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REVIEW ARTICLE

Evaluation of take home (para-occupational) exposure to asbestos and disease: a review of the literature

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Abstract

The potential for para-occupational (or "take-home") exposure to a number of chemicals has been recognized for over 60 years. 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. Ideally, a simulation study could be conducted in the future to better understand the relationships between the airborne concentrations in the workplace and the fiber characteristics that influence retention on fabric, as well as the concentrations that can be generated by handling the contaminated clothing by the persons in the home.

Keywords: Asbestos, para-occupational exposure, take-home exposure, family, fiber transport

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Introduction

Historically, asbestos in its various forms has been used in a variety of applications because of its unique physical properties (e.g. heat resistance) (Maines, 2005). Chrysotile, amosite and crocidolite asbestos were most frequently used, and were the only fiber types used in the vast majority of commercial setting applications. Chrysotile asbestos (a member of the serpentine mineral family) has been widely used in industry, particularly after the 1930s; however, shipyard industries, especially during World War II, extensively used amosite asbestos (an amphibole) (Balzer & Cooper, 1968; Bowles & Barsigian, 1951; Bowles & Stoddard, 1933; Virta, 2005). Despite the brief increase in amosite use associated with the war effort, the vast majority of asbestos used in the United States was chrysotile, mostly from Canada (Virta, 2006). Crocidolite was sometimes used in cements and building materials, in gaskets used in highly acidic environments, and occasionally in phenolic molding compounds. Because asbestos has been used in different applications in so many industries, workers producing, handling, or using asbestos-containing materials in these industries (i.e., manufacturing, construction, maritime) have had numerous opportunities to become exposed to it (Maines, 2005; OSHA, 1994).

As has been previously described in the published literature, knowledge regarding the health hazards posed by asbestos evolved considerably throughout the 20th century (Craighead & Gibbs, 2008; Paustenbach et al., 2004). Asbestos-related disease, specifically asbestosis, was initially discovered in settings in which large amounts of raw asbestos fibers were used and processed (Merewether & Price, 1930). With few exceptions, workers evaluated in the studies from the 1930s worked in mining or manufacturing settings, some of the dustiest environments at the time. By the mid-1940s, the focus of many studies expanded to include individuals who used asbestos-containing products. One of the most important worker groups studied during this time were those in the shipbuilding industry, which extensively used asbestos in pipe covering and insulation (Fleischer et al., 1946). Exposures in this industry were found to be high, particularly because large amounts of asbestos were often used in areas with limited space or ventilation (Marr, 1964). Recognition of insulation work hazards in the shipyard industry eventually broadened to include other trades that frequently used insulation, such as construction (Maines, 2005).

As the knowledge of the health hazards of asbestos expanded and asbestos-related diseases increased, case reports emerged describing asbestos diseases in members of households in which occupationally exposed persons lived. These types of cases were often thought to be a result of "secondary" exposure to asbestos, also referred to as "domestic," "family," "take home," or "para-occupational" exposure (Anderson, 1982; Anderson et al., 1976; 1979; Nicholson et al., 1980; Wagner et al., 1960). Para-occupational or "take home" exposure can be described as exposure to workplace chemicals that occurs in the worker's home. In the case of asbestos, such exposure is generally due to dust that has accumulated on the worker's clothing, shoes, or hair that is later inadvertently brought into the home. Thus, members of an asbestos worker's household could have been and were occasionally exposed to asbestos through physical contact with the worker or laundering his or her clothing.

The potential for para-occupational exposure to particles has been recognized for more than 60 years. Family member exposures have been identified among those working with a variety of dusts or fumes, including beryllium, asbestos, lead, arsenic, mercury, pesticides, pharmaceuticals and radionuclides (Aguilar-Garduno et al., 2003; Bradman et al., 2009; Curl et al., 2002; Hollins et al., 2009; NIOSH, 1995; Rao et al., 2006; Zirschky, 1996). With respect to asbestos, 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. Individual case reports continued throughout the 1960s, but it was not until the late 1970s that a sizeable cohort of family members of amosite asbestos factory workers was evaluated by researchers at the Mt. Sinai School of Medicine (Anderson, 1982; Anderson et al., 1976; 1979).

In the years that followed these studies, various additional published case reports and case control studies, as well as a few additional cohort studies, further identified disease in some family members of workers in industries with high exposure potential, where they were nearly always exposed to amphibole asbestos. As is discussed in some of the many studies involving take home asbestos exposures, the fiber type to which the worker was

exposed is an important consideration, since significant differences in mesothelioma potency exist among the various fiber types (and the differences could be even greater when fiber length is considered). For example, it has been reported that crocidolite may be 200–500 times more potent than chrysotile at inducing mesothelioma (if chrysotile alone can produce the disease) (Berman & Crump, 2008a, 2008b; Hodgson & Darnton, 2000; Pierce et al., 2008).

In the early 1990s, as a result of continued reports of contamination in workers' homes for a number of dusts (particles), two identical bills were proposed to Congress in the United States to study the issue. One of these bills was revised and eventually incorporated into the Fire Administration Authorization Act of 1992, as section 209, the "Workers' Family Protection Act" (NIOSH 1995). This Act included a directive for the National Institute for Occupational Safety and Health (NIOSH) to conduct a study on worker home contamination in cooperation with the Secretary of Labor, the Administrator of the Environmental Protection Agency (EPA), the Administrator of the Agency for Toxic Substances and Disease Registry (ATSDR) and other government agencies. The purpose of the study was to review past home contamination incidents as reported in the published literature or in governmental records for a variety of chemicals, and to evaluate the regulatory, statutory, and industrial hygiene measures being used by employers to prevent or remediate home contamination. Asbestos was one of approximately 15 chemicals (or chemical groups) that NIOSH specifically studied.

Based on its review of the available literature, NIOSH reported an increased risk of mesothelioma (pleural, pericardial, peritoneal), lung cancer, cancer of the gastrointestinal tract, non-malignant pleural and parenchymal abnormalities, and asbestosis among families of asbestos-exposed workers, citing a number of case reports, cohort studies, case-control studies, and a community survey (NIOSH, 1995). While the NIOSH report presented general information regarding the occupation(s) of the primary workers exposed to asbestos, the majority of the studies did not provide detailed quantitative information regarding the airborne asbestos concentrations experienced by those occupationally exposed, nor did they provide any estimates of airborne concentrations in the home due to the presence of asbestos-contaminated clothing or activities, such as laundering. Additionally, unless fiber type was specifically noted in the underlying literature such as in the case of amosite asbestos use in thermal insulation manufacturing (e.g. Anderson et al., 1976, 1982), asbestos fiber type was not directly addressed in the NIOSH document with respect to conclusions or recommendations. When asbestos fiber type was not mentioned, NIOSH simply indicated that it was "asbestos" or "asbestos dust." The majority of the primary workers, however, worked in industries in which exposures to airborne asbestos could be high, such as manufacturing, insulating, or shipbuilding. In many cases, the primary worker was noted to be an "asbestos worker," but additional details regarding possible airborne asbestos concentrations at the worksite were typically unavailable.

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 (e.g. workers cutting or tearing out insulation or handling raw asbestos) (Figures 1 and 2). There is no doubt that in earlier years, some workers went home with appreciable visible quantities of asbestos dust on their clothing; for example, the famous "snowmen of Grand Central Station," who received this name due to the large quantities of asbestos-containing dust on their clothing at the end of their work day (Metro North Commuter Railroad Company v. Michael Buckley (96–320), 521 U.S. 424, 1997; Gross, 1997). While it is possible to characterize the universe of

Figure 1. Worker cutting half-round using a band saw with no local exhaust ventilation. Photo Source: Carl Mangold. Previously published in Hollins et al., 2009.

Figure 2. Worker shaping a half-round with a knife, no protective clothing. Photo Source: Carl Mangold. Previously published in Hollins et al., 2009.

the plausible exposures of these workers based on historical knowledge of airborne asbestos concentrations in industry over time, there is little corresponding quantitative information regarding exposure levels experienced by their household contacts (Revell, 2002; Sawyer, 1977). Furthermore, even in two workplaces with identical airborne asbestos concentrations, the likelihood that the worker's clothing would be contaminated was dependent on the tasks that they performed using asbestos containing materials, as well as the duration. Those who removed old insulation with a hammer and installed insulation using equipment such as band saws, circular saws, and other mechanical devices, for example, had asbestos fibers directly projected onto their clothing, in addition to the ambient fibers that settled on their clothing (Hollins et al., 2009) (Figure 3). Conversely, workers may also have taken measures such as blowing themselves off with a high pressure air hose prior to leaving the work area, which could have substantially reduced the potential for fibers to be brought into the home.

In recent years, the number of legal cases involving alleged para-occupational or "take home" asbestos exposure has increased dramatically. Yet there are few, if any, available data that are useful for understanding the relationship between airborne asbestos concentrations in an occupational environment and the corresponding concentrations in the home associated with, for example, handling clothing that was contaminated in that environment. Furthermore, there are vast differences between the work conditions and resulting airborne concentrations of asbestos between 1930 and the 1960s and those experienced by workers after the creation of the Occupational Health and Safety Administration (OSHA) in 1970. The Occupational Health and Safety Act set forth the first asbestos exposure regulations in 1971, including the permissible exposure limit (PEL) for asbestos to 5 f/cc (OSHA, 1971a, 1971b). As knowledge regarding asbestos health hazards evolved, regulatory actions followed, further limiting exposures in the workplace. In 1972, OSHA began requiring change rooms for industries and locations that were in excess of exposure limits (OSHA, 1972). Such rooms included lockers or containers for storing street clothing to protect it from work clothing and uniforms. As stated in the regulation, "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). 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 exposures limits") (OSHA, 1972, p. 11321). These practices would have had a significant impact on the potential for take home exposures.

Figure 3. Work area of insulator sawing pipe insulation on a table. Photo Source: Carl Mangold. Previously published in Hollins et al., 2009.

Ultimately, exposure science and epidemiology studies will be necessary in order to properly characterize the take home risks of chrysotile and amphibole asbestos, since even for mesothelioma, studies have shown that this disease can also occur in persons not exposed to asbestos (Powers & Carbone, 2002; Price & Ware, 2004, 2009; Teta et al., 2008; Antman et al., 1997; Chahinian et al., 1982; Gibbs et al., 1989; Huncharek, 2002; Ilgren & Wagner, 1991; McDonald, 1985; McDonald & McDonald, 1994; Peterson et al., 1984; Powers & Carbone, 2002; Price & Ware, 2004, 2009; Roggli et al., 1992; Walker et al., 1983). Several etiological factors for mesothelioma other than asbestos have been identified over the past few decades, including therapeutic radiation, non-asbestos mineral fibers (e.g. zeolite, erionite), viruses, chronic inflammation, and genetic predisposition (Antman et al., 1997; Huncharek, 2002; Moore et al., 2008; Powers & Carbone, 2002; Teta et al., 2008). These factors may act independently or as co-carcinogens to induce mesothelioma. Recent articles have reported that 300–600 mesothelioma cases annually in the United States are likely unrelated to asbestos exposure (Powers & Carbone, 2002; Teta et al., 2008). Data from the National Cancer Institute's Surveillance, Epidemiology and End-Results (SEER) registry have been used in several recent publications to estimate background rates and projected rates of mesothelioma (Moolgavkar et al., 2009; Teta et al., 2008; Weill et al., 2004). Moolgavkar et al. (2009) estimated that the background rate of pleural mesothelioma in the US is between two and three cases per million individuals per year for all age groups combined. Thus, 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.

The purpose of this analysis, then, is to evaluate the published literature in order to characterize the reported cases of asbestos-related disease among household contacts of asbestos workers. A specific attempt was made to quantitatively understand the exposures that could occur among household contacts, when data were available. To

the extent possible, this analysis evaluates the occurrence of disease among household contacts as reported by the original authors. We did not perform a case-by-case analysis of the likelihood that the disease was truly due to the para-occupational exposure to asbestos, or some other factor such as a spontaneous tumor, radiation therapy, or unreported occupational exposure. Unless it was explicitly stated in the original article that a household case had another source of asbestos exposure, we did not exclude any cases from our review. We also grouped the cases by industry, which can provide information regarding the type of asbestos exposures that were likely experienced by the primary workers. Finally, our review also identifies and discusses papers that address fiber adhesion and/or re-suspension from clothing surfaces, as well as those that present more general models that discuss the adhesion or resuspension of particles from solid surfaces that could potentially be adjusted to account for fiber characteristics.

Methods

Literature search

We conducted a comprehensive search of publically available documents that directly or indirectly discussed disease in household contacts of persons who worked with asbestos occupationally. Several database search engines (e.g. PubMed, ToxNet) were used to identify relevant reports, literature, or conference proceedings. Government documents, such as NIOSH reviews and Human Health Evaluation (HHE) reports, and industrial hygiene textbooks were also considered. This search also included a review of published master's and Ph.D. theses, as well as, documents published in countries outside the United States. Various search terms were utilized in order to locate all pertinent literature and information, including: asbestos and "para-occupational," "take home," "family," "home," "household," "domestic," "secondary," "indirect," "non-occupational," "wife," "children," "clothing," and "laundry," among others. Some unpublished reports were obtained from other professionals, and efforts were made to locate unpublished studies, such as those conducted by corporations or universities. The review was limited to literature in English, unless translations from other languages were available.

Using the same search terms described above, a targeted review was also conducted of the Claims Resolution Management Corporation (CRMC) Asbestos Claims Research Facility in Aurora, Colorado, which houses a collection of more than 32,000 boxes of documents, 7200 rolls of microfilm, and 5000 subject-related and witness files turned over to the facility by the Johns-Manville (JM) Corporation. The search results yielded many different types of documents, including scientific articles published in the peer-reviewed literature, product specifications, US Navy memoranda, safety handbooks, internal JM documents, and other surveys, reports, and letters. Approximately 80 boxes of health, safety, and environment documents, as well as two boxes of microfilm files, each containing 42 rolls of film, were reviewed in order to locate pertinent information. This search, however, did not result in any new information that was not already publically available.

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. Environmental or neighborhood exposures in areas where asbestos was widely used were not specifically considered, although, occasionally, these exposures were thought to also contribute to cases of disease. 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.

Publications and reports were considered relevant when they included reports of disease thought to be caused by para-occupational exposure to asbestos from occupational sources or any data that might provide insight on quantitative estimates of para-occupational asbestos exposure. Because of the scarcity of quantitative information, no effort was made to restrict the search based on sample type (i.e. personal or area sampling), or the analytical method used (i.e. phase-contrast microscopy [PCM] or transmission electron microscopy [TEM]). Similarly, we maintained no inclusion criteria for sample duration. Throughout our review of cases of disease believed to be associated with para-occupational exposure, we made an effort to determine the disease diagnosis, as well as the household contact's occupation or category of industry, the time period when exposure occurred, and the fiber type with which the primary contact worked, when possible.

Results

Evolution of the reporting of para-occupational cases

A summary of the published studies of asbestos-related disease among household contacts, some of which are described in more detail below, is presented in Table 1. Table 2 provides a breakdown of the cases by industry group and disease status.

1960s

During the 1960s, the first case reports emerged indicating a possible risk of asbestos-related disease among persons who had grown up in asbestos mining or manufacturing areas, as well as among family members of workers at asbestos manufacturing or mining facilities (Newhouse & Thompson, 1965; Wagner et al., 1960). 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

Table 1. Summary of studies reporting disease thought to be caused by para-occupational exposure.

Industry Group	Study	Location	No. of Cases	Diagnosis	Disease Confirmed?	Family Member Occupation	Fiber Type	Time Period of Exposure or Diagnosis Date
Cement Factory	Magnani et al. 2000	Casale, Turin, Florence, Barcelona, Cadiz, Geneva (Italy)	4	Pleural mesothelioma	No	Asbestos industry worker	NR	Diagnosed in 1995
	Magnani et al. 2001	Casale Monferrato, Italy	23	Pleural mesothelioma	No	Asbestos cement factory workers	Crocidolite and chrysotile	Diagnosed in 1995
	Magnani et al. 1993; Ferrante et al. 2007	Casale Monferrato, Italy	12	Malignant neoplasm of the lung	No	Asbestos cement factory workers	Crocidolite and chrysotile	Exposed during 1907–1985 (plant operation)
	Magnani et al. 1993; Ferrante et al. 2007	Casale Monferrato, Italy	3	Malignant neoplasm of the peritoneum	No	Asbestos cement factory workers	Crocidolite and chrysotile	Exposed during 1907–1985 (plant operation)
	Magnani et al. 1993; Ferrante et al. 2007	Casale Monferrato, Italy	21	Malignant neoplasm of the pleura	No	Asbestos cement factory workers	Crocidolite and chrysotile	Exposed during 1907–1985 (plant operation)
	Ampleford and Ohar 2007	NR	1	Pleural mesothelioma	Yes	Cement manufacturing worker	Crocidolite	Exposed 1939–1967
	Peretz et al. 2009	Petach Tikvah, Israel	2	Bilateral thickening/ pleural plaques	X-ray	Raw asbestos debagger in an asbestos cement plant	Crocidolite and chrysotile	Exposed 1952–1962
Other Factory	Newhouse and Thompson 1965	London	1	Peritoneal mesothelioma	Yes	Asbestos factory foreman	NR	Exposed 1921–1942
	Newhouse and Thompson 1965	London	1	Pleural mesothelioma	Yes	Asbestos factory spinner	NR	Exposed 1928–1930
	Newhouse and Thompson 1965	London	1	Pleural mesothelioma	Yes	Asbestos factory worker	NR	Exposed 1925–1936
	Newhouse and Thompson 1965	London	1	Pleural mesothelioma	Yes	Asbestos factory worker	NR	Exposed 1919–1921
	Newhouse and Thompson 1965	London	1	Pleural mesothelioma	Yes	Asbestos factory worker	NR	Exposed 1941–1946
	Rusby 1968	NR	1	Pleural mesothelioma	NR	Asbestos factory worker	NR	NR
	Milne 1969	Victoria, Australia	1	Pleural mesothelioma	Yes	Asbestos factory worker	NR	Exposed in 1930
	Von Bittersohl and Ose 1971	Kreis Merseburg, Germany	1	Pleural mesothelioma	Yes	Watchman at an asbestos factory	"Serpentine" and "blue" asbestos	NR
	Navratil and Trippe 1972	Czechoslovakia	4	Pleural calcifications	X-ray	Asbestos plant workers	Chrysotile	NR
	Knappman 1972	Hamburg, Germany	1	Pleural mesothelioma	Yes	Asbestos factory worker	NR	Exposed approximately 1920–1930

(Continued)

Table 1. *(Continued)*.

Industry Group	Study	Location	No. of Cases	Diagnosis	Disease Confirmed?	Family Member Occupation	Fiber Type	Time Period of Exposure or Diagnosis Date
	Greenberg and Davies 1974	England, Scotland, Wales	1	Mesothelioma	No	Asbestos factory worker	NR	Exposed for two unspecified years
	Whitwell et al. 1977	England	1	Mesothelioma	Yes	Gas-mask factory worker	Crocidolite	NR
	Vianna and Polan 1978	New York, USA	1	Pleural mesothelioma	Yes	Asbestos factory worker	NR	Exposed for 5 years
	Vianna and Polan 1978	New York, USA	1	Pleural mesothelioma	Yes	Brake-lining worker	NR	Exposed for 15 years
	Vianna and Polan 1978	New York, USA	1	Pleural mesothelioma	Yes	Brake-lining worker	NR	Exposed for 10 years
	Epler 1980	Boston, MA	1	Pleural plaques, calcification, benign asbestos pleural effusion and fibrosis	X-ray	Asbestos factory worker	NR	Exposed 1939–1967
	McDonald and McDonald 1970; 1973; 1980	USA, Canada	3	Malignant mesothelioma	Yes	Asbestos production worker	Chrysotile	NR
	Epler 1980	Boston, MA	1	Pleural mesothelioma	Yes	Asbestos factory worker	NR	Diagnosed in 1973
	Anderson et al. 1976, 1979, 1982[a]	New Jersey, USA	239	Small opacities and/or pleural abnormalities	X-ray	Asbestos factory worker	Amosite	Exposed 1941–1954
	Kane et al. 1990	New York, USA	1	Pleural mesothelioma	Yes	Asbestos and glass factory worker	NR	Diagnosed between 1974 and 1986
	Kane et al. 1990	New York, USA	1	Pleural mesothelioma	Yes	Asbestos plant worker	NR	Diagnosed between 1974 and 1986
	Joubert et al. 1991[a]	New Jersey, USA	12	Lung cancer	No	Insulation factory worker	Amosite	NR
	Joubert et al. 1991[a]	New Jersey, USA	4	Pleural mesothelioma	No	Asbestos factory worker	Amosite	Exposed 1941–1954
	Schneider et al. 1996	Germany	1	Pleural mesothelioma	Yes	Asbestos cardboard worker	NR	Exposed 1969–1976
	Roggli et al. 1997	NR	1	Pleural mesothelioma	Yes	Asbestos plant manufacturer	NR	NR
	Roggli et al. 1997	NR	1	Pleural mesothelioma	Yes	Papermaker	NR	NR
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Asbestos product manufacturer	NR	Exposed 1935–1955
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Manufactured asbestos product	NR	Exposed 1930–1949
	Whitehouse et al. 2008	Montana, USA	1	Pleural mesothelioma	Yes	W.R. Grace Zonolite Facility	Tremolite	Exposed 1963–1965

(Continued)

Table 1. *(Continued)*.

Industry Group	Study	Location	No. of Cases	Diagnosis	Disease Confirmed?	Family Member Occupation	Fiber Type	Time Period of Exposure or Diagnosis Date
Dock or Shipyard	Newhouse and Thompson 1965	London	1	Pleural mesothelioma	Yes	Dock laborer	NR	Exposed 1930–1934
	Newhouse and Thompson 1965	London	1	Pleural mesothelioma	Yes	Shipyard engine room worker	NR	Exposed 1919–1960
	Lieben and Pistawka 1967	Pennsylvania, USA	1	Peritoneal mesothelioma	Yes	Shipyard insulators	NR	Diagnosed between 1958 and 1963
	McEwen et al. 1971	Scotland	1	Mesothelioma	Yes	Dock laborer	"Blue and white" asbestos	Diagnosed between 1950 and 1967
	Li et al. 1978	NR	2	Pleural mesothelioma	Yes	Shipyard insulator	NR	Exposed 1940–1965
	Edge and Choudhury 1978	Barrow-in-Furness, UK	1	Pleural mesothelioma	Yes	Shipyard plumber	Crocidolite	Diagnosed between 1966–1976
	Bianchi et al. 1981	Monfalcone, Italy	13	Pleural plaques and/or asbestos bodies	Yes	Shipyard worker	NR	Necropsy performed 1979–1980
	Kilburn et al. 1985; 1986	Los Angeles, CA	40	Asbestosis	X-ray	Shipyard worker	NR	Diagnosed in 1981
	Hammar 1989	Vancouver, WA	1	Peritoneal mesothelioma	Yes	Shipyard worker	NR	Exposed in early 1940s
	Huncharek et al. 1989	Canada	1	Pleural mesothelioma	Yes	Shipyard machinist	NR	Exposed 1935–1969
	Kane et al. 1990	New York, USA	1	Pleural mesothelioma	Yes	Shipyard insulator	NR	Diagnosed between 1974 and 1986
	Roggli and Longo 1991	NR	1	Pleural mesothelioma	Yes	Shipyard Insulator	Amphibole detected in lungs	NR
	Roggli and Longo 1991	NR	1	Pleural mesothelioma	Yes	Shipyard worker	Amphibole detected in lungs	NR
	Dodoli et al. 1992	Leghorn and La Spezia, Italy	9	Pleural mesothelioma	No	Shipyard worker	NR	Died between 1975–1988
	Roggli et al. 1997	NR	3	Peritoneal mesothelioma	Yes	Shipyard worker	Commercial and non-commercial amphiboles	NR
	Roggli et al. 1997	NR	5	Pleural mesothelioma	Yes	Shipyard worker, merchant marine	Commercial and non-commercial amphiboles	NR
	Magnani et al. 2000	Casale, Turin, Florence, Barcelona, Cadiz, Geneva (Italy)	2	Pleural mesothelioma	No	Shipyard worker	NR	Diagnosed in 1995 and 1996
	Miller 2005	NR	1	Peritoneal mesothelioma	Yes	Shipyard worker	NR	Exposed 1941–1947

(Continued)

Table 1. *(Continued).*

Industry Group	Study	Location	No. of Cases	Diagnosis	Disease Confirmed?	Family Member Occupation	Fiber Type	Time Period of Exposure or Diagnosis Date
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker	NR	Exposed 1942–1950
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker	NR	Exposed 1942–1982
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker	NR	Exposed 1943–1944
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker	NR	Exposed 1951–1957
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker	NR	Exposed 1941–1946
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker	NR	Exposed 1955–1975
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker	NR	Exposed 1942–1945
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker	NR	Exposed 1942–1945
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker	NR	Exposed 1935–1963
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker	NR	Exposed 1941–1944
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker	NR	Exposed 1941–1965
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Shipyard worker/ boilermaker	NR	Exposed 1942–1963 (SY), 1962–1980 (BM)
	Peretz et al. 2009	Washington, USA	2	Calcified pleural plaques	X-ray	Shipyard carpenter/ pipe lagger	NR	NR
	Bianchi et al. 2009	Trieste-Monfalcone, Italy	34	Pleural mesothelioma	Yes	Shipyard worker	NR	Diagnosed between 1968 and 2008
Insulation	Lieben and Pistawka 1967	Pennsylvania, USA	1	Peritoneal mesothelioma	Yes	Insulation plant engineer	Amosite and chrysotile	Diagnosed between 1958 and 1963
	Lieben and Pistawka 1967	Pennsylvania, USA	1	Pleural mesothelioma	Yes	Insulation plant worker	NR	Diagnosed between 1958 and 1963
	Champion 1971	Glasgow, Scotland	1	Pleural mesothelioma	Yes	Insulator	NR	Exposed approximately 1940–1950
	Vianna and Polan 1978	New York, USA	1	Peritoneal mesothelioma	Yes	Heat insulation worker	NR	Exposed for 15 years
	Vianna and Polan 1978	New York, USA	1	Pleural mesothelioma	Yes	Heat insulation worker	NR	Exposed for 18 years

(Continued)

Table 1. *(Continued).*

Industry Group	Study	Location	No. of Cases	Diagnosis	Disease Confirmed?	Family Member Occupation	Fiber Type	Time Period of Exposure or Diagnosis Date
	Vianna and Polan 1978	New York, USA	1	Pleural mesothelioma	Yes	Heat insulation worker	NR	Exposed for 5 years
	McDonald and McDonald 1970; 1973; 1980[b]	USA, Canada	5	Malignant mesothelioma	Yes	Asbestos/insulation factory worker	NR	NR
	Martensson et al. 1984	NR	2	Pleural mesothelioma	Yes	Foundry worker	NR	Exposed in childhood
	Sider et al. 1987	Chicago, IL	18	Pleural thickening and calcification	X-ray	Insulator	NR	Exposed 1936–1963
	Roggli and Longo, 1991	NR	1	Small/large cell carcinoma	Yes	Insulator	Amphibole detected in lungs	NR
	Roggli and Longo, 1991	NR	1	Small cell carcinoma	Yes	Insulator	Amphibole detected in lungs	NR
	Roggli and Longo, 1991	NR	1	Bronchioalveolar cell carcinoma	Yes	Insulator	Amphibole detected in lungs	NR
	Roggli and Longo, 1991	NR	1	Pleural mesothelioma	Yes	Insulator	Amphibole detected in lungs	NR
	Schneider et al. 1996	Germany	2	Pleural mesothelioma	Yes	Insulation-mat manufacturer	NR	Exposed 1950–1959
	Schneider et al. 1996	Germany	1	Pleural mesothelioma	Yes	Insulator	NR	Exposed 1964–1974
	Roggli et al. 1997	NR	10	Pleural mesothelioma	Yes	Insulator	Commercial and non-commercial amphiboles	NR
	Magnani et al. 2000	Casale, Turin, Florence, Barcelona, Cadiz, Geneva (Italy)	1	Pleural mesothelioma	No	Asbestos industry working with insulation in wagons	NR	Diagnosed in 1995
	Miller 2005	NR	1	Peritoneal mesothelioma	Yes	Insulator	NR	Exposed 1954–1961
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Insulator	NR	Exposed 1956–1964
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Insulator	NR	Exposed 1947–1987
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Insulator	NR	Diagnosed in 1992
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Insulator	NR	Exposed 1955–1988
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Insulator	NR	Exposed 1953–1976
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Spray asbestos insulator	NR	Exposed 1954–1977
Mining	Wagner et al. 1960	Cape Province, South Africa	1	Pleural mesothelioma	Yes	Asbestos miner	Crocidolite	Diagnosed in 1958
	Rubino et al. 1972	Northwestern Italy	3	Pleural mesothelioma	Yes	Asbestos mining industry workers	NR	NR

(Continued)

Table 1. *(Continued)*.

Industry Group	Study	Location	No. of Cases	Diagnosis	Disease Confirmed?	Family Member Occupation	Fiber Type	Time Period of Exposure or Diagnosis Date
	Magee et al. 1986	Corsica	1	Pleural mesothelioma	Yes	Miner	Chrysotile, tremolite and actinolite	Exposed 1943–1954, intermittent after
	Reid et al. 2008	Wittenoom, Australia	26	Pleural mesothelioma	No	Miner	Crocidolite	NR
	Whitehouse et al. 2008	Montana, USA	1	Pleural mesothelioma	Yes	Zonolite miner	Tremolite	Exposed 1941–1959
	Mirabelli et al. 2008	Balangero, Italy	1	Pleural mesothelioma	Yes	Asbestos packer in a mine	Potentially contaminated chrysotile	Exposed 1948–1973
Trades - Various	Newhouse and Thompson 1965	London	1	Peritoneal mesothelioma	Yes	Railway carriage builder	NR	Exposed 1912–1930
	Newhouse and Thompson 1965	London	1	Pleural mesothelioma	Yes	Boiler coverer	NR	Exposed 1925–1939
	Heller et al. 1970	Massachusetts, USA	1	Pleural mesothelioma	Yes	Pipefitter	NR	Diagnosed between 1960 and 1967
	Vianna and Polan 1978	New York, USA	1	Pleural mesothelioma	Yes	Electric wire insulation worker	NR	Exposed for 15 years
	Vianna and Polan 1978	New York, USA	1	Pleural mesothelioma	Yes	Heat-electric wire worker	NR	Exposed for 14 years
	Vianna and Polan 1978	New York, USA	1	Pleural mesothelioma	Yes	Pipe fitter	NR	Exposed for 6 years
	Kane et al. 1990	New York, USA	1	Pleural mesothelioma	Yes	Construction laborer	NR	Diagnosed between 1974 and 1986
	Chellini et al. 1992	Tuscany, Italy	3	Pleural mesothelioma	Yes	Construction worker	NR	NR
	Chellini et al. 1992	Tuscany, Italy	1	Pleural mesothelioma	Yes	Plumber in chemical manufacturing	NR	NR
	Dodoli et al. 1992	Leghorn and La Spezia, Italy	1	Pleural mesothelioma	Yes	Oil refinery worker	NR	Died between 1975–1988
	Ascoli et al. 1996	Rome	1	Malignant mesothelioma	Yes	Construction worker	NR	Diagnosed between 1980 and 1995
	Ascoli et al. 1996	Rome	1	Malignant mesothelioma	Yes	Smelting furnace worker	NR	Diagnosed between 1980 and 1995
	Ascoli et al. 1996	Rome	1	Malignant mesothelioma	Yes	Printing house worker	Amosite	Diagnosed between 1980 and 1995
	Schneider et al. 1996	Germany	1	Pleural mesothelioma	Yes	Asbestos cement roofer	NR	Exposed 1954–1971
	Schneider et al. 1996	Germany	1	Pleural mesothelioma	Yes	Turbine revision worker	NR	Exposed 1961–1984
	Roggli et al. 1997	NR	3	Pleural mesothelioma	Yes	Pipefitter/welder	Commercial and non-commercial amphiboles	NR
	Roggli et al. 1997	NR	1	Pleural mesothelioma	Yes	Oil refinery worker	NR	NR

(Continued)

Table 1. *(Continued).*

Industry Group	Study	Location	No. of Cases	Diagnosis	Disease Confirmed?	Family Member Occupation	Fiber Type	Time Period of Exposure or Diagnosis Date
	Roggli et al. 1997	NR	1	Pleural mesothelioma	Yes	Construction worker	NR	NR
	Roggli et al. 1997	NR	1	Pleural mesothelioma	Yes	Machinist	NR	NR
	Roggli et al. 1997	NR	1	Pleural mesothelioma	Yes	Auto mechanic	NR	NR
	Roggli et al. 1997	NR	1	Pleural mesothelioma	Yes	Tire presser	NR	NR
	Roggli et al. 1997	NR	1	Pleural mesothelioma	Yes	Steamfitter	NR	NR
	Miller 2005	NR	1	Peritoneal mesothelioma	Yes	Construction worker	NR	Diagnosed in 1994
	Miller 2005	NR	1	Peritoneal mesothelioma	Yes	Steel mill worker	NR	Exposed 1947–1972
	Miller 2005	NR	1	Peritoneal mesothelioma	Yes	Railroad brake worker	NR	Diagnosed in 1996
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Boilermaker	NR	Exposed 1945–1969
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Boilermaker	NR	Exposed 1940–1959
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Refractory bricklayer	NR	Exposed 1948–1990
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Steel mill worker	NR	Exposed 1950–1980
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Pipefitter	NR	Exposed 1942–1975
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Railroad blacksmith/ pipefitter	NR	Exposed 1935-NR (RR), 1954–1992 (PF)
	Miller 2005	NR	1	Pleural mesothelioma	Yes	Refinery worker	NR	Exposed 1941–1955
	Ampleford and Ohar 2007	NR	1	Peritoneal mesothelioma	Yes	Electrician, insulator, mechanic, machinist, miller, pipe fitter, plumber, steel worker, and welder at an aluminum can company; later a furnace operator	NR	Exposed 1980–2002
	Patel et al. 2008	NR	1	Pleural mesothelioma	Yes	Construction worker/insulator	Crocidolite	NR

(Continued)

Table 1. *(Continued).*

Industry Group	Study	Location	No. of Cases	Diagnosis	Disease Confirmed?	Family Member Occupation	Fiber Type	Time Period of Exposure or Diagnosis Date
	Patel et al. 2008	NR	1	Pleural mesothelioma	Yes	Pipefitter	NR	NR
Unknown Family Member Occupation	Kiviluoto 1965	Russia and Finland	4	Slight fibrosis	X-ray	Unknown	Mixed dusts	NR
	Kiviluoto 1965	Russia and Finland	1	Pulmonary fibrosis	X-ray	Unknown	Mixed dusts	NR
	Ashcroft and Heppleston 1970	Tyneside, UK	1	Pleural mesothelioma	Yes	Unknown "Asbestos worker"	NR	Exposed for 3 unspecified years
	Greenberg and Davies 1974	England, Scotland, Wales	1	Mesothelioma	No	Unknown	NR	Exposed for three unspecified years
	Lillington et al. 1974	NR	1	Pleural mesothelioma	Yes	Unknown	NR	Exposed 1941–1949
	Gibbs et al. 1989; 1990	United Kingdom	13	Pleural mesothelioma	Yes	NR	Suggestive of amphiboles	Diagnosed between 1979 and 1986
	Kane et al. 1990	New York, USA	1	Pleural mesothelioma	Yes	Unknown	NR	Diagnosed between 1974 and 1986
	Roggli et al. 1997	NR	1	Peritoneal mesothelioma	Yes	Unknown	NR	NR
	Roggli et al. 1997	NR	3	Pleural mesothelioma	Yes	Unknown		
	Roggli et al. 1997	NR	2	Pleural mesothelioma	Yes	Unknown	NR	
	Howel et al. 1997; 1999	England	17	Mesothelioma	Yes	NR	NR	NR
	Magnani et al. 2000	Casale, Turin, Florence, Barcelona, Cadiz, Geneva (Italy)	1	Pleural mesothelioma	No	Asbestos industry (possible foundry)	NR	Diagnosed 1997
	Leigh et al. 2002	Australia	42	Mesothelioma	Yes	Asbestos and/or products worker	NR	Diagnosed between 1945 and 2000
	Ascoli et al. 2003	Latium, Italy	2	Pleural mesothelioma	Yes	NR	NR	Diagnosed between 1993 and 2001
	Rake et al. 2009	England, Wales and Scotland	24	Mesothelioma	Unknown	NR	NR	NR

NR, Not reported.

[a]Anderson et al. studies (1976, 1979, 1982) were based on x-ray abnormalities found in household contacts of factory workers. Joubert et al. (1991) reported mortality of this cohort; 4 mesotheliomas and 12 lung cancer deaths were identified. [b]McDonald et al. 1970 and 1973 presented preliminary results from a survey of Canadian pathologists of histologically confirmed mesothelioma diagnosed between 1960 and 1968. The McDonald and McDonald 1980 study presented the ascertainment of 668 cases from 7400 pathologists in both the US and Canada.

Table 2. Cases of disease reported in the literature to be due to para-occupational exposure, by industry and disease classification.

Industry Classification[a]	No. of Cases	Pleural Mesothelioma (Confirmed[b])	Peritoneal Mesothelioma (Confirmed[b])	Unspecified Mesothelioma Subtype (Confirmed[b])	Asbestosis	Fibrosis/Pleural Plaques	Lung Cancer
Cement factory	66[c]	49 (1)	3 (0)	0	0	2	12
Other factory	286	24 (20)	1 (1)	5 (4)	0	244	12
Dock/Shipyard	133	71 (60)	6 (6)	1 (1)	40	15	0
Insulation	56	27 (26)	3 (3)	5 (5)	0	18	3
Mining	33[d]	33 (7)	0	0	0	0	0
Trades	39	31 (31)	5 (5)	3 (3)	0	0	0
Unknown	114	24 (23)	1 (1)	84 (59)	0	5	0
Sum:	727	259 (168)	19 (16)	98 (72)	40	284	27

[a]Industry classification was based on descriptions in the original studies. [b]Number of mesothelioma diagnoses that were stated by the original authors to have been confimed at autopsy, by biopsy or histopathological review. [c]The studies of the families and workers in Casale Montferrato (Magnani et al. 1993, 2000 and Ferrante et al. 2007) may have had some overlap in the reporting of cases of domestic exposure from Casale Montferrato. [d]Reid et al. (2008) reported 30 deaths from malignant mesothelioma of the pleura in the cohort, 26 lived with a worker. Death certificates indicated other potential causes of death in 10 of the 30 cases. There were no peritoneal mesotheliomas in the cohort; thus, all 26 mesothelioma cases were classified as pleural.

(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).

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. The reported years of first exposure ranged from 1912 to 1941, while the duration of exposure experienced by the household contacts ranged from 2 to 41 years. In general, the primary workers in this study were involved in occupational activities with considerable potential for high asbestos exposures. The authors described the case of a housewife, for example, who indicated that her husband was "white with asbestos" when he returned home from work (Newhouse & Thompson, 1965, p. 264). At the time that the study was conducted, only four of the mesothelioma cases were alive and could therefore be interviewed personally about their occupational exposure history. For the remainder of the study group, exposure histories were ascertained from medical records, interviews with family members, as well as an investigation of historical employment records at the nearby asbestos factory. In a later publication by Whitwell et al. (1977), it was noted that many of the cases in Newhouse and Thompson (1965) lived near sack repair factories and that "the patients, or more often their relatives, who were questioned by Newhouse and Thompson, were asked about employment in asbestos factories, not sack-repair factories, so many of the cases described as home-environment mesothelioma may, in fact, have been sack repairers" (Whitwell et al., 1977, p. 384).

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.

1970–1990

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, insulators, asbestos factory employees, pipefitters, and shipyard workers (Ashcroft & Heppleston, 1970; Champion, 1971; Heller et al., 1970; Knappmann, 1972; Li et al., 1978; Lillington et al., 1974; McDonald & McDonald, 1973; McEwen et al., 1971; Rubino et al., 1972; Vianna & Polan, 1978; Von Bittersohl & Ose, 1971). Many reports also indicated that the primary worker had other asbestos-related diseases, including asbestosis and mesothelioma (Champion, 1971; Li et al., 1978; Lillington et al., 1974). One report indicated that the primary worker had visible "asbestos dust on his hair and shoes," (Ashcroft & Heppleston, 1970, p. 177). No quantitative exposure information was available for this particular case, however, or for any of the other reported cases, and fiber types were often not specified.

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. The authors noted that the "actual intensity of household

asbestos contamination and length of each family contact's exposure to such fibers is unknown" (Anderson et al., 1976, p. 313). Of this group, 239 (35%) were reported to have radiographic abnormalities associated with asbestos exposure, including small opacities, pleural thickening, pleural calcification, or pleural plaques (Anderson et al., 1979). 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. Mortality data of 878 household contacts of the same factory workers were reported in 1991 (Joubert et al., 1991). Each household contact had more than 20 years since onset of exposure and no known personal occupational exposure to asbestos. While the various publications by the Mt. Sinai group indicate that smoking history was collected, there is no additional information regarding smoking rates among household contacts with reported radiographic abnormalities, nor do the authors discuss possible alternative factors that could be responsible for the observed effects in the lung. This is also true for the last publication (Joubert et al., 1991), which reported four mesothelioma and 12 lung cancer deaths among the household contacts.

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. The household contacts in this study were reported to have hand-laundered their family member's clothing for periods of 5 to 22 years. While the husband's occupation was reported to be the most important risk factor, the authors also reported that residential exposures (i.e., proximity of residence to an asbestos industry) may have been a risk factor for some patients that developed mesothelioma (Vianna & Polan, 1978).

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).

In 1986, Seixas and Ordin conducted an evaluation at the Friction Division Products plant in Trenton, New Jersey (Seixas & Ordin, 1986). Various issues related to using chrysotile asbestos were investigated, including the presence of asbestos on the clothing of workers before they left the plant each day. Vacuum samples collected on clothing positively identified chrysotile on all five workers evaluated; however, quantitative values were not included (Seixas & Ordin, 1986). Similarly, Driscoll and Elliott conducted a Health Hazard Evaluation at the Chrysler Friction Products and Chemical Plant in Trenton, Michigan in 1990. Among other samples collected throughout the facility, vacuum samples were taken from the clothing and the vehicles of employees who held various positions at the plant. Although no quantitative values were presented, asbestos was positively measured in 11 of the 13 samples (Driscoll & Elliott, 1990).

1990–2011

Throughout the 1990s, additional case reports and case–control studies described asbestos-related diseases thought to be caused by para-occupational exposures. 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). Many of these exposure evaluations were based on histological examination of lung tissue and/or questionnaires completed by family members or acquaintances after death (Chellini et al., 1992; Dodoli et al., 1992).

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). Birth and marriage data were collected from town records, while factory employment records of the husbands were used to determine domestic exposure durations. Wives with direct occupational exposure from the plant were excluded from the cohort. 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). A previous case–control study that included parents and spouses (both sexes) who worked at the plant also reported statistically significant SMRs for mesothelioma (Magnani et al., 2001). While no exposure data from the plant were available, (Magnani et al., 2000; Magnani et al., 1993, p. 783) given the potency of crocidolite for causing mesothelioma, a far lower cumulative dose would be required to induce disease in household contacts compared to chrysotile.

After the 1990s, case reports and case series continued to report asbestos-related disease thought to be caused by domestic exposure (Ampleford & Ohar, 2007; Bianchi et al., 2001a; 2001b; Mirabelli et al., 2008; Patel et al., 2008; Peretz et al., 2009; Whitehouse et al., 2008). More recent literature has utilized data from well-established mesothelioma and cancer registries in various countries, and even from records from law firms participating in asbestos litigation (Bianchi & Bianchi, 2009; Marinaccio et al., 2010; Miller, 2005; Powers & Carbone, 2002; Rake et al., 2009; Reid et al., 2008). 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.

Summary of studies that provide estimates of risk of asbestos-related disease due to take home exposures

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). The lowest estimates of RR were those for cancer of the respiratory system (O/E ratio 1.7 among males, 1.25 among females) calculated from data presented in Anderson et al. (1982) and for lung cancer (SMR 1.17) presented in Ferrante et al. (2007).

In almost all studies where a mesothelioma RR was presented or could be calculated, exposure to crocidolite or amosite asbestos was known to have occurred. Three cohorts have been studied with regard to cancer in family members of asbestos workers. The first cohort, studied by Anderson (1982), reported eight respiratory cancer deaths vs. 4.7 expected, two of which were mesotheliomas. The second cohort, studied by Magnani et al. (2000, 2001, 1993) and Ferrante et al. (2007), consisted of the wives of the Eternit factory in Casale Monferrato, Italy, where asbestos cement goods were manufactured using both chrysotile and crocidolite. The most recent update of the cohort, which includes more than 40 years of follow up, presented an SMR of 18 for pleural mesothelioma, representing 21 observed deaths (95% CI: 11.14, 27.52). Non-statistically significant SMRs were presented for peritoneal mesothelioma and for lung cancer. Third, family members of crocidolite miners in Australia have been studied by Reid et al. (2008); 26 mesothelioma deaths were reported to have occurred in women who lived with a former worker at the mines and it was concluded that the risk of mesothelioma increased, but not statistically significant, among women known to have lived with or washed the clothes of an asbestos miner or miller (hazard ratio 2.67, 95% CI: 0.77, 9.21). A review of the epidemiology studies on risk of pleural mesothelioma and household or neighborhood exposure to asbestos presented a meta-RR of 8.1 (95% CI: 5.3, 12) for household exposure (Bourdes et al., 2000). The review concluded that there is a strong association between high exposures to asbestos, whether environmental or household, and mesothelioma; in addition, it was suggested that there exists a higher risk due to exposure to amphiboles than for chrysotile (Bourdes et al., 2000).

Use of fiber-type specific asbestos burden in the lung to characterize para-occupational exposure

Beginning in the late 1980s and continuing throughout the 2000s, a greater number of studies began to include data on the lung burden of specific fiber types in household contacts diagnosed with asbestos-related disease. Several of these analyses provide comparisons of lung fiber burdens of household contacts to those occupationally exposed or internal reference populations with no known asbestos exposures. While lung burden analyses have provided information that has advanced the understanding of asbestos disease, differences in sampling, preparation, and counting techniques have made it difficult to make comparisons across studies (De Vuyst et al., 1998). For example, some researchers examine the lung tissue samples using light microscopy. Others use

Table 3. Studies that present relative risk estimates for asbestos-related disease among para-occupationally exposed subjects.

Study	Disease	RR (95% CI)	Type of Study	Notes
Newhouse & Thompson 1965	Mesothelioma	OR = 23.7 (4.7, 120)	Case control	Calculated post-hoc, OR not presented in study
Vianna & Polan 1978	Pleural and peritoneal mesothelioma	OR = 10 (1.42, 37.40)	Case control	
McDonald & McDonald 1980	Malignant mesothelioma	OR = 4.0 (0.8, 19)	Case control	Calculated post-hoc, OR not presented in study
Anderson 1982	Cancer of the respiratory system (females)	O/E = 1.25	Cohort	Presents observed/expected deaths of family contacts . CI calculated post-hoc.
	Cancer of the respiratory system (males)	O/E = 1.70		
Howel et al. 1997	Mesothelioma	OR = 5.8 (1.7, 19.2)	Case control	
Bourdes et al. 2000	Pleural mesothelioma	Meta-RR = 8.1 (5.3, 12)	Meta-analysis	Summary risk across 8 studies of both environmental and household exposure
Magnani et al. 2000	Pleural mesothelioma	OR = 4.92 (1.78, 13.61)	Case control	Domestically exposed, not environmentally or occupationally exposed. Domestic exposures included asbestos containing materials in the home or yard.
Magnani et al. 2001	Pleural mesothelioma	OR = 4.5 (1.8, 11.1)	Case control	Any relative (mother, father, spouse, other) exposed
Ferrante et al. 2007	Malignant neoplasm of the pleura	SMR = 18 (11.14, 27.52)	Cohort mortality	
	Malignant neoplasm of the peritoneum	SMR = 2.51 (0.52, 7.35)		
	Malignant neoplasm of the lung	SMR = 1.17 (0.6, 2.04)		
Reid et al. 2008	Pleural mesothelioma	HR = 2.67 (0.77, 9.21)	Cohort mortality	Women who lived with or washed clothing of a crocidolite miner
Rake et al. 2009	Mesothelioma (females)	OR = 1.9 (1.1, 3.2)	Case control	Domestic exposure before 30 years of age
	Mesothelioma (males)	OR = 2.1 (1.0, 4.5)		

RR, relative risk; OR, odds ratio; O/E, observed/expected; Meta-RR, meta relative risk; SMR, standardized mortality ratio; HR, hazard ratio.

scanning electron microscopy (SEM) at various magnifications, while others use transmission electron microscopy (TEM), also at various magnifications, and others may report concentrations in the lymph nodes. To add to the difficulties of comparing labs, one must be aware that some labs report the presence of only fibers five microns in length while others report all visible fibers. Table 4 presents the available lung fiber burden data for household contacts published to date. Individual results are presented for each study to facilitate comparisons by fiber type or occupation of the primary worker within a study, but care should be taken when looking at values across several studies, as these results are not comparable for the reasons mentioned.

Huncharek et al. (1989) were the first to provide these data from a household contact with asbestos-related disease and no known direct exposures. Their subject was a 77-year-old woman with no known occupational exposure to asbestos, but whose husband was a shipyard machinist from 1935 until 1969. He regularly dismantled boilers and other machinery associated with insulation. The subject laundered her husband's clothing, which was described as being "covered with dust" (Huncharek et al., 1989, p. 354). The authors noted that this fiber content was similar to that seen in those occupationally exposed, although this conclusion was based on an analysis of lung tissue in a completely different population that was published by another group (Mowe et al., 1985), and should be interpreted with caution.

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. Asbestos exposure occurred through laundering activities involving the clothing of workers in the shipyard, lagging, building, and ordnance industries. Lung burden analysis demonstrated elevated amosite and crocidolite in the majority of cases; however, two cases showed normal concentrations of all types of fibers. Because concentrations in some cases were highly elevated, the authors suspected that exposures aside from just para-occupational exposure had occurred (Gibbs et al., 1990; Gibbs et al., 1989).

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. The occupations of the "asbestos worker" were reported as either insulator or shipyard workers; five of the six workers had been diagnosed with asbestosis, and three also had lung cancer. Among their household contacts, the median

Table 4. Summary of published lung fiber burden data from household contacts.

Study	Disease	n	Age	Relation	Family Worker Occupation (Duration of Exposure)	Fiber Type	No. of fibers/g wet lung tissue ($\times 10^3$)	No. of fibers/g dry lung tissue ($\times 10^6$)	AB/g wet lung tissue ($\times 10^3$)
Huncharek et al., 1989	PM	1	76	Wife	Machinist at shipyard (34 yrs)	Chrysotile	172	2.5	–
						AC	59	0.8	–
						TAA	221	3.2	–
Gibbs et al., 1989, 1990	PM	1	–	Wife	Shipyard workers, laggers, builders, ordnance workers, other	Chrysotile	–	26.1	–
						Amosite	–	1	–
						Crocidolite	–	4.3	–
	PM	1	–	Wife[a]		Chrysotile	–	135.1	–
						Amosite	–	4.6	–
						Crocidolite	–	7.6	–
	PM	1	–	NA[a]		Chrysotile	–	2507	–
						Amosite	–	0	–
						Crocidolite	–	29.5	–
	PM	1	–	Wife[a]		Chrysotile	–	28.5	–
						Amosite	–	2.5	–
						Crocidolite	–	9	–
	PM	1	–	Wife[a]		Chrysotile	–	31	–
						Amosite	–	0	–
						Crocidolite	–	251.1	–
	PM	1	–	Wife		Chrysotile	–	9.2	–
						Amosite	–	1.2	–
						Crocidolite	–	1.2	–
	PM	1	–	NA[a]		Chrysotile	–	62.2	–
						Amosite	–	0	–
						Crocidolite	–	108.8	–
	PM	1	–	Wife		Chrysotile	–	1.9	–
						Amosite	–	0	–
						Crocidolite	–	0	–
	PM	1	–	Wife		Chrysotile	–	2.9	–
						Amosite	–	2.2	–
						Crocidolite	–	0	–
	PM	1	–	Wife		Chrysotile	–	6.7	–
						Amosite	–	6.1	–
						Crocidolite	–	1.6	–
	PM	1	–	NA		Chrysotile	–	2	–
						Amosite	–	0.3	–
						Crocidolite	–	0.3	–
	PM	1	–	Wife		Chrysotile	–	25.8	–
						Amosite	–	2.2	–
						Crocidolite	–	0	–
	PM	1	–	Daughter		Chrysotile	–	7.2	–
						Amosite	–	0	–
						Crocidolite	–	0	–
Roggli and Longo, 1991	PM	1	62	Wife	Shipyard insulator (29 yrs)	–	ND	–	8.2
	PM	1	33	Daughter	Insulator (25 yrs)	–	17	–	2.3
	LC, A	1	63	Wife	Insulator (yrs)	–	120	–	3.7
	LC, PPP	1	59	Wife	Insulator (23 yrs)	–	57	–	1.1
	LC	1	73	Wife	Insulator (yrs)	–	23.7	–	0.4
	PM	1	57	Wife	Shipyard worker (1–2 yrs)	–	24.3	–	0.002

(Continued)

Table 4. *(Continued)*.

Study	Disease	n	Age	Relation	Family Worker Occupation (Duration of Exposure)	Fiber Type	No. of fibers/g wet lung tissue (x10^3)	No. of fibers/g dry lung tissue (x10^6)	AB/g wet lung tissue (x10^3)
Howel et al., 1999[b]	MM	13	NA	NA	NA	Chrysotile	–	1.7 (<0.1 – 40.9)	–
						Amosite	–	<0.1 (<0.1 – 2.5)	–
						Crocidolite	–	5.1 (<0.1 – 228)	–
Roggli et al., 2002[c]	PM	79	59 (25–93)	NA	NA (median exposure duration 20 yrs)	–	–	–	0.13 (0.002–14.1)
						Chrysotile	1.8	–	–
						AC	3.4 (0.45–116)	–	–
						TAA	5.2 (0.98–22.4)	–	–
Dodson et al., 2003[d]	PM	1	59	Wife/ Daughter	Coast guard/shipyard worker	Chrysotile	–	0	–
						Amosite	–	0.013	–
						Crocidolite	–	0	–
						Tremolite	–	0.255	–
						Actinolite	–	0.013	–
						Anthophyllite	–	0	–
	PM	1	63	Wife/ Daughter	Maintenance systems worker (32 yrs)/ painter, plasterer & guard for shipbuilding & drydock company	Chrysotile	–	0.011	–
						Amosite	–	0	–
						Crocidolite	–	0	–
						Tremolite	–	0.011	–
						Actinolite	–	0	–
						Anthophyllite	–	0.011	–
	PM	1	66	Wife	Laborer, shipscaler, longshoreman, warehouseman, plumber's helper, burner helper & cement worker (45 yrs)	Chrysotile	–	0	–
						Amosite	–	0.026	–
						Crocidolite	–	0	–
						Tremolite	–	0	–
						Actinolite	–	0	–
						Anthophyllite	–	0	–
	PM	1	69	Wife	Crocidolite concrete pipe manufacturer	Chrysotile	–	0	–
						Amosite	–	0	–
						Crocidolite	–	0.521	–
						Tremolite	–	0	–
						Actinolite	–	0	–
						Anthophyllite	–	0	–

PM, Pleural mesothelioma; MM, Malignant mesothelioma; AB, Asbestos bodies; PPP, Pleural parietal plaques; A, Asbestosis; ND, Not done; AC, Commercial amphiboles (Amosite + crocidolite); TAA (noncommercial amphiboles (tremolite actinolinte anthopholite).

[a]It was noted in Gibbs et al. (1989) that it was "highly unlikely that [these cases] were only exposed paraoccupationally, despite their histories, since the mineral fibre analysis of these cases is very similar to that obtained in a unique group of Nottingham gask-mask workers who were exposed to considerable quantities of crocidolite (p. 223). [b]Results reported as median value for 13 cases. [c]Results reported as median value for 79 cases, with the range in parentheses. [d]Results reported for uncoated asbestos fibers.

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). It is noteworthy that in 8% of the household exposure mesothelioma cases, histologically confirmed

asbestosis was also present; however, no cases of asbestosis were present in other non-occupational exposure categories (i.e. building occupants, environmental exposure).

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). Fiber concentrations in the para-occupational group were similar to concentrations found in the occupationally exposed cohort.

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.

Studies reporting quantitative data on airborne asbestos concentrations during handling of clothing

Airborne asbestos concentrations during laundering (after ceiling abatement)

In 1977, Sawyer published the results of airborne samples collected during the abatement of an asbestos-containing ceiling on the Yale campus. In order to remove the ceiling, the asbestos-containing material was sprayed with a solution containing water, polyoxyethylene ester, and polyoxyethylene ether, and was subsequently scraped from the suspended ceiling before the sheetrock was removed. After all materials and debris were collected, the area was cleaned prior to the project's completion. Average airborne concentrations during each step of the removal process ranged from non-detects during final cleaning involving wet mopping, to 6.5 f/cc during gross cleanup, which involved placing debris in drums for disposal. The entire process lasted for 18 days (Sawyer, 1977).

After work was concluded each day, workers went through a decontamination facility, which included a changing room, shower room, equipment area, and laundry space. Workers were required to completely change and shower, and work clothing was collected for washing, drying, and folding. The average airborne asbestos concentrations from samples collected five feet away from the area where clothing was handled ranged from non-detected values while loading the dryer to 0.4 f/cc while picking up clothing (see Table 5). Sample collection times varied.

Asbestos concentrations in the homes of miners and millers

Nicholson et al. (1980) attempted to characterize the potential exposure to household contacts of chrysotile mine and mill employees; they published the results of 13 samples taken from the homes of mine and mill employees. These workers were employed in mine operations in California and Newfoundland, did not have access to shower facilities, and did not change clothing before going home. Sampling took place between 1973 and in 1976. Chrysotile asbestos concentrations ranged from over 50 to over 2000 ng/m^3, although exact measurements were not reported. In comparison, concentration measurements taken in the homes of non-miners were 32–65 ng/m^3. All reported samples were under 5000 ng/m^3 (Nicholson, 1983; Nicholson et al., 1980). Using a conversion factor of 30 μg/m^3 per f/cc (EPA, 1986), the values reported in Nicholson et al. (1980) and Nicholson (1983) correspond to a range of 0.0015–0.06 f/cc in the homes of the miners and 0.001–0.002 f/cc in the homes of the non-miners.

Various industrial operations

Contaminated work coveralls were collected by Mangold (1982) from various industrial operations during which asbestos was used. The clothing was then placed on the workmen in clean environments during an 8-h period during which no asbestos material was handled in order to determine the exposure from contaminated clothing alone. Although this study was not designed specifically to estimate para-occupational exposures, it provides some insight into the amount of exposure that can occur from contact with contaminated clothing (Mangold, 1982).

Mangold collected heavily contaminated coveralls worn while ripping out pipe insulation, and medium and lightly contaminated clothing that were worn for three days, although the source of this contamination was indicated only to be "various industrial operations where asbestos was in use" (Mangold, 1982, p. 26). Clothing worn during gasket handling and handling of other small parts containing asbestos was also tested. It is not surprising that the clothing worn during insulation removal provided the highest airborne concentrations in the breathing zone of the person handling the contaminated clothing, with an average 8-h TWA of 1.4 f/cc. The medium and lightly contaminated clothing resulted in average 8-h TWAs of 0.5 f/cc and 0.1 f/cc, respectively, while coveralls worn during work with gaskets and other small asbestos-containing parts resulted in an average 8-h TWA of 0.05 f/cc (see Table 5) (Mangold, 1982). Analytical methods were not reported. The authors noted that the sampling technique used during the experiment, particularly the placement of the sampling cassette, created a baffling effect of the edge of the coveralls to the open-faced cassette and may have caused the cassette to collect more fibers because of its proximity to the clothing. If the samples were analyzed using PCM, which does not distinguish between fiber types, it would not be possible to determine what percent of the airborne fibers measured were actually fibers from the clothing. Thus, these results could be an overestimate of the actual airborne concentrations.

Table 5. Reported concentrations in studies related to para-occupational exposure.

Activity	No. of Samples	Sample Type	Sample Time	Average Fiber Concentration (f/cc)	Average Primary Worker Exposure (f/cc)	Analytical Method	Reference
Laundering clothing worn during various abatement activities:					0-6.5 for various abatement activities	US Public Health Service Method	Sawyer 1977
General laundering activities	12	Personal	Variable[a]	0.4			
Picking up clothing	4	Area	Variable	0.4			
Loading washer	5	Area	Variable	0.4			
Loading dryer	6	Area	Variable	0			
Wearing heavily contaminated coveralls used during rip-out of pipe insulation	3	Personal	8 hours (TWA)	1.4	NR	P&CAM 239	Mangold 1982
Wearing medium contaminated coveralls worn three days before change	3	Personal	8 hours (TWA)	0.5	NR		
Wearing lightly contaminated coveralls worn three days before change	3	Personal	8 hours (TWA)	0.1	NR		
Wearing coveralls worn during gasket handling and other small asbestos parts	3	Personal	8 hours (TWA)	0.05	NR		
Wearing new coveralls in controlled area	3	Personal	8 hours (TWA)	0.003	NR		
Shaking, folding and stacking amphibole contaminated towels after laundering	1	Personal	18 minutes	0.05	NR[b]	MDHS 39	Revell 2002
	1	Area	18 minutes	0.04			

NR, Not reported.
[a]Sample times not reported by task, but overall sampling times ranged from 5 minutes to 10 hours. [b]Asbestos fiber density on contaminated towels: 15–99.8 f/mm^2.

Industrial laundering

Investigators have also looked at other factors indirectly related to para-occupational exposures. Revell (2002), for example, investigated the effectiveness of laundering towels and coveralls contaminated with amphibole asbestos fibers (see Table 5). This study was conducted by analyzing the fiber contamination of the fabric and evaluating any remaining contamination subsequent to laundering. The authors then evaluated potential exposures to asbestos remaining on towels after laundering through shaking, folding, and stacking the laundered towels (Revell, 2002). Revell found that, over an 18-min period, persons who shook, stacked, and folded laundered towels with asbestos fiber densities ranging from 15.3 to 99.8 f/mm^2 (analytical detection limit was 10 f/mm^2) were exposed to 0.05 f/cc (TEM), while the corresponding area sample had an airborne concentration of 0.04 f/cc (TEM).

Evaluation of clothing worn during work with friction products

In 2001, a simulation study was conducted during the arcing of automotive brake shoes (Weir et al., 2001). Auto mechanic tasks were re-constructed to create the probable maximum use of an arcing machine during full and half workdays. Following completion of the workday, the operator's one-piece suit was carefully removed and evaluated for the presence of fibers potentially generated during the arcing work. A non-rigid freeform dynamic flow chamber was constructed to allow agitation of the clothing while extracting an air sample from the chamber. Filtered air was introduced into one end of the chamber containing the clothing at a rate of 2.5 L/min. After the chamber was inflated to approximately 100 L, a pump drawing 2.5 L/min of air was started at the opposite end of the chamber, maintaining a constant volume of about 100 L throughout the study. Air from the exhaust was drawn through the sampling apparatus. The experiment was conducted for 30 min, during which time the clothing was agitated in alternating 5-minute intervals. The airborne fiber concentration generated from agitating the operator's coveralls was 0.72 f/cc (PCM) during the 30 min of testing (see Table 6). As noted by the authors, "the results support little reason to conclude that the persons handling such clothing might be exposed to chrysotile fiber concentrations above the [current] acceptable standards," particularly because the majority of fibers found were not asbestiform, and included cotton fibers (Weir et al., 2001, p.1145).

Three recent simulation studies reported personal and area samples taken during handling of clothing that had been worn throughout simulations involving chrysotile-containing friction products. Results are summarized in Table 6. In 2009, Madl et al. measured airborne asbestos concentrations during brake removal and disassembly activities on 12 pieces of heavy equipment. Before brake work was performed, mechanics were fitted with

Table 6. Reported concentrations in simulation studies during agitation or handling of contaminated clothing.

Activity	No. of Samples	Sample Type	Sample Time (Minutes)	Average Fiber Concentration During Clothing Handling PCM (f/cc)	Average Fiber Concentration During Clothing Handling PCME (f/cc)	Average Primary Worker Exposure (PCME, f/cc)*	Analytical Method	Reference
Clothing agitation after servicing drum brakes and arcing brake shoes	1	Area (in chamber)	30[a]	0.72[b]	–	0.4[c]	P&CAM 239	Weir et al. 2001
Clothing shake out after handling automotive clutches and packaging	4	Personal	15	0.0782	0.0013	0.001–0.231	NIOSH 7400/ NIOSH 7402	Jiang et al. 2008
	2	Area (bystander)	30	0.0097	0			
	1	Area (remote)	30	0.0092	0			
Clothing shake out after handling automotive brake shoes, pads and packaging	4	Personal	15	0.0369	0.0083	0.012–0.657	NIOSH 7400/ NIOSH 7402	Madl et al. 2008
	4	Area (bystander)	30	0.0136	0.0025			
	2	Area (remote)	30	0.0135	0.0008			
Clothing shake out after heavy equipment brake removal	4	Personal	NR	0.2308	0.0179	0.001–0.090	NIOSH 7400/ NIOSH 7402	Madl et al. 2009
	2	Area (bystander)	NR	0.0930	0.0105			
	2	Area (remote)	NR	0.0400	0.0000			

*With the exception of Weir et al., values represent average concentrations for various tasks; however, clothing from all tasks was collected and used during the shaking task. [a]Sampling occurred in 5 min alternative intervals of shaking and rest, over a 30-min period. [b]The authors note that the sample contained primarily non-asbestiform fibers, including cotton. [c]Concentration measured by PCM only.

new coveralls (Madl et al., 2009). The coveralls were collected after the mechanic completed work on each piece of equipment. Simulated clothes handling involved repeated shaking, folding, and turning clothes inside out for approximately 1–2 min per each pair of coveralls (Madl et al., 2009). Personal sample results during clothing related tasks ranged from non-detect to 0.039 f/cc, presented as phase contrast microscopy equivalent (PCME) measurements. A PCME measurement includes analysis by both phase contrast microscopy (PCM) and transmission electron microscopy (TEM). The ratio of asbestos fibers to non-asbestos fibers found through TEM is used to adjust the total fibers found through PCM.

Similar clothing handling samples were collected in a related study after handling new, chrysotile-containing automotive brake components and packaging (Madl et al., 2008). The primary worker's initial tasks included unpacking and repacking up to 20 boxes of brake pads and shoes and associated cleanup activities. The primary worker's coveralls were collected upon completion of the tasks, and were subsequently shaken, folded, and turned inside out for 1–2 min to simulate typical clothes handling tasks during laundering. Personal sample results during these tasks ranged from 0.007 to 0.015 f/cc (PCME).

Using nearly the same protocol as Madl et al. (2008), Jiang et al. (2008) collected samples during clothes handling tasks on clothing worn during automotive clutch handling. Primary worker tasks included stacking, unpacking, and repacking boxes of clutches containing chrysotile asbestos, as well as cleanup activities. The clothing handling tasks involved shaking and folding three different pairs of coveralls worn during clutch handling for approximately 45 sec. Personal samples collected during clothing collection ranged from non-detect to 0.006 f/cc (PCME) (Jiang et al., 2008). These results were again not directly comparable to those of the primary worker, as various tasks were completed prior to clothing shake-out.

Adhesion of fibers to clothing

Because the available field data could not resolve the relationship between workplace airborne asbestos concentrations, the number of fibers that adhere to a worker's clothing, and airborne asbestos concentrations in the home, we also evaluated whether basic research, including mathematical modeling, had been conducted to assess the adhesive properties of fibers and clothing. The re-suspension of particulates from clothing has been characterized for other agents such as beryllium and lead, but, to the best of our knowledge, the release of fibers from asbestos-contaminated clothing has never been quantitatively evaluated (Cohen & Positano, 1986; Winegar et al., 1977).

Research regarding particle adherence has focused primarily on spherical particles adhering to flat, solid surfaces. As many researchers have shown, van der Waals, electrostatic, and surface tension interactions prevent the vast majority of very small particles from being resuspended, since there is too little mass to generate sufficient inertia to overcome these attractive forces (Corn & Stein, 1966; Esmen, 1996; Hinds, 1999; Lam & Newton, 1992). Other factors that have been reported to influence the adhesion of particles to substrates include ambient air humidity, contact area between the particles and the surface, electrical charge of the particle, particle and surface type and texture, particle-surface contact time, and air stream velocity (Corn, 1961a, 1961b; Hu et al., 2008; Ibrahim et al., 2004; Mullins et al., 1992). In their study of spherical glass particles adhering to metal

substrates, Corn and Stein (1965) found that adhesion forces increase with particle size and ambient air relative humidity, but decrease with increased surface roughness (Corn & Stein, 1965). Other researchers, however, have suggested that high ambient relative humidity may reduce adhesive force by weakening hydrogen bonds or by reducing electrostatic attraction via diminished surface charge (Lamb et al., 1990; Walton, 2008).

In a study that specifically addressed fibrous particles, Esmen performed theoretical calculations to predict the air speeds required to re-suspend glass fiber threads from a smooth surface, then provided an experimental verification of his calculations (Esmen, 1996). There was general agreement among the results, although the measured air speeds required for fiber re-suspension were, on average, higher than calculated values. Esmen concluded that at about 10 m/s [approximately 2000 feet per minute], biologically relevant [9 μm in diameter] fibers would not be expected to be re-suspended under optimal conditions (Esmen, 1996, p. 382). It should be noted, however, that the glass fiber threads used in the experiment (diameters of 10 μm and 16 μm, with lengths ranging from 36 to 400 μm) were generally much larger than typical asbestos fiber lengths and diameters considered relevant by OSHA (>5 μm with an length:diameter aspect ratio of 3:1), thus it would be expected that greater force and/or wind speed would be required to re-suspend OSHA fibers.

The differences in the ability of different types of asbestos fibers to adhere to surfaces of any type have not been directly studied to our knowledge. The basic morphological differences between chrysotile and amphibole fibers (curly and flexible vs. straight and rigid, as described by (Berman & Crump, 2008a)) would likely impact the amount of surface contact between the fiber and a surface which could, in turn, affect the adhesive attraction. Specifically, van der Waals and electrostatic adhesion forces would be diminished with less surface contact, since both forces require close contact between surfaces (Esmen, 1996; Hinds, 1999). It is also important to note that flattening, or a decrease in separation distance between two surfaces due to mechanical deformation, also affects the adhesive force between two surfaces (Hinds, 1999). Flattening occurs to a lesser degree for harder materials, and since amphiboles are harder than chrysotile fibers, less flattening may occur for amphiboles (Virta, 2005).

Because of the physical characteristics of small fibers, respirable particles would be expected to, in large measure, remain attached to flat surfaces and to clothing. Some factors that could influence the degree of fiber release from clothing include the diameter, length, density, and type of the fibers, whether there is a single layer or multiple layers of fibers, the surface characteristics of the clothing fabric, whether the fabric is new or worn, and the vigor of shaking. It is also possible that in more humid environments, or for clothing that is wet with perspiration, a higher air speed would be required to re-suspend fibers (a trend observed by Esmen (1996)). From the various studies, one can conclude that wind alone will generally not be sufficient to resuspend an appreciable quantity of small (respirable) particles or asbestos fibers from clothing or a flat surface. However, vigorous shaking can clearly release some quantity of fibers from the clothing, and these could be measured in the room if the clothing was sufficiently contaminated.

Discussion

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. Further, in many of these cases, the primary workers were employed for decades or for their entire careers (which usually started well before 1970), resulting in a potential for chronic exposure to household contacts. Only one of the case reports involved an individual who lived with a primary worker that appeared to have worked exclusively with encapsulated end products: the woman was the wife of an auto mechanic who had also undergone radiation for lymphoma (Roggli et al., 1997).

While very few of the case studies reported airborne asbestos concentrations (for either the home or the workplace), based on what is known about the types of asbestos used in manufacturing or insulation work up through the 1960s, it is likely that exposures in these settings would have involved amphiboles, and potentially at high airborne concentrations. This finding, coupled with the workplace data, is consistent with the fact that 98% of lung tissue samples collected from household contacts that were analyzed for fiber type indicated the presence of amphibole asbestos, including amosite and crocidolite, or a combination of amphiboles and chrysotile (Gibbs et al., 1990; Gibbs et al., 1989; Howel et al., 1999; Huncharek et al., 1989; Roggli & Longo, 1991; Roggli et al., 2002). It is well-established that amphiboles have greater potency

for causing mesothelioma than does chrysotile, at an approximate ratio of 1:100:500 for chrysotile, amosite, and crocidolite, respectively (Hodgson & Darnton, 2000; Hodgson et al., 2005). A more recent evaluation provided estimates of the relative potency of chrysotile ranging from zero to about 1/200th that of amphibole asbestos (depending on metric) (Berman & Crump, 2008a, 2008b). This is consistent with the work of Pierce et al. (2008), which suggests that it takes very high lifetime doses of chrysotile to increase the risk of mesothelioma. Others have suggested that it is unlikely that chrysotile alone can cause mesothelioma (Hodgson et al., 2005; Yarborough 2006). 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.

There are several areas of uncertainty across the various studies identified in this review. First, it is always possible that household contacts had additional sources of asbestos exposure of which they were not aware. Exposure and occupational histories are often taken from family members who are unaware of other exposure incidents. Even when histories are taken from the patients themselves, recall bias may exist, as well as the possibility that the patient did not know he or she was exposed to asbestos occupationally. Many investigators have noted the difficulties and unreliability of linking certain asbestos-related diseases to household exposure alone, particularly when lung burdens are dramatically above background concentrations, or are at levels similar to or exceeding those who have been occupationally exposed (Dawson et al., 1992; Gibbs et al., 1990; Gibbs et al., 1989; Howel et al., 1999; Huncharek et al., 1989; Roggli et al., 2002). For example, Butnor et al. (2003) reported such a concern when he studied auto mechanics with malignant mesothelioma who claimed no other exposures (other than to brakes) but then found considerable concentrations of amphiboles in their lungs (these are not present in brakes), suggesting recall bias.

Second, it is possible that many of the reports of lung cancers, fibrosis, and mesotheliomas among the household contacts were not due to asbestos exposures. It is notable that our review identified 27 cases of lung cancer, a disease known to be caused primarily by smoking, but also by other factors. Information regarding smoking status was not provided for any of these cases, with the exception of one case of bronchioalveolar cell carcinoma that reportedly occurred in a nonsmoker (Roggli & Longo, 1991). It was also noted in Ferrante et al. (2007) that while there were 12 cases of lung cancer among household contacts, this value was not significantly higher than the expected value of 10.3. Additionally, 285 household cases where fibrosis and/or pleural plaques was the disease associated with para-occupational exposure were identified. Interstitial lung disease is known to be related to a variety of autoimmune diseases, where a person's immune system inappropriately targets its own tissues, often leading to progressive damage and tissue fibrosis. Examples include diseases such as lupus and rheumatoid arthritis (Duke Health, 2010). Occupational exposures to several other types of dusts can cause interstitial lung fibrosis, including coal dust, silica (sand blasting, stone crushing, foundry workers) and cotton dust (Dugdale III, 2012). Pulmonary fibrosis can also arise when none of these risk factors is present, a condition known as "idiopathic pulmonary fibrosis" (IPF); however, there is no scientific consensus for the mechanism of IPF (Dugdale III, 2012).

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.

Third, we acknowledge that the available studies that report airborne asbestos concentrations during various clothing handling scenarios have several limitations. Mangold et al. (1982), for example, characterized airborne asbestos concentrations experienced by workers wearing clothes that had previously been worn during work involving asbestos, but did not provide information regarding concentrations in the original work environment. Further, these values would not be directly relatable to those that would be experienced by a person handling laundry for ten minutes or less. Nonetheless it is interesting to note that the reported value for wearing clothes that had previously been worn during ripping out pipe insulation was approximately 30 times higher than a sample collected from clothing that had been worn during gasket and other small part handling (1.4 f/cc vs. 0.05 f/cc for 8-h samples), indicating that the airborne concentration experienced by the worker, as expected, influences the number of fibers that adhere to the clothing. The airborne asbestos concentrations reported by Mangold (1982) were relatively consistent with data collected by Sawyer et al. (1977), who reported a value of 0.4 f/cc for personal samples (short term) collected during laundering of clothing that had been worn during an asbestos abatement project, but

given that the Mangold (1982) data were not collected during clothing handling tasks that would be typical of a household contact, it is difficult to directly relate the two results. Revell (2002) also collected air samples during various laundering activities (including shaking), but no information was available regarding the airborne asbestos concentrations that were present at the time that the fabric was contaminated. Lastly, Nicholson et al. (1980) reported airborne asbestos concentrations in the homes of chrysotile miners in California and Newfoundland; these were found to be approximately 10–100 times greater than what was found in the homes of non-miners, but no information was available on health outcomes in either population group, nor were exposure data from the mines available for comparison.

Only a handful of recent simulation studies provide detailed information regarding both the workplace (simulation) environment and the subsequent handling of clothing that had been contaminated in that environment. One important point regarding these data is that all of the studies involved encapsulated end products containing only chrysotile asbestos (see Table 6). Routine use of these products generates low airborne asbestos concentrations, often several orders of magnitude less than what was seen in the industries in which many of the spouses or family members of reported household cases worked. Many of the reported results (workplace and clothing handling alike) are below 0.01 f/cc (or reported as non-detect). The comparability of analytical methods must also be considered. For example, the Weir et al. (2001) study had higher concentrations during clothing agitation compared to that measured during the actual work (0.72 f/cc vs. 0.4 f/cc), but noted that in the sample collected during clothing handling, the majority of fibers measured were non-asbestiform, including cotton. In contrast, because they reported only the presence of asbestos fibers, in the studies conducted by Madl et al. (2008) and Jiang et al. (2008), concentrations measured during clothing shake out were lower than concentrations experienced by the primary worker.

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.

Nonetheless, if one were to assume that a person who worked between 1930 and 1960 in an environment where airborne asbestos concentrations were around the prevailing TLV of five mppcf (approximately 30 f/cc) and brought home clothing, and it is assumed that the threshold dose for amosite and mesothelioma is somewhere between one and 10 f/cc-year, then it is plausible (based on a simple calculation) that a person laundering clothing contaminated in this environment could be at an increased risk for asbestos-related disease. As a very rough approximation, if it is assumed that the airborne asbestos concentration during handling of clothing in the home is approximately 1/10th that of the airborne asbestos concentration in the workplace, and clothing is shaken three times per week for 5 minutes each time, then the possible intake would be [3 f/cc × (5 min/day) × (1 h/60 min) × (3 days/week) × (50 weeks/year) × 30 years]/(2000 h/occupational year) = 0.56 f/cc-year. This would suggest that such doses of chrysotile would be unlikely to cause an increased risk of mesothelioma to persons in the home; however, given the relative potency of crocidolite compared to amosite (approximately 100 times greater) and chrysotile (approximately 500 times greater), it is entirely possible that a person laundering clothing of a worker who experienced these concentrations of crocidolite could well be at increased risk of developing mesothelioma. More work is necessary, however, to better characterize the input variables, in particular the relationship between airborne concentrations in the home relative to the workplace, such that screening calculations could be performed in order to assess the reasonableness of a "cause-effect" relationship between laundering clothing and the likelihood of disease.

Ideally, in the future, a simulation study could be conducted where clothing is contaminated through a broad range of simulated "workplace" exposures to asbestos, ranging from 0.1 f/cc, to perhaps as high as 10 or 25 f/cc. Once the clothing was contaminated, a second person could simulate the household contact by handling and shaking the contaminated clothing. Such a study would provide the information necessary to relate airborne asbestos concentrations in a hypothetical workplace to concentrations generated during laundering or handling the contaminated clothing in the indoor environment, and to quantitatively characterize the likely risks of take-home exposures. Although these data would not address the fraction of household contacts who shook the clothing outdoors, it would go a long way in helping risk assessors to understand the cumulative doses among household contacts and compare those values to published "no observed effect" levels for disease, and perhaps even help to determine, on a case by case basis, whether disease is likely to be related to asbestos exposure or may have occurred spontaneously (e.g. two to three per million per year).

Conclusion

In recent years, toxic tort litigation involving asbestos claims has reached record levels; an increasing number of these cases involve claims of para-occupational

exposure. Our analysis indicates that, to date, the vast majority of household cases reported in the literature occurred among individuals living with one or more family members who worked in industries characterized by high exposures, nearly always to amphibole fibers, and frequently during the 1930s–1960s. Thus, using these cases to characterize the risks associated with exposures that have occurred since about 1975 is not recommended, since not only have workplace asbestos concentrations decreased as a whole, but the use of amphiboles has also dropped dramatically and regulations that forbid removal of contaminated clothing from the workplace have been in place since the early 1970s. While basic research conducted on particle or fiber adhesion to clothing indicates that most respirable fibers would (on a mass basis) generally be expected to remain attached to the fabric, more research is needed to understand the influence of fiber size, type, fabric surface characteristics, degree of fiber loading on the fabric (f/cm^2), presence of multiple layers of fibers, and the vigor of shaking of the clothing on fiber release.

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Declaration of interest

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