-related diseases. Accordingly, this theory indicates that the chrysotile group of asbestos fibers, which accounts for more than 90% of the world’s production of asbestos, is far less pathogenic than had previously been thought. “There is fairly clearcut evidence that amphiboles are more dangerous than chrysotile” in the formation of disease in humans. Since chrysotile asbestos accounts for 95% of American in place asbestos, the implication of the Amphibole Hypothesis is that widespread abatement is unnecessary.

A. Historical Origins of the Hypothesis

Mossman’s Amphibole Hypothesis was not the result of a single study; rather, it is the recognition that, contrary to earlier beliefs, there are marked differences in the ability of different types of asbestos to produce disease in humans. Indeed, there is now good evidence that amphibole asbestos is much more dangerous than chrysotile asbestos. This “good evidence” that the Hypothesis recognizes is the work of many different researchers in many different studies conducted throughout the last thirty years.

The association of mesothelioma with asbestos exposure was first described in 1960, in South Africa. There, exposure was to

123. Mossman et al., supra note 4.
124. See supra note 39 and accompanying text.
125. Churg, supra note 65, at 223; see also Talcott & Antman, supra note 1, at 144 (categorizing chrysotile asbestos as less potent than crocidolite and amosite fibers, the most commonly used amphibole asbestos).
126. See supra note 26 and accompanying text.
127. See Churg et al., supra note 4, at 296.
128. Churg, supra note 40, at 621.
129. See supra note 81.
130. See J.C. Wagner et al., Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province, 17 BRIT. J. INDUS. MED. 260 (1960).
long, narrow crocidolite fibers of the amphibole group. Since that 1960 study, an increased incidence of mesothelioma has been documented in the workplace. These numerous reports investigated
various occupational settings and examined both amphibole and chrysotile exposure.

Within the past decade, however, transmission electron microscopy\(^\text{133}\) has allowed the examination of the types of fibers in the lung tissue of workers exposed to asbestos, revealing an important clue leading to the development of the Hypothesis.

**B. Lung Burden**

The results of studies employing TEM, surprisingly, have revealed "that many chrysotile-exposed workers showed an appreciable lung burden of amphibole fibers,"\(^\text{134}\) to which the workers had been exposed for only short periods. Simplistically, for purposes of this Note, the term "lung burden" indicates the quantity of asbestos fibers deposited in lung tissue.

Recent studies clearly indicate that amphibole fibers tend to collect in human lungs to a much greater extent than chrysotile fibers.\(^\text{135}\) These studies reveal that chrysotile lung burdens are lower than researchers would have expected to find in the lungs of asbestos workers and others exposed primarily to chrysotile asbestos.\(^\text{136}\) For example, in one significant study, the lungs of Canadian chrysotile miners possessed greater quantities of tremolite asbestos, a trace contaminant of the amphibole family, than chrysotile asbestos.\(^\text{137}\)

In another study, Le Bouffant studied workers at a spinning mill that dealt mostly with chrysotile asbestos. Examinations of the worker's lungs revealed only traces of chrysotile, and heavy burdens of amphibole fibers.\(^\text{138}\) Le Bouffant concluded that the "relative content of chrysotile and of amphiboles does not correlate with what is known about the actual exposures of the subjects."\(^\text{139}\)

An examination of the lungs of thirty-six workers dying of as-

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(finding the incidence of mesothelioma in South African workers to be the highest ever reported for a national population). See generally INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, BIOLOGICAL EFFECTS OF ASBESTOS (P. Bogovski ed., 1973) (presenting clinical studies of asbestos-induced disease in humans).

\(^{133}\) See supra notes 116-20 and accompanying text.

\(^{134}\) Mossman et al., supra note 4, at 296.

\(^{135}\) Id.

\(^{136}\) See e.g., J.C. Wagner et al., Pathological and Mineralogical Study, supra note 132, at 423.

\(^{137}\) F.D. Pooley, An Examination of the Fibrous Mineral Content of Asbestos Lung Tissue From the Canadian Chrysotile Mining Industry, 12 ENVTL. RES. 281 (1976).

\(^{138}\) Le Bouffant, supra note 77, at 26.

\(^{139}\) Id.
bestos-related diseases revealed no correlation between lung burden of chrysotile fibers and disease. At the same time, amphibole lung burdens were strongly associated with asbestosis, mesothelioma, and lung cancer. A similar study revealed that, in ninety-three cases of mesothelioma, all subjects had significantly more amphibole fibers than chrysotile. The same study found that chrysotile lung burdens were similar between mesothelioma subjects and healthy controls.

In a 1988 French study, the lung burden of asbestos and non-asbestos fibers was compared in cases of mesothelioma, lung cancer, and a control group. The cohort was taken from a coastal city with shipyards. Using TEM to examine the fibers, significantly higher amounts of amphibole fibers than chrysotile fibers were found in the mesothelioma cases. However, all three groups had equivalent levels of chrysotile fibers and non-asbestos fibers. These data suggest that the lung burden of chrysotile fibers bears no relation to the occurrence of mesothelioma or lung cancer.

C. Lung Clearance

A suggested reason that lung burden studies reveal fewer chrysotile fibers than amphibole fibers, even where exposure was only to minor amounts of amphiboles, is the ability of the human lung to clear chrysotile fibers. Two means by which chrysotile clearance may occur are dissolution and the elimination of fibers by way of the lymphatic system.

As a result of the unique physical characteristics of chrysotile asbestos, it is more readily cleared from the lungs. This physical cleansing may occur via the mucociliary escalator, and by the macrophage, where fibers are removed from the conducting airways.

140. Wagner et al., *Pathological and Mineralogical Study*, supra note 132, at 444.
142. See Mossman et al., *supra* note 4, at 296 (reporting the 1988 French study).
144. See *supra* notes 75-80 and accompanying text.
146. See *supra* notes 75-79 and accompanying text.
147. Burdett et al., *supra* note 145, at 278; Churg, *supra* note 65, at 223; A. Morgan,
Chrysotile "dissolves" with time, most likely because magnesium and silica are leached from the fibers.\textsuperscript{148} The persistence of amphibole fibers in human lungs is attributable to its insolubility, durability, and increased ability to penetrate the lung wall as a result of its needle-like shape.\textsuperscript{149}

**D. Differing Toxicity Between Chrysotile and Amphibole Asbestos**

Many scientists, including those in the Mossman camp, now agree that exposure to amphibole asbestos poses a much greater risk than exposure to chrysotile asbestos.\textsuperscript{150} The characteristic differences between fiber types\textsuperscript{151} tend to account for the increased pathogenicity, or toxicity, of amphibole fibers as compared to chrysotile fibers. Many studies support this conclusion.

A study of workers in the same industries, but working with different fibers, indicated that amphibole workers had significantly higher risks of developing asbestos-related diseases than those exposed to chrysotile asbestos.\textsuperscript{152} In a similar study, Australian crocidolite asbestos was found to be 800 times more potent than Canadian chrysotile asbestos as a factor for mesothelioma risk, and 200 times more potent as a factor for lung cancer risk.\textsuperscript{153}

In a study comparing female gas mask manufacturers, researchers found again that, where different fibers were used, different levels of disease followed.\textsuperscript{154} The group that worked with the amphibole fiber experienced a statistically significant increase in excess lung and ovarian cancers, whereas the chrysotile group did not.\textsuperscript{155} With re-
gard to mesothelioma, one study indicated that the amphibole group has a two order of magnitude greater potential for inducing disease than chrysotile fibers.\footnote{156} A 1989 study of asbestos workers in London further indicates the increased toxicity of amphibole fibers.\footnote{157} The data revealed that the severity of asbestosis and lung cancer, as well as mesothelioma, was in direct correlation to the amount of amphibole fibers found in the lung. But, a group of British workers exposed to chrysotile fibers at airborne concentration levels of between 0.5 \textit{f/cc} and 1.0 \textit{f/cc}\footnote{158} for more than 15 years, showed no level of deaths in excess of standard mortality rates for asbestosis, mesothelioma, lung cancer, or other asbestos-related diseases.\footnote{159}

Other studies, contrary to those cited in footnote 132, have also found that cohorts with long-term occupational exposure to only chrysotile asbestos have suffered no excess mortalities.\footnote{160}

\textbf{E. Chrysotile and Mesothelioma}

Andrew Churg, from the Department of Pathology and Health Science Centre Hospital, University of British Columbia, reviewed the literature reporting chrysotile-induced mesothelioma, to study the incidence of amphibole exposure with mesothelioma.\footnote{161} Of the 142 me-
sothelioma cases in this set of reports, Churg concluded that at most, only 53, about one third, could be considered chrysotile-induced tumors. Of the 53 accepted cases, Churg found that 41 were in workers exposed directly to chrysotile mining dust contaminated with tremolite, an amphibole asbestos. Churg concluded that tremolite is the actual causative agent of most chrysotile-induced mesotheliomas. Ultimately, Churg's review revealed that the total number of chrysotile-induced mesothelioma cases is very small, and that the dosage required to induce disease from chrysotile asbestos is several hundred times greater than that required by amphibole fibers.

These data, as well as other studies not discussed, lead to the conclusion that amphibole fibers are more potent than chrysotile fibers in the induction of asbestos-related diseases. Additionally, studies similar to one conducted by J.C. Wagner indicate that the contraction of asbestosis is associated with significantly heavier lung burdens than mesothelioma or lung cancer. Considering that chrysotile is not believed to be associated with mesothelioma in the workplace, and that concentrations of chrysotile asbestos such to cause asbestosis are unheard of in the non-occupational setting, it is reasonable to conclude that there is not a health risk that merits abatement of intact, in-place asbestos.

VI. Levels of Ambient Asbestos in Buildings

Since chrysotile asbestos accounts for 95% of in place asbestos in U.S. buildings, it would seem that non-occupational exposure to asbestos in buildings, in light of the above, is not a significant health risk. Further, the airborne levels of chrysotile asbestos that occur in buildings has not been shown to pose a health risk. In fact, many

305 (1985); F. Rubino et al., Mortality of Chrysotile Asbestos Workers at the Balangero Mine, Northern Italy, 36 BRIT. J. INDUS. MED. 187 (1979); Thomas et al., supra note 160, at 273.
162. Churg, supra note 40, at 622.
163. Id.
164. Id. at 627.
165. See Churg, supra note 40, at 627-28; accord McDonald, supra note 161, at 678 (concluding that the risk of mesothelioma after exposure to crocidolite is many times greater than after chrysotile exposure).
166. Wagner et al., Correlation, supra note 132, at 305.
167. See supra notes 161-65 and accompanying text; see also A.R. Gibbs et al., Non-occupational Malignant Mesotheliomas, in NON-OCCUPATIONAL EXPOSURE TO MINERAL FIBRES, supra note 2, at 219, 227 (concluding that chrysotile asbestos has no potential for mesothelioma induction on its own).
studies indicate that indoor airborne asbestos levels are equal to, and in some instances lower than, environmental levels. Additionally, current data do not indicate that airborne asbestos levels in buildings with asbestos-containing materials are any higher than buildings without asbestos-containing materials.

Specifically, in a 1988 study, the EPA concluded that its results “indicate no difference between [ambient] levels found in buildings with [asbestos-containing materials and] ambient levels [of buildings without asbestos-containing materials], when compared at the 0.05 level of statistical significance.” The EPA reached this opinion based on a study of forty-nine buildings occupied by the General Services Administration. A private study, using the data from the EPA report reached a similar conclusion. The authors found that the average indoor concentration of asbestos from the forty-nine buildings was 0.00073 f/cc for all fibers, and 0.00007 f/cc for fibers longer than 5 microns. The OSHA permissible exposure limit, recall, is 0.2 f/cc. Differences between indoor and outdoor levels were statistically insignificant, and differences between buildings without asbestos-containing material (“ACM”) and buildings with damaged ACM were also statistically insignificant. The average indoor concentration, therefore, of asbestos fibers greater than 5 microns was 2,500 times less than the OSHA permissible exposure level, and tens of thousands of times lower than occupational exposure during the 1950s and 1960s.

The EPA, in April 1990, admitted that the health risks from exposure to in place asbestos “could be negligible or even zero.” The EPA concluded that based on current data, “very few of us, given existing controls, have contracted or will contract, an asbestos-

168. See, e.g., Crump & Farrar, supra note 96, at 51 (concluding that no statistically significant difference exists between indoor and outdoor ambient asbestos concentrations).
170. EPA, STUDY OF ASBESTOS-CONTAINING MATERIALS IN PUBLIC BUILDINGS: A REPORT TO CONGRESS (Feb. 1988).
171. Id. at 12.
173. Id. at 56.
174. Id.
related disease at these low prevailing levels."176 The New York Times, similarly, quoted a Congressional Report, stating that "[u]ndisturbed and undamaged asbestos is likely to expose office workers and most building occupants to no more health risk than if they were outdoors . . . ."177 "[R]ecent epidemiologic studies of persons with low exposure to [chrysotile] asbestos . . . provide little support for the concept that there is an increased risk of lung cancer when asbestos concentrations are at levels several hundred or thousand times lower than those found in workplace situations in the past."178

The above data clearly establish that ambient indoor asbestos levels are not significantly high so as to pose a health risk to building occupants. This is established even more clearly when it is recalled that 95% of the in place asbestos consists of chrysotile fibers. However, despite the above, one fiber phobia persists, and building owners continue to abate asbestos at extremely high costs. These costs not only include the money spent on removal and the disruption to building life, but studies indicate than in most instances abatement of even damaged ACM increases the health risks to building occupants.

VII. POST-ABATEMENT AMBIENT ASBESTOS LEVELS

The largest threat to building inhabitants is poor work practices by the abatement contractors.179 During abatement, tremendous amount of asbestos fibers are disrupted and become respirable. If care is not taken during the abatement, post-removal ambient asbestos levels can be higher than at any time prior to abatement. However, even where the abatement is conducted at the highest professional levels, there is still a likelihood that post-abatement levels will equal pre-abatement levels.180

There have been five reliable studies that have compared pre- and post-abatement ambient asbestos levels.181 In an EPA-sponsored

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176. Id. at 78-79.
177. Stevens, supra note 25, at D24.
178. Mossman & Gee, supra note 4, at 1724.
180. According to Crump, even in a best case scenario, abatement is projected to increase rather than decrease risk in a significant percentage of buildings. Id. at 192-93; see also Burdett et al., supra note 145, at 288-89 (setting forth guidelines and risk estimates against which to weigh abatement).
study, researchers measured asbestos levels at twenty-two sites in four schools, both before and after the removal of asbestos-containing acoustical plaster.\textsuperscript{182} Samples were collected before removal during periods of regular activity, during removal, immediately after removal, and five months after removal during periods of regular activity. The geometric average of fiber concentrations measured by TEM, expressed in mass, were at the same 0.2 ng/m\textsuperscript{3} level both before and five months after abatement.\textsuperscript{183} This, of course, was not statistically significant, nor a reduction in fibers.

In an 1988 EPA school study, researchers measured asbestos levels at thirty-nine sites in six schools, both before and after the removal of asbestos-containing acoustical plaster from the ceilings and walls.\textsuperscript{184} Samples were collected before removal, during periods of regular activity, during removal, immediately after removal, and four months after removal during periods of regular activity. The geometric average mass concentrations for samples taken at asbestos sites before removal measured 22.2 ng/m\textsuperscript{3}. Immediately after abatement the mean fell to 0.7 ng/m\textsuperscript{3}. However, within four months, concentrations were at 28.7 ng/m\textsuperscript{3}. Overall airborne levels were 32\% higher after four months, and four of the six schools had higher average airborne concentrations than before abatement.\textsuperscript{185}

In a study conducted by the Ontario Royal Commission on Asbestos, researchers measured asbestos levels at twenty-four sites in eight buildings, both before and after the removal of asbestos-containing fire-proofing.\textsuperscript{186} Samples were collected before removal during periods of regular activity, during removal, immediately after removal, and at two buildings several months after removal during periods of regular activity. In building one, pre-abatement levels of air concentration were recorded at 0.703 ng/m\textsuperscript{3}, but several months later levels were found to exceed 1 ng/m\textsuperscript{3}. In building eight, pre-abatement levels were 0.13 ng/m\textsuperscript{3}. However, post-abatement levels were more than 660 ng/m\textsuperscript{3}, or 6000\% higher. A complete recleaning of the building

\textsuperscript{182} J. Chesson et al., Evaluation of Asbestos Abatement Techniques, EPA 560/5-85-019 (1985).
\textsuperscript{183} Id.
\textsuperscript{184} Crump, supra, note 49, at 173-75 (citing non-published report, R. Tuckfield, Evaluation of Asbestos Abatement Techniques, EPA 560/5-88-008 (1988)).
\textsuperscript{185} Id. at 174.
\textsuperscript{186} Crump, supra note 49, at 174-75 (citing D. Pinchin, Asbestos in Buildings (1982)).
was required.187

In a study in the United Kingdom, researchers measured asbestos levels at multiple sites in two buildings, both before and after the removal of asbestos-containing insulation.188 Samples were collected before removal, during periods of regular activity, and about five months after removal during periods of regular activity. Pre-abatement ambient asbestos levels in building one were less than 0.0002 f/ml (measured by direct TEM).189 Approximately eighteen weeks later, ambient levels doubled. Pre-abatement ambient asbestos levels in building two were less than 0.0001 f/ml, or undetectable. Approximately thirteen weeks later, ambient levels jumped to 0.001 f/ml; after twenty-one weeks, ambient levels on the first floor were 0.003 f/ml, on the second floor were 0.065 f/ml, and on the third floor 0.104 f/ml.190

Finally, another study191 measured the ambient levels at nine sites following abatement. Samples taken several months after removal revealed that only one of the nine met EPA cleanup levels.192

These studies reveal that, at best, asbestos abatement in buildings has a negligible effect on ambient fiber levels for up to six months after removal. Where removal is done poorly, however, ambient fiber levels can greatly exceed pre-abatement levels and significantly increase the health risks to building occupants. Even where exposures begin to decrease after time, residual concentrations can be present for some months or even years.193

Again, despite the mounting evidence that in place asbestos is not a significant health risk to building occupants, that chrysotile asbestos fibers are not harmful at the low levels found in buildings, and that abatement often merely increases ambient fiber levels, building owners continue to abate and seek damages. In addition to the above scientific evidence, to date, the most compelling data concerns comparative risk.

187. Id.
188. Burdett et al., supra note 145, at 277-290.
189. Id. at 279.
190. Id. at 279-88.
192. Id.
VIII. COMPARATIVE RISK

At this point it is clear that a defendant in an asbestos-related property damage litigation employing the above information is attempting to convince a jury, that not only is chrysotile asbestos in buildings not dangerous, but that it is more dangerous to remove the asbestos fibers than to contain it. The building owner is likely pursuing the litigation on one of the following theories of liability: negligence, strict products liability, restitution, breach of warranty, or nuisance. The Amphibole Hypothesis defense, however, addresses these theories well with the argument that the risk posed by the asbestos is insignificant. In addition to defending on the scientific evidence presented above, defendants have found it persuasive to introduce comparative risk evidence.

One of the central battlegrounds of these litigations, in fact, is the admissability of the evidence of comparative risk. Plaintiffs have attempted to exclude this powerful evidence as irrelevant, but without it a jury may be misled into perceiving a disproportionate risk.

As a matter of law, comparative risk evidence is gaining acceptance in the courts. Most recently, a Pennsylvania trial court, while granting a motion for a new trial, affirmed its decision allowing comparative risk evidence. At trial, a defense expert witness in the field of bio-statistics testified, comparing the risk of exposure to airborne asbestos fibers to the risks of ordinary everyday activities. The court reasoned that the testimony was helpful to the jury and provided an important counterweight to the plaintiff’s experts who “routinely explain the dangerous and serious health hazards which can result from exposure to asbestos fibers, including horrendous suffering from long-term illness and even ultimate death.” In short, the judge believed that the comparative risk evidence, in light of new scientific developments, was necessary to “even the playing field” for the defense.

Comparative risk evidence is as it sounds: it compares the risk from exposure to asbestos fibers in buildings to other risks. Before

194. Id. at 192-93.
195. Hardy, supra note 175, at 70.
196. Id.
198. Id. at 9.
risks can be compared, however, the risks must be established. This is done by quantitative assessment of risk.\textsuperscript{199} Risk is established by a formula that incorporates exposure studies with the standardized mortality ratio ("SMR") for the particular ailment. SMR is the ratio of the observed ("O") number of cases to the number of expected ("E") in the absence of the ailment, multiplied by 100. Algebraically, this is represented by the equation \( \text{SMR} = 100(O/E) \). Since the number of observed cases and expected cases, in the absence of the ailment, should be zero, \( \text{SMR} = 100 \). The risk equation, therefore, is written as: \( \text{SMR} = 100 + bx \), where \( b \) is the slope of the line and \( x \) is the exposure to the ailment.

The data used to calculate asbestos exposure risk originates from studies that suggest asbestos-related diseases is linearly related to cumulative asbestos exposure.\textsuperscript{200} The equation, expressed for asbestos risk would be: \( \text{SMR} = 100 + bx \), where \( b \) remains the slope of the line and \( x \) is the cumulative exposure (f/ml - yr) to asbestos. Thus, if the slope were two and exposure were 10 f/ml - yr, then the SMR would be 120\%, i.e., the observed amount of cases is 120\% of the number cases expected.\textsuperscript{201}

The slope, \( b \), is determined from epidemiological studies that have demonstrated a dose-response relationship, by calculating risk for a population on the basis of cumulative exposure.\textsuperscript{202} Errors in risk quantification can occur as a result of an incomplete population trace, inaccurate causes of death, and the use of an inappropriate comparison population for the extended mortality rates. Although measuring worker exposure is difficult, there are seven industrial cohorts for which a pattern of risk has been observed for categories of increasing cumulative exposure.\textsuperscript{203} The industrial cohorts, however, are difficult to extrapolate down to the low level of non-occupational exposure. Although the models are built on the assumption of a linear progression, "the evidence to date is quite limited,"\textsuperscript{204} and cannot address the question of a difference in risk between chrysotile fibers and amphibole fiber.


\textsuperscript{200} Id.

\textsuperscript{201} Id.

\textsuperscript{202} Id.

\textsuperscript{203} Id.

\textsuperscript{204} Id. at 7.
In school asbestos exposure, which is similar to public building exposure, among a cohort of one million students exposed to mixed asbestos fibers, a total of five life-time excess deaths can be expected, or 1.5 life-time excess deaths if exposure is to only chrysotile fibers. The researchers reached these numbers by basing their equation on the recent report of the Ontario Royal Commission of school ambient fiber levels. Based on this report, a mean exposure level of 0.003 f/ml was selected. Students will be enrolled in schools with asbestos-containing materials for varying numbers of years, but an average enrollment term is estimated at six years. A school year (thirty-six weeks per year, thirty-five hours per week) is equivalent to 0.656 of a work year (forty-eight weeks per year, forty hours a week). Accounting for exposure factors, a slope of 0.5 was assessed to be the closest estimate, although admittedly an overestimate. Thus, the equation is: \[ SMR = 100 + \frac{0.5}{0.003} \].

Employing the quantitative assessment of risk model, researchers have analyzed the risk of death from many activities. Mossman compared annual risk of death from exposure to 0.000024 f/cc (the mean asbestos airborne concentration in schools) for five school years, beginning at age ten, to various activities. In descending order of risk, the annual rate of death per one million Americans, will be 1,200 from smoking, sixty (ages one to fourteen) from home accidents, thirty-two pedestrians (ages five to fourteen) from car accidents, twenty-seven (ages five to fourteen) from drowning, ten from high school football injuries, six from aircraft accidents, one to six from whooping cough vaccines, and finally, 0.005 to 0.093 from five years of average school-age asbestos exposure.

A Harvard University asbestos symposium, however, chose to compare risk in a different manner. The symposium expressed risk of death from several activities by calculating how many per 100,000 Americans would die from the activity before age sixty-five. Expressed in this format, in descending order of risk, the number of Americans per 100,000 that will die prematurely are 21,900 from smoking (all causes), 8,800 from smoking (cancer only), 1,600 from motor vehicle accidents, 730 frequent flyers, 441 from coal mining accidents, 400 from indoor radon, 290 pedestrians from car accidents, 200 from second-hand smoke, seventy-five from diagnostic x-rays, seventy-five from bicycle accidents, seven from consuming New York

205. Id.
206. Mossman et al., supra note 4, at 299.
City tap water, three from contact with lightening, three from hurricanes, and one from asbestos in school buildings.\footnote{Slutsker, supra note 47, at 303.}

The risk associated with exposure to asbestos in buildings must be put into its proper perspective. Expressed in either format, comparative risk evidence is powerful and places the health risk from asbestos exposure in a new light. Words may express the different morphological characteristics between asbestos fiber types; however, telling a jury that common, ordinary diagnostic x-rays are seventy-five times more likely to kill than exposure to in place asbestos, gets the point across. Armed with this information, a juror will be likely to think twice before returning a multi-million dollar abatement verdict.

**IX. CONCLUSION**

The available experimental and epidemiological data supports the proposition that both asbestos fiber type and fiber size are important factors in the pathogenicity of asbestos. While the public’s insight into the effects of asbestos are cloaked by the history of occupational asbestos exposure,\footnote{Commins, supra note 14, at 476.} recent studies employing enhanced microscopy technology bolster the conclusion that exposure to chrysotile asbestos at current ambient levels does not constitute a significant health risk.

In direct contradiction to the accumulated evidence, building owners are abating at a $3 billion a year pace, and suing all healthy corporations associated now or in the past with the manufacture, production, or distribution of asbestos-containing materials. However, building owners’ traditional causes of action, sounding in negligence and strict products liability, are now being met head-on by the Amphibole Hypothesis. This defense theory relies on recent scientific evidence that shows that asbestos fiber concentrations inside buildings, even those with damaged asbestos-containing materials, are comparable to environmental levels, are extremely low in absolute terms, and are orders of magnitude below the historical levels that were associated with occupational disease. Further, this line of defense is supported in easy-to-understand terms of comparative risk evidence, which allows jurors to compare the risks associated with asbestos in buildings with the risks of being struck by lightening, exposed to cosmic radiation, or injured during the routine task of driving a car.

Taken together, this information simply means that asbestos
property damage litigations will continue to be costly, complicated, and time consuming, and that the defense has a substantial case that may persuade a jury. Asbestos-related property damage litigations will no longer be about the sole issue of damages: now, there will be a new focus on the merits of the liability claim.

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