Defending "Household," Building, and Neighborhood Exposure Cases with Tenuous Nonoccupational Exposure Histories

A Review of Medical and Scientific Literature

Bruce Bishop
Kevin Greene
Eric Cook
Michele Dallman
Willcox & Savage, P.C.
440 Monticello Avenue
Suite 2200
Norfolk, Virginia 23510
(757) 628-5500
Fax (757) 628-5566
bbishop@wilsav.com
kgreene@wilsav.com
ecook@wilsav.com
mdallman@wilsav.com
BRUCE BISHOP is a member with Willcox & Savage, P.C., and is an active member of the firm's Product Liability/Toxic Torts and Environmental Litigation Practice Teams. Bruce has extensive experience in toxic tort and other environmental litigation, primarily in the asbestos personal injury and property damage area, dating from 1977.

Bruce is a member of the Federal Bar Association, Tidewater Chapter (President 1980–81), and is also a member of the Defense Research Institute, the Virginia Association of Defense Attorneys, the Virginia Bar Association, the International Association of Defense Counsel, the American Board of Trial Advocates, and the American Bar Association. He also served on the faculty of the 1996 National Trial Academy in Boulder, Colorado, sponsored by the International Association of Defense Counsel. He has been listed in The Best Lawyers in America (Personal Injury Section) since 1991. In addition to the Bar of the Commonwealth of Virginia, Bruce is a member of the bars of the State of West Virginia and the Commonwealth of Pennsylvania.

Bruce received his B.A. from Old Dominion University in 1973 and his J.D. from The University of Virginia in 1976.
## Table of Contents

I. Introduction.................................................................................................................................3

II. "Traditional" Household Exposure: Wives and Children of Asbestos Workers in Homes of Asbestos Workers .................................................................3

A. Household Exposures Causing Mesothelioma Are Usually in Wives or Children of Persons Who Worked Directly with Asbestos and Who Had Lengthy Exposures .................................................................4

B. Mesotheliomas in Children Are Not Asbestos-Related .....................................................12

C. A Significant Percentage of Mesothelioma Cases Have No Known Cause and Are Termed “Idiopathic” ......................................................................................13

D. The Lack of Pleural Plaques May Indicate That a Mesothelioma Is Not Asbestos-Related ..........................................................................................................................20

E. Pleural Plaques Can Be Found in a Majority of Persons with Mesothelioma .................................................................................................................................21

F. To Attribute a Mesothelioma to Asbestos, the Household Member’s Exposure Must Be Significant .................................................................................................22

G. Household Exposures in Current Personal Litigation Cases are Usually Quite Low .................................................................................................................................23

III. Exposures in Buildings ...........................................................................................................25

IV. Neighborhood Exposures .......................................................................................................28

V. Ambient Air/Background Exposures .........................................................................................40

VI. Special Approaches in Female Cases ......................................................................................44

VII. Genetic Predisposition ...........................................................................................................47

VIII. Fiber Counts and Types ..........................................................................................................50

IX. Legal .........................................................................................................................................57

X. Exposures From Home Remodeling and Brake Work ...........................................................61

XI. Bibliography ..............................................................................................................................65

XII. Appendix ....................................................................................................................................88
(A) Table 1 from Donovan, et al., "Evaluation of take-home (para-occupational) exposure to asbestos and disease: a review of the literature" Critical Reviews in Toxicology 42:703-731(2012) ..................................................................................................................88

(B) Household Exposure/Mesothelioma Literature Excerpts .................................................................97

(C) State-by-State Survey of "Take Home" Asbestos & Toxic Tort Exposure Cases by Craig T. Liljestrand, Hinshaw & Culbertson LLP (2016)................................................................................................................................145


(E) Outline of Suggested Exposure Questions from an IH.................................................................145

(F) Key Questions to Ask Household Members and/or Workers Allegedly Carrying Asbestos Home.........................................................................................................145
I. Introduction

Defendants continue to be besieged by thousands of asbestos cases, but the types of exposures claimed have changed considerably. There has been an increase in mesothelioma cases filed claiming nontraditional exposures in settings outside the workplace, including homes, schools, and neighborhoods. This presentation deals with such nonoccupational claims.

Consider the following scenarios:

Case A – The wife of a shipyard electrician sues for asbestosis on the basis that she washed her husband's work clothes on a weekly basis for 20 years.

Case B – A male office worker, aged 35, develops a plural mesothelioma and sues numerous asbestos manufacturers, claiming that his disease was caused by exposure to in-place asbestos in elementary and secondary schools he attended as a child.

Case C – A 38-year-old woman develops what appears to be peritoneal mesothelioma. She claims that her disease was caused by exposure to her grandfather, a mechanic at a boiler plant, during visits to her grandfather's home as an infant and primary school child.

Case D – A 52-year-old woman with no history of occupational exposure develops mesothelioma and files a claim based in part on exposure to asbestos from an asbestos manufacturing facility within one-half mile of her childhood residences.

Case E – A 43-year-old woman diagnosed with peritoneal mesothelioma claims exposure to joint compound when a kitchen and bathroom in the house in which she grew up was remodeled in the mid-1970s when she was 7 years old.

These are not "slam dunk" cases for plaintiffs' counsel, in that they face a considerable challenge proving such remote exposures. However, these are all high profile cases. They tend to involve younger people, often with small children. In the case of mesotheliomas, they frequently undergo aggressive surgical, chemotherapeutic, and radiological treatments. These are emotional cases for juries. These cases, where appropriate, require use of every available medical and scientific method to clarify for the jury the lack of a link to asbestos exposure.

This presentation will outline the medical and scientific literature available to construct a defense in such cases.

II. "Traditional" Household Exposure: Wives and Children of Asbestos Workers in Homes of Asbestos Workers

Mesothelioma is an extremely rare disease. There are approximately 3,000 cases diagnosed annually in the United States in a population of 260 million. By way of comparison, there are 160,000 lung cancer deaths in the U.S. each year. Mesothelioma, although rare, is seen in persons with nonoccupational exposures. Indeed, the very first report by J.C. Wagner et al. associating pleural mesothelioma with crocidolite asbestos in South Africa included a number of individuals who never had direct occupational exposure to crocidolite fiber, but who either lived with persons who worked in asbestos mines, or who lived in the vicinity of the mines.
A. Household Exposures Causing Mesothelioma Are Usually in Wives or Children of Persons Who Worked Directly with Asbestos and Who Had Lengthy Exposures

Selikoff's 1965 article, "Relation Between Exposure to Asbestos and Mesothelioma" in the New England J. of Medicine, 291(1965):583-84, offered examples of an individual with mesothelioma who denied ever having seen, used, or handled any asbestos product. Despite this, both asbestos bodies and fibers were found in his lung at autopsy. Another case involved an individual who had worked in a dry cleaning plant as a "spotter." About the same time, Newhouse and Thompson in Britain published a study of 83 male and female patients with pleural and peritoneal mesothelioma. While many had been employed in occupations directly related to asbestos, nine had relatives who worked with asbestos. They found that:

"the most usual history was that of the wife who washed her husband's dungarees or work clothes. In one instance a relative said that the husband, a docker, came home "white with asbestos" every evening for three or four years and his wife brushed him down." (p. 264)

Both male domestic exposures were boys of nine or ten years old when their sisters were working at an asbestos factory. One of the sisters, who worked as a spinner from 1925 to 1936, died of asbestosis in 1946. Her brother, who was exposed when she returned home with dust on her clothes and who never had any occupational exposure himself, died of pleural mesothelioma in 1956. In 1965, Newhouse and Thompson were able to state, "there seems little doubt that the risk of mesothelioma may arise from both occupational and domestic exposures to asbestos." Newhouse and Thompson, "Mesothelioma of Pleural and Peritoneum Following Exposure to Asbestos in the London Area," British J. of Indus. Medicine, 22(1965):261-66, at 266.

Numerous reports began to enter the medical literature of wives developing mesothelioma after washing the asbestos-laden clothes of their husbands. See, for instance, Lillington et al "Conjugal Malignant Mesothelioma" in the New England Journal of Medicine in 1974 (where both husband and wife developed mesothelioma), and Anderson et al from Mt. Sinai whose report "Household-Contact Asbestos Neoplastic Risk" in the Annals of the New York Academy of Sciences in 1976 lists case reports up to that time. The Anderson et al article also reported on a clinical study of household contacts of asbestos workers to assess current health status, including chest x-rays and pulmonary function studies. Of the 326 household contacts, including wives, daughters, sisters, sons, brothers, mothers, fathers and cousins, 35 percent or 114 had some x-ray finding. Forty-two of the 114 only had pleural thickening. Since pleural thickening alone is not diagnostic of asbestos exposure, this finding in 42 persons may overstate the impact of asbestos in these household contacts. Nonetheless, this article demonstrates that household contacts of workers at a factory producing amosite asbestos products show asbestos-related pleural and parenchymal disease. In addition to the x-ray finding, two pleural mesothelioma deaths were identified: the daughter of a man who had worked with asbestos for more than 13 years, and the daughter of a man with five years' of asbestos work at the plant. Both women had latency periods of more than 30 years. At the time of the writing of the article, there were also two living household members of amosite workers who were being treated for mesothelioma. Anderson, H.A., et al., "Household-Contact Asbestos Neoplastic Risk," Annals of the N. Y. Academy of Sciences, 271(1976):311-23. A follow up report in 1979 by Anderson et al dealt with asbestosis among household contacts of amosite

Vianna and Polan's study of 52 females with malignant mesothelioma from New York showed a large number of husbands and fathers who had various types of asbestos exposure, including heat insulation workers, and a heat-electric wire worker. The authors concluded that the husband's occupation was clearly the most important risk factor. ("Nonoccupational Exposure to Asbestos and Malignant Mesothelioma in Females," *The Lancet*, May 20, 1978:1061-68.)

Epler *et al* reported on four cases of various asbestos-related diseases from household exposure. One wife of an asbestos worker developed a mesothelioma, and the other developed plaque calcifications and a benign asbestos pleural effusion, as well as subpleural parenchymal fibrosis. The wife with mesothelioma had brushed white dust from her husband's work clothes twice weekly for many years. The second wife had been married to a man who had worked in an asbestos product factory beginning in 1939. He developed severe asbestosis and died of a pleural mesothelioma in 1967. The wife's only contact with asbestos occurred while cleaning her husband's work clothes, which were so laden with white powder that dusting had to be performed outside the house at certain times. The two male cases in this study were sons of a lagger and asbestos worker at a chemical plant who had also brought home asbestos sheets and "fluff" from the chemical plant; the father had a small business at home for repairing burned out mufflers with asbestos sheets. The sons had played in the material for many years. These household contacts were clearly intense, and in fact the childhood exposures described by the two male cases were more likely the equivalent of an occupational exposure. Both male cases demonstrated calcified plaques on x-ray. Epler, G.R., *et al*. "Asbestos-Related Disease from Household Exposure," *Respiration*, 39(1980):229-40.

In 1985, Kilburn *et al* reported a study entitled, "Asbestos Disease and Family Contacts of Shipyard Workers," in the *American Journal of Public Health*, 75:615-17, in which they claimed that 11.3 percent of the wives of shipyard workers had asbestosis, 7.6 percent of the sons had asbestosis, and 2.1 percent of the daughters had asbestosis. This study is very misleading, however, because the term "asbestosis" was defined as an x-ray reading of a profusion of 1/0 or greater, or pleural findings (pleural thickening, plaques, or calcification). In actuality, the numbers of true asbestosis (by x-ray only) were three wives and one son.

In contrast to the findings of Kilburn *et al.*, a study by Sider, L. *et al.* of 17 wives of insulation workers did show pleural abnormalities in the form of pleural thickening, plaques, and calcifications, but there was no evidence of parenchymal opacities or thoracic malignancies. Furthermore, pulmonary function tests revealed no significant "functional" impairment. The authors concluded that "of all the factors in our survey, the presence of pleural plaques appears to be most dependent on the interval since the household contacts' first exposure." In their study, the mean latency for development of plaques was 32.8 years, compared with 23.8 years for persons with occupational exposure. An interesting finding in this study was that three women had more severe radiographic abnormalities than their husbands. However, each of the three women gave a family history of at least one household contact in addition to her husband (a brother, a father, and a father and son). These additional contacts may have contributed significantly to the household exposure of these three women. Sider, L., Holland, E.A., Davis, T.M., Jr., and Cugell,

Up until the late 1980s, most of the evidence for household exposure came from case reports and, to a lesser extent, epidemiological studies. However, with the development of fiber analysis techniques to determine the quantity of asbestos fiber in human lungs, determination of etiology in a specific mesothelioma case could be more precise. Huncharek et al reported a lung fiber burden of the wife of a shipyard machinist who had dismantled boilers throughout his 34-year employment. She developed mesothelioma at age 76. The authors found that her lung asbestos fiber burden was similar to that seen in cases of mesothelioma associated with occupational exposure to asbestos by bystanders in the work setting. (Huncharek et al., "Domestic Asbestos Exposure, Lung Fibre Burden, and Pleural Mesothelioma in a Housewife, "*British J. of Indus. Medicine*, 46(1989):354-55.)

In the same year, in a letter to the editor, Li et al reported on the case of a 32-year-old woman who died of mesothelioma. In her case, cotton cloth sacks in which molded asbestos insulation had been wrapped were used by her parents to make diapers for her. The mother of this woman had laundered the diapers and her husband's work clothes, and she also died of mesothelioma at age 49. The father had been an insulator who died with asbestosis and cirrhosis of the liver at age 53. Thus, the daughter's contact with asbestos was not only through exposure to her father's clothing, but also to diapers made from the material surrounding the asbestos insulation, surely not a use intended by the manufacturer. Nonetheless, the exposure was clearly substantial in this family. Li, F.P., Dreyfuss, M.G., and Antman, K.H., "Asbestos Contaminated Nappies and Familial Mesothelioma, The Lancet, Apr. 22, 1989:909-10.

Another study by Kane et al. was published in 1989 focusing on malignant mesothelioma in young adults. Two of these individuals had occupational exposure, three have reported no asbestos exposure, and the remainder were exposed through their households. The two peritoneal mesotheliomas in the group had reported occupational exposure. Some of the occupational exposures were questionable, however, including one who had worked for a schoolteacher for 14 years in a building later "cited for asbestos." The meaning of "cited for asbestos" is unclear, and there was no attempt to quantify the number of asbestos fibers in these patients' lungs, calling into question the assumption of occupational exposure. Kane, M., Chahinian, A.P., and Holland, J.E, "Mesothelioma and Young Adults" *Cancer*, 65 (1990):1449-55.

A rather unique clustering of mesotheliomas in one family was reported by Danish researchers in the *British Journal of Industrial Medicine* in 1990. A father, mother, and son all died of mesothelioma between 1984 and 1987. The family produced in the basement of the home a special asbestos cement consisting of amosite, gypsum, and sand during the years 1944 to 1961. The basement had no ventilation and no protective equipment was used. The authors termed this "a massive exposure," and justifiably so. One could certainly not term this a typical "household" exposure. Otte, K.E., Sigsgaard, T.I., and Kjaerulf, J., "Malignant Mesothelioma: Clustering in a Family Producing Asbestos Cement in Their Home, "*British J. of Indus. Medicine*, 47(1990):10-13.
The first in-depth study which analyzed asbestos fiber content of lungs of persons exposed in the typical household setting was by Gibbs et al. Cases were chosen because the history of asbestos exposure was absent, indirect, or ill defined. Mineral content of the lungs were analyzed. The authors noted:

Since exposure to asbestos may precede the appearance of malignant mesothelioma by several decades, the exposure history may be unreliable; equally, if the persons involved are influenced by considerations of compensation in particular cases, then exposure to asbestos maybe overestimated.

The study pointed out that the mere assertion of exposure to asbestos may not be borne out by a scientific determination of asbestos content in lungs. Gibbs et al., "Nonoccupational Malignant Mesotheliomas," in Nonoccupational Exposure to Mineral Fibers, IARC Science Publication, 90(1989):219-28. This study will be explored more fully in Section VIII.

A brief summary of the literature on home contamination by asbestos may be found in Report to Congress on Workers' Home Contamination Study Conducted under the Workers Family Protection Act (29 U.S.C. 671(a)), published in September 1995 by the Public Health Service, the CDC, and NIOSH (NIOSH Publication No. 95-123).

A German study of six fatal pleural mesothelioma cases (five wives and one son of asbestos industry workers) detailed the exposure histories and latencies of each. Two of the husbands had developed asbestosis. In one case, the son regularly delivered a hot meal to his father at his work place between 1950 and 1959. The son's exposure, therefore, was intensified not only by exposure at home (his mother died also from mesothelioma), but by his exposure at the insulation mat manufacturing facility where his father was employed. Schneider et al, "Pleural Mesothelioma and Household Asbestos Exposure," Reviews on Env'tl. Health, 11(1-2)(1996):65-70.

After the 1990s, case reports and case series continued to report mesotheliomas as a result of take home/domestic exposure (Ampleford and Ohar, 2007, Bianchi, et al., 2001; Mirabelli, et al., 2008; Patel, 2008; Peretz, et al., 2009; Whitehouse, et al. 2008). More recently, a series of data from cancer registries have also been published (Langhoff, et al., 2014; Mensi, et al., 2015; Rake, et al., 2009; Reid, et al., 2008; Reid, et al., 2013). The consistent thread that runs through all of the studies published since Newhouse and Thompson in 1965 is that mesotheliomas from take-home exposure have been largely linked with occupations with high exposure to amphibole asbestos, including shipyard workers, miners, millers, asbestos cement factory workers, and insulators. A superb analysis of the take-home literature can be found in Donovan, McKinley, Cowan and Paustenbach, “Evaluation of take-home (para-occupational exposure) to asbestos and disease: a review of the literature,” Critical Reviews in Toxicology 42(9):703-731(2012). With the gracious permission of the authors, Table 1 from the article which summarizes all of the studies reporting disease (primarily mesothelioma) from para-occupational exposure is included as Appendix A. A 2013 review by Goswami, Craven, Dahlstrom, Alexander, and Mowat evaluated epidemiological studies of asbestos-related disease or conditions among domestically exposed individuals and exposure studies that provided either direct exposure measurements or surrogate measures of asbestos exposure. Goswami, et al., “Domestic Asbestos Exposure: A Review of Epidemiologic and Exposure Data,” Int. J. Environ.
It found a relative risk estimate of 5.02 (95% confidence interval: 2.48-10.13) for persons domestically exposed via workers involved in occupations with a traditionally high risk of disease from exposure to asbestos (i.e., asbestos product manufacturing workers, insulators, shipyard workers, and asbestos miners).

It should be emphasized that the opportunity for take-home exposures diminished greatly in the early 1970s in the United States with the advent of OSHA as well as the removal of asbestos from thermal insulation. The 1972 OSHA requirements required change rooms for industries in locations where exposures were in excess of the OSHA permissible exposure limit for asbestos:

“The employer shall provide two separate lockers or containers for each employee, so separated or isolated as to prevent contamination of the employee’s street clothes from his work clothes.” (OSHA, 1972, p. 11321).

As stated in the Donovan article supra, “The standard also instructed the employer in the handling/transport of contaminated clothing (“in sealed impermeable bags”) and in laundering practice “shall be done so as to prevent the release of airborne asbestos fibers in excess of the exposure limits.” (OSHA, 1972, p. 11322). *Id.* at 706.

A number of studies have calculated relative risk estimates for asbestos-related disease (principally mesothelioma) among individuals who have experienced take-home exposures to asbestos. Once again, the excellent review article by Donovan, et al., provides a very useful summary in table form, a copy of which is included below.

The follow-up of the cohorts of wives of Casale, Monferrato (Italy) asbestos-cement factory workers (where Crocidolite was used in addition to chrysotile in the production of asbestos cement pipe), found an increased risk of pleural mesothelioma among the workers’ children (odds ratio = 7.4; CI = 1.9-28) (Magnani, *et al*, 2001). A meta-analysis of 8
mesothelioma studies (which included not only para-occupational but also environmental exposures) yielded a summary relative risk of 8.1 (CI = 5.3-12) for domestic exposure (Bourdes, et al, 2000). However, consistent with the fact that most household exposure mesothelioma cases have been the result of take-home exposure from individuals occupationally exposed to heavy amphibole asbestos,

"It is encouraging that the number of reports in recent years (Bianchi, et al, 2004; Miller, 2005; Ampleford and Ohar, 2007; Ferrant, et al 2007; Patel, et al, 2008) has seemingly decreased, which may be attributed to societal changes (i.e., the number of women who wash their husband's clothes has declined during the past 30 years). While there are more case reports detailing MM in women, due to their anecdotal nature, not all of them are reported here. It should be noted that the levels of indirect exposure where particularly high during the first half of the well before measures to reduce exposure and the risk that workers carried asbestos home were implemented in the workplace in the U.S. and Europe." Carbone, Dodson et al, "Malignant Mesothelioma: Facts, Myths, and Hypotheses," J Cell Physiol., 227:44, 49(2012)

Marchevsky, Harber, Crawford and Wick in their review article, "Mesothelioma in Patients with Non-Occupational Asbestos Exposure – An Evidence-Based Approach to Causation Assessment," Annals Diagnostic Pathology, 10:241-250 (2006) performed a systematic literature review yielding 1,028 cases of putative non-occupational asbestos exposure and mesothelioma. According to the authors, only 287 of those reports had a defined single exposure to a household, building occupant, or neighborhood/community asbestos source. There was insufficient information to perform a meta-analysis. However, the "available 'evidence' was used to develop semi arbitrary evidence-based causation guidelines for the assessment of putative association between MM and NOAE (non-occupational asbestos exposure)" at p. 241. In analyzing the household group, the 4 largest occupations of asbestos workers noted in the study were NOS (not otherwise specified) asbestos worker (61), asbestos product manufacturing (26), shipyard (27) and insulator (10). This was followed by cement worker (7) and pipefitter (6). Id. at 244. In identifying the type of asbestos noted in the studies, they found that 16 involved chrysotile only, 2 amosite only, 1 chrysotile only, 23 to mixed fiber types an 108 which were unspecified. Id. at 244. The authors emphasized the inadequacy of the information in most of the literature reviewed.

"This review shows that the literature supplies only limited and incomplete information on the possible association between MM and NOAE. As indicated in Table 3, most of the MMs reported in this context have been encompassed in small case series that frequently lacked control groups, details on asbestos exposure, asbestos fiber type(s), and other data needed to distinguish NOAE-related MM and idiopathic mesothelial neoplasms. Moreover, NOAE-associated MMs in epidemiological reports often were vaguely defined with regard to modes of pathologic diagnosis . . . Information on the duration of exposure, tumor latency, and fiber type(s) was represented in a minority of HOE and BOE (building occupant exposure), MM patients, but fiber type information was available in most of the NCE (neighborhood/community group) cases. Approximately 70% of the latter appeared to be associated with exposure to
Crocidolite. Chrysotile-only asbestos exposure was reported in only 2 of 106 NCE patients. Only 13 cases of peritoneal MM were present among all of the citations in this review; those neoplasms are generally believed to be either idiopathic, or they are associated with extremely high tissue burdens of amphiboles." *Id.* at 247.

The authors also examined the Helsinki criteria and found that "those criteria are much too general for use in assigning attribution of causation in individual cases of MM." *Id.* at 248

An analysis of all the cases of mesothelioma in the Italian Mesothelioma Registry from 1993 through 2008 identified 1,232 individuals who were not occupationally exposed to asbestos, of which 530 (4.4%) of malignant mesothelioma cases had familial exposure, i.e., they lived with a person who was occupationally exposed and 514 mesothelioma cases (4.3%) with environmental exposure to asbestos, i.e., they lived near sources of asbestos pollution and had not been occupationally exposed. The researchers estimated that the proportion of malignant mesothelioma cases due to non-occupational asbestos exposure (familial, environmental or related to leisure activities) was 10.2% at the national level, although the figures may be misleading because individuals could have more than one source of exposure. Table 2 is included below which identifies the cases of malignant mesothelioma collected by the National Mesothelioma Register divided into familial, environmental or leisure activity exposure and gender:

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Modalities of exposure of malignant mesothelioma (MM) cases (N, %) collected by the National Mesothelioma Register (ReNaM) disentangled by familial, environmental or leisure activity exposure categories and gender (Italy, 1993–2008)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Number of exposures*</td>
</tr>
<tr>
<td>Parents</td>
<td>74</td>
</tr>
<tr>
<td>Husband/wife</td>
<td>3</td>
</tr>
<tr>
<td>Son/daughter</td>
<td>5</td>
</tr>
<tr>
<td>Other cohabitants</td>
<td>18</td>
</tr>
<tr>
<td>Overall</td>
<td>100</td>
</tr>
<tr>
<td>Environmental (514 MM cases)</td>
<td></td>
</tr>
<tr>
<td>Asbestos cement plant</td>
<td>109</td>
</tr>
<tr>
<td>Railways</td>
<td>18</td>
</tr>
<tr>
<td>Rail stock building, repair and demolition plant</td>
<td>10</td>
</tr>
<tr>
<td>Docks</td>
<td>8</td>
</tr>
<tr>
<td>Shipbuilding and repair</td>
<td>8</td>
</tr>
<tr>
<td>Steel industry plants</td>
<td>2</td>
</tr>
<tr>
<td>Chemical or petrochemical plants</td>
<td>7</td>
</tr>
<tr>
<td>Mines or mills</td>
<td>7</td>
</tr>
<tr>
<td>Others</td>
<td>73</td>
</tr>
<tr>
<td>Overall</td>
<td>236</td>
</tr>
<tr>
<td>Use of asbestos materials containing asbestos</td>
<td>29</td>
</tr>
<tr>
<td>Home mortality</td>
<td>25</td>
</tr>
<tr>
<td>Thermal insulation at home</td>
<td>9</td>
</tr>
<tr>
<td>Leisure (188 MM cases)</td>
<td></td>
</tr>
<tr>
<td>Plumbing or electric repair at home</td>
<td>2</td>
</tr>
<tr>
<td>Car repair</td>
<td>4</td>
</tr>
<tr>
<td>Other activities</td>
<td>15</td>
</tr>
<tr>
<td>Overall</td>
<td>84</td>
</tr>
</tbody>
</table>

*The number of exposures exceeds the number of mesothelioma cases due to the possibility of multiple exposures for a single case, including exposures due to the presence of asbestos in objects not used in a working context (eg, ironing boards, rural tool sheds).

As you can see, the largest percentage among men was household exposure from a parent whereas in women, the largest source of household exposure was a spouse.

While there is no doubt that significant household exposure has been a cause of pleural plaques and mesothelioma, the asbestos exposures reported in the medical literature are by and large quite high and cover long periods of time. The workers who brought home the fibers on their clothes were persons with very substantial exposures at work. These are not the exposures of persons with light bystander or building environment exposures. The best proof of this is found in fiber analyses of the lungs of persons with mesothelioma. (See Section VII.) When fiber counts demonstrate a fiber burden at background levels, the mesothelioma should be attributed to other causes or viewed as idiopathic. This is an especially likely finding in women, since the number of asbestos cases in women has stayed flat over the past few decades, and only about a quarter of mesotheliomas in women is thought to be asbestos-related. (See Section VI.) It should be emphasized that only a very small proportion of female mesotheliomas can be attributed to domestic asbestos exposure. For example, Spirtas (1994) estimated that 7.9% of female mesotheliomas are attributable to living with a co-habitant who was ever exposed to asbestos. Rake (2009) estimated that 16% of female cases of mesothelioma are attributable to living with a worker who held a job with a potential for significant asbestos exposure.

While only a few studies have addressed the issue of lung cancer and take-home asbestos exposures, the study of the cohort of the wives of workers at an asbestos cement factory that utilized both Crocidolite and chrysotile asbestos did not detect a significantly increased incidence of lung cancer among the household contacts (12 observed versus 10.3 expected; SMR = 1.17; 95% CI 0.60-2.04). Ferrante, Dodesco, Miribelli and Magnani, "Cancer Mortality and Incidence of Mesothelioma in a Cohort of Wives of Asbestos Workers in Casale, Monferrato, Italy," Environ Health Perspect, 115:1401-1405 (2007). See also Reid, Heyworth, N De Klerk, and Musk, "The Mortality of Women Exposed Environmentally and Domestically to Blue Asbestos at Wittenoom, Western Australia," Occup Environ Med, 65:743-749 (2008) which found mortality from lung cancer was not significantly associated with cumulative asbestos exposure in a cohort of women environmentally and domestically exposed to Crocidolite fiber in Wittenoom, Australia. The authors note that "other studies that have examined communities environmentally exposed to asbestos have also failed to report an excess risk of mortality from lung cancer." Id. at 748 (citing Hammond, Garfinkel and Selikoff, "Mortality Experience of Residents in the Neighborhood of an Asbestos Factory," Annals of NY Acad Sci, 330:417-22 (1979); Camus, et al, "Non-Occupational Exposure to Chrysotile Asbestos and the Risk of Lung Cancer," N Eng J Med, 338(22):1565-71 (1998)). Additionally, Reid, A., Franklin, P., Olsen, N., Sleith, J., Samuel, L., Aboagye-Sarfo, P., de Klerk, N., Musk, A., “All-Cause Mortality and Cancer Incidence Among Adults Exposed to Blue Asbestos During Childhood,” American Journal of Industrial Medicine 56(2):133-145 (2013) found that there was no increased lung cancer incidence or mortality in a cohort of men and women who were environmentally and domestically exposed to Crocidolite fiber in Wittenoom, Australia as children.

In summary, the following points should be emphasized:
1) There clearly have been mesotheliomas caused by household exposure to asbestos brought home on the work clothes of asbestos workers. In the vast majority of instances, the occupations involved are those associated with heavy exposure to asbestos, including asbestos cement factories, insulators and shipyard workers.

2) The vast majority of household exposure mesotheliomas are pleural and not peritoneal. For example, in the cohort of women and girls documented to have lived in the blue asbestos mining and milling township of Wittenoom between 1942 and 1992 who were not involved in asbestos mining and milling, all of the mesotheliomas in the females were pleural. In contrast, there were a number of peritoneal mesotheliomas in the miners and millers of blue asbestos at Wittenoom.

3) The number of asbestos-related household exposure mesotheliomas is dramatically declining.

4) Almost all asbestos-related household contact/mesotheliomas involved exposure to commercial amphibole fiber.

5) The majority of asbestos-related household contact mesotheliomas occurred in female spouses, although an appreciable number occurred in daughters as well. A much smaller number occurred among sons, and any alleged household contact mesothelioma in a male today should be approached with a considerable amount of skepticism.

6) Given the low percentage of female mesotheliomas which are asbestos-related in the United States, "it can be very difficult to conclusively link the incidence of disease in a household member with a para-occupational exposure to asbestos, unless lung burden data are collected and linked with workplace exposures of the primary worker." Donovan, et al, supra at 706.

7) There is no proof that lung cancer risks are elevated as a result of household asbestos exposures.

8) Always keep in mind that "it is likely that the vast majority of asbestos-induced diseases in the industrialized world are caused by occupational rather than non-occupational asbestos exposure (International Agency for Research on Cancer, 1987; McDonald and McDonald, 1996; Committee on Asbestos, 2006)." Goldberg and Luce, "The Health Impact of Non-Occupational Exposure to Asbestos: What do we Know?" European Journal of Cancer Prevention, 18:489,490 (2009)

For quotes in chronological order from the most relevant literature addressing household exposures, please see Appendix B.

**B. Mesotheliomas in Children Are Not Asbestos-Related**

Mesothelioma in children is extremely rare but does exist. Children are by definition too young to have had the required minimum latency for development of asbestos-related mesothelioma. Existence of these tumors in childhood demonstrates the fact that mesotheliomas do happen outside exposure to asbestos. An interesting study by Cooper et al provided a
population based incidence rate of childhood mesothelioma in the United States. The study discussed other potential etiologic factors in the development of mesothelioma. Radiation for prior malignancies is the predominant etiology. The article listed 15 radiation-related mesothelioma cases which have been reported in adults, three of which followed radiation therapy for Wilms tumor during childhood. (Wilms tumor is a tumor of the kidneys which usually develops by age five, but may occur rarely in the fetus and in later life.) Unfortunately, the “precise role of radiation in the induction of mesothelioma remains unknown.” The paper also notes that INH administered in high doses to rats and mice has shown to induce tumors of the lung, liver, lymph nodes, and other sites, and may induce pulmonary tumors in offspring when administered to pregnant animals. There is one report of a mesothelioma reported in a nine-year-old boy whose mother had been given INH during pregnancy. This article concluded that:

Mesothelioma in children, as well as in adults, is likely to have a multi-factorial etiology. Radiation, prenatal medications, and genetic factors are all possible etiologic agents in childhood mesothelioma. In addition, other, as of yet unspecified environmental factors may play a role in this disease. (Italics in original)


See also Berk, S., Yalcin, H., Dogan, O., Epozturk, K., Akkurt, I., Seyfikli, Z., “The assessment of the malignant mesothelioma cases and environmental asbestos exposure in Sivas province, Turkey,” Environ Geochem Health 36:55-64, 59 2014 (“The reasons for such cases were suggested to be related to genetic predisposition and other unknown factors other than asbestos exposure.”); Fraire, Cooper, Greenberg, Buffler and Langston, “Mesothelioma of Childhood,” *Cancer*, 62:838-847(1988) (“The available evidence does not support a causal relationship between malignant mesothelioma and asbestos . . .”) at p. 838.

C. A Significant Percentage of Mesothelioma Cases Have No Known Cause and Are Termed “Idiopathic”

It is undisputed that idiopathic mesotheliomas exist. For instance, "Roughly 20% to 40% of patients with malignant mesotheliomas have experienced no recognized asbestos exposure, and the disease is believed to be spontaneous." Craighead, J.E., *Pathology of Environmental and Occupational Disease*, 468, (St. Louis: Mosby, 1995).

The present report intends to demonstrate that mesotheliomas can be, and are caused by substances other than asbestos and, indeed, may also arise spontaneously.

Accepted causes of mesothelioma are listed in Table 10.6.

Table 10.6

Accepted Causes of Malignant Mesothelioma:

Asbestos
Erionite (only in parts of Turkey)
Therapeutic radiation
Severe pleural scarring
Idiopathic


Between 60 and 95% of pleural mesotheliomas in women cannot be attributed to either occupational or domestic (non-occupational) asbestos exposure (Spirtas 1994; Rake 2009; Gun 1995; McDonald 1980; Muscat 1991; Tuomy 1991; Wright 1984). For example, the National Cancer Institute’s case-control study published in 1994 estimated that the Sugarbaker risk for exposure to asbestos and mesothelioma was 23% for both pleural and peritoneal mesothelioma combined. Today, very few, if any, peritoneal mesotheliomas in women are asbestos-related. For example, a case-control study of peritoneal mesotheliomas at the Washington Cancer Institute, a major referral center for peritoneal mesothelioma, found no association between asbestos exposure and peritoneal in women. (Sugarbaker 2003).

Set forth below are some quotable quotes from the medical and scientific literature on idiopathic incidence rates of mesothelioma, both pleural and peritoneal:


- “It is estimated that about 50-80% of pleural MM in men and about 20-30% in women developed in individuals whose history indicates asbestos exposure(s) above that expected from most background setting.” pg. 44.


- ”The association between asbestos exposure and peritoneal disease is less strong and in the case of pleural mesothelioma, with only 50% of patients with peritoneal mesothelioma having a history of asbestos exposure, as opposed to 80% in pleural disease (Bridda et al., 2007).” (p. 290)


- “This observation suggests that asbestos exposure was responsible for only a minor fraction of peritoneal mesotheliomas in SEER over the period 1973 – 2005. Spirtas, et al.
reported that about 58% of peritoneal mesotheliomas among men in their study population were attributable to asbestos exposure. For females, they were unable to estimate separate attributable fractions for pleural and peritoneal mesotheliomas, but reported that the attributable fraction for both sites combined was 23%. Our results here suggest that, at least in the SEER data over the period of observation, the attributable fraction for male peritoneal mesotheliomas was lower than that reported by Spirtas.” pg. 7

• “With the standard population used in this paper, the age-adjusted background peritoneal mesothelioma rate is approximately 1 per million individuals per year, as indicated in Figure 1.” pg. 8

• “For pleural mesotheliomas, background rates are more difficult to estimate because there have been secular trends among both men and women. The secular trends among men are clearly dominated by the use of asbestos in the work place, although other factors could also be at play. Among women, birth cohort effects have risen modestly while period effects have declined leading to age-adjusted incidence rates that have remained more or less constant over the period of this study at about 3 per million individuals per year. This observation suggests that, even if some fraction of female cases can be attributed to asbestos exposure, the background rates are between 2 and 3 per million individuals per year. If the pathogenesis of spontaneous pleural mesotheliomas is similar in men and women, a not unreasonable assumption, this range of estimates can be taken to represent estimates of the background rate of pleural mesothelioma in men as well. Thus, the background rates of pleural mesotheliomas appear to be approximately 2-3 times higher than the background rates of peritoneal mesothelioma.” pg. 8

• “Background incidences and life-time probabilities of mesothelioma at both sites appear to be similar in men and women with these rates for pleural mesothelioma being 2-3 times higher than those for peritoneal mesothelioma.” pg. 9


• “Although a strong link between malignant mesothelioma and amphibole asbestos exposure is established, not all cases are etiologically related to asbestos. In the adult male population, 20-40% of malignant mesothelioma are idiopathic, and in women in the United States, the incidence of spontaneous idiopathic malignant mesothelioma exceeds 50%.”

• “It has been estimated that about 20%-40% of MM occurring in the United States are idiopathic and appear to develop spontaneously despite an intensive search for the cause.” (p. 196).

• “Pathologists recognize spontaneously developing MM long before the commercial exploitation of asbestos; the first idiopathic legion was diagnosed in the mid-1800s.
There is evidence for a background rate of MM and this has been estimated at about 1-8 cases per million per year. Support for this conclusion comes from several sources: (1) the constancy of risk over time for MM in females in the United States; (2) cases of MM in which there is no history of exposure to asbestos and lung asbestos fiber burdens are not elevated; (3) the spontaneous occurrence in children in adolescence.” (p. 196)

- “Overall, attributable risk of MM due to asbestos exposure was lower for the peritoneum (58%) than the pleura (88%) in men in the United States. Cases of peritoneal MM in men are documented when there is no recognized amphibole asbestos exposure.” (p. 212).


- “Rates for persons with little or no opportunity for occupational asbestos exposure were 1.15 (95% confidence interval: 0.90-1.45) for men and 0.94 (95% confidence interval: 0.87-1.24) for women.” pg. 525

- “In Tables 2 and 3, we restrict mesothelioma rates to the 1953-1972 birth cohort and to diagnoses made in 1983-2002, which fixes age to be 30-50 years at diagnosis. This permits an examination of rates for men and women who were unlikely to have experienced asbestos exposure and therefore are demonstrating a background rate for this age group of about 1 per million, similar to the estimate of McDonald and McDonald (1985).” pg. 533

- “Whereas McDonald (1985) examined rates before the onset of asbestos-related diseases to estimate background, we examined rates that represent background upon the termination of asbestos exposures.” pg. 533

- “The slightly higher estimated background rates in males may be because of residual occupational asbestos exposures, such as remodeling of old homes, replacement of old furnaces, or demolition work without following safe work practices after 1972. The background rates for those under the age of 30 years seem to be well under 1 per million, suggesting increasing background rates with age.” pg. 533

- “Determination of whether the background rate of mesothelioma at older ages (50+ years) is also about 1 per million or perhaps greater because of susceptibility of an ageing population must await future SEER statistics.” pg. 533

- “Given the estimated 3300 cases of mesothelioma per year in the US population of approximately 300 million... upwards of 300 (1 per million) annually may be unrelated to asbestos exposure. Cases unrelated to asbestos exposure may reflect spontaneous cases, a small proportion of cases because of therapeutic radiation (Teta, et al, 2007), and/or cases because of unrecognized causes.” pg. 534

- “About 50% to 70% of mesotheliomas are associated with exposure to asbestos.” pg. 1301


- “It should be appreciated that not all malignant mesotheliomas are associated with asbestos exposure. The vast majority of mesotheliomas in men in the USA (approximately 90%) are of pleural origin, and 90% or more of these tumours are caused by asbestos.” pg. 440
- “Even in men, only 50-60% of peritoneal tumours are related to asbestos exposure.” pg. 440
- “In women the ratio of pleural to peritoneal tumours is about 2:1 and only about 20% of all mesotheliomas in women in the United States can be reasonably linked to past asbestos exposure.” pg. 440
- “It is likely that in fact most cases of mesothelioma in women represent the background (non-asbestos related) incidence of this disease.” pg. 440


- “If all female cases of mesothelioma were unrelated to asbestos exposure, our analysis indicates that . . .the current annual risk would be approximately 4 per million (over 5 per million if the population at risk is aged ≥ 20 years). These background risk levels would be upper bounds if a portion of female cases of mesothelioma were due to occupational, domestic, or unique high environmental exposures.” pg. 111
- “On the basis of the differences between cases of mesothelioma in males and females, an adjustment to the background rates suggested above for females would be required if they were to be applied for males. For example, if all peritoneal mesotheliomas in females were excluded, the annual background rate for males would be 83 percent of the background rate for females.” pg. 111


- “It is known that approximately 20% of MM cases occur in individuals with no history of asbestos exposure and only a small percent of exposed individuals develop the disease.” pg. S26

- “The association between asbestos exposure and pleural mesothelioma (PM) is well established. The background incidence of PM (without asbestos exposure) is estimated to be about 1-2 cases per million per year.” pg 791

- “In the industrialized world, about 80% of malignant PM develop in individuals with higher than background levels of exposure to asbestos.” pgs. 791-792

- “Although asbestos is considered to be the principal cause of mesothelioma, no exposure to asbestos fibres is detectable in a proportion of mesothelioma patients. It was estimated by Roggli, et al. that some 20% of mesotheliomas occur in persons with no history of asbestos exposure; however, we can not exclude that the long latency time may play a role in the masking of past exposures.” pg. 795


- A major concern is the significance of mesothelioma cases without any definite occupational asbestos exposure, which represent around 25-30% of the total. pg. 37


- “There might exist a background level of mesothelioma occurring in the absence of exposure to asbestos, but there is no proof of this and this ‘natural level’ is probably much lower than the 1-2/million/year which has been often cited.” pg. 505


- Roughly 20% to 40% of patients with malignant mesotheliomas have experienced no recognized asbestos exposure, and the disease is believed to be spontaneous. (p. 468)


- “Among men with pleural mesothelioma the attributable risk (AR) for exposure to asbestos was 88%...” pg. 804

- “For men, the AR of peritoneal cancer was 58%...” pg. 804

- “For women (both sites combined), the AR was 23%...” pg. 804
• “The large differences in AR by sex are compatible with the explanations: a lower background incidence rate in women, lower exposure to asbestos, and greater misclassification among women.” pg. 804

• “As the incidence of mesothelioma among women (about three cases per million women per year for all primary sites combined) is much lower than among men and has remained reasonably constant over time, it is possible that the incidence in women may be close to the background level. Alternatively, exposure to asbestos is lower and misclassification of exposure may be greater among women, which would also reduce their AR.” pg. 810

• “Mesothelioma was not significantly associated with asbestos exposure among women in our study, although the OR forever exposed women was 2.7. Although over 50% of the female cases reported some exposure to asbestos, the number of female cases was small, and women were less likely than men to be employed in jobs expected to have the highest exposure to asbestos, such as shipbuilding and insulation work.” pgs. 809-810


• Case involved 42 year old woman with a diagnosis of diffuse malignant mesothelioma of pleura and peritoneum, with metastases to lymphatics, lymph nodes, and lungs.

• “When cases of mesothelioma from large institutions are reviewed, however, 20 to 30 percent of the patients give no history of exposure to asbestos or have no evidence of asbestos exposure at autopsy or at biopsy.” (p. 663).

• “Several of my colleagues and I have reviewed the cases of mesothelioma seen at this hospital since that diagnosis was first made by the Department of Pathology in the early 1950s. We have reviewed 115 cases of pleural or peritoneal mesothelioma, or both. There has been a marked increase in the number of cases over the years; there were only 3 cases before 1950, 2 or 3 cases were diagnosed per year in the 1960s, and at present 10 or more cases are diagnosed annually. In our review, the outstanding finding was the fact that 30 percent or more of the patients with a mesothelioma gave no history of exposure to asbestos and on pathological examination had no evidence of asbestos fibers in the lungs or other changes consistent with exposure to asbestos. Therefore, one must conclude that not all mesotheliomas result from exposure to asbestos.” (p. 663) (emphasis added).

• “It is not possible to say whether the patient had appreciable exposure to asbestos, but on the basis of our experience, it is probable that the mesothelioma of the pleura was not related to asbestos.” (p. 662).

“The study population includes all 49 patients seen at the Massachusetts General Hospital through 1972. Cases were identified by computer review of the diagnosis during discharge of the study period and included 17 cases in which an associated mesothelioma had been previously reported. Nineteen patients with mesothelioma and had no exposure to asbestos by history and who, on histological examination had no evidence of asbestosis were not included.” (p. 649).

D. The Lack of Pleural Plaques May Indicate That a Mesothelioma Is Not Asbestos-Related

Pleural plaques, when asbestos-related, are a sensitive marker of asbestos exposure, even at low levels of exposure. A number of researchers have opined that plaques occur after light exposures. For instance:

Distinctive circumscribed plaques of dense fibrous tissue, deposited at specific sites in the parietal pleura are a unique and sensitive marker of asbestos exposure. The exposure threshold for plaque development is relatively low.

Craighead, *supra*, at 468.

The autopsy serves an additional important function in that it allows examination of the lungs for evidence of exposure to asbestos and asbestosis.... The presence or absence of fibrous plaques on the parietal or diaphragmatic pleura should be noted since this is also closely linked with exposure to asbestos and has been dubbed the “visiting” (or “calling”) card of asbestos. Evidence based on these observations is of great epidemiologic importance and may be crucial in litigation.


Pleural plaques are regarded as the “visiting card” of asbestos, and they serve as a useful marker to draw the attention of radiologist and pathologist to the possibility of asbestos exposure in those patients in whom they are found.


These data agree well with the epidemiological observations that pleural plaques often occur in individuals with brief, intermittent, or low-level asbestos exposure.


Plaques occur in workers with exposure to amphiboles and to chrysotile. Studies of fiber burden in such cases consistently show that plaques occur at fiber levels considerably greater than that carried by the general population but much lower than are seen in cases of asbestosis.
E. Pleural Plaques Can Be Found in a Majority of Persons with Mesothelioma

A number of researchers’ findings show that over 50 percent of persons who have mesothelioma also have plaques. These opinions are useful when a plaintiff with mesothelioma shows no asbestos signs, including plaques. For instance, in a British study of workers in a shipyard, the authors found 28 mesotheliomas:

Pleural plaques were present on the radiographs of 20 patients and at autopsy in two more. Edge, J., "Asbestos Related Disease in Barrow-in-Furness, "Environmental. Research, 11(1976):244-47.

Hirsch et al.’s study covered 36 mesothelioma cases with exposures ranging from definite heavy to persons without past asbestos exposure.

Controlateral asbestos-related radiological signs were present in 13 out of the 17 asbestos exposed cases, and absent in 9 out of the 10 unexposed cases.


In a study of asbestos-related and non-asbestos related mesothelioma patients at Massachusetts General Hospital, all of the patients with a history of asbestos exposure had some indicia of exposure to asbestos. Seventy-six percent (76%) had pleural plaques.


In their published paper on the analysis of asbestos fiber burden in the lung tissue of 55 mesothelioma patients, Drs. Dodson and Hammar, et al. found pleural plaques in 39 of 44 cases in which such information was available (the information was unavailable in 11 cases). Dodson, et al., "Analysis of Asbestos Fiber Burden in Lung Tissue from Mesothelioma Patients," Ultrastructural Pathology, 21:321, 327(1997).

There is also evidence that pleural plaques, when asbestos-related, are likely related to amphibole exposure rather than chrysotile exposure. See generally, Churg and DePaoli, "Environmental Pleural Plaques in Residents of a Quebec Chrysotile mining town," Chest, 94:58(1988). ("These observations suggest that environmental pleural plaques in this region of Quebec are probably caused by exposure to tremolite derived from local soil and rock . . ."); Graham W. Gibbs, "Etiology of Pleural Calcification: A Study of Quebec Chrysotile Asbestos Miners and Millers," Arch Environ Health, 76-82(March/April 1979) ("It appears highly unlikely that chrysotile asbestos itself is responsible for the pleural calcifications, but rather a mineral closely associated with it." – p. 82)
F. To Attribute a Mesothelioma to Asbestos, the Household Member’s Exposure Must Be Significant

Wives of insulators who washed their husbands’ clothes for many years and developed mesothelioma had significant exposures. In Churg’s chapter, "Asbestos-Related Diseases," which appears at page 890 in Churg, Myers, et al., *Thurlbeck's Pathology of the Lung*, 3rd Ed., (New York: Thieme, 1995), he states,

> It should be appreciated that these types of exposures are not “low level” in the sense this term is currently used; in fact, mineral analysis of some reported cases has indicated that the exposures may have been fairly substantial.

The literature in this section profiles exposures which are not trivial or occasional. At trial, the issue is likely to be whether the exposure was “significant.” If the plaintiff has plaques, or elevated fiber counts in lung tissue, the exposure clearly was significant.

Does a daily visitor to a home of a worker in a bystander trade receive a "significant" exposure? Does the daughter of a woman whose mother worked at an auto parts store selling (among other things) asbestos brake linings receive a significant exposure? The plaintiffs’ attorney would say yes, but the literature on household mesothelioma cases generally supports the opposite view.

However, some literature is problematic because it fails to define “significant.” For instance, the so-called “Helsinki Criteria,” framed at the International Expert Meeting on Asbestos, Asbestosis, and Cancer in January 1997 is such a document. This meeting, attended by many Finnish researchers, as well as Dr. John Dement, Dr. Victor Roggli and others, produced a paper with “state-of-the-art criteria” for diagnosis of disorders of the lung and pleura and their attribution with respect to asbestos. For mesothelioma, the criteria are:

A lung fiber count exceeding the background range for the laboratory in question or the presence of radiographic or pathological evidence of asbestos-related tissue injury (e.g., asbestosis or pleural plaques) or histopathologic evidence of abnormal asbestos content (e.g., asbestos bodies in histologic sections of lung) should be sufficient to relate a case of pleural mesothelioma to asbestos exposure on a probability basis. In the absence of such markers, a history of significant occupational, domestic, or environmental exposure to asbestos will suffice for attribution. There is evidence that peritoneal mesotheliomas are associated with higher levels of asbestos exposure than pleural mesotheliomas are. In some circumstances, exposures such as those occurring among household members may approach occupational levels.


These criteria leave open the question of what “significant” means, and it fails completely to address the issue of fiber type.
As previously discussed, most household contacts are wives or children of insulators or others who had heavy asbestos exposure at work. Roggli et al.’s chapter, “Mesothelioma in Women,” in Anatomic Pathology, 2(1997):147-63, has a table on page 150 profiling women with mesothelioma. The household contacts have husbands or fathers with jobs like “insulator” “pipecoverer” “pipefitter,” and “amosite asbestos plant worker.” See also Miller (2005); Marchevesky, et al (2006).

Summary

Just because a plaintiff claims exposure through household exposure by a parent or sibling does not make the allegation true. The vast majority of the medical and scientific literature shows that:

1) the person bringing the fibers into the household needs to have had a substantial exposure;
2) there are other causes of mesothelioma;
3) a certain number of mesotheliomas at this time appear to be “background” and idiopathic, especially in women;
4) even when the person who allegedly brought the fibers home worked in an occupation which gave him or her substantial exposures to asbestos fibers, defenses such as inadequate latency, lack of fibers above background, and other causes should be explained. (See Sections VI, VII, VIII and IX.)

G. Household Exposures in Current Personal Litigation Cases are Usually Quite Low

The Goswami, et al. review article previously identified above at page 7 contains an excellent summary of studies that have analyzed either qualitatively or quantitatively exposures in the home environment. The results are summarized in Table 4, a copy of which is included with permission as Appendix (D).

None of the studies are particularly relevant to the kinds of exposures we are seeing in litigation cases today as the Nicholson, et al., 1980 study addressed homes of chrysotile miners, the Selikoff and Lee 1978 article addressed settled dust levels of amosite asbestos in the homes of unibestos factory workers employed in the 1940s and 1950s, and the WHO 1986 study addressed residences of asbestos miners in South Africa.

The Goswami review article identifies one study of clothing and laundering - the Sawyer, et al., study from 1977 which measured air samples from the breathing zone of individuals laundering clothes of asbestos abatement workers. The mean of the personal samples was 0.4 fiber per cc.

However, there have been a number of exposure simulation studies - once again many of them identified in Table 4 from the Goswami review paper which are quite helpful and more relevant to the cases we see in litigation today. Two of the studies involved measuring air samples from clothing handling after unpacking and repacking clutches or brakes whereas other
studies measured exposures from clothing handling from performing brake repair on heavy equipment. All of the results were quite low. For example, the Madle, et al., 2009 study simulating mechanics performing brake repair on heavy equipment and subsequent clothes handing estimated a 30 minute PCM-adjusted mean of 0.0365 f/cc for the primary worker (range:0.032-0.039 f/cc) and for bystanders, 0.010 f/cc (range 0.003-0.018 f/cc).

There have been two simulation studies published subsequent to the Goswami review article which are extremely helpful and relevant to counsel representing low dose chrysotile product manufacturer/distributors - Sahmel, et al., "The evaluation of take-home exposure and risk associated with the handling of clothing contaminated with chrysotile asbestos," Risk Analysis 34:1448-1468(2014) and a follow-up article Sahmel, et al., "Airborne asbestos take-home exposures during handling of the chrysotile contaminated clothing following simulated full shift workplace exposures," Journal of Exposure Science and Environmental Epidemiology 1-15(2015). In Sahmel (2014), the study was designed to measure the relationship between airborne chrysotile concentrations in the workplace, contamination of work clothing and take-home exposures. The study included air sampling of handling and shaking out the clothes prior to laundering. The clothes were contaminated at three different airborne chrysotile concentrations - 0-0.1 f/cc, 1 to 2 f/cc, and 2 to 4 f/cc. The clothing exposed to those various concentrations of chrysotile were subjected to active handling and shakeout for 15 minutes and an additional 15 minutes of no handling. Airborne concentrations for the clothes handler were found to be 0.2 to 1.4% of the 8 hour TWA and 0.03 -0.27% of the 40 hour TWA. The investigators concluded that "cumulative chrysotile doses for clothes handling at airborne concentrations tested were estimated to be consistent with lifetime cumulative chrysotile doses associated with ambient air exposures (range for take-home or ambient doses 0.00044 - 0.105 f/cc years)." Id. at 1448.

The subsequent study by Sahmel, et al., published in 2015 expanded the analysis to measure airborne chrysotile concentrations associated with laundering of contaminated clothing worn during a full shift work day with "work clothing fitting onto mannequins exposed for 6.5 hours to an airborne concentration of 11.4 f/cc (PCME) of chrysotile asbestos, and then subsequently handled and shaken. The TWA airborne concentrations for clothes handling activity were approximately 1% of workplace concentrations and similarly, weekly 40 hour TWAs for clothes handling were approximately 0.20% of workplace concentrations. The investigators estimated that "take-home cumulative exposure estimates for weekly clothes handling over a 25 year working duration were below 1 f/cc for handling work clothes contaminated in an occupational environment with full shift airborne chrysotile concentrations of up to 9 f/cc - h(TWA)." Id. This article also is extremely helpful in including a discussion of the potential for resuspension exposure in a household. When confronted with statements from a plaintiff industrial hygienist regarding resuspension of asbestos into the air of residences and allegations that such exposures continue indefinitely for the life of the house, please see the detailed discussion at page 13 which provides an effective counter.
III. Exposures in Buildings

A number of asbestos-related claims have arisen, especially in mesothelioma cases, based on supposed exposure to in-place asbestos in buildings. These cases include allegations that mesotheliomas were caused by the mere presence of persons in a school containing asbestos building materials of one form or another. The same is true in office buildings.

In these cases there is no substitute for thorough discovery into the identity and condition of the claimed products. In schools, ever since the AHERA requirements have been in existence, school administrations have been required to do inventories and inspections of their potential asbestos containing products and are required also to submit periodic reports. In other environments, frequently OSHA inspection reports are available. Without this type of information, defendants are at a clear disadvantage.

However, once the lawyer has knowledge of the claimed products, and a history of installation, abatement, disturbance, and reconstruction is in hand, a defense attorney can go forward with a great deal of helpful material in the medical, scientific, and government literature. Lawyers in the asbestos property damage litigation have much to offer here as well.


EPA administrators from that time on began to back off from their very drastic statements about asbestos levels in buildings. The statement by administrator L.J. Fisher acknowledging that there are levels of exposure which can be negligible or even zero, and that “asbestos on an auditorium ceiling no more implies disease than a potential poison in a medicine cabinet or under a sink implies poisoning,” are very helpful in bringing a sense of perspective.

The rationale for the move back to rationality is best summarized by Edward Gaensler, M.D., in Clinics in Chest Medicine, 13(2)(1992):231-42. This article also contains excellent quotes for use at trial. For instance,

Asbestos-related diseases are dose-related. Among these, asbestosis has occurred only with the heavy exposures of the past, is a disappearing disease, and is of no concern with the very small exposures from building occupancy. (p.239)

In that there is neither clinical nor epidemiologic support for asbestos-related disease from building occupancy, risk estimates have been based on extrapolation from past experience with generally high-dose occupational exposure. However, only a few epidemiologic studies have contained quantitative estimates of
exposure, and these have been measured in terms of all particles, with conversion to asbestos fibers uncertain and the fiber type and dimension largely unknown. To these uncertainties must be added the unproved assumption of a linear dose-response down to very low levels of exposure with no threshold. (p. 240)

Comparative risk analyses have shown that most other involuntary risks, e.g., exposure to side-stream smoking, diagnostic radiography, or indoor radon, are many times more hazardous than possible risks from building asbestos exposure. Indeed, a committee of the Royal Society has called a lifetime risk of one per 100,000 negligible, while others have suggested that most recent data do not indicate that asbestos-related malignancy or functional impairment will result from airborne concentrations of asbestos in schools and other public buildings. (p.240)

Another helpful document is the extensive Health Effects Institute-Asbestos Research’s (HEI-AR), Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge, Cambridge, Mass., 1991. This nonprofit independent organization was formed to support research to determine the airborne exposure levels prevalent in buildings. It was sponsored jointly by the EPA and a broad range of other parties that “have an interest in asbestos.” Members of the organization’s asbestos literature review panel include such persons as Margaret Becklake, Arthur Langer, Brooke Mossman, William Nicholson, and Jonathan Samet. Chairman of its board of directors was Archibald Cox. Acting under a congressional mandate, the organization reviewed and synthesized the state of knowledge from scientific articles, reports and additional unpublished data on four issues:

- the concentrations of airborne asbestos fibers found in public and commercial buildings; the concentrations of such fibers to which building occupants, including custodial workers, maintenance workers, abatement workers, and other occupants are exposed;
- the situations causing such exposures and the potential for adverse health effects resulting therefrom;
- the possible impact that different asbestos remediation strategies may have on the exposure of building occupants to airborne asbestos and, in turn, on the risk of health effects in those exposed; and
- the significance of each form of asbestos in terms of its potential ill health effects and its implications for different remediation options in buildings.

The report gives detailed summaries of studies of average airborne asbestos concentrations in buildings in Canada, the U.K., and the U.S., including data on the numbers of asbestos fibers found inside, and if available, outside. There were a number of helpful conclusions in this tome:
It is almost certain that the overall excess of [lung cancer] cases that could be attributed to asbestos exposure in buildings will be relatively small and probably undetectable. (p. 8-2)

The risk elements discussed in Section 6.2... imply that the excess deaths [from mesothelioma] that may occur from exposure in buildings will not cause a sufficient increase in the mesothelioma rate to be readily detected. (p. 8-2)

In the United States, evidence suggests that cementitious sprayed insulation and acoustic plaster in buildings under normal repair rarely give rise to airborne concentrations above the outside ambient. (p. 4-81)

Damaged friable sprayed asbestos, particularly with visible debris, has often been associated with elevated airborne levels (above the outside ambient) in occupied buildings. On the other hand, undamaged friable sprayed asbestos in buildings not of recent construction is rarely associated with elevated levels. (p. 4-81)

Asbestos fibers longer than five microns represent a small proportion of the total number of airborne asbestos fibers. (p. 4-82)

Overall rates for mesothelioma incidence in women have been stable since the 1970s in some countries, including the United States... and therefore provide no evidence of an impact on the public health from environmental exposure to asbestos, including exposure in buildings containing ACM. (p. 6-54)

The EPA has given guidance to persons responsible for maintaining buildings. In 1985, they issued what was called the “Purple Book” entitled, Guidance for Controlling Asbestos-Containing Materials in Buildings. EPA 560, June 1985. In July 1990, the EPA issued a document later called the “Green Book” entitled Managing Asbestos in Place: A Building Owner’s Guide to Operations and Maintenance Programs for Asbestos-Containing Materials. This book displayed prominently five "facts." The first two are particularly pertinent to our discussion:

**Fact One:** Although asbestos is hazardous, the risk of asbestos-related disease depends upon exposure to airborne asbestos fibers.

In other words, an individual must breathe asbestos fibers in order to incur any chance of developing an asbestos-related disease. How many fibers a person must breathe to develop disease is uncertain. However, at very low exposure levels, the risk may be negligible or zero. (p. vii)

**Fact Two:** Based on available data, the average airborne asbestos levels in buildings seem to be very low. Accordingly, the health risk to most building occupants appears also to be very low.

A 1987 EPA study found asbestos air levels in a small segment of federal buildings to be essentially the same as levels outside these buildings. Based on that limited data, most building occupants (i.e., those unlikely to disturb asbestos-containing
building materials) appear to face only a very slight risk, if any, of developing an asbestos-related disease. (p. vii)

There are numerous studies of asbestos concentrations in buildings. Some of the most helpful include Corn et al’s “Airborne Concentrations of Asbestos in 71 School Buildings,” *Regulatory Toxicology and Pharmacology*, 13(1990:99-114, in which a total of 473 air samples from 71 schools scheduled for abatement were analyzed by transmission electron microscopy techniques. The authors concluded that

There is no conclusive evidence in the present study that indicates that asbestos-containing materials make a significant contribution to airborne asbestos levels in schools.


Roggli and Longo’s paper, "Mineral Fiber Content of Lung Tissue in Patients with Environmental Exposures: Household Contacts Versus Building Occupants," *Annals of the New York Academy of Science*, 643(1991):511-18, is instructive. This study compared the fiber content of persons claiming asbestos exposure in buildings with persons exposed as household contacts. The building occupants had much smaller absolute numbers of fibers of any type than the household contacts. Of the four building occupants studied, they averaged 4.4 percent commercial amphiboles, 20 percent noncommercial amphiboles, two percent chrysotile and 73 percent other (talc, silica, etc.). They concluded that, "Our studies of four occupants of buildings with asbestos-containing materials indicated that these individuals often have pulmonary asbestos burdens indistinguishable from the general non-occupationally exposed population." *Id.* at 517.

**Summary**

There is abundant literature to support the concept that asbestos-containing materials, if properly maintained, pose a negligible threat to the health of building occupants. If confronted with a building exposure case, get as much information as possible about the building, its maintenance records, AHERA reports, and so forth. Find the as-built plans for the building to determine to what extent there were asbestos-containing materials installed in the first place, and look for maintenance records. Talk to building maintenance personnel. Then use the ample medical, scientific, and government documentation to support your case.

**IV. Neighborhood Exposures**

Occasionally asbestos-related cases (predominantly mesothelioma) arise with a plaintiff claiming that his or her disease is attributable to exposures years before in the neighborhood of an asbestos manufacturing plant or other facility. These cases are relatively rare, although occasionally a claim of neighborhood exposure is added to other claimed exposures.
The early research on asbestos-related disease did indeed establish that in certain unusual circumstances with very high neighborhood amphibole asbestos contamination, asbestos-related diseases did occur.

Once again, the 1960 article by J.C. Wagner et al, which suggested a link between mesothelioma and crocidolite asbestos, included a number of mesothelioma patients who lived in the vicinity of the crocidolite mines, but who never had occupational exposure. The majority of those patients had not actually worked with asbestos but had lived in the vicinity of the mines and mills and some had left those areas of exposure even as young children. Wagner, J.C., Sleggs, C.A., and Marchand, P., "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," British J. of Indus. Medicine, 17(1960):260-71. By 1962, Wagner had found 75 persons whose exposure came from living in the vicinity of the mines and dumps. Wagner, J.C., "Epidemiology of Diffuse Mesothelial Tumors: Evidence of an Association from Studies in South Africa and United Kingdom," Annals of the N. Y. Academy of Sciences, 132(1965):575-78. Such exposures, however, were undoubtedly quite high, inasmuch as the Northwestern Cape Province had an extremely arid climate, crocidolite tailings were played in by children on a regular basis, and crocidolite tailings were used to “pave” the roads.

Newhouse and Thompson studied 76 persons with pleural and peritoneal mesothelioma from London Hospital. They concluded that from their data there was evidence that neighborhood exposures may be important. However, they indicated that “more evidence is required of an increased risk to the population living in the neighborhood of asbestos factories or other areas, such as dock yards where asbestos is used in quantity.” Newhouse, M.L. and Thompson, H., "Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area," British J. of Indus. Medicine, 22(1965):261-69.

In 1967, Borow et al studied autopsy material of 17 cases of mesothelioma at Somerset Hospital, which was located two miles from the Johns Manville plant in Manville, New Jersey. According to the authors, “approximately 75% of all the asbestos fiber which is mined in North America is converted to commercial use in an industrial facility which is located only two miles from Somerset Hospital.” The authors reported that two of their patients contracted mesothelioma with only environmental exposure consisting of living in a community adjacent to the asbestos mill. However, they also pointed out that as of the time of writing of the article, in South Africa no mesotheliomas had been reported from the Transvaal asbestos field where both crocidolite and amosite were mined. In this early stage of the development of scientific understanding of mesothelioma, researchers were groping for an understanding of exposures which truly caused the disease. Borow, M., et al, “Mesothelioma and Its Association with Asbestosis,” J. of the Am. Medical Ass’n, 201(8)(1967):587-91.

These same authors in 1973 published a study of 72 cases of mesothelioma. In this group they failed to find patients with neighborhood exposure. They observed that,

The failure to find patients with neighborhood exposure has been surprising to us as there has been, until the last several years, significant air pollution, with asbestos fiber over certain sections of a densely populated area in the vicinity of the asbestos mill. (p.644)
They also noted that the asbestos mill where the patient series originated used mainly chrysotile, although some of the processes utilized crocidolite. They concluded that “at present, there does not seem to be any health hazard for the general public.” Borow, Maxwell et al., “Mesothelioma Following Exposure to Asbestos: A Review of 72 Cases,” Chest, 64(5)(1973):641-46.

A 1967 study of 42 cases of mesothelioma from 152 hospitals produced eight persons who lived or worked close to asbestos manufacturing facilities. Histories of exposure varied tremendously in this study. One patient had lived for 30 years across the street from a plant manufacturing acoustic tile and linoleum. Another had lived within one-half mile of an insulation plant. Another claimed exposure for one year while he worked across the street from an insulation plant, and similarly another had worked for 26 years across the street from the same insulation plant. Two others had lived within three-fourths of a mile of two asbestos plants. The last two worked at a storage battery plant located less than one-half mile from an asbestos textile plant (an exposure the authors classified as “questionable”). Lieben, J., et al, “Mesothelioma and Asbestos Exposure,” Archives Envtl. Health, 14(1967):559-63.

Hammond, et al (including Selikoff), published a study comparing death certificates from persons living in the neighborhood surrounding an amosite asbestos factory in Patterson, New Jersey (the neighborhood of “Riverside”), with persons living in another Patterson neighborhood known as Totowa, located several miles from Riverside. They had collected samples from settled dust from the attics of houses in the Riverside neighborhood near the factory and found that they contained appreciable numbers of amosite asbestos fibers. Those collected from houses located at a greater distance from the factory contained fewer fibers. They acknowledged that it is safe to assume that people living in the neighborhood of the factory were exposed to asbestos dust, obviously at an extremely light exposure as compared with the exposure of men working in the factory.” They wished to determine “whether very light non-occupational exposure to amosite asbestos dust produces adverse effects to a measurable degree.” They found that, with respect to total deaths, deaths from cancer (all sites combined), and lung cancer, mortality experience was slightly worse in Totowa than in Riverside. No deaths from peritoneal mesothelioma, and just one death from pleural mesothelioma occurred during the 15-year period of death certificate counting. Ironically, this one death was of a Riverside subject (an electrician), who died of pleural mesothelioma in 1966. All of the persons from Riverside lived within one-half mile of the factory, and prevailing winds from the factory blew dust from the factory in the direction of most of the dwellings. Hammond, E.C., et al, “Mortality Experience of Residents in the Neighborhood of an Asbestos Factory,” Annals of N. Y. Academy of Sciences, 417-22, 1979.

A case report by Fischbein and Rohl in 1984 reported on a microchemical analysis of the lung tissue of a mesothelioma patient who had been employed in a factory adjacent to the Brooklyn Navy Yard. The male patient apparently developed mesothelioma in 1970, but did not die until January 1980. His employment adjacent to the Navy Yard was from 1957 to 1966. The authors indicated that there is “typically a long latency period between asbestos exposure and the resulting disease, but the requisite latency period does not appear to be sufficient here” The authors reported finding a “large number of amphibole asbestos fibers” but give no actual numbers. Furthermore, they claimed that amosite asbestos is not found in the lungs of persons from the general population, and its occurrence, therefore, indicates either an occupational exposure or an exposure to a specific environmental source.” The conclusions, therefore, based on this one case


J. Corbett McDonald, “Health Implications of Environmental Exposure to Asbestos,” Environmental Perspectives, 62 (1985):319-28, not only summarizes the literature up until 1985 but also points up the difficulty of separating neighborhood exposure from occupational exposure in the same neighborhood. This has been especially true of studies of the populations in Thetford Mines and Asbestos, Quebec, the largest chrysotile mining area in the Western Hemisphere.

Singh and Thouez attempted to measure ambient air concentrations of asbestos fibers during one summer in 1980 in three townships in Quebec to determine factors which might influence the number of fibers in the air in the three areas which include the town of Asbestos and two others west and east of the mine in that town. Their main conclusion was that there were large variations in the amount of fibers in the air depending upon windiness and turbulence, as well as wind direction. Rain also has an effect on the number of fibers in the air. Singh, B., and Thouez, J.P., “Ambient Air Concentrations of Asbestos Fibers near the Town of Asbestos, Quebec,” J. of Env. Research, 36 (1985):144-59.

In the 1980s, Churg and DePaoli discovered that tremolite in the soil in the chrysotile mining town of Thetford Mines, Quebec, was the cause of pleural plaques in certain persons not employed in mining but working the soil. In autopsy materials, plaques were found in three farmers and one road construction worker—all persons who had worked in the soil for years. They performed lung burden analyses on these four persons and on nine controls in the same town who had never been employed in the chrysotile mines or mills and who had no plaques. Both groups had similar concentrations of chrysotile fibers, reflecting the concentrations in the ambient air. As to tremolite, the farming/construction group with plaques had elevated tremolite concentrations. The authors concluded that the short tremolite fibers in the Thetford region were able to produce plaques at fairly low exposure levels, but that it had “very little potential to induce mesothelioma in man unless exposure occurs at extraordinarily high levels.” Churg, A., DePaoli, L.,” Environmental Pleural Plaques in Residents of a Quebec Chrysotile Mining Town,” Chest, 94(1988):58-60.


Inase et al, Japanese researchers, reported a case of pleural mesothelioma after what they called “neighborhood exposure” to asbestos during childhood. Even a cursory reading of the description of the exposure, however, makes it clear that there was more than “neighborhood exposure”: The mother of the young woman with mesothelioma worked in a cement plant and
usually took her daughter to the factory and let her play on the white dust-covered hills. It would appear, therefore, that the patient’s childhood exposure was more akin to a bystander or frankly occupational exposure. An examination of the pulmonary tissue revealed 517 asbestos bodies per gram by light microscopy. Inase, N., et al, “Case Report: Pleural Mesothelioma after Neighborhood Exposure to Asbestos during Childhood,” *Japanese J. of Medicine*, 30(4)(1991):343-45.

Another case report appeared in the *British Journal of Industrial Medicine* by Cazzadori et al in 1992. A 37-year-old woman developed pleural mesothelioma and her medical history included no occupational exposure to asbestos, but revealed that she had lived from birth until ten years of age in a house next to an asbestos processing factory. Seven asbestos bodies were identified in bronchoalveolar lavage (0.3 asbestos bodies/1M by light microscopy. The authors compared this finding with the determination by Dodson et al that one asbestos body/ml was the limit value to discriminate occupationally from nonoccupationally exposed persons. Cazzadori, A., et al, “Malignant Pleural Mesothelioma Caused by Non-Occupational Childhood Exposure to Asbestos,” *British J. of Indus. Medicine*, 49(1992):599. (See Dodson et al, “The Usefulness of Bronchoalveolar Lavage in Identifying Past Occupational Exposure to Asbestos: A Light and Electron Microscopy Study,” *Am. J. of Indus. Medicine*, 19 (1991):619-28.) The use of bronchoalveolar lavage is not common. The more reliable and recognized source of information on fiber burden is the use of fiber analysis from tissue digestion.

Australian researchers attempting to determine the magnitude of the population at risk from “nonoccupational exposure to crocidolite” analyzed a cohort of 4,890 residents of Wittenoom, Western Australia. Wittenoom was the site of a crocidolite asbestos mine during the period 1943 to 1966. Among these residents (none of whom actually worked for the mines), by the date of this study there had been 24 cases of mesothelioma: nine males and 15 females. While the authors termed this exposure “environmental,” nine cases had occurred in wives and nine cases in children of workers at the mine. These therefore would more appropriately be termed “household exposures.” In addition, it is important to note that tailings from the mill were used for the paving of roads, driveways, car ports, and school playgrounds, and in the yards of houses to suppress dust and reduce muddiness, as well as on the race course: These exposures sound much more similar to exposures in the original Wagner study, and of course the fiber involved is crocidolite, as in the Wagner study. It is very unlikely that plaintiffs in the United States were subjected to similar neighborhood exposures, and it is extremely unlikely that such exposures would have been to crocidolite. Hansen, J., et al., “Malignant Mesothelioma after Environmental Exposure to a Blue Asbestos,” *Int’l J. of Cancer*, 54 (1993):578-81.

In 1997, Hansen and colleagues analyzed a cohort of former residents of Wittenoom that comprises all people who lived in Wittenoom between 1943 and 1993 for at least one month and did not work with crocidolite at Wittenoom, either directly in the mining and milling of crocidolite or indirectly in other occupations. The objective was to assign individual airborne crocidolite exposure levels to former residents at the Town of Wittenoom, Western Australia. All environmental monitoring programs carried out in the town since 1966 were reviewed and assessed for their suitability for use in estimating individual exposure to crocidolite. Duration of residence was determined from questionnaires, work history sheets of the Australian Blue Asbestos Company, and the various sources used to assemble a cohort of previous residents. Most of the cohorts stayed at Wittenoom longer than one year and had a median estimated
intensity of exposure of 0.5 fiber per milliliter and a median estimated cumulative exposure of 2.8 fiber years per milliliter. As a result of the crocidolite mining operations at Wittenoom, the residents of the town, even as late as 1992, have been continuously exposed to higher levels of airborne crocidolite than other populations where no such source of contamination exists.

Hansen and colleagues published in 1998 regarding environmental exposure to crocidolite and mesothelioma: Exposure – Response Relationships. The study aimed to estimate exposure-response relationships for mesothelioma and environmental exposure to crocidolite. All 4,659 former residents of Wittenoom, Western Australia who lived there between 1943 and 1993, for at least one month, and were not directly employed in the crocidolite industry, were followed up through the Western Australia Death, Cancer and Mesothelioma Registries, electoral rolls, and telephone books. To the end of 1993, 27 cases of mesothelioma were diagnosed. Mesothelioma cases stayed longer at Wittenoom, had a higher average of intensity of exposure, and a higher cumulative exposure to crocidolite than control subjects. The rate increased significantly with time from first exposure, duration of exposure and cumulative exposure. At these levels of crocidolite exposure, there is a significantly increased risk of mesothelioma, which is dose-dependent. This cohort study of Wittenoom residents shows that the incidence of mesothelioma increased significantly with increasing time following first residents of Wittenoom and with increased level of exposure to crocidolite. This result holds whether level of exposure is measured by duration of residents or by cumulative exposure. Cases of mesothelioma in this cohort of Wittenoom residents have arisen in subjects with durations of crocidolite exposure as short as two months and estimated cumulative exposure as low as 0.53 fiber per milliliter.

An interesting study by Arblaster et al highlights the need to be thorough in attributing mesothelioma deaths to neighborhood-type exposure. In 1988, the coroner of Leeds, England, expressed concern about the number of mesothelioma deaths being referred for inquest. While it was known that some of the cases had been individuals who had worked at an asbestos factory, it was feared that many of the cases were persons who had lived near the factory, although they had never been employed there. The factory had used crocidolite asbestos and had made asbestos mattresses for the insulation of steam boilers and later a spray asbestos product. The coroner’s concern made it into the national television network news and this study was instituted. A large proportion of the people who had no occupational exposure at the asbestos factory worked in other occupations elsewhere with likely exposure to asbestos. Of 210 mesothelioma deaths, only five possible neighborhood (here termed “environmental”) were identified. Of those five, the diagnosis was only confirmed histologically in three. Four of the cases had resided within 250 yards of the factory for between five and 51 years. The fifth case had worked at a laundry near the factory for “some years.” The authors concluded that “most cases of Leeds’ mesothelioma deaths occurred in people directly or indirectly occupationally exposed to asbestos.” Arblaster et al.,” Occupational and Environmental Links to Mesothelioma Deaths Occurring in Leeds during 1971-1987,”1. Of Pub. Health Medicine, 297-304 (1995).

A much less thorough study of Manville, New Jersey, was published by Michael Berry in 1997. The author looked at pleural and peritoneal mesothelioma diagnoses in the New Jersey State Cancer Registry between 1979 and 1990. He concluded that there was “a strong relationship between past exposure from living in Manville and eventual development of mesothelioma.’ However, there was no information on risk factors such as the extent of occupational exposure or personal lifestyle habits. No personal interviews or other means of data collection for these factors were utilized in this study. Furthermore, this author specifically assumed that the likelihood of employment in other high-risk occupations or facilities would
not differ from that of the state as a whole which was used as a comparison population. Author Berry also seemed to be ignorant of the fact that a plant owned by Asbestos Ltd. Was located approximately three kilometers from the Johns-Manville facility. The Asbestos Ltd. Plant utilized crocidolite fiber from South Africa and manufactured magnesia block insulation from “blue” asbestos. Berry, M., “Mesothelioma Incidence and Community Asbestos Exposure,” *Envtl. Research*, 75 (1997):34-40.

According to Langer and Nolan,” In retrospect, the mesothelioma experience reported [at the Somerset Hospital in Somerville, New Jersey] was attributed to exposure at the J-M facility, but likely contained cases exposed at the Asbestos Ltd. Facility as well.” (See Langer and Nolan, “Asbestos in the Lungs of Persons Exposed in the U.S.A.,” *Monaldi Arch. Chest Dis.*, 53(1998):168-80.) There is certainly no substitute for careful interviewing, either on the individual or cohort level.

Camus, Siemiatycki, and Meek’s study of mortality of women in two chrysotile mining areas of Quebec compared with mortality of women in 60 control areas showed that there was no measurable excess risk of death due to lung cancer among the women in the asbestos mining regions. They also determined that the EPA's model predicting relative risk of cancer overestimated the risk of lung cancer by at least a factor of ten. Camus, M., Siemiatycki, J., and Meek, B., “Nonoccupational Exposure to Chrysotile Asbestos and the Risk of Lung Cancer,” *New England J. of Medicine*, 338(1998):1565-71.


Camus and colleagues published again in 2002, “Risk of Mesothelioma Among Women Living Near Chrysotile Mines Versus U. S. EPA Asbestos Risk Model: Preliminary Findings.” The authors tested the EPA Mesothelioma Risk Model in a population having experienced relatively high and mostly non-occupational chrysotile exposures. Namely, they analyzed female mesotheliomas first diagnosed from 1970 to 1989 in chrysotile asbestos mining districts (Asbestos and Thetford). An international expert panel estimated historical ambient exposure levels in these districts. Ambient airborne asbestos exposures ranged between 0.1 and 3 fibers per milliliter before 1970. The EPA Asbestos Risk Model predicted 150 (range 30-750) female mesotheliomas in Asbestos while only one case (peritoneal) was observed; 500 cases (range 100-2500) were predicted in Thetford Mines, while 10 cases (pleural) were observed. These large discrepancies could not be explained by random or systematic errors. While the authors observed an excess of mesotheliomas among the subject population relative to the female population of Quebec, the observed incidents was orders of magnitude smaller than that predicted by the U. S. EPA model.


**Italy** – In Casale Monferrato, Italy, 64 cases of malignant mesothelioma were identified in which patients had no occupational or familial exposure to asbestos. The incidents of
histologically confirmed malignant mesothelioma among local residents was 4.2 in men and 2.3 in women compared to rates from the Cancer Registry of Varese (a nearby town) and Italian Cancer Registries which was 1.0 and 1.8 in men and 0.3 and 0.6 in women, respectively. Two more recent case-controlled epidemiological studies by the same authors provide further evidence linking the association between residential exposure and the risk of malignant mesothelioma development. Magnani, et al., (1995), (2000), and (2001).

In 2007, Maule, reported on a population-based case control study that included 103 incident cases of mesothelioma and 272 controls in 1987-1993 in the area around Casale Monferrato, Italy, where an important asbestos cement plant had been active for decades. Information collected included life-long occupational and residential histories. Residents at the location of the asbestos cement factory had a relative risk for mesothelioma of 10.5 (95% confidence interval, 3.8 – 50.1), adjusted for occupational and domestic exposures. Risk decreased rapidly with increasing distance from the factory, but at 10 kilometers, the risk was still 60% of its value at the source. The study provides strong evidence that asbestos pollution from an industrial source greatly increases mesothelioma risk. Furthermore, relative risks from occupational exposure were underestimated and were markedly increased when adjusted for residential distance.

A spatial case-control study was conducted of residents living near another asbestos cement plant in Italy who did not have occupational, domestic or household exposure. (Musti, et al., 2009) The plant used 20% amphibole (15% crocidolite and 5% amosite). Again, a strong inverse relationship was found between the risk of mesothelioma and distance of the residents from the plant. Results in this study supported the evidence of an increased disease instance around the Eternit asbestos cement factory in Bari, Italy. From 2003 on, another 15 new malignant mesothelioma cases with environmental exposure have been registered among residents close to the asbestos cement factory.

The authors concluded, “we believe that the intersection of all our results provides a little but decisive evidence in support of the existence of an association between malignant mesothelioma incidence and asbestos environmental pollution around the asbestos cement plant. We have also verified the lack of strong potential confounders, no other known sources of asbestos pollution were present in the urban area.” Musti, et al, "The relationship between malignant mesothelioma and an asbestos cement plant environmental risk: a spatial case-control study in the city of Bari (Italy)," Int. Arch. Occup. Health, 82:489,496(2009).

In 2014, Fazzo, et al. published an ecological study at the micro-geographic level of mesothelioma incidence in the neighborhood of an asbestos cement plant in the industrial area of Coroglio-Vagnoli in Naples, Italy. An Eternit asbestos cement factory was located in that area and it had been in operation from 1939 to 1986. As in the case of virtually all the asbestos cement plant studies where crocidolite was utilized, mesothelioma incidence in the study area was reported to be increased: 46 cases were observed versus 22.3 expected (SIR 2.02). When those cases with any occupational exposure were eliminated, and the increase was once again confirmed with 19 cases among men (SIR=12.4) and 11 cases among women (SIR=1.34).
In 2015, a study based on data collected by the Lombardy Mesothelioma Registry compared the number of observed versus expected malignant mesothelioma cases among workers, their cohabitants, and people living near another asbestos cement factory located in Broni, Italy. The factory used 10-15% crocidolite in the asbestos cement sheets and 30% or more crocidolite in asbestos cement pipes (in addition to using some chrysotile and small quantities of amosite). The authors found an increased number of observed cases of malignant mesothelioma compared to the number of expected cases among the workers (38 observed v. 2.33 expected), their cohabitants (37 observed v. 4.23 expected), and the people living in the area (72 observed v. 10.89 expected). Results of this study illustrated that “[m]en and women with environmental exposure had comparable relative risks (SIR 11.2 and 14.8, respectively).” Menzi, C., Riboldi, L., De Matteis, S., Bertazzi, P., Consonni, D., “Impact of an asbestos cement factory on mesothelioma incidence: Global assessment of effects of occupational, familial, and environmental exposure,” Environment International 74:191-199, 2015.

Japan – Kurumatani and Kumagai (2008) proposed that the distance from the place of residence to an asbestos plant was related to the risk of malignant mesothelioma. These authors calculated standardized mortality ratios of mesothelioma from 1995 to 2006 among the estimated population at risk that lived around a former large asbestos cement pipe plant in Amagasaki City, Japan, between 1957 and 1975, the time when the plant had used crocidolite and chrysotiles. The distance between the plant and homes and relative asbestos concentrations obtained by diffusion equations involving meteorological conditions were used to determine asbestos exposure levels among residents. Among persons who had lived within a 300 meter radius of the plant, the standardized mortality ratio of mesothelioma was 13.9 (95% confidence interval, 5.6 – 28.7) for men and 41.1 (95% confidence interval, 15.2 – 90.1) for women. The authors concluded that the mesothelioma outbreak among residents was causally associated with asbestos, in particular, crocidolite, that the plant had used, and that the affected area spread as far as 2,200 hundred meters from the center of the plant in a dose-dependent way. Kurumatani (2008)

West Bank, Louisiana – In 2009, Case and Abraham updated the literature with respect to the potential neighborhood exposures to asbestos in the communities of the West Bank of the Mississippi River. Case and Abraham recognized two trends gaining relative importance as the mesothelioma risk has begun to decline in the United States. “Legacy” exposures causing mesothelioma are most important in locales having past asbestos industry, shipyards, and/or local distribution of asbestos amphibole-containing material as a result. The authors used Jefferson Parrish, Louisiana as an example of this trend. Jefferson Parrish, Louisiana was chosen as the prototype of “legacy” exposures on the basis of historical evidence of asbestos plants with known mesotheliomas in the workforce, known shipyards in the same area, EPA records of distribution of crocidolite-containing scrap to and remediation of over 1,400 properties, NIOSH published data on mesothelioma by county, and exposure data including lung-retained fibre analyses in victims, where available. The authors noted that, with some exceptions, highest concentrations of cases were likely to be noted in counties having either asbestos-using industrial plants, shipyards, or both. Twenty-two Jefferson Parrish medicolegal mesothelioma cases (10 female and 12 male) were identified from Dr. Case’s files. At least 6 of the 22 were related; 2 sets of siblings and one parent and child. All were resident at some time in their lives (most frequently for most of their lives) in Jefferson Parrish. All but one of the cases were pleural. The peritoneal case was a 39-year old female with no definite history of asbestos exposure. All
other cases were pleural and had a definite history of asbestos exposure including one or more of direct occupational exposure in asbestos manufacturing plants or shipyards in the area; indirect exposures; or environmental exposures through living near asbestos manufacturing plants or at home addresses where asbestos-containing materials had been directly deposited.

The latest figures for Jefferson Parrish, Orleans Parrish and St. Bernard Parrish combined show the region to have the highest age-adjusted (to the 2000 U.S. population) annual incidence rate of mesothelioma in the state at 2.1 per 100,000 for the five years from 2000 to 2004, based on 104 recorded cases, as compared to 1.3 for the state as a whole. Case and Abraham describe in detail the Johns-Manville plant located in Jefferson Parrish, which included a pipe-manufacturing facility which used crocidolite “steadily” in the manufacture of “transite” cement pipe. According to the U.S. EPA and Louisiana Department of Environmental Quality documents obtained through Freedom of Information requests, through the 1950s and 1960s, in addition to atmospheric pollution from the plants, plant personnel produced an asbestos-containing aggregate which was distributed throughout the communities of the surrounding area (the “West Bank” of the Mississippi River). This contained both chrysotiles and crocidolite asbestos, and a proportion varying from 1:3 respectively to 1:1, and determined by testing to be 35% to 45% total asbestos by weight. The asbestos aggregate and filler formed “a concrete-like material when mixed with water and therefore was considered by many local residents to be concrete substitute for construction purposes.” It was distributed to local residents free of charge, often by pick up or dump trucks. The material was “located in residential yards and driveways, school playgrounds, around daycare centers, and in other areas easily accessed by the public” and “children were seen playing on driveways composed of friable ACM with toys and basketballs. . .vehicles were observed creating dust clouds when passing over areas that contained the ACM. All of these routine activities (were) expected to increase ACM friability and dramatically increase human exposures.”

Recently, NIOSH published an up-to-date supplement of this data for the five years from 2000 to 2004, finding 75 deaths from mesothelioma in that time period (age-adjusted rate 42.1 deaths per million, of which 34.7% were female) in Jefferson Parrish. This is by far the largest current number of mesothelioma victims in high-rate counties in the U.S.A., exceeding by 15-fold for example of the five deaths seen in the same period in Lincoln County, Montana, where the Libby “epidemic” is situated.

Similar to the situation in the West Bank, a 2003 report indicates a similar problem in the vicinity of the Certainteed plant located at the boundary of the Bellefontaine Neighbors, Riverview, and the city of St. Louis. Geri L. Dreiling, “Left Behind: Bellefontaine Neighbors residents have lived—and died—with asbestos for years. The problem’s an open secret, but little has been done.” Riverfront Time, January 8, 2003. As reported by the article, “Government-led efforts to clean up asbestos at the seventeen-acre Certainteed/GAF site failed in 1979, and the most recent efforts to address the problem at Maline Creek left contamination near homes, schools, and playgrounds because state officials say it would simply be too dangerous to dig out the asbestos and move it.” In addition, there were multiple reports of residents taking scrap from the plant and using it in their neighborhoods and yards.

**Review article** – In 2000, Bourdes, Boiffetta, et al., conducted a literature search on articles included in MedLine and current contents that appeared between 1966 and 1988. The
authors conducted a review of epidemiological studies of pleural mesothelioma and environmental (household and neighborhood) exposure to asbestos. The review also included a quantitative meta-analysis. For neighborhood exposure, relative risks ranged between 5.1 and 9.3 (with a single RR of 0.2) and the summary estimate was 7.0 (95% confidence interval, 4.7 – 11). The main result of the analysis was a “strong relationship between pleural mesothelioma and high environmental exposure to asbestos, whether the source of exposure is domestic or neighborhood.” Bourdes, Supra at 411.

**Neighborhood exposure to anthophyllite asbestos** – Rom, Hammar, Dodson, et al., reported on a 38-year old man who presented with an epithelial type diffuse malignant pleural mesothelioma with a neighborhood exposure up to age 18 years to anthophyllite asbestos. The patient lived ¾ mile from an asbestos product manufacturing plant. The asbestos plant in Finland manufactured various tanks and piping systems. It imported 4.5 million pounds of Finnish anthophyllite asbestos from December 1964 to January 1972. This period overlapped the patient’s age of 8-16 years when he resided, played, attended school, and delivered newspapers near the plant. The authors include a brief review of the literature on neighborhood anthophyllite exposures that are linked to the development of mesothelioma. The authors conclude that anthophyllite asbestos has been associated with neighborhood environmental exposure and pleural plaques. See also Letter to the Editor by Michael Kottek, American Journal of Industrial Medicine, 41:514(2002).

**California** – In 2005, Pan and colleagues conducted a cancer registry-based case control study of residential proximity to naturally occurring asbestos with malignant mesothelioma in California. The adjusted odds ratios and 95% confidence interval for low, medium and high probabilities of occupational exposures to asbestos were 0.71, 2.51 and 14.94, respectively. The data supported the hypothesis that residential proximity to naturally occurring asbestos is significantly associated with increased risk of malignant mesothelioma in California. The authors noted that an association with domestic or neighborhood exposure to asbestos or other mineral fibers and an increased risk of mesothelioma has been found in several locations including Australia, South Africa, Italy and New Jersey. An association between environmental exposure to naturally occurring asbestos and mesothelioma has been observed in Cyprus, Greece, New Caledonia, Corsica, China and Italy. Results of the study demonstrated that residential proximity to geologic sources of naturally occurring asbestos in California was independently and positively associated with case status. The odds of being a mesothelioma case are 6.3% lower than the odds of being a control subject for every 10 kilometers farther from the nearest asbestos source. The association was observed in both men and women, although the association was statistically significant in men only.

The article gave rise to two letters to the editor. Dr. Michael Kelsh pointed out important study design limitations of the Pan study, and opined that the study design limitations and defects of exposure conditions should be further addressed. Firstly, Kelsh wrote that “the fact that many of the regions classified as exposed have experienced a substantial influx of population migration strongly suggests exposure misclassification when relying on current residents’ information.” Other limitations included important problems with exposure misclassification. Dr. Schenker, and colleagues, responded that confounding by prior occupations was an unlikely explanation of their findings, based on a survey of union workers’ migration patterns and analytical control for previous occupation. Dr. Carl Andrew Brodkin also wrote to the editor and noted that the study design does not account for the important principle of latency. Secondly, Dr. Brodkin noted that while the measurement of distance from residence to a naturally occurring asbestos source provides data at an individual level, it does not provide a direct measure of asbestos exposure. In conclusion, Dr. Brodkin suggested that “given these limitations, the findings of Pan and associates should be considered
hypothesis generating.” “On the other hand, the findings are of sufficient concern that caution should also be applied to the large-scale development of homes and naturally occurring asbestos source areas in California.”

In 2011, Case and Abraham published a state of the science review document created in support of the NIEHS workshop, “Asbestos: A Science-Based Examination of the Mode of Action of Asbestos and Related Mineral Fibers.” In their discussion of legacy exposures, Case and Abraham “remind us of past neighborhood environmental exposures that occurred when the industries that produced them were still extant and that often resulted in community disease, particularly mesothelioma.” Case and Abraham cite three instructive examples of studies of environmentally exposed groups: (a) the extensive studies of Quebec Chrysotile Miners and Millers and of residents of that region; (b) Australian (Wittenoom) Miners and Millers of Crocidolite and residents of that region (citations omitted), and (c) studies conducted around the asbestos cement plant using crocidolite in Casele Monferrato, Italy (citations omitted). “In each of these situations, environmental and/or domestic exposures lead to asbestos-related disease, most seriously mesothelioma, and always (even when chrysotiles was also used or primarily mined) in the presence of asbestiform amphiboles. The current epidemic of mesothelioma in Jefferson Parish, Louisiana, was predictable on the basis of the presence of asbestos manufacturing plants and shipyards in the area.” Table 7-10 records Malignant Mesothelioma: Counties with Highest Age Adjusted Death Rates (Per Million Population), U. S. Residents Age 15 and Over, 2000-2004. Because a high male ratio is most likely to indicate an occupational origin, areas with greater female proportions are of particular concern for possible environmental (or domestic) exposures. For instance, in Jefferson Parish, Louisiana, where there were asbestos plants and shipyards and the use of ACM (including crocidolite) to cover driveways, school yards, and even daycare centers (Case and Abraham, 2009), there were 75 deaths at a rate of 42.1 deaths per million population, of which 34.7% were female.

True environment exposures – that is, exposures from either natural or legacy industrial sources – can also be a result of misclassification of occupational or household exposure. One example is provided by a misinterpretation of exposures to women in the Quebec mining regions. Camus and colleagues enlisted an international panel of experts to estimate occupational, household and environmental (“neighborhood”) exposure in the area; lifetime cumulative exposure was estimated (with considerable uncertainty) at an average of 16 fibers per cc – year. Camus and colleagues found no measurable excess risk of death due to lung cancer among women in two chrysotiles asbestos-mining regions; the U.S. EPA model thus overestimated the risk of asbestos-induced lung cancer by “at least a factor of 10.” However, 7 deaths from pleural cancer were identified during the same time frame (Camus 1998), all in the higher tremolite Thetford Mines area. An accompanying commentary (Landrigen, 1998) assumed these were due to “environmental” exposure, but ascertainment of individual data by questionnaire determined that of 10 cases in the area among women, all but one had occupational and/or household exposure.

Case and Abraham emphasized two additional points: first, that it is difficult to separate household (and often occupational) exposures from environmental or neighborhood exposures, and second, that all the studies have been performed in areas of high levels of exposures. Bourdes (2000) addressed this issue in their meta-analysis.

In 2011, Adgate, et al., published “Modeling community asbestos exposure near a vermiculite processing facility: Impact of human activities on cumulative exposure,” Journal of Exposure Science and Environmental Epidemiology 21; 529-35 (2011). The article notes that contaminated vermiculite ore from Libby, Montana was processed in Northeast Minneapolis from 1936 to 1989 in a densely populated residential neighborhood, resulting in non-occupational exposure scenarios from plant stack and fugitive
emissions as well as from activity-based scenarios associated with use of the waste rock in the surrounding community. Fiber emissions form the plant were the largest source of exposure for the majority of the cohort, with geometric mean cumulative exposures of .02 fibers/cc x month.

In 2013, Ryan, et al., published “Childhood Exposure to Libby amphibole during outdoor activities,” Journal of Exposure Science and Environmental Epidemiology, 1-8 (2013). Vermiculite from the Libby mine was the source of more than 70% of all vermiculite sold in the United States from 1919 to 1990. The study objectives were to describe available data from the US EPA preremediation activities for Libby amphibole (LA) exposure in Libby MT and develop an approach to characterize outdoor residential exposure to LA among children. Homes in Libby, MT included in the US EPA preremediation Contaminant Screening Survey (CSS) were categorized by the presence of interior and/or exterior visible vermiculite and concentrations of LA were measured in samples of dust and soil. Airborne exposure to LA while digging/gardening, raking and mowing were estimated using US EPA activity-based sampling (ABS) results. A total of 3154 residential properties participated in the CSS and 44% of these had visible exterior vermiculite. Airborne concentrations of LA where there was visible vermiculite outdoors were 3-15 times higher than during digging/gardening, raking and mowing activities compared with homes without visible outdoor vermiculite.

A 2013 study aimed to determine whether residential distance and meteorological conditions are related to differences in the risk of developing pleural mesothelioma. Tarres, et al., “Pleural Mesothelioma in relation to meteorological conditions and residential distance from an industrial source of asbestos,” Occup. Environ. Med. 70:588-590 (2013). The authors reported the incidence rate of environmental pleural mesothelioma was higher in the population living within 500 m of the plant than in those living in a radius of 500-2000m and much higher than those living 2000-10,000m. The highest incidence rate for pleural mesothelioma was found in the southeast quadrant of the 500m area, coinciding with the predominant wind direction.

Summary

In conclusion, while the medical and scientific literature has reported some cases of “neighborhood” exposed patients with mesothelioma, the bona fide neighborhood exposure cases appear to come almost exclusively from a few sites where amphibole asbestos, and particularly crocidolite, was abundant not only in the plant but outside the plant environment. Defense counsel seeking to deal with the “neighborhood” case would be well advised to learn as much as possible about the type of asbestos utilized in the particular facility, the work practices and product manufacturing processes there, and to search the historical record (including local newspapers, state environmental documents, and other such materials) to gain a very good understanding of the facility and of the neighborhood. In some localities there may be data on cancer deaths which may be useful. A good industrial hygienist may be essential in defending against a neighborhood claim.

V. Ambient Air/Background Exposures

In the year 2016, we know that asbestos is ubiquitous on earth and in its atmosphere, although at lower levels than in the 1950s, 60s and 70s. “Samples collected in remote locations of the earth indicate that asbestos is a ubiquitous atmospheric pollutant that has been present for thousands of years, as evidenced by Antarctic ice sample analyses for chrysotile.” Kohyania, N.,
Remote and rural locations have airborne asbestos concentrations of less than 1 ng$^{-3}$. According to Corn, these concentrations are "generally equivalent to fiber concentrations less than 0.0005 f/cm$^3$, or phase contrast microscopy equivalent fibers (calculated) greater than 5 microns in length of 0.0005 f/cm$^3$ (HEI-AR 1991). Urban area fiber concentrations of up to 0.002 f/cm$^3$ greater than 5 microns in length have been reported. Corn, M., “Airborne Concentrations of Asbestos in Non-Occupational Environments,” Annals of Occupational Hygiene, 38(1994):495-502.

In fact, ambient air concentrations for asbestos have been reported for various localities. Use of these background levels may be very helpful in defending against a claim of asbestos-related cancer. Where an industrial hygienist has calculated a plaintiff’s exposure over time, if that exposure can be shown to be at or below background, a much stronger defense case is made.

Virtually all expert witnesses agree that asbestos fibers are found in the lungs of persons living in most American environments. Very early on, Thompson and Graves examined the lungs of 500 Miami residents and found asbestos bodies in 31.6 percent of the men and 20.4 percent of the females. In the great majority of the cases, including all the females, the asbestos bodies were scanty, were not associated with any pulmonary changes, and were regarded as the result of contamination of the urban atmosphere by asbestos. Thomson, J.G., Path, P.C. and Graves, W.M., “Asbestos as an Urban Air Contaminant,” Archives of Pathology, 81(1966):458-64.

A study of T.R. Fears, “Cancer Mortality and Asbestos Deposits,” appearing in American Journal of Epidemiology, 104(1976):523, found no significant relationship between deaths caused by cancers in states containing deposits of asbestos (both amphibole and chrysotile) and deaths in peripheral states.

Wigle similarly found that there was no increase in the number of deaths attributable to the presence of asbestos fibers in the drinking water of 20 cities in Quebec, including Asbestos and Thetford Mines, even though the concentration of asbestos fibers in the nonfiltered drinking waters of these places was high. Wigle, D.T., “Cancer Mortality and Relation to Asbestos in Municipal Water Supplies,” Archives of Environmental Health, 185, July-August 1977.


We are all exposed to asbestos because it is in the water we drink and the air we breathe. Counts of up to four million fibers per litre of drinking water have been found in Southern Ontario municipalities (e.g., Sarnia and Metropolitan Toronto) and of up to 22 million fibers per litre in Northern Ontario municipalities (Thunder Bay). The fibers are most invariably extremely short, that is below one micron in length. The small dimension of these fibers, coupled with our finding that
asbestos disease is occasioned by inhalation rather than ingestion, leads us to conclude that asbestos in drinking water is not a health hazard. (p. 15)

The report further observed:

We conclude that an ambient air quality objective such as the 0.04 fibers/cc guideline of the Ministry of the Environment remains important, not so much because of any health hazards that might be posed at such levels of exposure, but so that any unusual level of contamination can be identified and its source sought out. (p. 16)

The report also described surveys taken in 1975 and 1976 by the Ontario Ministry of the Environment. They surveyed the asbestos fiber concentrations in the air at a number of locations around Ontario. The 1975 survey of seven areas, taking several samples in each area, contained no area with median sample concentrations of fibers longer than five microns greater than 0.01 f/cc.” In fact, the median count in each area of fibers longer than five microns was usually zero or nondetectable. The count of fibers longer than five microns in any single sample, if not zero or nondetectable, ranged as high as 0.054 f/cc.” The number of fibers of all lengths was considerably greater than the number of fibers longer than five microns, but still always less than one f/cc. A later study by Dr. Eric Chatfield for the Royal Commission also found what they termed “extremely low” asbestos fiber concentrations in rural areas. One set of samples taken adjacent to an expressway ramp in downtown Toronto and in two suburban locations were also very small. The greatest asbestos fiber concentrations in the study were found in the expressway ramp, but “even there the maximum concentration was only 0.0042 fibers/cc over five microns in length, and the median concentration was less than 0.0033 fibers/cc. The report concluded that ambient asbestos fiber concentrations present no health risk.

The Royal Commission report also has helpful observations about brake linings and dust emission levels from them. They conclude that “the weight of [the] evidence is that it is likely that automotive brakes cause only a fraction of the airborne urban asbestos detected, although it is possible that the fraction may be greater than one-half. In any event, we have found urban airborne asbestos levels to be extremely low.’


Little, if any, direct evidence is available on the risk of mesothelioma (or of other asbestos related diseases) as a consequence of exposure to asbestos in the general environment in countries where asbestos has been used industrially for many years. Although asbestos fibres are widely found in the ambient environment, the concentrations are very low, as compared with most present, and certainly most past, occupational levels of exposure.

The only nonindustrial ambient asbestos exposures producing mesotheliomas are those areas in Turkey where erionite is found, as well as some tremolite and chrysotile. This exposure
is actually more than environmental, but also occupational, since the asbestos was used in various ways for building or whitewashing houses. The report concluded on page 393 that:

No direct epidemiological evidence that mesothelioma and lung cancer have resulted from general environmental exposure to asbestos in industrialized countries is available.

Two Greek researchers who studied environmental and domestic exposures in Greece and Turkey concluded that:

Asbestos is not a macro-pollutant. Thus, the terms “nonoccupational mesothelioma” and “environmental mesothelioma” are not synonymous. There is no environmental mesothelioma in the sense of urban environmental pollution from asbestos. There is, on the contrary, nonoccupational/domestic mesothelioma from domestic sources of asbestos. This is different from the paraoccupational/domestic exposure occurring in family members of asbestos workers, e.g. wives washing clothes covered with asbestos. The non-occupational domestic exposure has occurred in many areas of the Mediterranean countries where small villages are located inside or near naturally occurring asbestos ores and inhabitants have traditionally used this asbestos-containing soil for domestic uses (mainly whitewashing).


Summary

You need to know the literature on ambient air concentrations for a number of reasons. Juries must understand that asbestos is everywhere in the ambient environment, from natural and man-made sources, but is not a health threat. For this reason, the plaintiffs’ “every exposure contributes” theory is not credible. Also, the fiber concentrations measured in many buildings with asbestos in place are equal to or less than the ambient air. (Indeed, in one case in our experience, a DuPont plant demonstrated fewer fibers per cubic foot than at the stop sign outside the plant.) Finally, an understanding of ambient levels helps the jury understand that if a plaintiff’s lung burdens are at background levels, his mesothelioma is idiopathic.
VI. Special Approaches in Female Cases

There are relatively few cases of asbestos-related disease in females. This is largely because most women in the generations during which asbestos-containing materials were prevalent, did not work in asbestos-related industries. The most famous exceptions are, of course, the women who worked in the asbestos textile industry in the 1920s and 1930s, and the World War II crocidolite gas mask manufacturing workers in Britain. The textile workers had such incredibly high exposures that they often developed asbestosis within ten years of their initial exposure. The gas mask workers utilized crocidolite in the manufacture of the product, and significant percentages of them developed mesothelioma two or three decades later.

Claims for asbestosis by women are very suspect, unless the woman actually was occupationally exposed. Because asbestosis requires a long and intense exposure for development of the disease, persons defending such cases should be very wary of the validity of the claim for asbestosis from household or neighborhood exposure. It is most likely that, if the medical record does demonstrate decrements in lung function or abnormal x-ray findings, that there is another cause. A good medical history taken by a very competent pulmonologist, as well as a solid set of B-readings and pulmonary function studies, are very likely to provide a more than adequate defense to such a case.

Mesotheliomas are more difficult to defend because it has been established that household exposures may cause mesothelioma. (See Section II.) In evaluating the case, defense counsel should first determine whether the diagnosis is incorrect. Most pathologists agree that mesothelioma is not an easy diagnosis. It goes without saying that defendants should obtain a consultation from a pathologist experienced in diagnosing mesotheliomas and especially one familiar with the various immunohistochemical stains which distinguish mesothelioma from a number of other malignancies. In women, and especially in women with peritoneal mesothelioma, there are a number of conditions which present real diagnostic challenges. Ovarian tumors and primary and secondary serous papillary carcinomas of the peritoneum must be distinguished from true malignant mesotheliomas.

According to Roggli, the well-differentiated papillary mesothelioma of the peritoneum is a special variant characterized by “an indolent clinical course in prolonged survival.” Roggli also mentions decidual peritoneal mesothelioma as another uncommon variant that affects young women. Roggli, V., et al., “Malignant Mesothelioma in Women,” Anatomic Pathology, 2 (1997):147-63.

Goldblum and Hart pointed out in their 1995 article that:

The diagnosis of mesothelial tumors in women is fraught with difficulties for many reasons. Chief among them is the recognition that the peritoneum is a major site of serous epithelial tumors, either metastatic from the ovary and other genital organs or arising as primary neoplasms directly from the peritoneum. Their article is an exhaustive review of the differential diagnoses in potential peritoneal mesotheliomas in women. The authors concluded that, “Unlike pleural mesotheliomas, peritoneal mesotheliomas in women do not appear to be closely related to asbestos exposure.”

Assuming the diagnosis of mesothelioma is confirmed, and also assuming that the plaintiff did not have a significant household or occupational exposure, the mesothelioma is most likely due either to some other known or suspected cause of mesothelioma (radiation treatments for a Wilms tumor, breast cancer, or some other malignancy some years prior, perhaps SV-40), or is unknown (idiopathic). The investigation for other causes is no different than that for men. However, in women, only approximately 20 to 50 percent of mesotheliomas has been termed asbestos-related, depending on the author. Most experts will agree that there are certain percentages of all malignancies for which there is no known cause. A thorough examination of the background incidence of the particular type of mesothelioma is in order. A most important fact to get across to the jury is that the numbers of mesotheliomas in women have not increased over the past few decades. This is not true for men.

Price, in “Analysis of Current Trends in United States Mesothelioma Incidence,” *American Journal of Epidemiology*, 145(1997):211-18, demonstrates that the growth rate of mesothelioma in females is zero between 1973 and 1993. However, the growth rate for men has increased in that time period. The age group with the greatest growth rate is those over 75.

Charts in the Price article are a dramatic demonstration of the lack of change in the incidence in women versus men. Figure 4 on page 216 of the Price article shows the projected number of mesothelioma cases in toto based on SEER data. The numbers in women, according to his projections, decrease only infinitesimally, while those in men demonstrate a steep decline from 1997 and approach that of women in 2047. Price stated that:

For females, the average historical mesothelioma rate was applied to all cohorts after the 1955-1959 cohort. This assumption is not controversial because the trend for females has been virtually constant for the past 20 years. Spirtas *et al* came to similar conclusions.

In our study nearly 90% of incidences of pleural mesothelioma among men were directly attributable to past exposures to asbestos. Although there were only a small number of peritoneal mesothelioma cases among men available for study, it seems that a substantial percentage of these cases, perhaps 60%, also could be attributed to asbestos exposure. Among women, however, only about 20% of the cases were attributable to asbestos exposure. As the incidence of mesothelioma among women (about three cases per million women per year for all primary sites combined) is much lower than among men and has remained reasonably constant over time, it is possible that the incidence in women may be close to the background level. Alternatively, exposure to asbestos is lower and misclassification of exposure may be greater among women which would also reduce their attributable risk.

In 2004, Price, *et al* published an update on analysis of the SEER data including the years from 1993 through 2003. Compared with their earlier analyses based on data through 1992, a slower decline was found in male cases immediately after a peak in 2000-2004, but "no other
notable changes in the time pattern were detected." Id. at 107. According to the authors, "The constancy alone of the mesothelioma risks for females over time supports the existence of a threshold exposure." Id. at 110. The authors found that the age-adjusted mesothelioma rate for females was constant at an average of approximately 0.30 per 100,000 between 1973 and 1982, when it showed a one-time increase to 0.40 per 100,000.

Drs. Moolgavkar, Meza and Turim published their analysis of the SEER data from 1973 to 2005 and reached a similar conclusion regarding the female rate of mesothelioma. Moolgavkar, Meza and Turim, "Pleural and Peritoneal Mesotheliomas in SEER: Age Effects and Temporal Trends, 1973-2005," Cancer Causes Control, 220:935-944(2009). Moolgavkar et al, found that "age-adjusted rates of pleural mesothelioma among women have remained more or less constant at about 2.5 per million persons-years over the period 1973-2005 and that age-adjusted rates for peritoneal mesothelioma in both men (1.2 per million person-years) and women (0.8 per million person-years) exhibit no temporal trends over the period of the study." Id. at 935.


“In women the ratio of pleural to peritoneal tumours is about 2:1 and only about 20% of all mesotheliomas in women in the United States can be reasonably linked to past asbestos exposure. Indeed, recent data suggest that, in contrast to the dire prognosis of pleural mesotheliomas, a substantial proportion of peritoneal mesotheliomas in women are curable, implying that this fraction may represent a different disease. It is likely that in fact most cases of mesothelioma in women represent the background (non-asbestos related) incidence of this disease. These are the reasons that we have confined this analysis to mesothelioma trends in men.” Id. at 440

It appears that few, if any, peritoneal mesotheliomas in women are asbestos-related. For example, Dr. Paul Sugarbaker, et al, in their case-control study of all cases of peritoneal mesothelioma at the Washington Cancer Institute (a major referral center for peritoneal mesothelioma) did not find a causal relationship between asbestos exposure and peritoneal mesothelioma in women.

"Some studies would link asbestos exposure as a significant etiologic factor in peritoneal mesothelioma, but implication of asbestos exposure and the causation of peritoneal mesothelioma is far less obvious than in pleural mesothelioma [6,7]. To further investigate the relationship between asbestos exposure and the development of peritoneal mesothelioma, a case-control study was conducted on all patients with confirmed diagnosis of peritoneal mesothelioma at the Washington Cancer Institute [8] . . . Although there was a strong relationship between asbestos exposure and peritoneal mesothelioma in men, our data did not support a relationship between asbestos exposure and peritoneal mesothelioma among women. In men there was a relationship between occupational exposure alone and peritoneal mesothelioma; this association was not seen in women, our study suggested that the epidemiology of peritoneal mesothelioma does differ
between men and women, but the current data was insufficient to tell how and why."

Sugarbaker, Welch, Mohamed, and Glehen, "A Review of Peritoneal Mesothelioma at the Washington Cancer Institute," *Surg Oncol Clin N Am*, 12:605,606(2003). Note that one of the coauthors of this study was a prominent plaintiff’s expert in asbestos litigation, Laura Welch, M.D.

One important fact to keep in mind regarding peritoneal mesotheliomas in women is the majority agreement among experts that peritoneal mesotheliomas result from heavier asbestos exposures, if they are indeed asbestos-related. Oury, Hammar, and Roggli studied tissue asbestos content in 40 cases of peritoneal mesothelioma compared to 170 pleural mesothelioma cases. They concluded that “more asbestos is needed to induce peritoneal as compared to pleural mesothelioma” Oury, Hammar, Roggli, “Asbestos Content of Lung Tissue in Patients with Malignant Peritoneal Mesothelioma,” Presentation 923 at the 8th World Conference on Lung Cancer, Dublin, Ireland, August 1997. This fact buttresses the argument that a nonoccupational peritoneal mesothelioma in a woman is very likely either due to some other cause or is idiopathic. See Coggon et cd., “Differences in Occupational Mortality from Pleural Cancer, Peritoneal Cancer, and Asbestosis,” *Occupational and Envtl. Medicine*, 52(1995):775-77.

**Summary**

Asbestosis among women with household exposure is very rare; obtaining the plaintiff’s treating medical records, a good medical history, and IME are likely to eliminate asbestos as a cause. The medical literature demonstrates only a few cases of asbestosis in women with “household” exposures, and their actual exposures approached bystander occupational levels.

In mesothelioma cases, it is vital to exclude other similar malignancies especially in peritoneal mesothelioma. Search for other possible causes, and emphasize to the jury that at least 50 percent to 80 percent of mesotheliomas in women is not related to asbestos. Make sure the jury understands that there is a background level of mesotheliomas, and that the rate of mesotheliomas has been flat among women, unlike in men. Emphasize that many experts believe that the mesothelioma rate in women is close to the background level. Go over Roggli’s table on Mesotheliomas in Women showing that the household mesotheliomas in that article were wives of pipecoverers, asbestos factory workers, and others with long direct exposures to asbestos. Emphasize the need for extra heavy exposures to attribute a peritoneal mesothelioma to asbestos.

**VII. Genetic Predisposition**

There is some evidence that a tendency to mesothelioma runs in families. There have been a few studies suggest this possibility. Vianna *et al* studied the occupational histories of 52 females with malignant mesothelioma and certain of their relatives. The study revealed that the frequency of parental cancer was also significantly greater for cases than for their controls. The authors suggested that it raises the possibility of a genetic predisposition to malignant mesothelioma. Vianna and Polan, “Non-Occupational Exposure to Asbestos and Malignant Mesothelioma in Females,” *The Lancet*, May 20, 1978:1061-63.
A 1984 article by Swedish researchers profiled two pairs of siblings with malignant mesothelioma. One sister and a brother experienced “slight household asbestos exposure during childhood.” Their father had worked at a foundry where asbestos was used for insulation purposes and he brought his work clothes home where they were hung on a hook in the kitchen where the children played. Both developed and died from mesothelioma. The other case involved identical twin brothers who were occupationally exposed to asbestos as shipyard platers and draftsmen in the shipyard office. One developed a pleural effusion in 1981 and by the next year, he was diagnosed with malignant mesothelioma. The other developed symptoms of mesothelioma in 1981 and died one year later. Martensson, Larson, Zettergren, “Malignant Mesothelioma in Two Pairs of Siblings: Is There a Hereditary Predisposing Factor?” European J. of Respiratory Disease, 65(1984):179-84.


Krousel et al reported on a family in which three persons developed pleural mesothelioma. Their mother also died of malignant mesothelioma. Her husband had worked as a laborer for a lumber and shingle company for ten years. The family lived within one mile of the company, which used asbestos insulating material as wrapping for its steam pipes. The son, who developed mesothelioma at age 35, was a torpedo man aboard a nuclear submarine for five years beginning at age 18 and then worked as a shipyard helper for one month and a cement pipe maker for two months. The authors reported that there was no microscopic evidence of asbestos fibers in either the daughter, the son, or the mother. The authors further found that other types of cancer also occurred in this family. The brother of the mother died at age 52 of renal carcinoma and her fraternal twin sister died at age 58 of a metastatic carcinoma of uncertain origin. Her elder sister died at age 66 with an infiltrating bronchiolar carcinoma. This may be the strongest case for genetic predisposition in the literature, since the asbestos exposure appears to be very light and/or brief and in the case of the son, lacking in sufficient latency. Krousel, T., Garcas, N., and Rothschild, H., “Familial Clustering of Mesothelioma: A Report on Three Affected Persons in One Family,” Am. J. of Preventative Medicine, 2 (4) (1986):186-88.

Heineman et al compared reported histories of cancer in first degree relatives obtained from telephone interviews with the next of kin of 196 patients who had a pathologic diagnosis of mesothelioma, and with those from 511 deceased controls. Among men, they found a statistically significant two-fold elevation in the risk of mesothelioma for patients reporting cancer in two or more first degree relatives. They found no significant elevation in women or among the small number of men without asbestos exposure. The next-of-kin of three patients (but no controls) reported a possible mesothelioma in a first degree relative. However, asbestos exposure could not be ruled out among those relatives. The authors concluded that the results provided “suggestive, but limited evidence that a family history of cancer may be a risk factor for mesothelioma, or may indicate an increased susceptibility to mesothelioma given asbestos exposure.” Heineman et al, “Mesothelioma, Asbestos and Reported History of Cancer in First-Degree Relatives,” Cancer, 77(1996):549-54.

In the same year, Huncharek et al published a case control study on the same topic. They examined parental cancer history in a cohort of 39 cases of pleural mesothelioma and 259 age-
matched controls. Twenty-eight (71 percent) of the cases reported a parental history of cancer versus 114 (44 percent) in the control group. The authors concluded that the data suggest a possible role for family history in the development of pleural mesothelioma and that “genetic predisposition may be important in the etiology of this tumor.” Huncharek, M., et al., “Parental Cancer and Genetic Predisposition in Malignant Pleural Mesothelioma: A Case Control Study,” Cancer Letters, 102 (1996):205-08.

Roggli et al in their chapter, “Malignant Mesothelioma in Women,” in Anatomic Pathology, 1997, also suggest that the discovery of a Wilms tumor suppressor gene and the identification of the gene product in a large proportion of mesotheliomas may provide a lead in the study of familial or hereditary predisposition to mesothelioma.

More recently, the review article by Carbone, Dodson, et al, supra provide an excellent summary of the current state of knowledge regarding genetic predisposition to malignant mesothelioma. They argued that there is substantial evidence supporting genetic predisposition to malignant mesothelioma as a result of studies of mesothelioma in the Cappadocian region of Turkey where malignant mesothelioma "causes up to 50% of deaths in the villages of Karain, Sarihidir, and Tuzkoy as a result of the inhabitance building their homes from stories containing levels of erionite, a very carcinogenic mineral fiber that specifically causes malignant mesothelioma (Roushy-Hammady, et al, 2001; Dogan, et al, 2006). Id. at 51. According to Carbone, et al, "Incidences of MM similar to those observed in the Cappadocian, families have also been detected in some US and European families. In these cases, genetic predisposition and exposure to asbestos appears to be the most likely cause. In other words, there are some families who are very sensitive to mineral fiber carcinogenesis and whether the fiber is asbestos or erionite does not appear to make any difference." Carbone, M., et al, "Malignant Mesothelioma: Facts, Myths, and Hypotenes," Journal of Cellular Physiology, 2227: 44-58. 2012.

Some of these same researchers (including Joseph Testa and Michele Carbone) have discovered that Germline BAP1 mutations predispose individuals to developing a number of cancers, including uveal melanoma and mesothelioma. Testa, et al, "Germline BAP1 mutations predisposed to malignant mesothelioma," Nature Genetics, 10:1022-1026(Oct. 2011). While the researchers demonstrated the existence of a BAP1-related cancer syndrome characterized by mesothelioma uveal melanoma and possibly other cancer types, there was insufficient information to determine whether "individuals with BAP1 mutations are exposed to asbestos, mesothelioma predominates," or alternatively, "BAP1 mutations alone may be sufficient to cause mesothelioma." Id. at 1025. In a more recent update, some of the same researchers, including Dr. Joseph Testa, described a new family with BAP1 mutations leading to a number of cancers, including mesothelioma, in numerous family members. They noted that "the fact that several members of the family manifested 2 or more different types of cancer suggests widespread BAP1 related tumor susceptibility targeting tissues of multiple organs. In addition, a review of BAP1 cancer syndrome families reported to date indicates that the location of the BAP1 mutation does not have any bearing on the spectrum of cancer types observed, either for mesothelial or melanocytic tumors." Cheung, et al, "Further evidence for Germline BAP1 mutations predisposing to melanoma and malignant mesothelioma," Cancer Genetics, 206:210(2013).

See also Huncharak, "Non-Asbestos Related Diffuse Malignant Mesothelioma," Tumori, 88:1-9(2002) ("Available information suggests that genetic factors may play a larger role in the
ideology of this disease than currently appreciated" at page 1). Hunchak also concluded that "biology of mesothelioma is an enigma. Although this disease appears to occur in the absence of asbestos exposure, the genetic and biological differences between asbestos-related and non-asbestos related tumors is unclear. Additional epidemiological and laboratory studies are needed to provide a better understanding of the relationship between environmental and non-environmental causes of mesothelioma." (Id. at 1).

In 2013, de Klerk et al analyzed subjects and their families from Wittenoom, Australia (discussed supra) because of their detailed follow-up over many years and their exposure assessments which provided a unique opportunity to study the interaction of genetic and environmental changes which resulted in asbestos-related diseases. This was the first study that was able to adjust properly for degree and timing of asbestos exposure when evaluating genetic risk of mesothelioma. They found a doubling of risk that may be attributable to genetic factors and “suggest that there is an important, but not large, genetic component in [malignant mesothelioma] similar to that found for other cancers.” de Klerk, N., Alfonso, H., Olsen, N., Reid, A., Sleith, J., Palmer, L., Berry, G., Musk, A., “Familial aggregation of malignant mesothelioma in former workers and residents of Wittenoom, Western Australia,” International Journal of Cancer 132:1423-1428, 2013.

Summary

It seems likely that there is a genetic component in the causation of malignant mesothelioma. This is obviously an area in which the defense lawyer must tread very carefully, but jurors are aware that genetics play a role in cancers. One can only hope that medical science will fully uncover the genetic links in the future.

VIII. Fiber Counts and Types

Often the most effective weapon in the arsenal of defense counsel in a mesothelioma case is the analysis of lung tissue for fiber analysis. There are three potential rewards:

1) the determination that the numbers of fibers in the lung are within the same range as persons who have background exposure, providing the evidence to conclude the mesothelioma is not asbestos related;

2) the finding that the specific type of fiber contained in one’s defendant’s product is either absent from the tissues or present in background level quantities; and

3) findings of an elevated amphibole fiber burden and a background or slightly elevated chrysotile burden supportive of a chrysotile defense.

A number of laboratories perform fiber counting and analysis using a variety of techniques. The lung tissue is digested in some manner and the remaining mineral fibers are counted by transmission electron microscopy (TEM), or scanning electron microscopy (SEM). Some studies are reported in wet weight and others in dry weight. To convert wet weight to dry weight for uniformity, multiply by ten. Because of differences in techniques, size of fibers counted, age of persons whose tissue were analyzed, and geographic location of the person studied, researchers come up with a variety of “background” levels. For instance, Dodson et al in 1999 published a study of 33 persons from a rural east Texas area. Of the 33 persons, ten were
females and 23 were males. Perhaps more importantly, six persons were aged 12 to 21 and demonstrated no fibers. Four others aged 26 to 41 also had no fibers. Therefore, the average number of total asbestos fibers found by Dodson was 84,000 per gram of dry lung. Compare this with the 1991 study by Gibbs et al, which reported an arithmetic mean of 15,600,000 total asbestos fibers among 55 controls from Cardiff. See Gibbs, 1991, in Section VIII. The length of fibers analyzed were as small as 0.5 microns. Comparison of quantitative findings from one lab with backgrounds from another may be of limited value because of these differences.

In addition to merely counting numbers of fibers to determine whether the plaintiff had an exposure beyond background, defense counsel may want to have an analysis and count of the specific types of fibers performed, depending on the type of fiber in the particular products in question. Most knowledgeable experts recognize a gradient of carcinogenicity among types of asbestos fibers. Crocidolite and amosite are amphiboles, which are straight, rigid, and needle-like fibers. Chrysotile, a “serpentine” fiber, is a completely different mineral. It has a curly structure which readily breaks down over relatively short periods of time into small units called fibrils. It has been shown that chrysotile is cleared from the lungs in a matter of weeks or months, whereas amosite and crocidolite remain in the lungs for decades.

Epidemiological studies have shown that crocidolite and, to a lesser extent, amosite are related to the development of mesothelioma. Many experts believe that among persons exposed only to chrysotile or to mixed fibers, only those with extremely high exposures to chrysotile in the range associated with asbestosis have even the potential of developing a chrysotile-related mesothelioma. Some experts believe that processed chrysotile, from which the potential amphibole contaminant tremolite has been removed, is not implicated at all in the causation of mesothelioma. In any event, defendants whose products contain only chrysotile are likely to benefit from analysis of the lung tissue, and counting of the fiber types in that tissue. There is a good likelihood that the tissues will contain no chrysotile at all, or chrysotile within background. A defendant in this position would also hope that the analysis would show no tremolite or only background levels.

For the chrysotile defendant, if the fiber analysis shows above background levels of amosite and/or crocidolite, his defense is advanced further because he can pin responsibility on the amosite product. Defendants whose product contained amosite, and even crocidolite, can benefit if the fiber analysis shows no trace of these types of fibers, or of background levels. In a case where the exposure is very suspect, it is almost always worth the effort to obtain a fiber analysis.

In the past decade, there have been a number of articles in the medical literature regarding the mineral fiber content of persons allegedly exposed in nonoccupational settings. In 1990, Gibbs et al published an investigation into ten “paraoccupational” cases of malignant mesothelioma and compared them to an analysis of seven cases of malignant mesothelioma in the crocidolite gas mask workers. Nine of the ten “paraoccupational” cases were thought to have developed their tumors because of exposure to asbestos on their husband’s work clothes, and one developed in a daughter of a man who had died of asbestosis. The husbands’ exposures varied greatly, as they had worked in shipyards, as laggers, in the building trades, and in “ordnance.” The lungs from the paraoccupational group showed a more variable fiber count than in the gas mask workers. However, there were above background concentrations of crocidolite or amosite or both in eight of the ten cases; chrysotile was nearly always within the range of controls. The authors
concluded that amphiboles are much more important in the genesis of malignant mesothelioma than chrysotile. Tremolite was not found in significant concentrations in either the paraoccupational cases or the controls. Two of the ten paraoccupational cases were below background, and both of them had husbands with nebulous exposure histories. One was a dockyard worker and the other was a "jobbing" builder. For the "paraoccupational" (household) exposed patients, the authors concluded that, "In general the fiber burdens in the paraoccupational group were similar to other groups of workers that we have seen with light or moderate direct industrial exposure to asbestos." Gibbs, A.R., et al., "Comparison of Fiber Types and Size Distributions in Lung Tissues of Paraoccupational and Occupational Cases of Malignant Mesothelioma," British J. of Indus. Medicine, 47(1990):621-26.

Roggli and Longo examined the mineral content of six household context of asbestos workers and four persons with claimed exposure to asbestos in buildings. All had various diseases alleged to be related to asbestos. Of the six household contacts, three had pleural mesothelioma and three had lung cancer. The individual with lung cancer had what was termed "mild asbestosis." Another lung cancer patient had parietal pleural plaques. Both these women were wives of insulators with asbestosis and lung cancer themselves. In the household contact cases, commercial amphiboles accounted for 48 percent of all fibers analyzed. Noncommercial amphiboles, including tremolite, anthophyllite and actinolite, were on average 10.5 percent, and chrysotile was only 4.2 percent. Other fibers, including talc, silica, and so forth, were 37 percent of all fibers found.

The findings were very different among persons claimed to be exposed in buildings. Only 4.4 percent of all fibers found was commercial amphiboles, 20 percent was noncommercial amphiboles, and only 2 percent was chrysotile. The vast majority (73 percent) was other fibers. Their study indicated that:

Building occupants have pulmonary asbestos burdens that are quite similar to individuals with no known occupational exposure to asbestos and it would be anticipated that their risks for developing an asbestos-related disease would be correspondingly low.

They further stated that:

We have no reason to believe that these individuals are anything other than representative of the background, "nonexposed" population for our area. Furthermore, not all mesotheliomas are related to asbestos exposure since spontaneous cases do occur, as do a few rare cases attributable to causes other than mineral fibers.

One case of a woman who worked in a building with asbestos-containing materials was found to have an unusual number of high aspect-ratio tremolite fibers in her lungs. The authors speculated that, especially because she demonstrated histologically confirmed pleural plaques, her pleural mesothelioma was asbestos-related, although they could only state that," Acoustical ceiling plaster... was 'the most likely source of the tremolite asbestos fibers that were identified:' They further indicated that, "Additional studies are necessary in order to determine whether [cases of exposures to acoustical ceiling plasters] such as these occur with sufficient frequency to be of
Following up on the examination of tremolite in the lungs, Srebro and Roggli published a 1994 study of asbestos body counts and mineral fiber analysis on pulmonary tissue from five mesothelioma cases and two asbestosis cases with pulmonary tremolite burdens greater than background levels. They found no uncoated amosite or crocidolite fibers in any of the cases. Three of the patients had been occupationally exposed to chrysotile asbestos; two patients had environmental exposures (one to vermiculite and one to chrysotile and talc); and one was a household contact of a shipyard worker. They found that the tremolite burdens for the asbestosis cases were one to two orders of magnitude greater than those for the mesothelioma cases. The authors noted that a potential source for the exposure for the three women in the study was cosmetic talc, which may be contaminated with tremolite asbestos. They also noted that uncoated chrysotile fibers greater than five microns were detected in only two of the seven cases, and that the tremolite concentrations for those two cases were four to five times the chrysotile concentrations. The authors implied that it is more likely the tremolite than the chrysotile which is the causative agent. They stated that:

The finding of relatively modest elevations of tremolite content in some of our mesothelioma cases suggests to us, that at least for some susceptible individuals, moderate exposures to tremolite-contaminated dust can produce malignant pleural mesothelioma.


While we are conditioned not to expect to find crocidolite in the tissues of persons exposed to asbestos-related disease, a paper by Langer and Nolan published in 1998 shows that there is evidence of more crocidolite in the U.S. than one would expect. The authors obtained tissue from autopsy or biopsy from 81 workers and two household persons who had died of various allegedly asbestos-related diseases. Thirty-three were mesotheliomas, 35 were lung cancers, 12 were asbestosis, and three died from other cancers. Twenty-three of these were insulators or pipecoverers, 28 were shipyard workers in various trades, 30 were persons in other trades, and only two were housewife family members of insulators. The main surprising finding in this study was the much higher incidence of crocidolite in the tissues of these people. Crocidolite was found in 39 percent of the tissue specimens obtained from individuals who had some history of shipyard work. This included one wife of an insulator who developed lung cancer. In that case, the wife had 700,000 crocidolite fibers per gram of lung tissue, but the counts for amosite and chrysotile were below the level of detection. There is a very interesting appendix on crocidolite consumption information pertinent to the United States which will be of interest to all whose products did not contain crocidolite. The authors further concluded that, “A worker must inhale many orders of magnitude more chrysotile fiber, as compared to amphibole fiber, to achieve the same risk for mesothelioma.” The authors also pointed out that tremolite occurs with the highest concentrations in lungs of plasterers. They noted that,
Patching, taping, and spackling compounds marketed in the U.S.A. are known to have contained tremolitic talc in addition to chrysotile, although the actual materials have been shown to contain cleavage fragments rather than asbestos.

The authors also pointed out that while tremolite may be associated with chrysotile, it is also associated with other types of commercial amphibole asbestos and that, “Calcic-amphibole contamination of amosite needs to be considered as well as other sources of tremolite unrelated to chrysotile.” Langer, A.M. and Nolan, R.P., “Asbestos in the Lungs of Persons Exposed in the USA,” Monaldi Arch Chest Dis., 53(2)(1998):168-80.

Dodson and others have published two reports on examination of other types of tissue for fiber burdens. No firm conclusions can be drawn from their data, but it is important to be aware of these developments. Dodson, R.F., Huang, J., and Bruce, J.R., “Asbestos Content and the Lymph Nodes of Nonoccasionally Exposed Individuals,” Am. J. of Indus. Medicine, 37(2000):169-74, examined a cohort of 21 persons previously defined as “nonoccasionally exposed to asbestos.” Tissue burden of asbestos obtained from lung analysis by analytical electron microscopy was compared with burden in the lymph nodes. Of the 12 individuals, no asbestos fibers were detected in the nodes of eight. The majority of the fibers found in lymph nodes were less than five microns in length and were most often noncommercial amphiboles. There were only two samples with ferruginous bodies in the lymph nodes. The authors concluded that the total asbestos burden in the lung tissue from these persons was quite low, although in 12 of the 13 cases that had positive lymph nodes, the tissue burden in the node was “appreciably heavier” per gram than in the lung. The authors concluded that lymph nodes provide useful “supplementary data” to data derived from lung analysis and “may in some individuals be a better indicator of levels of lifetime exposures than lung tissue.” The authors admitted, however, that more studies are necessary for clarification of relocation of fibers from the lung.

Dodson, and others, including Samuel P. Hammar, in “Asbestos in Extrapulmonary Sites: Omentum and Mesentery,” Chest, 117(2000):486-93, studied tissue from 20 persons with mesotheliomas, most with a history of asbestos exposure. They wanted to determine whether asbestos fibers would be found in the omentum and mesentery (parts of the peritoneum), and if they were, whether their presence would be predicted by various qualitative and quantitative features of asbestos bodies and asbestos fibers in lungs of the same persons. While 18 of the 20 had asbestos bodies in their lungs, asbestos bodies were found in the mesentery of only five patients, and in the omentum in two. In both the mesentery and omentum, the highest number of asbestos bodies were found in the person with the highest number of asbestos bodies in the lung tissues. All had amosite cores, with the exception of one tremolite asbestos core.

As to asbestos fibers, while 19 of the 20 had asbestos fibers in their lungs, 17 had fibers in at least one of the two extrapulmonary sites. Fourteen persons (70 percent) had asbestos fibers in both the mesentery and the omentum. Amosite was found most predominantly. The authors concluded that there was a correlation between the numbers of asbestos fibers found in the lung and in the mesentery and omentum. Asbestos fibers in the mesentery and omentum were predominantly longer amphiboles, particularly amosite. However, the authors did find chrysotile in the omentum and mesentery and observed that, “There was no apparent degradation of these fibers, and a portion of them were long fibers greater than five microns.” These results led them to conclude that the fact that long fibers of chrysotile reached the omentum in several cases,
indicates that chrysotile is also translocated from the lung to other sites and “could be potentially important in the pathogenesis of peritoneal mesothelioma.”


In the household contact mesothelioma cases, the mean exposure duration was 20 years, with the range being 1 to 45 years. The 57% of the household contact mesotheliomas also had pleural plaques while only 7.9% had asbestosis. Id. at 59. In the lung fiber burden analyses of household contact mesothelioma cases, "Household contacts of asbestos workers had tissue asbestos burdens that were similar to the median value for some occupational groups. For example, the median asbestos body count of household contacts (13 AB/G) was of the same order of magnitude as construction workers (190 AB/G). Hence, household contacts have tissue asbestos burdens that are on the average equivalent to mild to moderate occupational exposure. Wives tended to have higher lung asbestos burdens then daughters or sons." Id. at 61.

In 2003, Drs. Dodson and Hammar, et al, published a quantitative analysis of asbestos burden in 15 female mesothelioma cases. Five of the cases were household exposure mesotheliomas. No asbestosis was found in any of those cases. Four out of the 5 cases had elevated commercial amphibole fibers – principally amosite, although one of the cases had elevated Crocidolite fibers only. The remaining case had an estimated equal number of tremolite, anthophyllite and chrysotile fibers, although the total asbestos fiber concentration was well within the background range previously noted by Dodson. Dodson, O'Sullivan, Brooks and Hammar, "Qualitative Analysis of Asbestos Burden in Women with Mesothelioma," Am J Ind Med, 43:188-195(2003). The authors noted that "The three lowest concentrations of total uncoated asbestos fibers were found in individuals whose primary exposures were from household contacts (cases 5, 12, and 14) with 2 individuals (cases 5 and 12) not having ferruginous bodies detected in our analysis." Id. at 194.

Based on these most recent articles and developments from them, it is possible that in the future enough data will accumulate on the fiber counts in organs other than the lung in various levels of exposure to obtain information on peritoneal mesotheliomas in which there is no lung tissue to examine. At this point in time, however, these techniques are still in the experimental stage.

In the Third Edition of Dr. Roggli's textbook "Pathology of Asbestos-Associated Diseases" (Springer 2014), the analysis of tissue mineral fiber content chapter authored by Dr. Roggli and Dr. Sharma contains, as in the case of the earlier editions, a subsection specifically on household exposures (page 278). Table 11.15 reproduced below with permission provides the asbestos content of lung tissue by exposure category, and helps place the household contact fiber burden information in perspective:
These numbers stand in stark contrast to earlier fiber burden studies performed by Gibbs et al. of ten cases of malignant pleural mesothelioma among household contacts of asbestos workers where the total fiber count range from 5.3 to 320 million per gram of dry lung tissue with amosite and/or crocidolite found at elevated levels in 8 of the 10 cases (2 cases had fiber counts within the range of the reference population), or the case report by Huncharek et al., (1989) where a fiber burden count in a 75 year old wife of a shipyard machinist who dismantled boilers and other shipyard machinery for 34 years was found to have 6.5 million fibers per gram of dry lung as determined by TEM. In Dr. Roggli's fiber burden studies of household contacts with mesothelioma, amosite is the major fiber type identified in cases where asbestos levels are elevated. Roggli, et al. (2014):279. Interestingly, when Roggli compared his 13 household contacts of insulators with his series of 89 insulators, he found that the household contacts had about 4% of the asbestos body and total asbestos fiber content compared to the insulators. Id. at 279.

Most recently, Dr. Roggli's group published an update on an analysis of the 546 cases of mesothelioma where a fiber analyses were performed at Duke, dividing the cases into asbestos-related and non-asbestos-related based on whether the asbestos fiber counts were above background. Kraynie, et al., "Malignant mesothelioma not related to asbestos exposure: analytical scanning electron microscopic analysis of 83 cases and comparison with 442 asbestos-related cases," Ultrastructural Pathology (online 2016). Women were more often in the non-asbestos-related group (33% or 27 of 83 cases) then in the asbestos-related group (10% or 44 of 442 cases). Roggli, et al., also found that "the mean age of the asbestos-related group was 66 years (range 31-94 years), while the mean age of the non-asbestos-related group was 55 years (range 25-84 years). When comparing pleural versus peritoneal mesothelioma, "pleural mesotheliomas in women were asbestos-related in 44 of 71 cases (62%), but only 1 of 6 cases...
(16%) of peritoneal mesothelioma in women could be attributed to asbestos." (p. 3) What does this mean for your evaluation of household exposure cases"? The vast majority of such cases involve claims by women and the probability of it being asbestos-related is significantly lower than in male cases of pleural mesothelioma and the probability is extremely low for a female peritoneal mesothelioma to be asbestos-related.

Summary

Gibbs and Craighead sum up what information can be gleamed from fiber burden analysis when they stated "lung fiber burden analyses of cases with para-occupational exposure have been comparatively few but generally, they have shown commercial amphiboles in amounts comparable to workers with light to moderate direct occupational exposure (Dawson, et al, 1993; Gibbs, et al, 1990; Hunzharek, et al, 1989; Roggli, 2004, Chapter 4). Gibbs and Pooley, "Chapter 12 – Mineral Fiber Analysis and Asbestos-Related Diseases," Craighead and Gibbs, Asbestos and its Diseases 311 (Oxford University Press 2008).

It is almost always worth the effort to obtain lung fiber counts and probably fiber analysis in cases where exposure appears to be very low. Defendants having a chrysotile defense are advised to seriously consider utilizing lung fiber type analyses, when sufficient lung tissue is available, particularly when evidence of an alternative amphibole exposure is elusive.

IX. Legal

Household Exposure Cases

Duty of Premises Owners/Employers to Household Members of Employees

Courts are split on whether premises owners and employers owe a duty to plaintiffs who have never set foot on their premises, but allegedly were exposed to asbestos through household members who brought asbestos dust home on their clothing or belongings. The courts which reject such a duty generally focus on the absence of a special, or legal, relationship between employer or premises owner and the non-employee. These courts also look to the public policy issues that justify declining to extend the duty to warn to non-employees, such as the potential for limitless liability and an endless pool of plaintiffs. See, e.g., Gillen v. Boeing Co., 40 F.Supp.2d 534, 538-539 (E.D. Pa. 2014) (determining that the employer owed no duty to the wife of an employee for her take-home exposures to asbestos from laundering her husband’s clothes, even though the wife also worked for the employer, because the relationship between the wife and the employer “must be viewed in the context of the alleged tort”); Price v. E.I. DuPont de Nemours & Co., 26 A.3d 162, 169-70 (Del. 2011) (determining that the employer owed no duty to the non-employee without a “special relationship” between the employer and the employee’s spouse, which did not exist simply from her husband’s employment with DuPont, health insurance provided by DuPont, and the existence of a family friendly workplace); Boley, et al. v. Goodyear Tire & Rubber Co., et al., 929 N.E.2d 448, 450, 453 (Ohio 2010) (affirming the Eighth District Court of Appeals’ decision in Adams v. Goodyear Tire & Rubber Co. finding “a premises owner is not liable in tort for claims arising from asbestos exposure originating from asbestos on the owner’s property, unless the exposure occurred at the owner’s property”); Riedel v. ICI Americas Inc., 968 A.2d 17, 25-27 (Del. 2009) (affirming the trial court’s granting of
summary judgment to an employer in an employee’s wife’s negligence action as no legally significant relationship existed between wife and defendant; Van Fossen v. MidAmerican Energy Co., 777 N.W.2d 689, 699 (Iowa 2009) (affirming the granting of summary judgment concluding that the premises owner does not owe a duty to the wife of an independent contractor’s employee); CSX Trans., Inc., v. Williams, 278 Ga. 888, 892 (2005) (refusing to extend employer’s duty based only on mere foreseeability to encompass all who may come into contact with an employee or an employee’s clothing); In re New York City Asbestos Litig. (Holdamf, et al. v. A.C. & S., Inc. et al. and the Port Auth. of N. Y. and N. J., 840 N.E.2d 115, 122 (N.Y. 2005) (concluding there is no duty of care owed to an employee’s household member who has no relationship with the employer as it would lead to “limitless liability”); Campbell v. Ford Motor Co., 141 Cal. Rptr. 3d 390, 405 (Cal. Ct. App. 2012) (holding that “a property owner has no duty to protect family members of workers on its premises from secondary exposure to asbestos used during the course of the property owner’s business” as drawing a line between those non-employees to whom a duty is owed and those to whom no duty is owed is too difficult); Estate of Holmes v. Pneumo Abex, L.L.C., et al., 955 N.E.2d 1173, 1177 (Ill. App. Ct. 2011) (holding that no duty existed because there was no relationship between parties which by law would have imposed an obligation of reasonable conduct upon the defendant for the benefit of the plaintiff); In re Eighth Judicial Dist. Asbestos Litig. (Rinfleisch v. AlliedSignal, Inc.), 12 Misc. 3d 936, 815 N.Y.S 2d 815 (N.Y. Sup. Ct. 2006) (applying the “relationship” test to deny wife’s “take-home” claim as wives of employees had no relationship with their husband’s employer); Adams v. Owens-Illinois, Inc., 705 A.2d 58, 66 (Md. App. 1998) (holding that the defendant employer owed no duty to strangers simply from handling an employee’s clothing, as this could create liability to all who came in close contact with an employee, such as other family members, automobile passengers, and co-workers). But see Simpkins v. CSX Corp. and CSX Trans., Inc., 929 N.E.2d 1257, 1262-64 (Ill. Ct. App. 2010) (relying on the holdings in Satterfield v. Breeding Insulation Co., et al. and Olivo v. Owens-Illinois, Inc. to find that a relationship existed between a wife, allegedly exposed to asbestos through husband’s work clothes, and her husband’s employer).

An additional line of take-home cases involving premises owners and employers have examined foreseeability based on scientific knowledge about the potential harm to non-users at the time of the exposure. These courts have refused to extend a duty to warn to household members of employees based on the absence of evidence that the defendant was aware, or should have been aware, of the danger to household members at the time. Notably, the relevant dates of exposure in these cases all preceded 1972.

Most recently in Palmer v. 999 Quebec, Inc., the Supreme Court of North Dakota affirmed that defendant was not liable for injuries to a man who claimed he developed mesothelioma from asbestos brought home by his father who worked for defendant from 1961 through 1965. 874 N.W.2d 303 (ND 2016). The court held that the “evidence submitted by [plaintiff] fails to establish a special relationship between [defendant] and [plaintiff] or [defendant]’s knowledge of the dangers of asbestos while [plaintiff]’s father was employed by [defendant].” Id. at 310.

In Hoyt v. Lockheed Martin Corp., the Ninth Circuit affirmed that defendant was not liable for injuries to a woman who claimed she developed mesothelioma from asbestos brought home inadvertently by relatives from work, finding that the company could not have foreseen the
risk. 540 Fed.Appx. 590 (9th Cir. 2013). The court held that “no reasonable factfinder could conclude that harm from take-home exposure should have been foreseeable to Lockheed by 1958.” Id. at 592. In fact, plaintiff’s own expert testified that the first studies regarding take-home exposures were not published until the 1960s; therefore, Lockheed could not have known about the risks prior to that time. Id.

Additionally, in Martin v. Cincinnati Gas & Elec. Co., et al., 561 F.3d 439 (6th Cir. 2009), the Sixth Circuit Court of Appeals affirmed the United States District Court for the Eastern District of Kentucky’s decision that an employer/premises owner owed no duty to the son of an employee who brought asbestos home on his clothes during his employment from 1951 to 1963. Focusing on the foreseeability of harm at the time of injury, the court explained that the plaintiff must show that the employer knew or should have known of the danger of “take-home” exposure to asbestos during the time his father was employed there. Id. at 444-45. The court found that C.G. & E. did not owe a duty to plaintiff as there was no evidence that the employer had actual knowledge of the danger of “take-home” exposure. Further, C. G. & E. did not have constructive knowledge since the first studies of bystander exposure were not published until 1965. Id. at 446. See also Bootenhoff v. Hormel Foods Corp., 2014 WL 3744011, (W.D. Okla. July 30, 2014) (holding that “lack of foreseeability and additional policy considerations dictate that [defendant] did not owe a duty of care to” plaintiff who alleged take-home exposure to asbestos from her husband’s direct exposure to asbestos in 1959 through his work for defendant); In re Certified Question from Fourteenth Dist. Court of Appeals of Texas (Miller et al. v. Ford Motor Co.), 740 N.W.2d 206, 216 (Mich. 2007), reh’g denied, 739 N.W.2d 78 (Mich. 2007) (denying take-home exposure claim of stepdaughter of employee of independent contractor determining that “from 1954 to 1956, the period during which [stepfather] worked at defendant’s plant, we did not know what we know today about the hazards of asbestos”); Estate of Holmes, 955 N.E.2d at 1178-79 (finding no duty based on lack of foreseeability as the studies and evidence presented by plaintiff did not show a connection between asbestos fibers and “take-home” exposure until 1974 and plaintiff’s claims dated to 1962-1963; Rodarmel v. Pneumo Abex, L.L.C., 957 N.E.2d 107, 126 (Ill. App. Ct. 2011) (finding that the risk from asbestos carried home on work clothes was not foreseeable in the exposure years of 1953 to 1956, and therefore defendants cannot be held liable); Alcoa Inc. v. Behringer, 235 S.W.3d 456, 462 (Tex. Ct. App. 2007) (holding that foreseeability is the “foremost and dominant consideration” in a legal duty analysis, therefore Alcoa owed no duty to worker’s wife as “the danger of non-occupational exposure to asbestos dust on workers’ clothes was neither known nor foreseeable to Alcoa in the 1950s”).

The courts that have imposed such a duty on premises owners and employers also based their holdings on the foreseeability of the risk of harm to third parties. These courts found that there was sufficient evidence to support a finding that during the relevant time period the defendant knew or should have known of the potential for harm to household members of workers exposed to asbestos. However, these courts found a duty prior to 1972 based on foreseeability either by imputing knowledge upon companies without evidence of actual knowledge or by equating a company’s knowledge of general asbestos hazards with knowledge of hazards to household members. For exposures post-1972, the courts point to OSHA’s 1972 regulations.
In Condon v. Union Oil Co., 2004 WL 1932847 (Cal. Ct. App. August 31, 2004), review denied, (Nov. 17, 2004), the court upheld the lower court’s verdict in favor of the ex-wife of an employee of defendant who allegedly brought asbestos home on his work clothing, which the wife washed during the 1948-1963 time period. The court found that there was substantial evidence, including expert testimony, to support a finding that during the relevant time period, it was known that worker clothing could be a source of contamination to others; therefore, it was foreseeable that family members exposed to this clothing would also be in danger of exposure. Id. at *4-5. See also Bobo v. Tennessee Valley Authority, 2015 WL 5693609, *18 (N.D. Ala. Sept. 29, 2015) (holding employer liable for household asbestos exposures during decedent’s husband’s employment from 1975 to 1997 because of “the very nature of the relevant OSHA regulations and TVA’s internal standards”); Satterfield v. Breeding Insulation Co., et al., 266 S.W.3d 347, 367 (Tenn. 2008) (affirming appellate court and holding that a duty existed to daughter of worker allegedly exposed to father’s asbestos-containing clothing brought home from work since employer knew of danger during relevant years of 1970s through 1980s and failed to abide by OSHA’s 1972 regulations); Olivo v. Owens-Illinois, Inc., 895 A.2d 1143, 1149 (N.J. 2006) (holding employer liable for household asbestos exposures during decedent’s husband’s employment from 1947 to 1984 because Exxon knew exposure to asbestos created a threat of injury as early as 1937 and industrial hygiene texts as early as 1916 recommended workers change out of work clothing to avoid bringing contaminants home); Chaisson v. Avondale Indus., Inc., 947 So.2d 171, 183 (La. Ct. App. 2006) (holding that employer had duty to prevent foreseeable risks to household members created by worker’s clothing from 1976-1978 based on OSHA’s 1972 take home clothing regulations); Zimko v. Am. Cyanamid, 905 So.2d 465, 483 (La. Ct. App. 2005) (finding that a duty existed as employers have a general duty to act reasonably in view of foreseeable risks which includes risks to household members who predictably come into routine contact with employees’ clothing).

Notably, a court case applying Tennessee law expanded the take-home exposure duty beyond household members. See Millsaps v. Aluminum Co. of Am., et al., No. 10-358, (E.D. Pa. Oct. 10, 2012). U.S. Judge Eduardo C. Robreno of the Eastern District of Pennsylvania, overseeing the federal asbestos multidistrict litigation, held that the duty to prevent “foreseeable” take-home asbestos exposures is not limited only to household members. Defendant Alcoa moved for summary judgment arguing that Tennessee law did not impose a duty to warn the plaintiff because she did not live with her father-in-law who was alleged to have brought home asbestos on his clothing, relying on Satterfield v. Breeding Insulation Company, 266 SW 3d 347,366-67(Ten. 2008). Judge Robreno denied Alcoa’s motion, noting that "nothing in Satterfield requires that a person subjected to 'take-home' asbestos exposure be a resident of the same household as the defendant's employee in order for there to be a duty of care owed by the defendant to that person. Rather, Satterfield specifically holds that the class of 'foreseeable' people to whom a defendant such as Alcoa owes a duty include 'persons who regularly and for extended periods of time came into close contact with the asbestos-contaminated work clothes of Alcoa's employees', 266 SW 3d at 367." Id. at 7.
Duty of Manufacturer or Supplier of Asbestos-Containing Product

Courts have addressed the duty of a manufacturer of an asbestos-containing product to a household member of a person exposed to asbestos from the product much less frequently than the duty owed by premises owners and employers. Where the action is not based on the duty to provide a safe workplace or premises liability, but rather is against a manufacturer or supplier of the asbestos-containing product based on a duty to warn, the cases have turned almost exclusively on the foreseeability of harm to the person to whom the duty is alleged to owe. Thus, the focus of courts in determining a product manufacturer’s duty in product liability cases centers on the state-of-the-art issues discussed above in premises owner and employer household exposure cases.

The Court of Appeals of Maryland, in Georgia-Pacific, LLC v. Farrar, addressed this issue and held that in cases involving pre-1972 take-home asbestos exposure, product manufacturers had no duty to warn household members who had no relationship with the manufacturer, did not use the manufacturer’s product and were never physically present at the job site where the product was used. 69 A.3d 1028 (Md. 2013). Plaintiff contracted mesothelioma allegedly from exposure to asbestos fibers brought into the home in 1968-1969 on the clothes of her grandfather, who was exposed to asbestos during the course of his employment. Specifically, plaintiff alleges her grandfather worked in the immediate vicinity of workers installing Georgia-Pacific Ready-Mix joint compound. Id. at 1030. The court found that the record lacked evidence that it was foreseeable that asbestos dust from Georgia-Pacific joint compound posed a danger to a member of the household of a worker who was a bystander to work involving the product at issue. Id. at 1035. The court also offered its skepticism as to whether it was even feasible to warn non-users of dangers from its products. Id. at 1039.

See also Martin, 561 F.3d at 445 (finding that defendant G.E. did not owe a duty to plaintiff as (1) plaintiff could not present evidence that the risk was knowable in 1951 when G.E. supplied the asbestos products and (2) that the knowledge of the danger was available to defendant); Carel v. Fibreboard Corp., 74 F.3d 1248 (10th Cir. 1996) (citing Rohrbaugh v. Owens-Corning, 965 F.2d 844, 846 (10th Cir. 1992)) (holding that under Oklahoma law a product manufacturer does not have a duty to warn an injured party who “was never exposed to asbestos as a user or present where the product was used”); Rohrbaugh v. Celotex Corp., 53 F.3d 1181, 1183-84 (10th Cir.1995) (reaffirming its holding in Rohrbaugh I that the defendants did not have a duty to warn decedent because she was not a foreseeable user or purchaser of the product and because plaintiffs “had not produced any evidence that would show that [defendant] knew or should have known of the hazards associated with their product” prior to 1969, the date of decedent’s last exposure to appellants’ products).

X. Exposures From Home Remodeling and Brake Work

Some of the most common scenarios for low dose chrysotile exposures in the home include work as a “shade-tree” mechanic and tape joint compound work during home renovations. These exposures are far below that of their career-long counterparts, and often below cumulative background exposures. Furthermore, in most instances in which an individual has been exposed to asbestos in the home, there is typically an alternate exposure that far outweighs the low dose chrysotile exposure.
ALTERNATE EXPOSURES

It’s important to remember that in most instances in which an individual has been exposed to asbestos in the home, there is typically an alternate exposure that far outweighs the low dose chrysotile exposure at home. Select examples demonstrating this from the peer-reviewed literature follow for brake work and joint compound.

ALTERNATE EXPOSURE FOR BRAKE MECHANICS


In this extremely significant article by Dr. Victor Roggli and his colleagues, Roggli reported on his fiber burden analyses of 268 cases referred to his laboratory and determined the fiber content and type of fiber, concluding in part that:

commercial amphiboles are responsible for most of the mesothelioma cases observed in the United States. Id. at 55.

The cases were classified into 23 exposure categories, with the largest number of cases being shipbuilding, followed by U.S. Navy, construction industry and the insulation industry, each of which included 100 or more cases (there was substantial overlap).

While he found that 26% of the shipbuilding cases, 11% of the U.S. Navy, 17% of construction, and 58% of insulation had pathologic evidence of asbestosis, there was no evidence of asbestosis in any of the automotive mechanics examined.

Moreover, the lung burden analyses in automotive brake repair workers reflect either a normal range tissue asbestos content or elevated commercial amphiboles [which he attributed to unrelated commercial amphibole exposure]. Id. at 61.

Roggli comments specifically on the auto mechanic group:

Only one occupational exposure group contained on average more non-commercial amphibole fibers than commercial amphibole fibers. This was the category of automotive brake repair workers. In this group, fiber burden analyses performed on 11 cases showed either an elevated amosite content or an asbestos concentration indistinguishable from background. Workers in this category frequently were exposed to asbestos in other categories as well (TABLE 1) and none had asbestosis (TABLE 6). Among 15 workers exposed to brake dust who also had plaques, ten had additional exposures in jobs or industries where commercial amphiboles predominated, and three additional cases had elevated commercial amphiboles in their lung tissue samples. Lung tissue was not available for analysis in the other two. These findings are consistent with our prior observations of brake repair workers with mesothelioma, and with the nature of brake dust, which contains low levels of short chrysotile fibers. These observations in combination with a negative case control study indicate that brake dust is unlikely to cause mesothelioma. Id. at 64.
ALTERNATE EXPOSURE FOR JOINT COMPOUND


This is an article consisting of 3 case reports of individuals with no reported exposure to asbestos other than from joint compound. The authors implicitly acknowledge that work with joint compound has not been established as a cause of mesothelioma. “To the authors’ knowledge, this paper constitutes the first case reports of mesothelioma associated with exposure to asbestos-containing drywall finishing products. This report discusses joint compound as a potential source of asbestos exposure and cause of mesothelioma.” Id. at 338.

No fiber burdens were performed on the 3 individuals studied. Dahlgren admits he was unable to exclude the possibility of alternate exposures. “No other potential asbestos exposures were elucidated for the other cases; however, other exposures cannot be excluded in any of the three cases.” Id. Case 2 involved trades with elevated risk of developing mesothelioma. “Case 3’s exposure to asbestos began at birth and lasted most of his childhood years. His father frequently used joint compound from 1968 to 1974 while working in construction. The father recalls that sanding the joint compound created visible dust that settled on his clothes and on his person, which he then took home.” Id. Dahlgren admits Case 3’s take-home exposure from his father should be interpreted with caution due to numerous potential sources of asbestos at construction sites. Id. at 340.

Dr. Roggli and others examined 44 construction workers and determined that amosite was the main fiber type to which the workers were exposed. Dr. Roggli and his co-authors, as well as numerous other studies have examined household exposure cases and found that amosite was also the predominant fiber identified. Furthermore, women represent the overwhelming majority of those with asbestos-related diseases from take-home exposure, as wives, daughters and mothers historically have been the ones to handle the workers’ asbestos-laden clothing.


Dr. Roggli and others examined 405 patients with asbestos-related diseases and summarized their findings by occupational category compared with 19 patients with normal lungs at autopsy. Construction workers constituted one of the categories and encompassed a variety of occupations including drywall finisher, construction worker, painter, plasterer, and laborer. The authors studied 44 workers in this category with asbestos-related diseases and identified amosite as the main fiber type. Twenty-six of the construction workers had mesothelioma.


“We conducted a literature review in order to characterize reported cases of asbestos-related disease among household contacts of workers occupationally exposed to asbestos. Over
200 published articles were evaluated. Nearly 60 articles described cases of asbestos-related disease thought to be caused by para-occupational exposure. Over 65% of these cases were in persons who lived with workers classified as miners, shipyard workers, insulators, or others involved in the manufacturing of asbestos-containing products, with nearly all remaining workers identified as craftsmen. 98% of the available lung samples of the persons with diseases indicated the presence of amphibole asbestos.” Id. at 703.

“This review indicates that the literature is dominated by case reports, the majority of which involved household contacts of workers in industries characterized, generally, by high exposures to amphiboles or mixed mineral types. The available data do not implicate chrysotile as a significant cause of disease for household contacts.” Id.

“About 90% of these case reports included information about the occupation of the spouse or family member who was thought to be the source of the para-occupational exposure. Over 70% of the household cases were associated with workers classified as miners, manufacturers of asbestos or asbestos-containing products (typically involving raw asbestos), shipyard workers, or insulators. Among the remaining cases, common occupations of the primary worker included various types of crafts, such as steel mill workers, boilermakers, or construction workers; most of these exposures occurred between the 1930s and the 1960s. As such, it appears that these types of craftsmen were historically exposed to amphiboles. The remaining 10% of the case reports did not include specific information regarding occupations, but often made qualitative references to dusty conditions or higher exposures.” Id. at 725.
XI. Bibliography

Section II Bibliography: "Traditional" Bystander and Household Exposure: Wives and Children of Asbestos Workers in Homes of Asbestos Workers


Hillerdal, G., Mesothelioma: Cases Associated with Non-occupational and Low Dose Exposures, Occupational Environmental Medicine, 56(1999):505-513.


Langhoff, M., Kragh-Thomsen, M., Stanislaus, S., Weinreich, U., “Almost half of women with malignant mesothelioma were exposed to asbestos at home through their husbands or sons,” Dan Med 61(9):4902, 2014.


OSHA, National consensus standards and established federal standards, 29 CFR 1910.93. Air contaminants: US Department of Labor - Occupational Safety and Health Administration (OSHA) 1971b,


Reports to Congress on Workers' Home Contamination Study Conducted under the Workers' Family Protection Act (29 U.S.C. 671a), 1995.


Section III Bibliography: Exposures in Buildings


Section IV Bibliography: Neighborhood Exposures


Section V Bibliography: Ambient Air/Background Exposures


**Section VI Bibliography: Special Approaches in Female Cases**


**Section VII Bibliography: Genetic Predisposition**


Section VIII Bibliography: Fiber Counts and Types


Section IX Bibliography: SV-40


Section X Bibliography:


Kraynie, et al., "Malignant mesothelioma not related to asbestos exposure: analytical scanning electron microscopic analysis of 83 cases and comparison with 442 asbestos-related cases," Ultrastructural Pathology (online 2016).


Nicholson, et al., Investigation of Health Hazards in Brake Lining Repair and Maintenance Workers Occupationally Exposed to Asbestos, Environmental Sciences Laboratory, Mount Sinai School of Medicine, 1982.


XII. Appendix

(A) Table 1 from Donovan, et al., "Evaluation of take-home (para-occupational) exposure to asbestos and disease: a review of the literature" Critical Reviews in Toxicology 42:703-731(2012)
Table 1 from Donovan, et al., "Evaluation of take-home (para-occupational) exposure to asbestos and disease: a review of the literature" *Critical Reviews in Toxicology* 42:703-731(2012)

<table>
<thead>
<tr>
<th>Industry Group</th>
<th>Study</th>
<th>Location</th>
<th>No. of Cases</th>
<th>Diagnosis</th>
<th>Disease Confirmed</th>
<th>Family Member Occupation</th>
<th>Fiber Type</th>
<th>Time Period of Exposure or Diagnosis Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cement Factory</td>
<td>Magnani et al. 2000</td>
<td>Casale, Turin, Florence, Barcelona, Cadiz, Geneva (Italy)</td>
<td>4</td>
<td>Pleural mesothelioma</td>
<td>No</td>
<td>Asbestos industry workers</td>
<td>NR</td>
<td>Diagnosed in 1995</td>
</tr>
<tr>
<td></td>
<td>Magnani et al. 2001</td>
<td>Casale Monferrato, Italy</td>
<td>23</td>
<td>Pleural mesothelioma</td>
<td>No</td>
<td>Asbestos cement factory workers</td>
<td>Crocidolite and chrysotile</td>
<td>Diagnosed in 1995</td>
</tr>
<tr>
<td></td>
<td>Magnani et al. 1993; Ferrante et al. 2007</td>
<td>Casale Monferrato, Italy</td>
<td>12</td>
<td>Malignant neoplasm of the lung</td>
<td>No</td>
<td>Asbestos cement factory workers</td>
<td>Crocidolite and chrysotile</td>
<td>Exposed during 1907–1985 (plant operation)</td>
</tr>
<tr>
<td></td>
<td>Magnani et al. 1993; Ferrante et al. 2007</td>
<td>Casale Monferrato, Italy</td>
<td>3</td>
<td>Malignant neoplasm of the peritoneum</td>
<td>No</td>
<td>Asbestos cement factory workers</td>
<td>Crocidolite and chrysotile</td>
<td>Exposed during 1907–1985 (plant operation)</td>
</tr>
<tr>
<td></td>
<td>Magnani et al. 1993; Ferrante et al. 2007</td>
<td>Casale Monferrato, Italy</td>
<td>21</td>
<td>Malignant neoplasm of the pleura</td>
<td>No</td>
<td>Asbestos cement factory workers</td>
<td>Crocidolite and chrysotile</td>
<td>Exposed during 1907–1985 (plant operation)</td>
</tr>
<tr>
<td></td>
<td>Ampleford and Ohar 2007</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Cement manufacturing worker</td>
<td>Crocidolite</td>
<td>Exposed 1939–1967</td>
</tr>
<tr>
<td>Other Factory</td>
<td>Newhouse and Thompson 1965</td>
<td>London</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Asbestos factory foreman</td>
<td>NR</td>
<td>Exposed 1921–1942</td>
</tr>
<tr>
<td></td>
<td>Newhouse and Thompson 1965</td>
<td>London</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos factory foreman</td>
<td>NR</td>
<td>Exposed 1928–1930</td>
</tr>
<tr>
<td></td>
<td>Newhouse and Thompson 1965</td>
<td>London</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos factory spinner</td>
<td>NR</td>
<td>Exposed 1925–1936</td>
</tr>
<tr>
<td></td>
<td>Newhouse and Thompson 1965</td>
<td>London</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos factory worker</td>
<td>NR</td>
<td>Exposed 1919–1921</td>
</tr>
<tr>
<td></td>
<td>Newhouse and Thompson 1965</td>
<td>London</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos factory worker</td>
<td>NR</td>
<td>Exposed 1941–1946</td>
</tr>
<tr>
<td></td>
<td>Rusby 1968</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos factory worker</td>
<td>NR</td>
<td>Exposed 1930</td>
</tr>
<tr>
<td></td>
<td>Milne 1969</td>
<td>Victoria, Australia</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos factory worker</td>
<td>NR</td>
<td>Exposed in 1930</td>
</tr>
<tr>
<td></td>
<td>Von Bittersohl and Ose 1971</td>
<td>Kreis Merseburg, Germany</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Watchman at an asbestos factory</td>
<td>&quot;Serpentine&quot; and &quot;fibrous&quot; asbestos</td>
<td>Exposed approximately 1920–1930</td>
</tr>
<tr>
<td></td>
<td>Navratil and Tripp 1972</td>
<td>Czechoslovakia</td>
<td>4</td>
<td>Pleural calcifications</td>
<td>X-ray</td>
<td>Asbestos plant workers</td>
<td>Chrysotile</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Knappman 1972</td>
<td>Hamburg, Germany</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos factory worker</td>
<td>NR</td>
<td>Exposed approximately 1920–1930</td>
</tr>
</tbody>
</table>

(Continued)
Table 1 from Donovan, et al., "Evaluation of take-home (para-occupational) exposure to asbestos and disease: a review of the literature" Critical Reviews in Toxicology 42:703-731 (2012)

Table 1. (Continued).

<table>
<thead>
<tr>
<th>Industry Group</th>
<th>Study</th>
<th>Location</th>
<th>No. of Cases</th>
<th>Diagnosis</th>
<th>Disease Confirmed?</th>
<th>Family Member Occupation</th>
<th>Fiber Type</th>
<th>Time Period of Exposure or Diagnosis Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greenberg and Davies 1974</td>
<td>England, Scotland, Wales</td>
<td>1</td>
<td>Mesothelioma</td>
<td>No</td>
<td>Asbestos factory worker</td>
<td>NR</td>
<td>Exposed for two unspecified years</td>
<td></td>
</tr>
<tr>
<td>Whitwell et al. 1977</td>
<td>England</td>
<td>1</td>
<td>Mesothelioma</td>
<td>Yes</td>
<td>Gas-mask factory worker</td>
<td>Crocidolite</td>
<td>Exposed for 5 years</td>
<td></td>
</tr>
<tr>
<td>Vienna and Polan 1978</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos factory worker</td>
<td>NR</td>
<td>Exposed for 15 years</td>
<td></td>
</tr>
<tr>
<td>Vienna and Polan 1978</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Brake-lining worker</td>
<td>NR</td>
<td>Exposed for 10 years</td>
<td></td>
</tr>
<tr>
<td>Vienna and Polan 1978</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Brake-lining worker</td>
<td>NR</td>
<td>Exposed for 10 years</td>
<td></td>
</tr>
<tr>
<td>Epler 1980</td>
<td>Boston, MA</td>
<td>1</td>
<td>Pleural plaques, calcification, benign asbestos pleural effusion and fibrosis</td>
<td>X-ray</td>
<td>Asbestos factory worker</td>
<td>NR</td>
<td>Exposed 1939-1967</td>
<td></td>
</tr>
<tr>
<td>Epler 1980</td>
<td>Boston, MA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos factory worker</td>
<td>NR</td>
<td>Diagnosed in 1973</td>
<td></td>
</tr>
<tr>
<td>Anderson et al. 1976, 1979, 1982</td>
<td>New Jersey, USA</td>
<td>239</td>
<td>Small opacities and/or pleural abnormalities</td>
<td>X-ray</td>
<td>Asbestos factory worker</td>
<td>Amosite</td>
<td>Exposed 1941-1954</td>
<td></td>
</tr>
<tr>
<td>Kane et al. 1990</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos and glass factory worker</td>
<td>NR</td>
<td>Diagnosed between 1974 and 1986</td>
<td></td>
</tr>
<tr>
<td>Kane et al. 1990</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos plant worker</td>
<td>NR</td>
<td>Diagnosed between 1974 and 1986</td>
<td></td>
</tr>
<tr>
<td>Joubert et al. 1991</td>
<td>New Jersey, USA</td>
<td>4</td>
<td>Pleural mesothelioma</td>
<td>No</td>
<td>Asbestos factory worker</td>
<td>Amosite</td>
<td>Exposed 1941-1954</td>
<td></td>
</tr>
<tr>
<td>Schneider et al. 1986</td>
<td>Germany</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos cardboard worker</td>
<td>NR</td>
<td>Exposed 1969-1976</td>
<td></td>
</tr>
<tr>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos plant manufacturer</td>
<td>NR</td>
<td>Exposed 1935-1965</td>
<td></td>
</tr>
<tr>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Papermaker</td>
<td>NR</td>
<td>Exposed 1930-1949</td>
<td></td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos product manufacturer</td>
<td>NR</td>
<td>Exposed 1963-1965</td>
<td></td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Manufactured asbestos product</td>
<td>NR</td>
<td>Exposed 1963-1965</td>
<td></td>
</tr>
</tbody>
</table>

(Continued)
Table 1. (Continued):

<table>
<thead>
<tr>
<th>Industry Group</th>
<th>Study</th>
<th>Location</th>
<th>No. of Cases</th>
<th>Diagnosis</th>
<th>Disease Confirmed?</th>
<th>Family Member Occupation</th>
<th>Fiber Type</th>
<th>Time Period of Exposure or Diagnosis Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dock or Shipyard</td>
<td>Newhouse and Thompson 1965</td>
<td>London</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Dock laborer</td>
<td>NR</td>
<td>Exposed 1930-1934</td>
</tr>
<tr>
<td></td>
<td>Newhouse and Thompson 1965</td>
<td>London</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard engineer</td>
<td>NR</td>
<td>Exposed 1919-1960</td>
</tr>
<tr>
<td></td>
<td>Lieben and Pistawka 1967</td>
<td>Pennsylvania, USA</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Shipyard insulators</td>
<td>NR</td>
<td>Diagnosed between 1958 and 1963</td>
</tr>
<tr>
<td></td>
<td>McEwen et al. 1971</td>
<td>Scotland</td>
<td>1</td>
<td>Mesothelioma</td>
<td>Yes</td>
<td>Dock laborer</td>
<td>NR</td>
<td>Diagnosed between 1950 and 1967</td>
</tr>
<tr>
<td></td>
<td>Li et al. 1978</td>
<td>NR</td>
<td>2</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard insulator</td>
<td>NR</td>
<td>Exposed 1960-1965</td>
</tr>
<tr>
<td></td>
<td>Edge and Choudhury 1978</td>
<td>Barrow-in-Furness, UK</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard plumber</td>
<td>Crocidolite</td>
<td>Diagnosed between 1966-1976</td>
</tr>
<tr>
<td></td>
<td>Bianchi et al. 1981</td>
<td>Monfalcone, Italy</td>
<td>13</td>
<td>Pleural plaques and/or asbestos bodies</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Necropsy performed 1979-1980</td>
</tr>
<tr>
<td></td>
<td>Hammar 1989</td>
<td>Vancouver, WA</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed in early 1940s</td>
</tr>
<tr>
<td></td>
<td>Huncharek et al. 1989</td>
<td>Canada</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard machinist</td>
<td>NR</td>
<td>Exposed 1935-1968</td>
</tr>
<tr>
<td></td>
<td>Kane et al. 1990</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard insulator</td>
<td>NR</td>
<td>Diagnosed between 1974 and 1986</td>
</tr>
<tr>
<td></td>
<td>Roggli and Longo 1991</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard Insulator</td>
<td>Amphibole observed in lungs</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td>Roggli and Longo 1991</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>Amphibole detected in lungs</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td>Dodoli et al. 1992</td>
<td>Leghorn and La Spezia, Italy</td>
<td>9</td>
<td>Pleural mesothelioma</td>
<td>No</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Died between 1975-1988</td>
</tr>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed 1941-1947</td>
</tr>
</tbody>
</table>

(Continued)
Table 1 (Continued).

<table>
<thead>
<tr>
<th>Industry Group</th>
<th>Study</th>
<th>Location</th>
<th>No. of Cases</th>
<th>Diagnosis</th>
<th>Disease Confirmed?</th>
<th>Family Member Occupation</th>
<th>Fiber Type</th>
<th>Time Period of Exposure or Diagnosis Dates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed 1942-1950</td>
</tr>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed 1942-1982</td>
</tr>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed 1943-1944</td>
</tr>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed 1951-1957</td>
</tr>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed 1941-1946</td>
</tr>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed 1955-1975</td>
</tr>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed 1942-1945</td>
</tr>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed 1942-1945</td>
</tr>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed 1941-1944</td>
</tr>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Exposed 1941-1965</td>
</tr>
<tr>
<td></td>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker/boilermaker</td>
<td>NR</td>
<td>Exposed 1942-1963</td>
</tr>
<tr>
<td></td>
<td>Peretz et al. 2009</td>
<td>Washington, USA</td>
<td>2</td>
<td>Calcified pleural plaques</td>
<td>X-ray</td>
<td>Shipyard carpenter/pipe fitter</td>
<td>NR</td>
<td>Exposed 1942-1963</td>
</tr>
<tr>
<td></td>
<td>Bianchi et al. 2009</td>
<td>Trieste-Monfalcone, Italy</td>
<td>34</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Shipyard worker</td>
<td>NR</td>
<td>Diagnosed between 1968 and 2008</td>
</tr>
<tr>
<td></td>
<td>Lieben and Pistoia 1967</td>
<td>Pennsylvania, USA</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Insulation plant engineer</td>
<td>Asbestos and chrysotile</td>
<td>Diagnosed between 1968 and 1983</td>
</tr>
<tr>
<td></td>
<td>Lieben and Pistoia 1967</td>
<td>Pennsylvania, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Insulation plant worker</td>
<td>NR</td>
<td>Diagnosed between 1968 and 1983</td>
</tr>
<tr>
<td></td>
<td>Champion 1971</td>
<td>Glasgow, Scotland</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Insulator</td>
<td>NR</td>
<td>Diagnosed between 1968 and 1983</td>
</tr>
<tr>
<td></td>
<td>Vienna and Polan 1978</td>
<td>New York, USA</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Heat insulation worker</td>
<td>NR</td>
<td>Exposed for 15 years</td>
</tr>
<tr>
<td></td>
<td>Vienna and Polan 1978</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Heat insulation worker</td>
<td>NR</td>
<td>Exposed for 18 years</td>
</tr>
</tbody>
</table>

(Continued)
Table 1 from Donovan, et al., "Evaluation of take-home (para-occupational) exposure to asbestos and disease: a review of the literature" Critical Reviews in Toxicology 42:703-731(2012)

<table>
<thead>
<tr>
<th>Industry Group</th>
<th>Study</th>
<th>Location</th>
<th>No. of Cases</th>
<th>Diagnosis</th>
<th>Disease Confirmed</th>
<th>Family Member Occupation</th>
<th>Fiber Type</th>
<th>Time Period of Exposure or Diagnosis Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viana and Polan 1978</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Heat insulation worker</td>
<td>NR</td>
<td>Exposed for 5 years</td>
<td></td>
</tr>
<tr>
<td>McDonald and McDonald 1978; 1979; 1980</td>
<td>USA, Canada</td>
<td>5</td>
<td>Malignant mesothelioma</td>
<td>Yes</td>
<td>Asbestos/insulation factory worker</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>Martensson et al. 1984</td>
<td>NR</td>
<td>2</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Foundry worker</td>
<td>NR</td>
<td>Exposed in childhood</td>
<td></td>
</tr>
<tr>
<td>Sider et al. 1987</td>
<td>Chicago, IL</td>
<td>18</td>
<td>Pleural thickening and calcification</td>
<td>X-ray</td>
<td>Insulator</td>
<td>NR</td>
<td>Exposed 1938-1963</td>
<td></td>
</tr>
<tr>
<td>Roggli and Longo. 1991</td>
<td>NR</td>
<td>1</td>
<td>Small/large cell carcinoma</td>
<td>Yes</td>
<td>Insulator</td>
<td>Amphibole detected in lungs</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>Roggli and Longo. 1991</td>
<td>NR</td>
<td>1</td>
<td>Small cell carcinoma</td>
<td>Yes</td>
<td>Insulator</td>
<td>Amphibole detected in lungs</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>Roggli and Longo. 1991</td>
<td>NR</td>
<td>1</td>
<td>Bronchiolovascular cell carcinoma</td>
<td>Yes</td>
<td>Insulator</td>
<td>Amphibole detected in lungs</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>Schneider et al. 1996</td>
<td>Germany</td>
<td>2</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Insulation manufacturer</td>
<td>NR</td>
<td>Exposed 1950-1959</td>
<td></td>
</tr>
<tr>
<td>Schneider et al. 1996</td>
<td>Germany</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Insulator</td>
<td>NR</td>
<td>Exposed 1964-1974</td>
<td></td>
</tr>
<tr>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>10</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Insulator</td>
<td>Commercial and non-commercial amphibole</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>Magnani et al. 2000</td>
<td>Casale, Turin, Florence, Barcelona, Cadiz, Geneva (Italy)</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>No</td>
<td>Asbestos industry working with insulation in wagons</td>
<td>NR</td>
<td>Diagnosed in 1955</td>
<td></td>
</tr>
<tr>
<td>Miller 2003</td>
<td>NR</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Insulator</td>
<td>NR</td>
<td>Exposed 1954-1961</td>
<td></td>
</tr>
<tr>
<td>Miller 2003</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Insulator</td>
<td>NR</td>
<td>Exposed 1954-1961</td>
<td></td>
</tr>
<tr>
<td>Miller 2003</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Insulator</td>
<td>NR</td>
<td>Exposed 1947-1987</td>
<td></td>
</tr>
<tr>
<td>Miller 2003</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Insulator</td>
<td>NR</td>
<td>Diagnosed in 1992</td>
<td></td>
</tr>
<tr>
<td>Miller 2003</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Insulator</td>
<td>NR</td>
<td>Exposed 1953-1976</td>
<td></td>
</tr>
<tr>
<td>Miller 2003</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Spray asbestos insulation</td>
<td>NR</td>
<td>Exposed 1954-1977</td>
<td></td>
</tr>
<tr>
<td>Wagner et al. 1992</td>
<td>Cape Province, South Africa</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos miner</td>
<td>Crocidolite</td>
<td>Diagnosed in 1958</td>
<td></td>
</tr>
<tr>
<td>Rubino et al. 1972</td>
<td>Northwestern Italy</td>
<td>3</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos mining industry workers</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
</tbody>
</table>

(Continued)
Table 1 from Donovan, et al., "Evaluation of take-home (para-occupational) exposure to asbestos and disease: a review of the literature" Critical Reviews in Toxicology 42:703-731 (2012)

<table>
<thead>
<tr>
<th>Industry Group</th>
<th>Study</th>
<th>Location</th>
<th>No. of Cases</th>
<th>Diagnosis</th>
<th>Disease Confirmed?</th>
<th>Family Member Occupation</th>
<th>Fiber Type</th>
<th>Time Period of Exposure or Diagnosis Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trades - Various</td>
<td>Newhouse and Thompson 1965</td>
<td>London</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Railway carriage builder</td>
<td>NR</td>
<td>Exposed 1912-1930</td>
</tr>
<tr>
<td></td>
<td>Newhouse and Thompson 1965</td>
<td>London</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Boiler coverer</td>
<td>NR</td>
<td>Exposed 1925-1939</td>
</tr>
<tr>
<td></td>
<td>Heller et al. 1970</td>
<td>Massachusetts, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Pipefitter</td>
<td>NR</td>
<td>Diagnosed between 1960 and 1967</td>
</tr>
<tr>
<td></td>
<td>Vianna and Polan 1978</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Electric wire insulation worker</td>
<td>NR</td>
<td>Exposed for 15 years</td>
</tr>
<tr>
<td></td>
<td>Vianna and Polan 1978</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Heat-electric wire worker</td>
<td>NR</td>
<td>Exposed for 14 years</td>
</tr>
<tr>
<td></td>
<td>Vianna and Polan 1978</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Pipefitter</td>
<td>NR</td>
<td>Exposed for 6 years</td>
</tr>
<tr>
<td></td>
<td>Kane et al. 1990</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Construction laborer</td>
<td>NR</td>
<td>Diagnosed between 1974 and 1986</td>
</tr>
<tr>
<td></td>
<td>Chellini et al. 1992</td>
<td>Tuscany, Italy</td>
<td>3</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Construction worker</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Chellini et al. 1992</td>
<td>Tuscany, Italy</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Plumber in chemical manufacturing</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td>Dodoli et al. 1992</td>
<td>Leghorn and La Spezia, Italy</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Oil refinery worker</td>
<td>NR</td>
<td>Died between 1975-1988</td>
</tr>
<tr>
<td></td>
<td>Ascoli et al. 1996</td>
<td>Rome</td>
<td>1</td>
<td>Malignant mesothelioma</td>
<td>Yes</td>
<td>Smelting furnace worker</td>
<td>NR</td>
<td>Diagnosed between 1980 and 1995</td>
</tr>
<tr>
<td></td>
<td>Schneider et al. 1996</td>
<td>Germany</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos cement roofer</td>
<td>NR</td>
<td>Exposed 1954-1971</td>
</tr>
<tr>
<td></td>
<td>Schneider et al. 1996</td>
<td>Germany</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Turbine revision worker</td>
<td>NR</td>
<td>Exposed 1961-1984</td>
</tr>
<tr>
<td></td>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>3</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Pipefitter/welder</td>
<td>Commercial and non-commercial amphiboles</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Oil refinery worker</td>
<td>NR</td>
<td></td>
</tr>
</tbody>
</table>

(Continued)
Table 1 from Donovan, et al., "Evaluation of take-home (para-occupational) exposure to asbestos and disease: a review of the literature" Critical Reviews in Toxicology 42:703-731(2012)

<table>
<thead>
<tr>
<th>Industry Group</th>
<th>Study</th>
<th>Location</th>
<th>No. of Cases</th>
<th>Diagnosis</th>
<th>Disease Confirmed?</th>
<th>Family Member Occupation</th>
<th>Fiber Type</th>
<th>Time Period of Exposure or Diagnosis Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Construction worker</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Machinist</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Auto mechanic</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Tire presser</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Steamfitter</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Construction worker</td>
<td>NR</td>
<td>Diagnosed in 1994</td>
<td></td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Steel mill worker</td>
<td>NR</td>
<td>Exposed 1947-1972</td>
<td></td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Railroad brake worker</td>
<td>NR</td>
<td>Diagnosed in 1996</td>
<td></td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Boilermaker</td>
<td>NR</td>
<td>Exposed 1945-1969</td>
<td></td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Boilermaker</td>
<td>NR</td>
<td>Exposed 1940-1950</td>
<td></td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Refractory bricklayer</td>
<td>NR</td>
<td>Exposed 1948-1990</td>
<td></td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Steel mill worker</td>
<td>NR</td>
<td>Exposed 1950-1980</td>
<td></td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Pipefitter</td>
<td>NR</td>
<td>Exposed 1942-1975</td>
<td></td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Railroad blacksmith/ pipefitter</td>
<td>NR</td>
<td>Exposed 1935-NR (RR); 1954-1992 (PF)</td>
<td></td>
</tr>
<tr>
<td>Miller 2005</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Refinery worker</td>
<td>NR</td>
<td>Exposed 1941-1955</td>
<td></td>
</tr>
<tr>
<td>Ampleford and Ohar 2007</td>
<td>NR</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Electrician, insulator, mechanic, machinist, miller, pipe fitter, plumber, steel worker, and welder at an aluminum can company; later a furnace operator</td>
<td>NR</td>
<td>Exposed 1980-2002</td>
<td></td>
</tr>
<tr>
<td>Patel et al. 2008</td>
<td>NR</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Construction worker/insulator</td>
<td>Crocidolite</td>
<td>NR</td>
<td></td>
</tr>
</tbody>
</table>

(Continued)
Table 1 from Donovan, et al., "Evaluation of take-home (para-occupational) exposure to asbestos and disease: a review of the literature" Critical Reviews in Toxicology 42:703-731(2012)

<table>
<thead>
<tr>
<th>Industry Group</th>
<th>Study</th>
<th>Location</th>
<th>No. of Cases</th>
<th>Diagnosis</th>
<th>Disease Confirmed?</th>
<th>Family Member Occupation</th>
<th>Fiber Type</th>
<th>Time Period of Exposure or Diagnosis Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patel et al. 2008</td>
<td>NR</td>
<td>Russia and Finland</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Pipefitter</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Kiviluoto 1965</td>
<td>Russia and Finland</td>
<td>4</td>
<td>Slight fibrosis</td>
<td>X-ray</td>
<td>Unknown</td>
<td>Mixed dusts</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Kiviluoto 1965</td>
<td>Tyneside, UK</td>
<td>1</td>
<td>Pulmonary fibrosis</td>
<td>X-ray</td>
<td>Unknown</td>
<td>Mixed dusts</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Greenberg and Davies 1974</td>
<td>England, Scotland, Wales</td>
<td>1</td>
<td>Mesothelioma</td>
<td>Yes</td>
<td>Unknown</td>
<td>NR</td>
<td>Exposed for 3 unspecified years</td>
<td></td>
</tr>
<tr>
<td>Lillington et al. 1974</td>
<td>NR</td>
<td>England, Scotland, Wales</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Unknown</td>
<td>NR</td>
<td>Exposed 1941-1949</td>
</tr>
<tr>
<td>Gibbs et al. 1989; 1990</td>
<td>United Kingdom</td>
<td>13</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>NR</td>
<td>Suggestive of asbestosis</td>
<td>Diagnosed between 1979 and 1986</td>
<td></td>
</tr>
<tr>
<td>Kane et al. 1990</td>
<td>New York, USA</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Unknown</td>
<td>NR</td>
<td>Diagnosed between 1974 and 1986</td>
<td></td>
</tr>
<tr>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>England, Scotland, Wales</td>
<td>1</td>
<td>Peritoneal mesothelioma</td>
<td>Yes</td>
<td>Unknown</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Roggli et al. 1997</td>
<td>NR</td>
<td>England, Scotland, Wales</td>
<td>2</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Unknown</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Techet et al. 1976</td>
<td>England, Scotland, Wales</td>
<td>24</td>
<td>Mesothelioma</td>
<td>Unknown</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Magnani et al. 2000</td>
<td>Casale, Turin, Florence, Barcelone, Cadiz, Geneva (Italy)</td>
<td>1</td>
<td>Pleural mesothelioma</td>
<td>No</td>
<td>Asbestos industry (possible foundry)</td>
<td>NR</td>
<td>Diagnosed 1997</td>
<td></td>
</tr>
<tr>
<td>Leigh et al. 2002</td>
<td>Australia</td>
<td>17</td>
<td>Mesothelioma</td>
<td>Yes</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Ascoli et al. 2003</td>
<td>Latium, Italy</td>
<td>2</td>
<td>Pleural mesothelioma</td>
<td>Yes</td>
<td>Asbestos and/or products worker</td>
<td>NR</td>
<td>Diagnosed between 1945 and 2000</td>
<td></td>
</tr>
<tr>
<td>Rake et al. 2009</td>
<td>England, Wales and Scotland</td>
<td>24</td>
<td>Mesothelioma</td>
<td>Unknown</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
</tr>
</tbody>
</table>

NR, Not reported.

*Anderson et al. studies (1976, 1979, 1982) were based on X-ray abnormalities found in household contacts of factory workers. Johebert et al. (1991) reported mortality of this cohort: 4 mesotheliomas and 12 lung cancer deaths were identified. *McDonald et al. 1970 and 1973 presented preliminary results from a survey of Canadian pathologists of histologically confirmed mesotheliomas diagnosed between 1960 and 1968. The McDonald and McDonald 1980 study presented the ascertainment of 688 cases from 7400 pathologists in both the US and Canada.*
Household Exposure/Mesothelioma Literature Excerpts

**HOUSEHOLD EXPOSURE/MESOTHELIOMA LITERATURE EXCERPTS**

The literature dealing with household exposure to asbestos length was focused on persons who lived with asbestos workers who worked with or around industries characterized by high exposures to amphibole or mixed exposure to asbestos. To date, the available literature does not implicate chrysotile as a significant contributor to development of mesothelioma in household contacts. Moreover, of the lung samples available for review, the majority of cases indicated the presence of amphibole asbestos.

<table>
<thead>
<tr>
<th>NO.</th>
<th>CITE</th>
<th>QUOTE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lieben, J, and Pistawka, H, &quot;Mesothelioma and Asbestos Exposure,&quot; Arch Environ Health.</td>
<td>&quot;The group of nine, seven women and two men, whose relatives worked with asbestos, are of particular interest. The most unusual history was that of the wife who washed her husband’s dungarees or work clothes. In one instance, a relative said that the husband, a docker, came home “white with asbestos” every evening for three to four years and his wife brushed him down. The two men in this group, when boys of eight or nine years old, had sisters who were working at an asbestos factory. One of these girls worked as a spinner from 1925 to 1936. In 1946, she died of asbestosis. The press report of the inquest states: “She used to return home from work with dust on her clothes.” Her brother had apparently no other exposure to asbestos; he started work as a shop assistant, then became a sawer of iron girders until 1948 when he worked as a loader of groceries in the docks for five years (but never on dusty cargos) and then returned to sawing iron girders. He died in 1956 of a pleural mesothelioma.&quot; (at p. 264)</td>
</tr>
<tr>
<td>B.</td>
<td>&quot;Of the 42 patients, 10 had definite occupational exposure to asbestos during lifetime, three were family contacts of asbestos workers, eight either lived in the immediate neighborhood of asbestos plants or had been employed next to an asbestos plant. Ten patients had a questionable</td>
<td></td>
</tr>
</tbody>
</table>
2. "Mesothelioma Patients With Family Contacts Exposed to Asbestos. Patient 1-F, a 3-year-old child, was the daughter of a ceramic engineer who worked in an insulation plant that utilizes 400 tons of Canadian chrysotile and 1,500 tons of South African amosite annually.

Patient 2-F, a 40-year-old nurse, never had any occupational asbestos exposure, but her father had worked for 35 years in the insulation plant where patients 3-O, 4-O, and 5-O had worked and which was also credited with three neighborhood cases. The nurse's brother also had worked in this plant for one year.

Patient 3-F, a 67-year-old woman, never had any asbestos exposure nor had she lived near an asbestos plant. She had two sons who worked as insulators in a shipyard for six years. These sons lived at home until 15 years prior to their mother's death." (at p. 561)

3. "Patients 1-F and 3-F had peritoneal and patient 2-F pleural mesotheliomas." (at p. 561)

C. Newhouse, M.I., “The Medical Risks of Exposure to Asbestos,” Practitioner, 285 (1967). "Whilst these [household] exposures are less common than occupational exposures, careful investigation of the history of those suffering from asbestosis has revealed that more casual contacts may lead to heavy enough exposure to be of importance.

Elmes (1966) quotes the case of a woman who assisted her husband in building two bungalows: she held the asbestos sheeting while he, a seaman, sawed them up. She had clinical asbestosis and subsequent investigation of the husband revealed characteristic x-ray changes of this condition.

Domestic exposure through dust brought home on the clothes or overalls of asbestos workers, and exposure through living in the vicinity of a mine or asbestos factory, have also been found to be of importance by workers who have investigated the histories of those dying of mesothelial tumors. In these cases, exposure, which may have been
heavy, usually occurred more than 30 years ago. (Wagner et al., 1960; Newhouse and Thompson, 1965; Lieben and Pistawah, 1967).”  

D.  

"A study of the occupational histories of 52 females with malignant mesothelioma and certain of their relatives, carried out to measure the risk of this disorder attributable to indirect asbestos exposure, showed that a significantly greater number of husbands and fathers of cases than of controls worked in asbestos-related industries, and the relative risk for this factor was 10."  (at p. 1061)

4.  
"Sixty-five females were reported to have died from malignant mesotheliomas during the 11-year study period, but only 52 (32 pleural and 20 peritoneal) were histologically confirmed."  (at p. 1061)

5.  
"Ten patients (B, C, and G-N) had husbands and/or fathers who worked in asbestos-related occupations, whereas their matched controls did not. All these patients routinely hand-laundered their husbands’ (or fathers’) clothings. Patients G through N were either housewives or were employed as secretaries and clerks in non-asbestos related industries. In only one instance did the case’s husband and father not have positive occupational histories, while their matched control’s husband worked in the heating insulation industry for 20 years of their married life. The estimated relative risk of mesothelioma for this pattern of exposure to asbestos is 10 with a 95% confidence interval of 1.42 to 37.40."  (at p. 1062)

---

1 Wagner et al. (1960) discuss household exposure from crocidolite miners in Australia. Newhouse and Thompson (1965) discuss household exposures of a group of nine individuals whose relatives worked with asbestos. The authors described heavy exposures to asbestos from laundering clothes. Lieben and Pistawah (1967) describe mesothelioma patients with family contacts to asbestos. All of the patients had family members who worked with insulations products and were heavily exposure to asbestos.

2 In this series, there were 8 pleural mesotheliomas and one peritoneal mesothelioma. Patient K, who was diagnosed with peritoneal mesothelioma, had a husband who worked as a heat insulation worker and a father who worked as an elevator insulation worker.
<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>6.</td>
<td>&quot;The husband’s occupation was clearly the most important risk factor, but six of our patients also lived in environments where more than one source of exposure was likely. The importance of other non-occupational exposure varies with the type of industry (e.g., asbestos mines and factories, shipyards, etc.) present in an area and their proximity to homes.&quot; (at p. 1063)</td>
<td></td>
</tr>
<tr>
<td>E.</td>
<td>McDonald, A, and McDonald, J, &quot;Malignant Mesothelioma in North America,&quot; American Cancer Society, 46(4):1650-1656, (1980).</td>
<td>&quot;Of 557 cases reported to the end of 1972, 395 were male and 162 were female. In males, 307 were pleural and 88 were peritoneal including 14 also affecting the pleura. In females, 99 were pleural and 63 were peritoneal.&quot; (at p. 1651)</td>
</tr>
<tr>
<td>7.</td>
<td>&quot;Two male and six female subjects but no matched control had been exposed at home to the dusty clothing of an asbestos worker; two controls were so exposed and the paired cases were not. Of the eight subjects, five had been exposed in childhood and the remaining three plus two controls as adults. In three cases, and for one control, the clothing was that of a Quebec chrysotile production worker and, in five cases and for one control, an employee engaged in insulation or factory work. In the USA survey, an additional question was asked about exposure to asbestos in work or hobbies in the home.&quot; (at p. 1653)</td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>&quot;The association of mesothelioma with exposure to asbestos contaminated clothing in the home was again confirmed.&quot; (at p. 1655)</td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>&quot;Fifty-three patients (77%) had a history of asbestos exposure including 47 patients with pleural and six patients with peritoneal mesothelioma. Asbestos exposure was significantly more frequent in men than women</td>
<td></td>
</tr>
</tbody>
</table>
10. "Of particular interest is the occurrence of environmental exposure in three patients, and familial exposure in four others whose age was 40 years or under. Such indirect exposure has recently been emphasized particularly for women, and was present in our series in four of five women exposed to asbestos in contrast to three of 48 men exposed." (at p. 751-52)

11. "It has been suggested that heavier exposure to asbestos may cause peritoneal rather than pleural mesothelioma. Our findings are consistent with this hypothesis, because there was no case of peritoneal mesothelioma secondary to environmental or familial exposure in our series." (at p. 752)

G. The State of the Evidence on Major Health Questions," Chapter 5, Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario, Vol 1 (eds. Dupre, JS, et al) Ontario Ministry of the Attorney General (1984). "We recognize that the reported deaths from mesothelioma after what appeared to have been brief (for gasmask workers) or low (for family contact and neighborhood cases) exposures have caused considerable public anxiety concerning this disease. We cannot confirm the accuracy or otherwise of the proposition that there are cases of mesothelioma which have occurred from insignificant doses of asbestos. However, we think it unlikely. The doses to which the gasmask workers were exposed appeared to have been quite intense, albeit brief, in duration; pictorial evidence and subsequent experiments have indicated that household and neighborhood exposures where mesothelioma resulted were in fact considerably higher than originally thought and approached, or in many cases were equivalent to, corresponding occupational exposures." (at p. 288)

12. "Dr. Davis told the commission that the notion that mesothelioma occurs at low doses first arose from documented cases of mesothelioma among those who lived in the vicinity of the Cape Crocidolite mines in South Africa and from the cases occurring among the wives of asbestos workers who merely brushed their husband's overalls when they came home from work at
night. Dr. Davis is of the opinion that the wrong conclusion has been drawn from this evidence. He has seen pictures which demonstrate that in the past, crocidolite waste was used to surface the roads in South Africa and that vehicles driving along these roads produce dust clouds likely as high as in the asbestos mines and factories. Further, Dr. Davis is aware of experiments which have shown that exposing overalls to asbestos clouds of the sort that might have occurred years ago and then brushing them produced dust levels in the order of 200 fibers/cc." (at p. 288-289)

<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>13.</td>
<td>&quot;Despite the scarcity of environmental data, it is likely that household contacts of asbestos workers are much more heavily exposed than others who simply live in the area.&quot; (at p. 321)</td>
</tr>
<tr>
<td>13.</td>
<td>&quot;Measurements made by Nicholson in the homes of miners and non-miners in a chrysotile mining community in Newfoundland suggest that fiber concentrations were many-fold higher in the former than the latter.&quot; (at p. 321)</td>
</tr>
<tr>
<td>I.</td>
<td>&quot;Some portion of risk assessment is built on that which focuses on reports, incorporating anecdotal observations, which indicate that mesothelioma occurs in nonoccupational settings. These include reports of mesothelioma in bystander occupations, and household members of asbestos workers, and in people who live in the environment of an asbestos mine, mill, factory, or facility which used asbestos-containing products. These reports often carry the presumption of a low to ambient exposure to asbestos. Frequently cites studies are given in Table 8.&quot; (at p. 109)</td>
</tr>
</tbody>
</table>
103

14. “There are several features which require attention. The fiber type most cited in these exposures is crocidolite; asbestos borne amphiboles are virtually always implicated as a component exposure.” (at p. 109)

15. “The impression that non-occupational mesothelioma occurs after a low, invisible, or even ambient exposure, appears not to be the
| 16. | "The occupational history showed no direct occupational exposure to asbestos. An extensive interview with the patient’s husband established that he was employed for 34 years (1935-1969) as a machinist at a shipyard, where he dismantled boilers and other shipyard machinery throughout his employment. He stated that his work clothes became “covered with dust” and that his wife regularly laundered these clothes at home." (at p. 354) |
| 17. | "This patient married her husband in 1931 at the age of 25, after which she never worked outside the home. Her occupational history before 1931 was not given. Her only documented exposure to asbestos was by secondary exposure in laundering her husband’s asbestos contaminated work clothes." (at p. 354) |
| 18. | “As seen in the Table, the lung asbestos fiber content of this woman was similar to that in cases of mesothelioma associated with occupational exposure to asbestos. Since lung fiber concentrations exceeding 1 million fibers per gram of dry tissue are associated with an increased risk of mesothelioma, it appears that such household contacts, as described above, may represent a considerable risk of mesothelioma." (at p. 355) | universal case. Even household mesothelioma, with dusty clothing as the vector, may have been associated with exposures greater than 5 f/ml." (at p. 110) |

"The results of analysis of mineral fibres in lung tissues from 10 paraoccupational cases of malignant mesothelioma were compared with analysis obtained from seven cases of malignant mesotheliomas that had developed in gas mask workers." (at p. 621)

19. "Nine of the paraoccupational cases were considered to have developed their tumours because of exposure to asbestos on their husbands' working clothes and one cancer developed in the daughter of a man who had died of asbestosis." (at p. 621)

20. "The gas mask workers had direct exposure to asbestos while working in a factory that produced military gas masks." (at p. 621)

21. "The gas mask workers showed a consistent pattern with high crocidolite concentrations and normal or low concentrations of chrysotile and amosite. Fibre lengths for chrysotile were similar in both groups and predominantly less than 5 microns. Crocidolite fibres tended to be longer in the gas mask workers than in the paraoccupational group and longer than chrysotile in both groups. Amosite fibres tended to be more variable in width than those of chrysotile or crocidolite." (at p. 621)


"The tissue asbestos content of the six household contacts of asbestos workers is summarized in Table 1. All were women with ages ranging from 33 to 73. Three of these patients had pleural mesothelioma and 3 had lung cancer. One of the later also had mild asbestosis and one had
peritoneal parietal pleural plaques. Case 5 was a non-smoker. The husband in 4 cases and the father in 1 case had worked as asbestos insulators. Each have been diagnosed as having asbestosis and 3 also had lung cancer. The asbestos body (AB) counts among the 6 household contacts ranged from 2 to 8,200 AB/GM, with a median value of 1,700 AB/GM. The contents of uncoated fibers (UF) 5 microns or greater in length ranged from 17,000 to 120,000 UF per gram, with a median count of 24,300 UF per gram. In comparison, a normal range for asbestos bodies as determined in 84 cases with no evidence of asbestos exposure or an asbestos-related disease is 0 to 20 AB per gram. The median uncoated fiber count for 20 patients with macroscopically normal lungs at autopsy and no history of asbestos exposure was 3,100 UF per gram (and unpublished observations).” (at p. 513-514)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Exposure</th>
<th>Diagnosis</th>
<th>AB/gm (LM)</th>
<th>UF/gm (SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>62/F</td>
<td></td>
<td>Wife of shipyard insulator with asbestosis; 29 yr</td>
<td>Pleural mesothelioma</td>
<td>8,200</td>
<td>ND</td>
</tr>
<tr>
<td>2</td>
<td>33/F</td>
<td></td>
<td>Daughter of insulator with asbestosis; 25 yr</td>
<td>Pleural mesothelioma</td>
<td>2,330</td>
<td>17,000</td>
</tr>
<tr>
<td>3</td>
<td>63/F</td>
<td></td>
<td>Wife of insulator with asbestosis and lung cancer; yrs</td>
<td>Small cell/large cell carcinoma of lung; mild asbestosis</td>
<td>3,670</td>
<td>120,000</td>
</tr>
<tr>
<td>4</td>
<td>59/F</td>
<td></td>
<td>Wife of insulator with asbestosis and lung cancer; 23 yrs.</td>
<td>Small cell carcinoma of lung; PPP</td>
<td>1,060</td>
<td>57,000</td>
</tr>
<tr>
<td>5</td>
<td>73/F</td>
<td></td>
<td>Wife of insulator with lung cancer and asbestosis; yrs</td>
<td>Bronchioloalveolar cell carcinoma of LUL</td>
<td>400</td>
<td>23,700</td>
</tr>
<tr>
<td>6</td>
<td>57/F</td>
<td></td>
<td>Wife of shipyard worker; 1–2 yr</td>
<td>Pleural mesothelioma</td>
<td>2</td>
<td>24,300</td>
</tr>
</tbody>
</table>

"Table 3 compares to tissue asbestos contents in the instant cases with the environmental exposure with that of 161 occupational exposed individuals"
and 33 with no known occupational exposure. It can be seen that in terms of asbestos body concentrations, household contacts ranked fourth and have levels that are comparable to those of shipyard workers other than insulation workers and other asbestos workers (including asbestos cement workers, asbestos textile workers, chemical maintenance workers, welders, machinists, filter manufacturers, roofing plant workers, refinery workers, sheet metal workers, and industrial workers with exposure to asbestos not further specified).” (at p. 514)

**Table 3. Asbestos Content of Lung Tissue by Exposure Category**

<table>
<thead>
<tr>
<th>Exposure Category</th>
<th>n</th>
<th>AB/gm (LM)</th>
<th>UF/gm (SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulation workers</td>
<td>59</td>
<td>20,400</td>
<td>224,000</td>
</tr>
<tr>
<td>Shipyard workers (other than insulators)</td>
<td>60</td>
<td>3,600</td>
<td>37,000</td>
</tr>
<tr>
<td>Other asbestos workers</td>
<td>24</td>
<td>2,360</td>
<td>68,800</td>
</tr>
<tr>
<td>Household contacts</td>
<td>6</td>
<td>1,700</td>
<td>24,300</td>
</tr>
<tr>
<td>Railroad workers</td>
<td>10</td>
<td>55</td>
<td>28,800</td>
</tr>
<tr>
<td>Brakeline work or repair</td>
<td>8</td>
<td>50</td>
<td>15,400</td>
</tr>
<tr>
<td>Manual laborer</td>
<td>15</td>
<td>20</td>
<td>8,830</td>
</tr>
<tr>
<td>Other</td>
<td>18</td>
<td>2.9</td>
<td>2,910</td>
</tr>
<tr>
<td>Building occupants with ACM</td>
<td>4</td>
<td>1.9</td>
<td>9,680</td>
</tr>
</tbody>
</table>

*Data are presented as median values. For other abbreviations, see footnotes to TABLES 1 and 2.*

23. “Almost half of the fibers from the household contact cases were the commercial amphiboles, amosite or crocidolite, where as fewer than 5% of the fibers from the building occupants were commercial amphiboles. [N]on commercial amphiboles in chrysotile accounted for a minority of fibers in both groups (5 to 22%).” (at p. 515)

24. “The present study indicates that in general, household contacts have substantially elevated pulmonary asbestos burdens, often in the range of those individuals who were occupationally exposed to asbestos. That the exposures in these women’s homes were heavy is further supported by the observation that in 5 of the 6 cases, the occupationally exposed individuals in the household was in insulation worker with clinically diagnosed asbestosis. Three of these individuals also had lung cancer. The median
| M. | "Report to Congress on Workers' Home Contamination Study Conducted Under the Workers' Family Protection Act," 29 USCA 67(a), US Department of Health and Human Services Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health Publication No. 95-123 (September 1995). | "Most cases of asbestos disease among workers' family members occurred in households where information indicated that asbestos contaminated work clothing was brought into a home and women were exposed during home laundering of the contaminated work clothing." (at p. 7) |
| 25. | "Three review articles discussed the adverse effects in family members of asbestos workers and the basis for inferring that these adverse health effects result in transporting contaminated clothing and other articles into the home. Garandjean and Bach (1986) reviewed the literature and effects of asbestos exposure on workplace bystanders and family members and Rom and Lockey (1982) and Berry (1986) reviewed the association between asbestos exposure and mesothelioma." (at p. 8) |
| 26. | "Estimates of exposure levels that could have occurred during home laundering of beryllium and asbestos suggest that such exposures could have exceeded OSHA occupational exposure limits for these substances." (at p. 35) |
| N. | Schneider, Joachim; "Pleural Mesothelioma and Household Asbestos Exposure" Reviews on Environmental Health, 11(1-2): 65-70 (1996). | "This article discusses the development of asbestos induced malignant mesotheliomas after nonoccupational environmental exposure to asbestos through contact with occupationally exposed household members. In our policlinic, we have seen six fatal pleural mesothelioma cases (5 wives and one son of asbestos industry workers) with no history of occupational asbestos exposure. In 5 women, a causal relation was established between the fatal disease and inhalation of asbestos fibers while cleaning the asbestos body an uncoated fiber contents of 30 insulation workers with asbestos in the author's series are 109,000 AB program and 646,000 UF program of wet lung tissue, respectively." (at p. 516) |
contaminated work clothes and shoes of their husbands at home. The son had also been exposed to asbestos throughout his childhood during daily visits with his father at the workplace." (at p. 65)

27. "Detailed job histories revealed that neither the women nor the son had ever worked in an asbestos factory. Asbestos exposure was exclusively through residential inhalation of asbestos from contaminated work clothes or shoes that were brought home from the workplace by the husband. Throughout his childhood, the son had regularly delivered a hot meal to his father at the workplace. From 1950 to 1984, the husbands were employed in different companies that manufactured asbestos mats, asbestos textiles, and asbestos cardboard, or in such occupations as insulators, roofers, or turbine-revision workers who process asbestos products." (at p. 65-66)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Born</th>
<th>Died</th>
<th>Length of household exposure</th>
<th>Latency (years)</th>
<th>Occupation of household contact</th>
</tr>
</thead>
<tbody>
<tr>
<td>E.L.</td>
<td>1925</td>
<td>1993</td>
<td>1961–84</td>
<td>31</td>
<td>Turbine revision</td>
</tr>
<tr>
<td>M.L.</td>
<td>1931</td>
<td>1987</td>
<td>1969–76</td>
<td>17</td>
<td>Asbestos cardboards</td>
</tr>
<tr>
<td>G.K.</td>
<td>1933</td>
<td>1992</td>
<td>1964–74</td>
<td>26</td>
<td>Insulator</td>
</tr>
</tbody>
</table>

*Son of G.J.*


"Members of the households of former asbestos workers are at a higher risk from dying of mesothelioma resulting from asbestos exposure from cleaning asbestos contaminated working clothes of their relatives at home. The lung fiber burdens of some of those who developed mesothelioma are similar even to those of former asbestos workers. Such people are said to be paraoccupationally exposed." (at p. 67)
Table 1. Published case series of malignant mesotheliomas in wives and children or adolescents attributed to inhalative asbestos fibre exposure at home via their relatives' exposure at the job (contaminated work clothes)

<table>
<thead>
<tr>
<th>Ref.</th>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>Mesotheliomas</th>
<th>Occupational asbestos exposure of the relatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>Wagner et al.</td>
<td>1960</td>
<td>South Africa</td>
<td>1</td>
<td>crocidolite miner</td>
</tr>
<tr>
<td>30</td>
<td>Lieben and Piirnakowa</td>
<td>1967</td>
<td>USA</td>
<td>1</td>
<td>insulator</td>
</tr>
<tr>
<td>51</td>
<td>Champion</td>
<td>1970</td>
<td>England</td>
<td>1</td>
<td>cleaning cardio machines</td>
</tr>
<tr>
<td>52</td>
<td>McEwen et al.</td>
<td>1971</td>
<td>England</td>
<td>3</td>
<td>pipe lagger</td>
</tr>
<tr>
<td>53</td>
<td>Rubino et al.</td>
<td>1972</td>
<td>Italy</td>
<td>1</td>
<td>shipyard industry</td>
</tr>
<tr>
<td>54</td>
<td>Milne</td>
<td>1972</td>
<td>Australia</td>
<td>1</td>
<td>asbestos cement factory</td>
</tr>
<tr>
<td>55</td>
<td>Knappmann</td>
<td>1972</td>
<td>Germany</td>
<td>1</td>
<td>asbestos factory</td>
</tr>
<tr>
<td>56</td>
<td>Greenberg and Davies</td>
<td>1974</td>
<td>England</td>
<td>1</td>
<td>n.a.</td>
</tr>
<tr>
<td>57</td>
<td>Lilington et al.</td>
<td>1974</td>
<td>USA</td>
<td>1</td>
<td>insulator</td>
</tr>
<tr>
<td>58</td>
<td>Li et al.</td>
<td>1978</td>
<td>USA</td>
<td>1</td>
<td>machinist</td>
</tr>
<tr>
<td>59</td>
<td>Epler et al.</td>
<td>1980</td>
<td>USA</td>
<td>6</td>
<td>insulator, maintenance worker</td>
</tr>
<tr>
<td>60</td>
<td>Vianna et al.</td>
<td>1982</td>
<td>France</td>
<td>1</td>
<td>steam fitter</td>
</tr>
<tr>
<td>61</td>
<td>Hirsch et al.</td>
<td>1982</td>
<td>Sweden</td>
<td>2</td>
<td>foundry</td>
</tr>
<tr>
<td>62</td>
<td>Martensson et al.</td>
<td>1984</td>
<td>USA</td>
<td>1</td>
<td>steam fitter</td>
</tr>
<tr>
<td>63</td>
<td>Krouzel et al.</td>
<td>1986</td>
<td>USA</td>
<td>3</td>
<td>asbestos miners</td>
</tr>
<tr>
<td>64</td>
<td>McConnachie et al.</td>
<td>1987</td>
<td>Cyprus</td>
<td>1</td>
<td>n.a.</td>
</tr>
<tr>
<td>65</td>
<td>Wolf et al.</td>
<td>1987</td>
<td>USA</td>
<td>6</td>
<td>ceramic engineer</td>
</tr>
<tr>
<td>66</td>
<td>Faire et al.</td>
<td>1988</td>
<td>USA</td>
<td>1</td>
<td>insulator</td>
</tr>
<tr>
<td>67</td>
<td>Li et al.</td>
<td>1989</td>
<td>USA</td>
<td>1</td>
<td>shipyard machinist</td>
</tr>
<tr>
<td>68</td>
<td>Huncharek et al.</td>
<td>1989</td>
<td>USA</td>
<td>1</td>
<td>shipyard industry</td>
</tr>
<tr>
<td>69</td>
<td>Hammar et al.</td>
<td>1989</td>
<td>USA</td>
<td>1</td>
<td>n.a.</td>
</tr>
<tr>
<td>70</td>
<td>Shepherd et al.</td>
<td>1989</td>
<td>USA</td>
<td>9</td>
<td>shipyard industry, pipe lagger</td>
</tr>
<tr>
<td>71</td>
<td>Gibbs et al.</td>
<td>1990</td>
<td>USA</td>
<td>1</td>
<td>glass and shipyard industry</td>
</tr>
<tr>
<td>72</td>
<td>Kane et al.</td>
<td>1990</td>
<td>USA</td>
<td>5</td>
<td>railroad worker</td>
</tr>
<tr>
<td>73</td>
<td>Maltoni et al.</td>
<td>1991</td>
<td>Italy</td>
<td>1</td>
<td>maintenance worker</td>
</tr>
<tr>
<td>74</td>
<td>Anderson et al.</td>
<td>1991</td>
<td>USA</td>
<td>1</td>
<td>car machinist</td>
</tr>
<tr>
<td>75</td>
<td>Muskat and Winder</td>
<td>1991</td>
<td>USA</td>
<td>2</td>
<td>shipyard industry</td>
</tr>
<tr>
<td>76</td>
<td>Roggi and Longo</td>
<td>1991</td>
<td>Italy</td>
<td>5</td>
<td>shipyard industry</td>
</tr>
<tr>
<td>77</td>
<td>Giarelli et al.</td>
<td>1992</td>
<td>Italy</td>
<td>9</td>
<td>shipyard industry, oil refinery</td>
</tr>
<tr>
<td>78</td>
<td>Dodici et al.</td>
<td>1992</td>
<td>Italy</td>
<td>6</td>
<td>shipyard industry</td>
</tr>
<tr>
<td>79</td>
<td>Schneider and Woittowitz</td>
<td>1995</td>
<td>Germany</td>
<td>1</td>
<td>insulator, roofworker, asbestos textile industry</td>
</tr>
</tbody>
</table>

n.a. = Not available.


"Study results support previous evidence that occupational and paraoccupational exposure to asbestos is associated with developing mesothelioma." (abstract)

28. "It is generally accepted that there is a link between paraoccupational exposure to asbestos and mesothelioma. Case-control studies in London, New York, and North America found a history of paraoccupational exposure to asbestos in more cases than controls, although the numbers were small. This study supports previous
evidence about paraoccupational exposure, but the size and precision of the OR depends on whether we include those possibly exposed with those likely or unlikely to be paraoccupationally exposed." (at p. 407)

| 29. | "The few studies which have measured airborne asbestos concentrations in non-occupational settings indicate that paraoccupational exposure can lead to higher concentrations than residential exposure, but we cannot be sure that this is the case for all the subjects in this study." (at p. 408) |


|  | "By far the most common form of exposure was through household contact with an asbestos worker, from asbestos brought home on the clothes of the worker in the same household. Thirty-six (61%) of 59 women with mesothelioma were household contacts of asbestos workers. For 34 of these cases, this was the only identifiable exposure to asbestos...Nine of the women of this group had parietal plaques and 2 also had asbestosis. Three had peritoneal mesotheliomas and 1 patient’s father died of mesothelioma." (at p. 156) |
There was one case each of contact through a husband and a son; a father and uncles; and a father, a cousin, and uncles. The household contacts’ occupations were as follows: insulator (10 cases); shipyard worker (6 cases); pipefitter/welder (3 cases); oil refinery worker (2 cases); and steamfitter, papermaker, tire presser, machinist, construction worker, merchant marine seaman, auto mechanic, and asbestos plant manufacturer (1 case each). In 7 cases, the occupation of the household contact was not available." (at p. 156)

Fiber burden analysis was performed on lung tissue from 13 women who were household contacts of asbestos workers, and 10 (77%) of these had an elevated tissue asbestos burden. The median asbestos body count for these 13 samples was 3 AB per G and exceeded the background range in 8 (62%) of the 13 cases." (at p. 158)

"Among the 12 samples for which SEM and EDXA were performed, commercial amphiboles
were elevated in 7 cases, non-commercial amphiboles in 4 cases and chrysotile in 2 cases. In one unique situation, both the woman (case 18) and her husband presented with mesothelioma and pleural plaques. The husband also had asbestosis. Analysis of the husband’s lung tissue demonstrated about 80 times as many asbestos bodies and 30 times as many uncoated fibers as those found in his wife’s lungs.” (at p. 158)

33.  
"Among the 3 patients exposed through household contact but without an elevated tissue asbestos burden, 1 was a 29 year old woman (case 55) with pleural mesothelioma who was exposed to dust on the clothes of her father, cousin, and uncles over a 20 year period. A single amosite fiber was detected by SEM, which is considered an ambiguous result (none expected in controls). Another patient was a 45 year old woman (case 34) with pleural mesothelioma who was exposed to dust on her father’s clothes for 18 years; her father was an insulator who died of mesothelioma. The third patient was a 41 year old woman (case 39) with peritoneal mesothelioma who had been exposed to asbestos on the clothes of her father and uncles during the summers over a 20 year period.” (at p. 158)

34.  
"A history of exposure to asbestos through household contact with an asbestos worker was found in 61% of our cases. This type of exposure was first reported as a cause of mesothelioma by Newhouse and Thompson in 1965 and Anderson et al reported 5 cases of pleural mesothelioma in a study of 756 household contacts of asbestos factory workers in 1979. Gibbs et al. reported the results of tissue asbestos analysis in a series of 10 patients with mesothelioma who were household contacts of asbestos workers and found elevated counts beyond those of a reference population in 8 of the 10 cases. These finding are almost identical to those presented here, in which an elevated tissue asbestos burden noted in 10 (77%) of 13 of the household contacts with mesothelioma. One of the tumors (Case 31)\(^3\) was

\(^3\) Wife of shipyard worker
a sarcomatoid peritoneal mesothelioma. To our knowledge, this is the first case of peritoneal mesothelioma in a household contact has been demonstrated in support of an asbestos etiology." (at p. 160)

| 35. | "An elevated tissue asbestos burden was noted in 70% of women from whom lung tissue was available for analysis. The main fiber type identified was amosite, followed by tremolite and chrysotile." (at p. 161-162) |

| R. Bourdes, V, et al., "Environmental Exposure to Asbestos and Risk of Pleural Mesothelioma: Review and Meta-Analysis" Eur. J. Epidemiology, 16(5):411-417 (2000). | "A number of epidemiological studies have addressed the risks of pleural mesothelioma from environmental (household and neighborhood) exposure to asbestos, but no overall risk assessment is available. We reviewed the epidemiological studies on risks of pleural mesothelioma and household or neighborhood exposure to asbestos. We identified eight relevant studies; most were conducted in populations with relatively high exposure levels. We combined the risk estimates in a meta-analysis based on the random affects model. The relative risks of pleural mesothelioma for household exposure range between 4.0 and 23.7, and the summary risk estimate was 8.1. For neighborhood exposure, RR range between 5.1% and 9.3%. In the summary estimate lists 7.0%. This review suggests a substantial increase in risk of pleural mesothelioma following high environmental exposure to asbestos; however, the available data are insufficient to estimate the magnitude of the excess risk at the levels of environmental exposure commonly encountered by the general population in industrial countries." (at p. 411) |

| 36. | "The combined RR of pleural mesothelioma from household exposure was 8.1. All but one study was conducted in the areas of either predominant or co-contaminant amphibole exposure, a fact that limited the analysis according to fiber type." (at p. 412) |

| 37. | "Only two studies presented results on peritoneal mesothelioma following environmental asbestos exposure. Newhouse and Thompson reported two |
cases of peritoneal mesothelioma in women with household exposure, none with neighborhood exposure and seven cases, including three in men, without evidence of occupational or environmental exposure to asbestos. Vianna and Polan reported eight cases of peritoneal mesothelioma among women without occupational exposure: household exposure was reported for seven of them, and residential exposure for three, including the case without the household exposure." (at p. 413) (see analysis above re: these studies). (at p. 413)

38. Table 3 presents the results of the meta-analysis. The combined RR for neighborhood exposure was 7.0. There was a non-significant increased risk in the two studies considering mainly chrysotile exposure.

<table>
<thead>
<tr>
<th>Type of fiber</th>
<th>Neighborhood exposure</th>
<th>Household exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N RR 95% CI</td>
<td>N RR 95% CI</td>
</tr>
<tr>
<td>All studies</td>
<td>6 7.0 4.7-11</td>
<td>5 8.1 5.3-12</td>
</tr>
<tr>
<td>Type of fiber</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chrysotile</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amphiboles</td>
<td>1 8.7 6.7-11</td>
<td>1 2.1 2.8-157</td>
</tr>
<tr>
<td>Mixed/unspecified</td>
<td>3 6.7 4.4-10</td>
<td>3 8.2 5.2-13</td>
</tr>
</tbody>
</table>

N = Number of studies; RR = relative risk; CI = confidence interval.

39. "The main result of this analysis is a strong relationship between pleural mesothelioma and high environmental exposure to asbestos, whether the source of exposure is domestic or neighborhood. The results also suggest a higher risk from exposure to amphiboles than from exposure to chrysotile." (at p. 413-415)

40. "[T]he studies including in the meta-analysis addressed circumstances of exposure to relatively high levels of asbestos." (at p. 415)
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>41.</td>
<td>&quot;Insufficient evidence exists on the risk of pleural mesothelioma from non-occupational exposure to asbestos. A population based case-control study was carried out in six areas from Italy, Spain, and Switzerland. Information was collected for 215 new histological confirmed cases and 448 controls…In 53 cases and 232 controls without evidence to occupational exposure to asbestos, moderate or high probability of domestic exposure was associated with an increased risk adjusted by age and sex: odds ration 4.8, 95% competence interval 1.8-13.1. It is suggested that low dose exposure to asbestos at home or in the general environment carries a measureable risk of malignant pleural mesothelioma.&quot; (at p. 104)</td>
</tr>
<tr>
<td>42.</td>
<td>&quot;For both domestic and environmental exposure, a dose response relation was observed with intensity of exposure. Relative risk for environmental exposure seemed higher than for domestic exposure, but were based on smaller numbers, and confidence intervals overlapped.&quot; (at p. 107)</td>
</tr>
<tr>
<td>43.</td>
<td>&quot;The high probability of environmental exposure, defined as living within 2,000 meters of an asbestos mine or works such as asbestos cement plants, asbestos textiles, shipyards or brake factories, entailed an almost 12-fold increase in risk. Living between 2,000 and 5,000 meters of asbestos industries or within 500 meters of industries using asbestos products (low permeability) was associated with an increased, but not statistically significant risk.&quot; (at p. 107)</td>
</tr>
</tbody>
</table>

"In the present study, a 5-fold increase in risk has been estimated for high or moderate probability of being exposed to asbestos at home. This relative risk was higher in Barcelona than in Casale or Torino. The risk has long been recognized and has been mainly attributed to exposure to fibers brought home with the clothes of asbestos workers. The present study, however, suggests that exposure at home from handling asbestos material for maintenance and from presence of asbestos material susceptible to damage also
| T. | Magnani, C., et al. “Increased Risk of Malignant Mesothelioma of the Pleura after Residential or Domestic Exposure to Asbestos: A Case-Control Study in Casale Monferrato, Italy,” Environmental Health Perspectives 109(9): 915-919 (2001). | "Our study investigates environmental and domestic asbestos exposure in the city where the largest Italian asbestos cement factory was located. This population based case control study included pleural mesothelioma incidents in the area in 1987 to 1993, matched by age and sex, to two controls." (at p. 915) |
| 44. | "In 1981 the company reported the use of 15,000 tons of asbestos (10% crocidolite)." (at p. 915) |
| 45. | "The relative risk of 3.1 per spouses in the present study lies in the confidence interval of a previous independent estimate in the same area. Here, parents work in the AC factory was a stronger risk factor." (at p. 918) |
| U. | Camus, M., et al., “Risk of Mesothelioma Among Women Living Near Chrysotile Mines Versus US EPA Asbestos Risk Model: Preliminary Findings” Annals of Occupational Hygiene, 46 (Suppl. 1): 95-98 (2002). | "The average cumulative exposure of the study population from all sources was 25 fibers/ml-yr (168 hours per week) or a "worker equivalent exposure" of 105 fibers/ml-yr. Over the 1970-1989 study, the study base totaled 22,375 person years, for which the EPA model predicted 150 mesotheliomas in females in asbestos and 500 in females in Thetford mines. In comparison, a single mesothelioma (peritoneal) occurred in the Asbestos district. Ten mesotheliomas (all pleural) were observed in females in the Thetford mine district. More detailed exposure information is given in Case et al. The risk of mesothelioma (pleural and peritoneal) was overestimated 150-fold in the asbestos area, 35-fold in the Thetford area, and 50-fold in both areas combined. The mesothelioma incidence rates were 67.5 per million person year in the Thetford area and 13.7 million person year in the asbestos area. In comparison, mesothelioma incidence among Quebec women was about 4 per million person year." (at p. 97) |
| V. | Case, B.W., et al., “Preliminary Findings for Pleural Mesothelioma among Women in the Québec Chrysotile Mining Regions,” Annals of Occupational Hygiene, 46(Suppl. | "Mean cumulative exposure (residential, domestic, and occupational combined) was estimated as 22.61 fibers/ml-yr (range 84-525 for cases and 84.1 fibers/ml-yr (range 0-189 for controls, with a plausible 5-fold error on either
| 46. | "Ten pleural mesothelioma cases were identified in the study, all in the Thetford mines area. Seven had tissue available for review by the two pathologists and an eighth had been evaluated at the time of diagnosis by other members of the US Canada Mesothelioma Panel. Six cases were considered definite or probable mesothelioma and the seventh "possible" by pathologists. The eighth case had insufficient tissue for diagnosis according to both pathologists. Although available pathology records suggested a high probability of mesothelioma. This case, the case in which both pathologists agreed the diagnosis was "possible," and the two cases for which tissue was unavailable were retained after applying the algorithm we developed. The six definite were probable and four possible cases are grouped together for case control comparison." (at p. 129) |
| 47. | "All 10 cases lived for all of the years of their lives (other than the final 20 in one case) in the Thetford mines area, more specifically in the Western part. This is the portion adjacent to the "central mines" known to have the highest tremolite content. Domestic exposures in nine of the 10 cases were in part related to work by household members in one or more of the central mines, but no attempt was made to compare these in a quantitative fashion to the domestic exposures and the control population." (at p. 129-130) |
| 48. | "The case control comparison demonstrated the particularly high risk associated with having worked in the asbestos industry. Two cases worked as cobbers prior to World War II; this work was done by hand. Three additional cases were women who worked in a single unventilated asbestos bag fabrication and repair shop in the period immediately following World War II. Bags were made of burlap and came from locations both within and outside Thetford mines. Lung retained fiber analysis had been performed |
for two of these three cases, and for a third woman who died from lung cancer and asbestosis who worked in the same facility during the same years. In the two cases, lung levels of tremolite were 5.0 and 29.9 fibers per ug and chrysotile and 2.9 and 7.5 fibers per ug respectively. In one of the two lung tissue also contained crocidolite and amosite. For the woman with lung cancer and asbestosis who worked in the same facility, lung tissue contained 45 fibers per ug dry lung, 36 tremolite fibers per ug and 8.4 amosite fibers per ug. Tissue for lung burden analysis was not available for any of the other mesothelioma cases." (at p. 130-131)


"Autopsy material from a cohort of 15 women with the pathological diagnosis of mesothelioma was selected for this study...The history of exposure ranged from direct work with asbestos containing products to secondary exposure at home from contaminated work clothing. Lung tissue from the individuals was evaluated by one of the authors for pathologic asbestosis and when found, graded according to the College American Pathologists and the National Institute for Occupational Safety and Health Criteria." (at p. 189)

49. "Ferruginous bodies were found in 13 of the 15 individuals. Seven of the individuals had over 1,000 ferruginous bodies per gram of dry weight while no ferruginous bodies were detected in two individuals." (at p. 192-193)

50. "Crocidolite was found in only one individual (Case 15) and it represented the only type of asbestos found in that individual. That patient had a history of domestic bystander exposure from her spouse who worked at a plant that made crocidolite asbestos containing pipe products." (at p. 193)

51. "The three lowest concentrations of total uncoated asbestos fibers were found in individuals whose primary exposures were from household contacts with two individuals not having ferruginous detected in their analysis. Exposure history and
tissue burden can be inconsistent in that the third highest number of ferruginous bodies and uncoated asbestos fibers were in an individual who had no primary exposure to asbestos. Domestic bystander exposure to asbestos occurred within the home from contact with her husband's clothing. The husband worked at a plant that made crocidolite containing products. In contrast, the individual with the fifth lowest total burden of uncoated asbestos fibers had worked in a shipyard. The least commonly encountered types of asbestos in these females was crocidolite. However, it represented the only type found in the individual with the third highest tissue burden and as noted the source of the contact was in the household. While chrysotile has been reported to clear over time, it was found in three of the 15 individuals. Chrysotile as the major fiber type in one individual who had worked in a brake shoe factory. Her lungs also contained a substantial amount of amosite.” (at p. 194)

52. "The household exposures include Assay Numbers: 5; 12; 14; and 15. Assay Number 5 had a husband in the Coast Guard and a father who was a shipyard worker; Assay Number 12 had a husband who worked as a maintenance system worker for 32 years and a father who worked as a painter, plasterer, and a guard for shipbuilding and drydock company; Assay Number 14 worked as a clerical assistant for 30+ years and her husband worked as a laborer, shipscaler, longshoreman, warehouseman, plumber’s helper, bumer helper, and cement worker for 45 years; and Assay Number 15 is described above, her husband worked in a crocidolite concrete pipe manufacturer." (at p. 194) See Table I(A).
**TABLE I(A). Historical Data for Female Mesothelioma Cases**

<table>
<thead>
<tr>
<th>Assay number</th>
<th>Age*</th>
<th>Packs/ year*</th>
<th>Asbestos/ grade</th>
<th>Patient</th>
<th>Husband</th>
<th>Father</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>69</td>
<td>&lt;1</td>
<td>N</td>
<td>Shipyard laborer (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>67</td>
<td>23</td>
<td>N</td>
<td>Electronics component assembler (11)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>74</td>
<td>15</td>
<td>N</td>
<td>Brake shoe factory (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>78</td>
<td>U</td>
<td>Y/1</td>
<td>Shipyard worker during WWII</td>
<td>Coast guard,</td>
<td>Shipyard worker</td>
</tr>
<tr>
<td>5</td>
<td>59</td>
<td>25</td>
<td>N</td>
<td>Bathroom fixture manufacturer (8), airplane manufacturer (5), clerk subject to renovation dust (25)</td>
<td>Alpilene manufacturer</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>89</td>
<td>NS</td>
<td>N</td>
<td>Bathroom fixture manufacturer (8), airplane manufacturer (5), clerk subject to renovation dust (25)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>76</td>
<td>60</td>
<td>N</td>
<td>Shipyard worker (5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>81</td>
<td>U</td>
<td>Y/1</td>
<td>Shipyard worker, electrician helper (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>86</td>
<td>U</td>
<td>N</td>
<td>Shipyard worker, janitor (1–2)</td>
<td>Chipper &amp; caulkier (23)</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>93</td>
<td>NS</td>
<td>N</td>
<td>Shipyard worker during WW II (4), farm operator (40)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>78</td>
<td>U</td>
<td>U</td>
<td>Airplane insulator (2–5)</td>
<td>Pipefitter, Maintenance systems worker (32)</td>
<td>Pointer, platemaker, &amp; guard for shipbuilding &amp; drydock company</td>
</tr>
<tr>
<td>12</td>
<td>63</td>
<td>12</td>
<td>U</td>
<td>Airplane insulator (2–5)</td>
<td>Pipefitter, Maintenance systems worker (32)</td>
<td>Pointer, platemaker, &amp; guard for shipbuilding &amp; drydock company</td>
</tr>
<tr>
<td>13</td>
<td>76</td>
<td>NS</td>
<td>N</td>
<td>Machine operator, pipefitter, worker &amp; assembly worker (50)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>66</td>
<td>2</td>
<td>N</td>
<td>Clinical assistant (30+)</td>
<td>Industries worker, longshoreman, warehouseman, plumber's helper, bumer helper, &amp; cement worker (45)</td>
<td>Crocked concrete pipe manufacturer</td>
</tr>
<tr>
<td>15</td>
<td>69</td>
<td>U</td>
<td>N</td>
<td>-kind</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: U and None, unknown or unavailable; Y, yes; N, no.  
*Age at time of death.  
*NS for non-smoker, U for unknown, S for smoker listed as number of packs/year.  
*Exposure and number of years worked.

---


"The authors have had the opportunity to examine the pulmonary asbestos content from 28 household contacts of asbestos workers, including 22 with mesothelioma and 4 with lung cancer. Twenty-six of these cases were women, including 16 wives, 9 daughters and one mother of an asbestos worker. The other two cases were sons of asbestos workers. The occupation of the worker was known in 24 cases, and included 11 insulators, 5 shipyard workers, 3 power plant workers, 2 pipefitters, 1 chemical plant worker, 1 vinyl work installer, 1 toolfitter/glass plant worker." (at p. 340)

53. "The median asbestos body count for these cases was 260 AB per gram, with a range of 2.0 to 14,100 AB per gram. The median uncoated fiber content was 22,400 fibers 5 µ or greater in length per gram of wet lung, with a range of 2,920 to..."
162,000 fibers per gram. It should be noted that the tissue asbestos content in household contacts is of the same order of magnitude as that of molten metal workers, U. S. Navy mechanic/marine seaman, construction workers and oil and chemical refinery workers. Our findings are thus similar to those reported by Gibbs, et al. Asbestosis was confirmed histologically in three of 27 cases. Amosite was the major fiber type identified." (at p. 341)


"From the files of nine Plaintiff law firms throughout the United States, 32 cases of mesothelioma were analyzed who had no occupational, environmental or other exposure to asbestos than as a household member of a worker with a clear occupational exposure." (at p. 459)

54. "Occupations of the workers who lived with the present cases were: shipyard (13), insulator (other than shipyard) (7), railroad (2), manufacturer of asbestos materials (2), boilermaker (2), steel mill (2), refinery (1), pipelayer (1), refractory brick layer (1), and construction (1)." (at p. 459)

55. "Histological classification of the 32 cases is typical of mesothelioma in general: 27 pleural and 5 peritoneal. Among the 29 female cases, 24 were pleural and 5 were peritoneal; all three male cases were pleural. Diagnosis was confirmed by immunohistochemical reactions and was based on multiple specimens reviewed by pathologists in different institutions in the remaining 3." (at p. 459)

56. "Although information about other asbestos related disease was incomplete, pleural plaques were present in at least ten household mesothelioma cases, four of whom also had asbestosis." (at p. 459)

57. The household exposures that resulted in peritoneal mesotheliomas included: the daughter/sister of a father and son steel mill workers; the step-daughter of an insulator; the wife of a construction worker; the wife of a
<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Z.</strong></td>
<td>Reid, A., et al, “The mortality of women exposed environmentally and domestically to blue asbestos at Wittenoom, Western Australia,” <em>Occup Environ Med</em>, 65:743-749 (2008).</td>
<td>&quot;2,552 women and girls are documented to have lived in the blue asbestos mining and milling township of Wittenoom between 1943 and 1992 and were not involved in the asbestos mining or milling. Quantitative asbestos exposure measurements were derived from periodic dust surveys.&quot; (at p. 743)</td>
</tr>
<tr>
<td><strong>58.</strong></td>
<td></td>
<td>&quot;Of the 30 known cases of malignant mesothelioma of the pleura in this cohort, 14 were recorded on death certificates as having died from malignant neoplasm of the pleura, 6 had malignant mesothelioma of the pleura and, 1 had mesothelioma of other sites and 1 had mesothelioma site unspecified. They were recorded as having died from lung cancer, one from renal disease, one from malignancy with ill-defined or unspecified site, one from benign neoplasm of the pleura, one from asbestosis and one had no cause of death coded. There were no deaths from peritoneal mesothelioma.&quot; (at p. 746)</td>
</tr>
<tr>
<td><strong>59.</strong></td>
<td></td>
<td>&quot;Mortality from mesothelioma was significantly associated with cumulative asbestos exposure. The risk of mesothelioma tended to increase for those residents who were known to have washed clothes of, or lived with, an ABA asbestos worker.&quot; (at p. 746)</td>
</tr>
</tbody>
</table>
| **AA.** | Goldberg, M. and Luce, D., "The health impact of non-occupational exposure to asbestos: what do we know?" *Eur J Cancer Prev*, 1-15 (2009). | "The studies considered here concern mesothelioma cases in individuals with no known personal occupational exposure, but who lived with asbestos workers. Several case reports have attributed mesothelioma to paraoccupational exposure in diverse circumstances (Lieben and Pistawka, 1967; Milne, 1972; Vianna et al., 1981; Bianchi et al., 1982, 1987; Schneider et al., 1996). Population-based case–control studies in different countries have shown cases of pleural and/or peritoneal mesothelioma attributed to regular exposure to soiled work clothes, brought home from an exposed workplace (Newhouse and Thomson, 1965; Vianna and Polan, 1978; McDonald and McDonald, 1980). The levels of
asbestos exposure in these circumstances are probably high, but cannot be quantified (Nicholson, 1983; Langer and Nolan, 1988). A cohort study involving more than 2,000 individuals living with asbestos-exposed workers showed an excess rate of mortality from mesothelioma (Anderson, 1983). The follow-up of the cohort of wives of Casale Monferrato (Italy) asbestos-cement factory workers, where approximately 10% of the asbestos used was crocidolite, revealed an excess of pleural mesothelioma; 21 observed versus 1.2 expected (Ferrante et al., 2007). A case–control study in this same population showed an increase in the risk of pleural mesothelioma among the workers’ children. A population-based case–control study of pleural mesothelioma in a region of England suggested that purely household exposure might increase the risk of mesothelioma (Howel et al., 1997). A population-based case–control study of pleural mesothelioma in three European countries (Italy, Switzerland, and Spain) found an elevated risk for domestic exposure, with an exposure–response pattern (Magnani et al., 2000). Finally, a meta-analysis of eight mesothelioma studies yielded a summary relative risk of 8.1 for domestic exposure." (at p. 6)

| BB.  | Rake, C, et al, "Occupational, Domestic and Environmental Mesothelioma Risks in the British Population: A Case Control Study," Br J Cancer, 100:1175-1183 (2009). | "We obtained lifetime occupational and residential histories by telephone interview with 622 mesothelioma patients (512 men, 110 women) and 1420 population controls. Odds ratios were converted to lifetime risk estimates for Britains born in the 1940s. Male ORs relative to low risk occupations for greater than 10 years of exposure before the age of 30 years were 50.0 for carpenters, 17.1 for plumbers, electricians and painters, 7.0 for other construction workers, 50.3 for other recognized high risk occupations and 5.2 in other industries where asbestos may be encountered. The LR was similar in apparently unexposed men and women, and this was approximately doubled in exposed workers’ relatives. No other environmental hazards were identified. In all, 14% of male and 62% of female cases were not attributable to occupational or |
domestic asbestos exposure. Approximately half of the male cases were construction workers, and only four had worked for more than five years in asbestos product manufacture."  (abstract)

60. "The only significant non-occupational association was living with a potentially exposed worker before 30 years of age. The OR for living within a mile of a potential source before 30 years of age was 0.6. The OR for any type of DIY activity was 0.7, and no subgroup of DIY activity by frequency or possible asbestos exposure suggested any excess. No type of housing was significantly associated with risk. The OR for current smokers compared with nonsmokers was 1.5 in this unexposed subgroup, and 1.2 overall."  (at p. 1178)

61. "The ORs for domestic exposure before 30 years of age in Table 6 (2.1 for men, 1.9 for women; combined OR 2.0, 95% CI 1.3–3.2) are based on cases with no occupational exposure. Logistic regression analysis in all cases, adjusting for duration and main exposure group, gave an OR for domestic exposure before 30 years of age of 2. in women, significantly higher than the OR of 1.1 in men. Similar analyses based on the type of relative, irrespective of age, gave a combined estimate of 1.3 for living with a high-risk parent or sibling and an estimate of 2.1 for living with a high risk spouse. The latter was based largely on women, as only two male cases and one male control reported living with a high-risk spouse. Of controls, 20% reported living with a high-risk parent and 12% with a high-risk sibling, and 20% of female controls had lived with a high-risk spouse."  (at p. 1178-1179)
The only substantial risk factor in those with no direct occupational exposure was living with a high-risk worker, a hazard that has been recognized for many years. The excess risk, which was confined to those who lived with an exposed worker before 30 years of age and was similar in men and women, corresponds to an increase in LR of about 1 per 1000." (at p. 1181)
| CC. | Donovan, EP, et al, "Evaluation of take home (para-occupational) exposure to asbestos and disease: a review of the literature," Crit Rev Toxicol, (Epub ahead of print) (2012). | "We conducted a literature review in order to characterize reported cases of asbestos-related disease among household contacts of workers occupationally exposed to asbestos. Over 200 published articles were evaluated. Nearly 60 articles described cases of asbestos-related disease thought to be caused by para-occupational exposure. Over 65% of these cases were in persons who lived with workers classified as miners, shipyard workers, insulators, or others involved in the manufacturing of asbestos-containing products, with nearly all remaining workers identified as craftsmen. 98% of the available lung samples of the persons with diseases indicated the presence of amphibole asbestos. Eight studies provided airborne asbestos concentrations during (i) handling of clothing contaminated with asbestos during insulation work or simulated use of friction products; (ii) ambient conditions in the homes of asbestos miners; and (iii) wearing previously contaminated clothing. This review indicates that the literature is dominated by case reports, the majority of which involved household contacts of workers in industries characterized, generally, by high exposures to amphiboles or mixed mineral types. The available data do not implicate chrysotile as a significant cause of disease for household contacts. Also, our analysis indicates that there is insufficient information in the published literature that would allow one to relate airborne asbestos concentrations in a workplace to those that would be generated from subsequent handling of contact with clothing that had been contaminated in that environment." (abstract) |
| 63. | | "Overall, as evidenced by the majority of cases described in the published literature, para-occupational exposure to asbestos for family members of asbestos workers may have occurred when proper precautions were not taken, especially in industries with a potential for extremely high exposures to asbestos." (at p. 3) |
| 64. | | "As a result of both our general literature search and our review of the JM documents, we identified more than 200 relevant publications; all |
were publically available. We also confined our scope to para-occupational exposures to asbestos that occurred because a household contact had been occupationally exposed. Further, published papers that discussed exposures from asbestos-containing materials used in the home for construction or renovation projects were not included in our evaluation." (at p. 5)

| 65. | "Wagner et al. (1960) were the first to describe a case of pleural mesothelioma in a woman who did not have occupational exposure, but whose father was a crocidolite asbestos miner in South Africa. Based on this observation, the authors reasoned that exposure to crocidolite (an amphibole) in the ambient air and from para-occupational exposure may have resulted in an asbestos related disease in persons who had never worked in the mines or factories (Wagner et al., 1960)." (at p. 13-14) |
| 66. | "Several years later, in a case-control study in a London hospital, Newhouse and Thompson (1965) reported nine cases (two male, seven female) of domestic exposure among family contacts of those primarily working as insulators or in asbestos factories. In general, the primary workers in this study were involved in occupational activities with considerable potential for high asbestos exposures." (at p. 14) |
| 67. | “Additional case reports and case series in the mid- to late-1960s began to identify more cases of pleural and peritoneal mesothelioma that were believed to be a result of exposures from family members in occupations known for high asbestos exposures, such as insulators, asbestos factory workers, and shipyard workers (Kiviluoto, 1965; Lieben & Pistawka, 1967; Milne, 1969; Rusby, 1968). By the end of the 1960s, approximately 20 cases of disease in household contacts thought to be caused by asbestos exposure had been reported in the literature.” (at p. 14) |
| 68. | “Throughout the 1970s, additional case reports, case series, and case-control and cohort studies were published suggesting asbestos related diseases in household contacts of miners, millers,
| 69. | "In the late 1970s, the first large cohort study involving take home asbestos exposures was conducted by researchers at Mt. Sinai, who reported pleural or parenchymal abnormalities (noted on X-rays), lung cancer, and mesothelioma among household contacts (Anderson, 1982; Anderson et al., 1976; 1979; Joubert et al., 1991). Anderson et al. (1976) evaluated household contacts of those who were employed in a factory that produced amosite asbestos products from 1941 until 1954. Of the available household contacts, 679 were determined to have lived in the household of a factory employee, and had not had an occupational exposure to asbestos or fibrogenic dust themselves. They further indicated that individuals who were exposed between 1941 and 1946 had the highest prevalence of radiographic abnormalities; the lowest prevalence was among those exposed between 1950 and 1954." (at p. 14-15) |
| 70. | "In 1978, Vianna and Polan presented the results of their case–control study, in which they discussed nine cases of pleural and peritoneal mesothelioma with no known occupational exposure to asbestos. The occupations of the husbands in these cases included pipefitter, brake lining factory worker, and insulation worker; however, information regarding the types of asbestos used or exposure levels was not provided." (at p. 15) |
| 71. | "Case reports and case-control studies in the 1980s continued to focus on household contacts of miners, insulators, and workers in shipyards and other industrial locations containing historically high airborne asbestos concentrations and the potential for substantial clothing
contamination (Bianchi et al., 1981; Epler et al., 1980; Gibbs et al., 1989; Huncharek et al., 1989; Kilburn et al., 1985; Magee et al., 1986; Martensson et al., 1984; McDonald & McDonald, 1980). McDonald and McDonald (1980), for example, in one of the largest case-control studies of mesothelioma in Canada and the United States, reported eight cases thought to result from household exposures. Five of these eight cases were individuals exposed to the clothing of asbestos factory and insulation workers, while the remaining three were exposed to the contaminated clothing of a chrysotile production worker (McDonald & McDonald, 1980).

In 1981, Bianchi et al. published an autopsy series describing lung abnormalities and occupational histories in Monfalcone, an Italian shipping town. Of the 100 autopsies, 13 were thought to have potentially experienced domestic exposure, primarily due to relatives working in shipyards. After tissue sampling and digestion, eight of these cases were found to have between 10 and 10,000 asbestos bodies in the small lung portion sampled (Bianchi et al., 1981). Pathology evidence has indicated that cores of asbestos bodies primarily result from the presence of amphibole asbestos in the lungs (Mossman & Churg, 1998). Similarly, Kilburn et al. (1985; 1986) studied United States shipyard workers and their families by examining chest radiograms using the criteria established by the International Labour Organization (ILO) for classification of pneumoconiosis. Evidence of asbestos-related disease was determined by three experienced physicians with “B” reader qualifications who reviewed the chest radiograms. Chest radiograms that showed irregular opacities with a profusion rating of 1/0 or greater and/or pleural findings of thickening, plaques, and calcification were determined positive for asbestos-related disease. Of the 274 wives of shipyard workers examined, 11.3% had radiographic evidence of asbestosis, while 2.1% of the female children and 7.6% of the male children showed signs of asbestosis (Kilburn et al., 1985; Kilburn et al., 1986).” (at p. 15)
| 72. | "Many reports continued to focus on workers in the same high exposure trades, including shipyard workers, asbestos cement manufacturers and insulators who were exposed decades earlier (Dodoli et al., 1992; Magnani et al., 1993; Schneider et al., 1996). However, similar to the manner in which knowledge evolved regarding asbestos exposure to those considered end users in the building and construction trades, in the 1990s, more information emerged regarding disease in family members of those involved in construction, where there would have been exposure to asbestos insulation (Ascoli et al., 1996; Chellini et al., 1992; Kane et al., 1990)." (at p. 15-16) |
| 73. | "Magnani et al. (1993) and Ferrante et al. (2007) reported on a cohort of wives of workers at an asbestos cement factory in Italy that utilized both crocidolite and chrysotile asbestos (Ferrante et al., 2007; Magnani et al., 1993). Twenty-one pleural neoplasm cases were observed, as were three peritoneal neoplasms and 12 lung cancers…The only statistically significant increase when compared to women who were not exposed domestically was found for pleural neoplasms (SMR of 18; \( p < 0.01 \)) and an increasing trend with longer duration of exposure was observed (Ferrante et al., 2007). As was true in earlier studies, household contacts continued to be linked with occupations historically characterized by the potential for high exposures to asbestos (usually amphiboles); however, quantitative estimates of exposure levels experienced by the household contacts were unavailable." (at p. 16) |
| 74. | "In total, ten epidemiology studies estimated relative risks for asbestos-related diseases among para-occupationally exposed persons (see Table 3). The highest relative risk (RR) estimate for mesothelioma was calculated from data presented by Newhouse and Thompson (1965), the first study to estimate such a risk (OR of 23). Among the remaining studies, RRs ranged from approximately 2–18 for pleural and peritoneal mesotheliomas. Only one study presented a RR specific to peritoneal mesothelioma (Ferrante et al., 2007)… In almost all studies where a
75. "Gibbs et al. (1990) presented the results of mineral content analysis in 10 cases selected from mesothelioma registries in the UK that were thought to be caused by para-occupational exposure. Lung burden analysis demonstrated elevated amosite and crocidolite in the majority of cases; however, two cases showed normal concentrations of all types of fibers." (at p. 17)

76. "In a case series analysis conducted by Roggli and Longo (1991), fiber burden data were presented for six household contact cases; three were mesothelioma cases, and three were lung cancer cases… Among their household contacts, the median asbestos bodies counted per gram of wet lung tissue (AB/g), as determined by light microscopy, was 1700 AB/g. In comparison, the normal range as determined in 84 cases with no
known exposure to asbestos was 0–20 AB/g. The predominant fiber type was commercial amphibole (i.e. crocidolite and amosite) (Roggli & Longo, 1991). In a more recent report of 89 household exposure mesothelioma cases (79% were female; 10 reportedly also had occupational exposure to asbestos), the median lung fiber burden was reported to be 130 AB/g. Compared to 19 reference cases in which no history of asbestos exposure and no evidence of asbestos-related tissue injury at autopsy was present (median fiber burden of 3 AB/g), the lung burden appeared significant, and was described as being of the same order of magnitude as construction workers. In this second series, the predominant fiber type detected was noncommercial amphiboles (i.e. tremolite with some actinolite and anthophyllite) (Roggli et al., 2002)."

| 77. | “Similarly, Howel et al. (1999) examined fiber-specific lung burdens in various cohorts, including 13 cases of those possibly or likely para-occupationally exposed to asbestos. Although each case was not presented individually, the results indicated that median concentrations of amphibole and chrysotile fibers were higher in those thought to be para-occupationally exposed than in those in the control group (Howel et al., 1999).” (at p. 20) |
| 78. | “In 2003, Dodson et al. published analyses from lung tissues collected from 15 women who died from mesothelioma, four of whom had no occupational exposure but lived with one or more family members that worked in industries where asbestos was used regularly (Dodson et al., 2003). Lung tissues were analyzed for multiple fiber types. Measured concentrations were generally lower than what was seen in cases that had occupational exposure; however, no analyses were performed on an internal reference (unexposed control) population.” (at p. 20) |
| 79. | “Based on this review, it is clear that reports of asbestos related disease among household contacts in the published literature started in the |
1960s and, due to the long latency of asbestos-related disease, continue to be reported. Of the nearly 60 studies describing disease among household contacts, approximately 60% are case reports or case series, even throughout the 1990s. About 90% of these case reports included information about the occupation of the spouse or family member who was thought to be the source of the para-occupational exposure. Over 70% of the household cases were associated with workers classified as miners, manufacturers of asbestos or asbestos-containing products (typically involving raw asbestos), shipyard workers, or insulators. Among the remaining cases, common occupations of the primary worker included various types of crafts, such as steel mill workers, boilermakers, or construction workers; most of these exposures occurred between the 1930s and the 1960s. As such, it appears that these types of craftsmen were historically exposed to amphiboles. The remaining 10% of the case reports did not include specific information regarding occupations, but often made qualitative references to dusty conditions or higher exposures." (at p. 23)

80. "Overall, based on our review, the available data do not implicate chrysotile alone as a significant cause of disease among household contacts, but we acknowledge that one cannot rule out the possibility that chronic exposures to concentrations of chrysotile that are high enough to cause asbestosis, and involve very long fibers, may increase the risk of developing mesothelioma." (at p. 24)

81. "It is noteworthy that a small number of the mesotheliomas associated with household exposure were peritoneal (19 vs. 259 pleural mesotheliomas). Several researchers have reported cases of peritoneal mesotheliomas that have no identifiable history of asbestos exposure and are, consequently, of unknown etiology (Albin et al., 1990; Asensio et al., 1990; Goldblum & Hart, 1995; Spirtas et al., 1994; Huncharek, 2002; Ilgren & Wagner, 1991; McDonald, 1985; McDonald & McDonald, 1994; Price & Ware, 2004; Walker et al., 1983). In most
industrialized countries, the incidence rate of peritoneal mesothelioma ranges between 0.5 and three cases per million in men and between 0.2 and two cases per million in women (Boffetta, 2007). Recently, Moolgavkar et al. (2009) reported that the age-adjusted background rate of peritoneal mesothelioma in the US is one case per million individuals per year for all age groups combined and that the rate increases with age (Moolgavkar et al., 2009). Thus it is not certain whether these 19 cases are due to a spontaneous tumor or para-occupational exposure to asbestos." (at p. 24)

<p>| 82. | &quot;Based on our evaluation of the literature, then, we have found that it is difficult to accurately characterize the relationship between historical para-occupational exposures and the risk of adverse health effects to those handling and laundering the clothing of family members. The inability to offer quantitative guidance is due to various shortcomings in the current literature, including the fact that historical air data are poorly characterized with respect to particle size and respirability. We acknowledge that there could be differences between the adherence and release of amphibole versus chrysotile fibers from the clothing, but we have no reason to believe this is significant for a fiber of a given length. The impact of fiber type on adhesion to clothing is another area that requires further research.&quot; (at p. 25) |
| 84. | “Disentangling the effects of common exposure to asbestos from common genetic associations is rarely possible, but the Wittenoom subjects and their families, because of their detailed follow-up over many years and their exposure assessments, provide a unique opportunity to study the interaction of genetic and environmental changes which result in asbestos-related diseases (particularly MM, asbestosis and lung cancer).” (at p. 1423) |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th>for common exposure to crocidolite.” (at p. 1423)</th>
</tr>
</thead>
<tbody>
<tr>
<td>85.</td>
<td>“The risk ratio for blood relatives was 1.9 (95% confidence interval [CI] = 1.3-2.9, ( p = 0.002 )). (at p. 1423)</td>
<td></td>
</tr>
<tr>
<td>86.</td>
<td>“This is the first study that has been able to adjust properly for degree and timing of asbestos exposure when evaluating genetic risk of MM, indicating an approximate doubling in risk for relatives compared to no increase in spouses although based on small numbers.” (at p. 1426)</td>
<td></td>
</tr>
<tr>
<td>87.</td>
<td>“Our findings suggest that there is an important, but not large, genetic component in MM similar to that found for other cancers. This result indicates a doubling of risk that may be attributable to genetic factors and supports the contention of other studies that were based on less precise exposure information in case-control studies or descriptive data. The difference in relative risks between spouses and relatives also indicates that familial clustering in MM is unlikely to be caused solely by common infective agents such as SV40 virus or by common dietary factors.” (at p. 1427)</td>
<td></td>
</tr>
<tr>
<td>88.</td>
<td>“As all subjects were exposed only to crocidolite, extension to other forms of asbestos might not be justified.” (at p. 1427)</td>
<td></td>
</tr>
</tbody>
</table>
| 90. | “In 1993, a cohort of former Wittenoom residents was established and the health outcomes of this cohort have been published previously [Hansen, et al., 1993, 1998; Reid et al., 2007, 2008a]. Of the nearly 5,000 people in this cohort about half were children (<15 years old) when they were first exposed to asbestos. The former
“Wittenoom children” are now at an age when chronic adult diseases are becoming more prevalent and many have died. Although they have been routinely included in the mortality and cancer incidence studies that have been reported for the whole cohort, apart from their risk of MM [Reid et al., 2007] they have not been studied separately as a group. The aim of this study, therefore, is to document the cancer incidence and all-cause mortality of people exposed to blue asbestos as children.”  (at p. 134)

91. “Most (93.5%) of the former children had left before the age of 16 years, thereby only having exposure to asbestos during childhood.”  (at p. 135)

92. “After more than 30 years follow-up these ‘former Wittenoom children’ have increased overall mortality and cancer incidence rates compared with the local population, predominantly but not solely due to MM.”  (at p. 139)

93. “For this study, cumulative exposure assigned to each case was based on environmental levels of asbestos. Whether this was the main exposure route is uncertain. Some of the males had subsequent occupational exposure (see above) while the majority of the former Wittenoom children (35 of the 40 with known parentage) lived with an asbestos worker. Despite this, an exposure-response relationship based on assigned environmental levels was observed.”  (at p. 141)

94. “There was no increased lung cancer incidence or mortality in either the men or the women in these analyses.”  (at p. 141)

95. “Although childhood exposure seems to be associated with lower risk of developing MM than exposure in adults, children are considered to be more susceptible than adults to the effects of environmental exposures and similar exposures at different developmental periods may cause a different spectrum of disease [Selevan et al.,
<table>
<thead>
<tr>
<th>No.</th>
<th>Authors</th>
<th>Reference</th>
<th>Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>96. FF</td>
<td>Berk, S., Yalcin, H., Dogan, O., Epozturk, K., Akkurt, I., Seyfikli, Z.,</td>
<td>“The assessment of the malignant mesothelioma cases and environmental asbestos exposure in Sivas province, Turkey,” Environ Geochem Health 36:55-64, 2014.</td>
<td>“In total, 219 patients with MM who were diagnosed in our hospital between 1993 and 2010 were retrospectively analyzed in terms of demographical and clinical features.” (at p. 55)</td>
</tr>
<tr>
<td>97.</td>
<td></td>
<td></td>
<td>“The province of Sivas is located in the mid-eastern part of Anatolia. It includes 17 districts and 1,236 villages; its population is approximately 600,000, 34% of which live in rural areas. Despite a fall in its use in recent years, asbestos-contaminated soil has been used for many years as a whitewash and stucco (name locally as ‘white’ or ‘barren’ soil) in the wall of the houses in rural areas.” (at p. 56)</td>
</tr>
<tr>
<td>98.</td>
<td></td>
<td></td>
<td>“Rarely, MM occurs in patients younger than 20 years (Fraire et al. 1988). The reasons for such cases were suggested to be related to genetic predisposition and other unknown factors other than asbestos exposure. In our series, the average age at the time of diagnosis was 59 and the male-to-female ratio was 1.4:1. The youngest patient was diagnosed at the age of 18 and had no asbestos exposure.” (at p. 59)</td>
</tr>
<tr>
<td>99.</td>
<td></td>
<td></td>
<td>“Most of our patients declared to have lived in rural areas and had environmental asbestos exposure for many years. Furthermore, no difference in the duration of exposure was identified between men and women. Only a small proportion of our patients had a history for possible occupational exposure. These findings suggest that environmental exposure to asbestos is more important in our region compared to work-related asbestos exposure in urban areas and that women are exposed to asbestos as much as men.” (at p. 60)</td>
</tr>
<tr>
<td>100. G</td>
<td>Bourgault, M., Gagne, M., Valcke, M.,</td>
<td>“Lung cancer and mesothelioma risk assessment for a population environmentally exposed to</td>
<td>“Asbestos-related cancer risk is usually a concern restricted to occupational settings. However, recent published data on asbestos environmental concentrations in Thetford Mines, a mining city in</td>
</tr>
</tbody>
</table>
Quebec, Canada, provided an opportunity to undertake a prospective cancer risk assessment in the general population exposed to these concentrations.” (at p. 340)

“Depending on the chosen potency factors, the lifetime mortality risks varied between 0.7 and 2.6 per 100,000 for lung cancer and between 0.7 and 2.3 per 100,000 for mesothelioma. In conclusion, the estimated lifetime cancer risk for both cancers combined is close to Health Canada’s threshold for ‘negligible’ lifetime cancer risks.” (at p. 340)

“The main objectives of this study was to assess the cancer risk for a general population environmentally exposed to asbestos, using potency factors taken from previously published studies on dose-response models.” (at p. 344)

“The results of the present study showed that the lifetime mortality risk for lung cancer and mesothelioma combined varied between 1.4 and 4.9 per 100,000 persons continuously exposed to asbestos for 80 years, depending on the statistical descriptors considered (BE or UB) for the population-specific potency factors. These numbers slightly exceed Health’s Canada threshold for considering a lifetime cancer risk as negligible (i.e. 1 per 100,000) (Health Canada, 2010).” (at p. 344)

“In our evaluation exercise (see Methods), the population-specific potency factors taken from Berman and Crump (2008a) better reflected the past cancer risk in the mining area described by Camus et al. (1998, 2002), and more so for mesothelioma. Thus, these potency factors appeared more adequate for the purpose of our prospective cancer risk assessment as well.” (at p. 344)

“The originality of this study resides in the fact that it consists of a ‘population-specific’ asbestos risk assessment in which the predictive value of the risk estimates used was assessed rather satisfactorily. This allowed the determination of lung cancer and mesothelioma risk that is close to
1 per 100,000. Such population-specific analysis could facilitate risk management decisions by the relevant authorities in view of other risk management considerations that could be accounted for.” (at p. 345)

<p>| 107. | | “The findings indicate an increased incidence of pleural mesothelioma in the neighbourhood of asbestos-cement plant, and a possible etiological contribution of asbestos environmental exposure in detected risks.” (at p. 322) |
| 108. | | “The present study provides the estimation of pleural malignant mesothelioma incidence in a territory close to a national priority contaminated site, that included an asbestos cement plant.” (at p. 326) |
| 109. | | “The incidence rates of pleural malignant mesothelioma in the study area (5.9 among males and 1.9 in females x 100 000 inhabitants) were higher than the corresponding Italian national rates (3.49 for men and 1.25 for women per 100 000 inhabitants, in 2004, with a wide regional variability).” (at p. 326) |
| | | “The present study detected an increased incidence of pleural mesothelioma in the study area, with respect to the Regional and Municipal rates; the limitation of information about individual asbestos exposure, due to the low interview rate in COR database, does not allow to exclude a role of occupational exposure in the increases of mesothelioma incidence in the study area. The excess incidence of mesothelioma in the study area, anyhow, is present also in the analysis excluding cases with documented occupational exposure to asbestos. The observed increased IRR in the population below 55 resident in the subarea closer to the asbestos factory might indirectly support the notion of environmental exposure early in life.” (at p. 326) |</p>
<table>
<thead>
<tr>
<th>No.</th>
<th>Cite</th>
<th>Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>110.</td>
<td>[II] Langhoff, M., Kragh-Thomsen, M., Stanislaus, S., Weinreich, U., “Almost half of women with malignant mesothelioma were exposed to asbestos at home through their husbands or sons,” Dan Med 61(9):4902, 2014.</td>
<td>“This was a retrospective study in women with MM of the pleura. A total of 30 women were diagnosed with and treated for MM in Northern Jutland from 1996 to 2012. In all, 24 women were included.” (at p. 1)</td>
</tr>
<tr>
<td>111.</td>
<td></td>
<td>“A total of 12.5% of the study population were primarily exposed to asbestos. 46% had domestic exposure to asbestos through their husbands or sons.” (at p. 1)</td>
</tr>
<tr>
<td>112.</td>
<td></td>
<td>“Nearly 50% of the women affected by MM have been domestically exposed to asbestos through first degree relatives.” (at p. 1)</td>
</tr>
<tr>
<td>113.</td>
<td></td>
<td>“One of the main risk factors for people without occupational exposure is living with a high-risk worker, referred to as domestic exposure in this study.” (at p. 1)</td>
</tr>
<tr>
<td>114.</td>
<td></td>
<td>“A total of 13% (3/24) of the study population had primary exposure to asbestos. In all, 46% (11/24) of the women had domestic exposure to asbestos through their husbands, fathers or sons who worked with asbestos. Previously, the city of Aalborg had large industries with occupational exposure to asbestos. Many men worked at concrete and cement factories, and at shipyards. Furthermore, some men worked in isolation and plumbing businesses and as carpenters. In five cases, there were no known exposure to be found, and another five women had no information on asbestos exposure listed in their case records. Hospital records on occupational history showed three cases of employment in industries where asbestos was used, seven women had no information on their occupation and 14 women had occupations where asbestos exposure was very unlikely, e.g. cleaning, health personal, office assistants and childcare.” (at p. 2)</td>
</tr>
</tbody>
</table>
| 115. |  | “The high number of patients with domestic exposure may, of course, be explained by the very high degree of occupational exposure that has
<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>previously characterised this region.” (at p. 3)</td>
<td><strong>116.</strong></td>
<td>“In a larger female MM study population from Northern Jutland, significant differences in the female MM incidence rate may have appeared.” (at p. 3)</td>
</tr>
<tr>
<td>“The heavy occupational exposure is, however, reflected clearly in the domestic exposure of the women in this study population, given that 46% of the women included had secondary exposure through their father, husband or son.” (at p. 3)</td>
<td><strong>117.</strong></td>
<td>“In this retrospective paper, only 24 women with MM were included, which is a limitation to the study.” (at p. 4)</td>
</tr>
<tr>
<td>“We identified 147 MM cases (17.45 expected), 138 pleural and nine peritoneal, attributable to exposure to asbestos from the factory. Thirty-eight cases had past occupational exposure at the factory (2.33 expected), numbering 32 men (26 pleural, six peritoneal) and six women (four pleural, two peritoneal). In the families of the workers, there were 37 MM cases (4.23 expected), numbering five men (all pleural) and 32 women (31 pleural, one peritoneal). Among residents in Broni or in the adjacent/surrounding towns, there were 72 cases of pleural MM (10.89 expected), numbering 23 men and 49 women.” (at p. 191)</td>
<td><strong>120.</strong></td>
<td>“The largest MM burden was among women, from non-occupational exposure. Almost half of the MM cases were attributable to environmental exposure.” (at p. 191)</td>
</tr>
</tbody>
</table>
122. “Broni is a small town (<10,000 inhabitants) in the Province of Pavia in Lombardy, north-west Italy, where an asbestos cement factory (Fibronit) has been operating, employing about 2741 men and 714 women overall (Oddone et al., 2014). It was the second oldest and largest asbestos cement factory in Italy (Mirabelli et al., 2010), covering an area of 135,000 m² about 600 m west from the historical centre of the town. It produced cement in 1919–1931 and asbestos cement products in 1932–1993: Portland cement 325 was mixed with chrysotile and crocidolite, and small quantities of amosite were added to produce asbestos cement pipes and sheets. The percentage in weight of crocidolite was 10–15% in sheets and could be 30% or more in pipes.” (at p. 192)

123. “In this study we found 147 MM cases (138 pleural, nine peritoneal) between 2000 and 2011 attributable to occupational, familial, or environmental asbestos exposure from the asbestos cement factory in Broni, with an overall excess of about 130 cases above the regional average for non-exposed people. The absolute impact from occupational exposure was greatest in men, but the overall MM burden (excess cases) was higher in women.” (at p. 195)

124. “Men and women with environmental exposure in Broni had comparable relative risks (SIR 11.2 and 14.8, respectively), but in absolute terms there were more MM cases among women (28 against 20 in men). In the adjacent towns, women had higher relative risks (SIR 10.8) than men (SIR 1.3) and also a higher absolute MM burden (17 cases versus two in men). The largest contribution came from the town of Stradella (14 cases in women against two in men). There are at least two explanations for these gender differences. First, Stradella and Broni are by far the most populated towns. Second, men were in general much more frequently employed in industry/occupations involving asbestos exposure.” (at p. 195)
| 125. | “This study documents the large impact of an asbestos cement factory in Broni, Italy on MM incidence in the area, where there were about 130 excess cases in 2000–2011. In absolute terms, women suffered a greater MM burden than men, mainly because of diffuse familial and environmental exposure. This study underlines the importance of assessing the impact of asbestos exposure not only among workers, but also among their family members and in the community at large. In fact, approximately half of the MM cases were attributable to environmental exposure, a quarter to occupational exposure, and a quarter to familial exposure.” (at p. 198) |
(C) State-by-State Survey of "Take Home" Asbestos & Toxic Tort Exposure Cases by Craig T. Liljestrand, Hinshaw & Culbertson LLP (2016)


(E) Outline of Suggested Exposure Questions from an IH

(F) Key Questions to Ask Household Members and/or Workers Allegedly Carrying Asbestos Home
State-By-State Survey of "Take-Home" Asbestos & Toxic Tort Exposure Cases

Craig T. Liljestrand
312-704-3647
cliljestrand@hinshawlaw.com

© 2016 Hinshaw & Culbertson LLP, all rights reserved.
Alabama

Duty – Yes

2015 WL 693609 (N.D. Ala. 2015)
Alaska
Duty – ?
No Reported Cases

Arizona
Duty – ?
No Reported Cases

Arkansas
Duty – ?
No Reported Cases
California

Duty – No


Gregory C & Christian Calv al

Can et al

Duty – Yes


Duty – No

172 Cal. Rptr. 3d 771 (Ct. App. 2014), review filed (July 15, 2014)

331 P.3d 179 (Cal. 2014)

Duty – No

noncitable (Apr. 16, 2010)
Duty – Maybe


Duty – No


Can `bell,s u pa,

Duty – No


Can `bell, Rew land

Duty – Maybe

Duty – Yes

2013 WL 8103803 (Cal.Super.)

Can pbell

Can pbell

Can pbell

Duty – Maybe

, 2008 WL 8957253

Rw land

Duty – Maybe

2014 WL 1246400 (Cal.Super.)

Can pbell

citing

Rw land
Duty – Maybe

, 2012 WL 10646823 (Cal.Super.)

Duty – Maybe

, 2012 WL 10677931 (Cal.Super.)

do Co.

Duty – No

, 2013 WL 6148795 (Cal.Super.)

Duty – No

101 Cal. Rptr. 3d 867 (Cal. Ct. App. 2d Dist. Div. 8 2009)
Colorado

Duty – ?
No Reported Cases

Connecticut

Duty – Maybe

,, 2015 WL 4380102 (Not reported, Superior Court of Connecticut, Judicial District of Fairfield)
Duty – No

District of Columbia

Duty -- ?

No Reported Cases
Florida

Duty – ?

No Reported Cases on the issue of duty, but...
Georgia

Duty – No

608 S.E.2d 208 (Ga. 2005)

Duty – Yes, for manufacturers (but not employers)

773 S.E.2d 859 (Ga. 2015)
Hawaii

Duty – ?
No Reported Cases

Idaho

Duty – ?
No Reported Cases
Illinois

Duty — Yes

401 Ill. App. 3d 1109, 929 N.E.2d 1257 (2010)
   2012 Ill. 110662, 965 N.E.2d 1092

Duty — No

2011 Ill. App (4th) 100463, 957 N.E.2d 107

Duty — No

2011 Ill. App (4th) 100462, 955 N.E.2d 1173

Duty — No, not under a premises liability theory

909 N.E.2d 931 (2009)
Indiana

Duty – Yes

N.E.2d 974 (Ind.2002)
Iowa

Duty – No

777 N.W.2d 689 (Iowa 2009)
Kansas

Duty – No

Kentucky

Duty – No

CIV. A. 02-201-DLB, 2007 WL 2682064 (E.D. Ky. Sept. 5, 2007)
561 F.3d 439 (6th Cir. 2009)
Duty – Yes

2005-1511 (La. App. 4 Cir. 12/20/06), 947 So. 2d 171
2007-0411 (La. 4/5/07), 954 So. 2d 145

Duty – Yes

2003-0658 (La. App. 4 Cir. 6/8/05), 905 So. 2d 465
(La. 3/17/06), 925 So. 2d 538

Duty – Yes

2010 WL 4340019

Duty – Maybe

2012-1397 (La. App. 4 Cir. 5/8/13), 116 So. 3d 858, 869
2013-1321 (La. 9/20/13), 123 So. 3d 177
Duty – Yes


Duty –

933 So. 2d 843, 871-72 (La. App. 2006) (Tobias, J., concurring)
Maine

Duty – ?

No Reported Cases
Maryland

Duty – Yes

433 Md. 137, 142, 70 A.3d 328, 330 (2013)

Duty – No


Duty – No


Duty – No

879 A.2d 1088, 1096-97 (Md. 2005)
Massachusetts

Duty – ?

No Reported Cases
Michigan

Duty - No

Mich. 498, 740 N.W.2d 206 (July 2007)
Minnesota

Duty – ?
No Reported Cases

Mississippi

Duty – ?
No Reported Cases
Missouri

Duty – ?

2003 WL 25279798

Duty – Maybe

2014 WL 4654964 (Mo.Cir.), 2 (applying Wisconsin law)

Duty – Maybe

2014 WL 4654947 (Mo.Cir.), 2 (applying Wisconsin law)
New Jersey

Duty – Yes


Duty – Yes


by as upa
New Mexico

Duty – ?

No Reported Cases
New York

Duty – No

5 N.Y.3d 486, 806 N.Y.S 2d 146 (Oct. 2005)

Duty – Maybe

2011 WL 400198 (Sup. Ct. of N.Y.) (Trial Order)

Duty – No

815 N.Y.S.2d 815, 817 (N.Y. Sup. Ct. 2006)

Duty – No

611 N.Y.S.2d 569, 571 (N.Y. App. 2d Dist 1994)
North Carolina

Duty — ?
No Reported Cases

North Dakota

Duty — No, if defendant is a supplier and installer

Palmer v. 999 Quebec, Inc./874 N.W.2d 203 (2016)
Oklahoma

Duty – No

965 F.2d 844, 846 (10th Cir. 1992), citing 105, 765 P.2d 770, 774.

1988 OK
Oregon

Duty – Maybe

No reported cases. Oregon courts typically look to the state of Washington for guidance in asbestos cases. WA courts have found that such a duty does exist.
Pennsylvania

Duty – No

Feb. 21, 2012) (Memorandum Opinion)

Duty – No


Duty – No


Duty – No


, case number 1170 EDA 2012, in the Superior Court of Pennsylvania

HINSHAW & CULBERTSON LLP
Duty – No

40 F. Supp. 3d 734 (E.D. Pa. 2014)
Rhode Island
Duty – ?
No Reported Cases

South Carolina
Duty – ?
No Reported Cases

South Dakota
Duty – ?
No Reported Cases
Tennessee

Duty – Yes

266 S.W.3d 347 (Tenn. 2008)
Texas

Duty – No

No. 14-04-0113-CV, 2007 WL 1174447 (April 19, 2007),

Duty – No

256 S.W.3d 415 (Tex. App. 2008)

Duty – No

235 S.W.3d 456 (Tex. App. 2007)
Duty – Yes

401 S.W.3d 246, 259 n.13 (Tex. App.-Hous. 2012)
Utah
Duty – ?
No Reported Cases

Vermont
Duty – ?
No Reported Cases

Virginia
Duty – ?
No Reported Cases
Washington

Duty – Yes

(Wash.App.Div. 1), 2007 WL 2325214

Duty – Maybe

West Virginia

Duty – ?

No Reported Cases
Wyoming

Duty – ?

No Reported Cases
Federal Cases

Duty – No

, 540 F. App'x 590 (9th Cir. 2013)

Duty – No


Duty – Potentially, but in this case no duty

, 540 F. App'x 590 (9th Cir. 2013)
Duty – ?


Duty – Maybe


Duty – No


Duty – ?

Duty – Yes


Duty – No

965 F.2d 844 (10th Cir. 1992)

Duty – No

53 F.3d 1181 (10th Cir. 1995)

HINSHAW & CULBERTSON LLP

Duty – No

74 F.3d 1248 (10th Cir. 1996)

Duty – No


 adopted

cost denied

Duty – No
Duty – No


Duty – No

No. 15 C 10507 (N.D. Ill. 2016)

#### Table 4. Domestic exposure studies.

<table>
<thead>
<tr>
<th>Author and Year</th>
<th>Population or Task Studied</th>
<th>Asbestos Fiber Type</th>
<th>Reported Exposure Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nicholson et al. 1980 [81]</td>
<td>Homes of chrysotile miners in Copperopolis, California and Baie Verte, Newfoundland</td>
<td>Chrysotile</td>
<td>Homes of miners: 100 to &lt; 5,000 ng/m$^3$ (approx. 0.003–0.15 f/cc$^a$) (n' = 13) Homes of non-miners (Baie Verte): 32, 45, 65 ng/m$^3$</td>
</tr>
<tr>
<td>Selikoff and Lee 1978 [82]</td>
<td>Settled dust in asbestos workers' homes</td>
<td>Amosite</td>
<td>“…small amounts of amosite were found 20–25 years later in the settled dust of asbestos workers’ houses from factory operations over the period 1941–1954, and up to 400 yards downwind in the neighboring houses of non-asbestos workers”</td>
</tr>
<tr>
<td>WHO 1986 [83]</td>
<td>Asbestos miners’ homes</td>
<td>NR</td>
<td>Residences of asbestos miners in South Africa: Mean = 0.006 f/cc (range, 0.002–0.011 f/cc); Purr-occupational range: 0.01–1.0 f/cc</td>
</tr>
</tbody>
</table>

#### Study of clothing and laundering

<table>
<thead>
<tr>
<th>Author and Year</th>
<th>Population or Task Studied</th>
<th>Asbestos Fiber Type</th>
<th>Reported Exposure Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sawyer et al. 1977 [85]</td>
<td>Asbestos abatement workers</td>
<td>Chrysotile</td>
<td>Mean of personal samples (n = 12): 0.4 f/cc (max = 1.2 f/cc) Mean of area samples: During picking up clothing (n = 4): 0.4 f/cc Loading washer (n = 5): 0.4 f/cc Loading dryer (n = 6): 0.0 f/cc</td>
</tr>
</tbody>
</table>

#### Exposure simulation studies

<table>
<thead>
<tr>
<th>Author and Year</th>
<th>Population or Task Studied</th>
<th>Asbestos Fiber Type</th>
<th>Reported Exposure Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jiang et al. 2008 [88]</td>
<td>Unpacking and repacking clutches</td>
<td>Chrysotile</td>
<td>30 min PCM$^<em>$-adjusted mean following clothing handling = 0.002 ± 0.002 f/cc (n = 4) Estimated 8 h TWA$^</em>$ = 0.0001 f/cc</td>
</tr>
<tr>
<td>Madl et al. 2008 [89]</td>
<td>Unpacking and repacking brakes</td>
<td>Chrysotile</td>
<td>30 min PCM-adjusted mean (range) following clothing handling (n = 5): 0.011 f/cc (0.002–0.015 f/cc )</td>
</tr>
</tbody>
</table>
Not all questions will apply in every case, you will have to make adjustments and may have to add follow-up questions to drill down into the details.

The bottom line is that the industrial hygienist must know (to the extent possible):

- to what the plaintiff was exposed to,
- the intensity of each exposure,
- the duration of each exposure, frequency of exposure,
- the years in which the exposures occurred,
- the proximity of the plaintiff to the source of exposure
- the proximity of the plaintiff to other sources of exposure
- how the plaintiff’s job changed over time
- how the source of exposure changed over time

**Occupational Exposures**

1. Query all jobs (list employers)
2. Dates to/from for each job
3. What tasks were performed in each job?
4. Get a detailed (step by step) description of each task performed for each job
5. If deponent is a co-worker, obtain description of the plaintiff’s jobs and tasks as indicated below.
6. Determine the general operating/environmental conditions for each job and task—
   - indoors/outdoors
   - weather conditions
   - new construction/old construction
   - conditions that existed before task(s) performed
7. What was the frequency of each task?
8. What was the duration of each task?
9. How long was an average workday? Did it change over time?
10. Percentage of time task performed (breakdown for all tasks performed)
11. What was the total time at lunch and on other breaks each day?
12. What tools/machines were used for each task
13. What products/chemicals used/handled in each task?
14. What years were each product used?
15. How were these products or chemicals used?
16. How much time each day was spent working with these products?
17. Were there any “waste” materials created or produced during the task (eg., dust, chemical, etc.)
18. Who cleaned up after tasks completed?
19. How were things cleaned up—what techniques were used (eg. dry sweeping).
20. How were jobs and tasks learned (on job training, formal training, apprentice, etc)
21. Are there formal work procedures for each task—are copies available (generally available only when deponent worked for a large company?)
22. Did the job or tasks change over time/When did the changes take place?
23. What tools and machines were used for each task?
24. What exposure controls were established for each task (ventilation, natural or mechanical, respirators clothing, etc.)
25. What was the size of the work area(s) (H x L x W)?
26. How many other workers in area performing same or similar task?
27. What other trades working in area?
28. Detailed description of other trades (to the extent that the deponent knows)
29. How much time were other trades in area?
30. What tools or machines were used by other tradesmen
31. Number of other tradesmen (performing other tasks) working in those trades at the same time deponent was in area.
32. Chemicals/products used or handled by each of the other trades
33. Was the deponent aware of exposure sampling:
   - Taken of his work?
   - Ever taken in area?
   - Ever taken of other workers?
   - Ever see the results of exposure measurements?

34. What information were you given about the products you worked with:
   - Product name?
   - That it contained asbestos?
   - The hazards of the product?
   - Ways to protect oneself—use of respirators, etc?
   - Were respirators and other PPE supplied?
   - Were respirators or other protective devices actually used when supplied?

NON-OCCUPATIONAL EXPOSURES (You may want to enhance this section by using questions from above)

35. What hobbies did you have?
   - What kinds of substances or chemicals did you use?
   - How much of these substances did you use (sq ft, lbs, gallons, etc)
   - How often did you participate in these things?
   - What years did you participate in these activities?
   - Describe the activities?
   - What tools did you use?

36. Did you do any home repair or remodeling?
   - What did you do?
   - How often?
- How much time was spent doing these repairs or remodeling?
- What products did you use?
- How much (lbs, sq ft, gallons, etc) did you use?
- Over what period of time did these repairs or occur?
- How long did each project take?
- What specific tasks were you involved with?
- Did you hire a contractor to do some or all of the work?

37. What industries did your father/mother (and others in household) work in?

38. What jobs did they hold?

39. How long did they work in each job?

40. Do you know what materials they worked with?

41. Did they come home in their work cloths?

42. Did they wear their work cloths at home?
Key Questions to Ask Household Members and/or Workers Allegedly Carrying Asbestos Home

- You and your spouse generally kept a clean and well-maintained house?
  - Who cleaned the house?
  - In keeping a clean house, did you/your spouse use a vacuum? Wet mop? How often?
  - Didn’t want to dirty the house?
  - Do the best you could to brush the dirt and dust off your clothes before you came into the house?

- Laundry
  - Did you ever use a laundry service?
  - Who did the laundry?
    - Did you help your spouse with the laundry, or was that something he/she did on his/her own?
    - Were you present when your spouse did the laundry? [How many times?] What were you doing when the laundry was done?
  - Describe layout of residence, size and rooms. [Diagram floors]
    - Did you have a laundry room in the house? Where, layout, windows, connecting rooms?
  - Describe for me the process by which you/your spouse did laundry? [Duration for each task and room]
    - Take laundry out of basket/hamper and put in washing machine?
    - [If shake clothes off first] Kept a clean house? Would shake clothes outside to avoid dirtying the house?
    - Shaking out the clothes took a matter of seconds?
  - Did you have a dryer? Use a clothes line?
  - How often did you/your spouse do laundry?
  - What did you/your spouse wear to work?
• Uniform?

• Did your work have changing facilities? Did you/your spouse ever change out of work clothes at the beginning/end of the work day?

• Did your work have a laundry service? Did you use it?

• What did your clothes look like at the end of the day?
  ▪ You kept a clean home, so you would try to avoid trekking in dust and dirt?

• Did you ever visit your spouse at work?
  ▪ How often?
  ▪ Was your spouse working when you visited him/her?
  ▪ What work was he/she performing?