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Asbestos Exposure in the Research Laboratory

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by

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GLOSSARY

ACM = Asbestos-containing material(s)

ATSDR = Agency for Toxic Substances and Disease Registry

BDL = Below the Limit of Detection

CDC = Centers for Disease Control

CFR = Code of Federal Regulations

DHHS = Dept. of Health and Human Services

EMP = Elongated Mineral Particles

ERG = Eastern Research Group, Inc.

f/cc = Fiber per cubic centimeter (of air); same as f/ml.

Mt = million metric ton

NIOSH: National Institute for Occupational Safety and Health

OSHA: Occupational Safety and Health Administration

PBZ = Personal Breathing Zone sample

PCM = Phase Contrast Microscopy

PEL = Permissible Exposure Limit

SEER= Surveillance Epidemiology and End Results

TEM= Transmission Electron Microscope (/Microscopy)

TOXNET = Toxicology Data Network

TWA= Time-weighted average

ABSTRACT

Introduction: Asbestos has been employed in a myriad of industrial applications for more than a century. Of the 181 Mt of asbestos produced worldwide, over 31 Mt was consumed in the U.S. The production and consumption of this mineral eventually was found to cause asbestosis, lung cancer and mesothelioma. While the brunt of the human exposure to asbestos occurred in insulation work, many more workers in other jobs were expose to asbestos fibers. Very little is known about the potential exposure to asbestos in research labs.

Goal: In order to investigate potential research lab work exposures, we simulated lab work with various asbestos-containing items commonly found in research laboratories: 6 wire gauze pads, 3 gloves, 3 beaker tongs, and 3 Transite boards. All samples were analyzed by Phase Contrast Microscopy and, when appropriate, by Transmission Electron Microscopy and Polarized Light Microscopy.

Results: All tested items were confirmed by bulk sampling to have asbestos fibers in their composition. Exposures from the 7 wire gauze pads were significantly lower than the PEL and the excursion limit, the highest exposure concentration measuring 0.029 f/cc. For the 3 beaker tongs with asbestos sleeves, exposure was below the PEL for 2 of the 3, with one resulting in an exposure of 0.160 f/cc (8-h TWA = 0.01 f/cc). For the Transite boards had the highest exposures of all tested items, with a maximum concentration of 0.320 f/cc (8-h TWA = 0.02 f/cc).

Conclusion: Asbestos exposure in our simulated research lab work was significantly lower than any of the historical exposures associated with asbestos-related disease including cancer.

INTRODUCTION

Asbestos is a generic name given to the fibrous variety of six naturally occurring minerals that have been used in commercial products. Its importance in industry derives from its strength, heat resistance, flexibility, weavability, its ability to resist chemical and thermal degradation, and high electrical resistance. Though there is evidence of its use in pottery dating back to 2500 B.C., it was not until the 20th century that its use grew exponentially when new industrial applications for the mineral emerged. With technological developments such as the Hatschek machine in the 1900s and later technologies, asbestos could be made into flat and corrugated panels and into pipes, enabling its widespread use in construction and water-supply piping. The growing automobile industry incorporated asbestos in brakes, clutch components, and engine gaskets. These developments led to a rapid increase in asbestos use worldwide. Cumulative world production from 1900 through 2003 was about 181 million metric tons (Mt). Of this amount, the United States and Western Europe were the largest consumers, especially during the first two-thirds of the 20th century. However, the rest of the world soon caught up with Western countries in asbestos usage. Even though early consumption was massive and fast spreading worldwide, by the 1950s, it is estimated that close to half of the asbestos produced in the world was used in the U.S. By 1958, dubbed "the boon of humanity," asbestos was used in about 3,000 applications, including millboard and paper for electrical panels; textiles for insulating electrical wiring; spray-on products for protecting steel girders in buildings; reinforcing, heat-resistant fillers for plastics; fire-resistant roofing materials such as felts, shingles, and asphalt roofing compounds; inexpensive, durable, and dimensionally stable flooring products, such as vinyl tile and flooring

felts; heat- and acid-resistant gaskets and packings; thermal insulation on boiler systems for buildings and homes; fireproof suits for firefighters; reinforcement for plasters and caulking compounds; and filler, reinforcer in paints and asphalt road surfacing. Peak demand for asbestos was achieved around 1977, when about 25 countries were producing a total of almost 4.8 Mt annually**,** with about 85 countries manufacturing its products. In the United States, and to a lesser extent, in many European countries, due to public opposition, the use of asbestos-containing materials (ACM) waned in the early to late 1970s due to the growing research linking this wonder material to serious health effects (Virta, 2006). In general, of the six asbestos fibers, in the U.S., chrysotile was the predominant form used in the manufacture of ACM around the 1920s and 1930s; amosite gradually increased its presence in the market starting in the mid-1930s and was widely used by the U.S. Navy in marine construction and repairs between the 1940s and the 1970s. Starting in the1960s, chrysotile gradually re-emerged as the principal fiber type used in ACM, though it was often mixed with other forms of asbestos such as crocidolite and amosite. By the mid-1970s, as mentioned above, due to health concerns and increasing regulatory requirements, the asbestos contents in ACM, including insulation products, decreased significantly due to its being substituted by other insulation materials such as fiberglass (Williams *et al*, 2007).

EPIDEMIOLOGY OF ASBESTOS EXPOSURE AND ASBESTOS-RELATED DISEASE

The nature and extent of human exposures to asbestos in the United States have changed over the past 50 years. In the 1970s, most asbestos mining ceased in the United States, with the final mine closing in 2002. This ended more than a hundred years of production (1890s to 2003) of an estimated 3.29 Mt of asbestos. Additionally, in the 20th century, nearly 29.6 Mt asbestos were imported (mostly from Canada) into the United States for industrial uses (total U.S. asbestos consumption was > 31 Mt); about 98% of that amount being chrysotile, with amphibole asbestos being another two percent or so, ~1.2 % crocidolite, smaller quantities of amosite and anthophyllite (Virta, 2006). From epidemiological studies we know that insulation workers may have had the highest exposure to asbestos from among all asbestos workers, with this group reporting higher risk of lung cancer and mesothelioma. Based on time-motion analyses data, their exposure has been estimated to be close to 50% of their work time. It is also believed that non-insulators asbestos workers have been exposed less frequently and for shorter duration than insulators. In some crafts, the exposure would have been relatively low due to asbestos sometimes being encapsulated with protective coatings or embedded in adhesives, plastics, resins, and rubber matrixes that significantly decrease fibers air release. Such products include gaskets and phenolic molding materials, which were exempt from federal labeling requirement. Insulators working full-time with asbestos in heavy construction work had an estimated 8-h TWA of about 6 f/cc around 1965, and up to 15 f/cc during earlier periods; but the same group had a lower exposure during work at a (non-shipyard) steam-electric plant (6 f/cc) and at an oil refinery (~0.6 f/cc); for Navy shipyard workers (pipe coverers/insulators; at Puget Sound), the

annual asbestos exposure estimate was about 18 f/cc from 1962 to 1972. Some British studies have similarly large exposure estimates for insulator workers—between 7.3 to 256 f/cc; some of sampling for these studies occurred at the time of asbestos spraying. A summary of estimated exposures in shipyard and non-shipyard asbestos work is provided in Table 1 (modified from Williams *et al*, 2007). This is a heterogeneous group of studies spanning over a decade in the period covered. However, it can still be appreciated that there is a trend toward higher exposure in shipyard work compared to non-shipyard work.

Table 1. Historical Asbestos Exposures in Shipyard and Non-shipyard Work

	Approximate Exposure (f/cc)	
Non-shipyard Job Description	Pre-fab	>1, <20
	Mixing	>0.1, <10
	Application finishing	>0.1, < 100
	Removal	>0.1, <10
	Clean-up	>0.1, < 10
Shipyard Job Description	Pre-fab	< 40
	Mixing	>10, <600
	Application finishing	< 1
	Removal	>10, <800
	Clean-up	>0.1, <300

Currently, an undocumented amount of asbestos continues to be imported in products manufactured elsewhere, and a substantial amount of asbestos remains in existing buildings and manufactured products. An estimated 1.3 million construction and general industry workers in the United States are potentially exposed to asbestos each year, mainly from manipulation of asbestos during renovation or demolition activities. In terms of fiber-specific asbestos health effects, the National Institute for Occupational Safety and Health (NIOSH) estimates that 44,000 mine workers may have been exposed to asbestos or amphibole cleavage fragments during mining of some mineral commodities. In 1990, OSHA estimated that about 568,000 workers in

production and service industries, as well as 114,000 in construction industries might have been exposed to asbestos in the workplace. Health research conducted during the 1920s to 1940s revealed an association between exposure to asbestos and non-malignant lung disease including diffuse asbestosis (diffuse fibrosis of the lung), and localized or diffuse, acute or chronic pleural disease. Furthermore, in the late 1950s and early 1960s, a connection between asbestos exposure, lung cancer and mesothelioma, was established. National surveillance, cause-of-death data collected since the 1960s shows a positive trend over time for both, asbestosis and mesothelioma. During 1968--2005, *asbestosis* was identified as the underlying cause of death for 9,024 decedents, most of them male. The data shows that asbestosis mortality trails asbestos exposures due to the latency period between exposure and development of disease (usually one to two decades or longer) and because individuals can live with asbestosis for years before succumbing to it. Since the peak in exposure was late 1960s to 1970s, asbestosis deaths plateaued at 1,500 per year since 2000, with some already documenting a decrement since the late 1990s. *Malignant mesothelioma* deaths have been tracked by NIOSH since 1999 from death certificates from the National Occupational Respiratory Mortality System (NORMS). This data recorded a relatively steady increase in annual mortality in the period ending in 2005. There was a 9% relative increase when comparing deaths in 1999 (2,484) to those in 2005 (2,704). However, more recent data (from the Surveillance Epidemiology and End Results, SEER, 9 areas) shows a stable incidence rate for mesothelioma since 2006, again, indicating that new diagnoses for this malignancy have reached a plateau in the U.S. The later peak for mesothelioma compared to asbestosis has been explained by the cancer having a longer latency than the lung fibrosis. In addition to asbestosis and mesothelioma, lung cancer has been causatively linked in part to asbestos. However, surveillance cannot be done for lung cancer caused by asbestos due to the

absence of specific diagnostic criteria and a specific disease code for the subset of lung cancers caused by asbestos (Centers for Disease Control and Prevention, 2008, 2009; NIOSH, 2011; National Cancer Institute, 2011; Nelson, 2009). Based on historical trends and assumptions concerning current and projected exposure to asbestos (analogous to asbestosis), the annual number of male mesothelioma cases in the US is projected to decline to the background rate of approximately 500 cases per year around the year 2055. However, female cases (interpreted to represent background incidence) are projected to remain steady at approximately 500 cases per year (Price, 2004).

More recently, some authors have found links between asbestos and other cancers, including cancers of the larynx, pharynx, ovaries, stomach, colorectal tissue, and even some immune disorders. However, the link between asbestos and non-lung, non-mesothelioma malignancy is at best weak, to the point that many regard it as nonexistent altogether (Chan, 1998; Institute of Medicine, 2006; Williams *et al*, 2007; Gamble, 2008; Reid, 2011; Straif, 2012).

ASBESTOS DISEASE PATHOPHYSIOLOGY

Asbestos fibers can enter the body mainly after inhalation and minimally by the oral route. Ingested fibers are mostly excreted in the feces, some penetrate the gastric mucosa, and fewer are distributed to other tissues. The oral route poses no significant risk in those occupationally exposed to asbestos, and no study so far suggests ingested asbestos is carcinogenic. For this reason the rest of this section is predominantly dealing with inhalation exposure. Fibers deposited in the lung may have different fates depending on a number of factors, including health and comorbidities. For asbestos, the most important removal process is the mucociliary clearance. The clearance mechanism is adversely affected by cigarette smoke (and other airway toxicants). Chronic exposure to cigarette smoke has been shown to cause a prolonged impairment of particulate clearance from the bronchial region, increasing retention of asbestos fibers in the bronchi, leading to chronic inflammation of its epithelium. The risk of *lung cancer* is about ten times higher for smokers than for non-smokers, and is multiplicative to the risk from asbestos exposure alone. However, from the works of Muscat and Wynder (1991) and others, smoking does not influence the risk of *mesothelioma*. As alluded above, the results of human studies so far indicate that pneumoconiosis and cancer only occur after long-term inhalation of large amounts of asbestos fibers. These doses of asbestos were more common among asbestos products manufacturers and insulation workers (Muscat & Wynder, 1991; Nelson, 2009).

The mechanism at play in the pathogenesis of asbestos-induced disease, including malignant mesothelioma, is not well-characterized. For a particulate to cause long-term damage after inhalation, it must reach the alveoli (where it can have long retention times). Whether it

reaches respiratory lung tissue is determined mainly by its size. If the particle reaches terminal bronchioles, it causes a foreign body (inflammatory) reaction that would depend mostly on its dose, size and chemical composition. The lungs first try to remove the foreign substance by the action of resident macrophages. If the particle is within the size macrophages can phagocytize, it will be engulfed and removed by translocation to the airways (then expectorated or swallowed) or to the draining lymphatics. If the dose is too large for the local macrophages to handle, more macrophages are recruited to the site; acutely, if the system is overwhelmed, clinical signs develop. If the fiber is too large to be phagocytized (~13 μm in diameter in rats/hamsters; ~21 in humans), the fiber cannot be removed unless it is broken into shorter lengths or it dissolves. Whether by large size or dose, if macrophages are overwhelmed, an initial series of events occur in the location where most of the fibers are deposited (mostly at the junction of the terminal bronchioles/proximal alveolar duct). Additional macrophages (and neutrophils) are recruited; the local type II pneumocytes undergo metaplasia and become hyperplastic, leading to *bronchiolization* (with terminal airways appearance); this stage may be reversible. This results in increased mucous production and sometimes inspissation of the mucus-mix or granuloma formation. With time, local fibroblasts produce high amounts of collagen resulting in fibrosis of the lung tissue; this stage may be reversible in rodents (Eastern Research Group, 2003). Recent human tumor studies have described, among other cellular responses, altered cell signaling pathways, excessive activation of growth factor pathways, and molecular changes. Mechanistically, the pleural mesothelium may be involved in the following way. Macrophages recruited to eliminate the asbestos fibers are thought to induce an inflammatory cascade that may contribute to fiber translocation to the pleura. Once interacting with pleural mesothelial cells, fibers can be genotoxic (leading to base oxidation, DNA breaks, mutations, and aneuploidy);

may target cell proliferation (e.g. by loss-of-function mutations in p53); and could activate cellular surface receptors and increase growth factor expression. Translocation and retention of fibrous particulates from initial sites of pulmonary deposition to extrapulmonary sites such as the pleura are believed to be important aspects of their potential toxicity (Broaddus, 2011; Kane, 2006; NIOSH, 2011). Whether the fibrosis and other non-cancer changes provoked by asbestos are predictive of future cancer, the conventional answer is that there is no direct link. However, in animal inhalation studies, all fibers that have caused cancer have also caused fibrosis, while not all fibers that have caused fibrosis have caused cancer. Based on these facts, if we used fibrosis as a surrogate of significant dose intake, the minimum fiber burden required to cause cancer should be at least that required to lead to fibrosis.

Fiber size. In animal studies the incidence of malignant mesenchymal neoplasms was more strongly correlated with particles longer than 8 μm and no wider than 0.25 μm, especially for particles longer than 4 μm and no wider than 1.5 μm. This observation has led to the assertion that carcinogenicity of (durable) elongated mineral particles (EMPs) such as asbestos depends on dimension and durability (see below), rather than the physicochemical properties (the "*Stanton hypothesis*"). A review of data from animal models exposed by instillation or inhalation of EMPs of a defined size distributions, along with data on human lung fiber burden and associated effects concluded (consistent with Stanton's) that asbestosis is most closely associated with the *surface area* of retained EMPs; that mesothelioma is most closely associated with the number of EMPs *longer than ~ 5 μm and thinner than about 0.1 μm*; and that lung cancer is most closely associated with EMPs *longer than about 10 μm* (some regard the "critical" toxic length to be > 15 μm) and *thicker than about 0.15 μm*. A separate analysis concluded that fibers and bundles longer than 5 μm and thinner than 0.4 μm contributed to lung tumor risk; very long (>40 μm) and very thick (>5 μm) complex clusters and matrices possibly contribute to cancer. Although structures <5 μm long did not

contribute to lung tumor risk, potency of thin (<0.4 μm in diameter) structures increased with increasing length above 5 μm, and s*tructures >40 μm long were estimated to be about 500 times more potent than structures between 5 and 40 μm long*. Fibers with a length of 20 μm or longer and a diameter of 0.1-0.25 μm very likely have the highest relative carcinogenic potency, which decreases with decreasing length and/or increasing diameter. Further (indirect) evidence supporting that shorter fiber sizes (< 5-8 μm) are not carcinogenic comes from lymph node studies in animals. It has been observed that the hilar lymph nodes that drain the lungs of animals exposed via inhalation to asbestos get literally filled with macrophages containing short fibers and fiber fragments. However, there has been no evidence of pathology or neoplastic change in either the lymph nodes or adjacent tissues. The preponderance of the evidence supports the assertion that particles < 5 μm in length are cleared with similar kinetics and mechanism as isomorphous particles. Nonetheless, and for completion, a minority of researchers believes that short "fibers" (<5 μm) are also carcinogenic (some dispute calling < 5 μm long structures a "fiber," arguing this length structure behaves as a "particle"). One of those studies, the one by Suzuki, examining human tissue, concluded that short-thin fibers appear to contribute to the causation of mesothelioma. A review of the animal asbestos studies and some data from studies of human tissue concluded that asbestos fibers of all lengths induce pathological responses (NIOSH, 2011; Broaddus, 2011; Suzuki, 2005; Bernstein, 2006).

Fiber type, durability, and concentration in relation to potency. Though it is difficult to separate fiber size (esp. length) from the overall asbestos toxic profile, there appears to be significant differences in asbestos pathologic potential linked to the type of fiber (inhaled). Chrysotile is mineralogically distinct from the amphiboles, possessing a very different chemical structure. It has been shown that chrysotile is rapidly removed from the lungs following inhalation in experimental animals, and that lung fibrosis is better correlated with tremolite than

chrysotile asbestos concentrations. Consistent with the above, McDonald *et al* found the risk of mesothelioma was significantly related to concentrations of *amphibole* fibers longer than 8 μm and that fibers shorter than 8 μm accounted for none of the cancer risk**.** A second study, by Rogers *et al* reported that mesothelioma risk was greatest for *crocidolite* asbestos fibers longer than 10 μm, followed by *amosite* asbestos fibers longer than 10 μm, and then by *chrysotile* fibers less than 10 μm. The authors suspected that the relative risk for chrysotile fibers less than 10 μm resulted from *longer fibers breaking into shorter fibers* (McDonald, 1989; Rogers, 1991; Bernstein, 2013).

The risk of developing lung cancer (latency period ~ 10 years) is approximately linearly related to the duration and intensity of exposure. The risk of mesotheliomas is linearly related to the intensity of exposure but exponentially related to the time from onset of exposure. Distinct from fibers' exposure chronicity (at the worksite) is the time of fiber stay or biopersistence in human tissue. Biopersistence of fibers in the lung parenchyma influences the fiber dose that is ultimately translocated to the pleura. Persistence in the lung is dependent on (1) site and rate of deposition, (2) pulmonary clearance parameters, (3) solubility in lung fluids, (4) breakage rate and patterns, and (5) rates of fiber translocation (to farther locations, e.g. the pleura) and retention. Surface chemistry and diameter are important determinants of solubility. However, much of the knowledgebase concerning the role of biopersistence is actually derived from studies of synthetic vitreous fibers and not from asbestos fibers *per se* (Broaddus, 2011). The persistence of fibers in the lung and pleura is thought to be influenced by the structure of the asbestos itself. The thin, rolled or concentric sheets that form the chrysotile fibers leads to the ability of the lung macrophage system to decompose the chrysotile fibers once inhaled, a phenomenon seen in the biopersistence studies of commercial chrysotiles. This finding is

substantiated by mineralogical and *in vitro* studies (NIOSH, 2011). It is believed that in the environment created by macrophages (esp. its acidic pH), chrysotile fibers disintegrate to structures more akin to "amorphous" silica. In contradistinction, when amphibole fibers break down, their resulting structures are still fibers that are highly resistant to neutral or acid dissolution. Clearances half-lives have been recognized in the literature from various studies. For fibers either > 20 and 5-20 μm in length, respectively, clearance half-life has been fo**u**nd to be ~0.3 and 7 days for Calindia chrysotile, 0.3 and 2.4 days for Brazilian chrysotile, 11.4 and 29.7 days for Canadian chrysotile, 418 and 900 days for amosite, 536 and 262 days for crocidolite, while amosite persisted for the length of the evaluation (Bernstein, 2006).

ASBESTOS REGULATION

Many characteristics are used to identify asbestos minerals including their crystal structure, chemical composition, and growth habits. As mentioned above, the term *asbestos*, rather than a single chemical species, defines naturally formed silicated minerals and associated cations (e.g. Na, Mg, Ca, and Fe), with a particular fibrous form (the *asbestiform* crystalline "*habit*") from the serpentine and the (double-chain) amphibole mineral groups. There is only one *serpentine* asbestos: chrysotile; the *amphibole* group has five members: crocidolite (riebeckite), amosite (cummingtonite-grunerite), actinolite, anthophyllite, and tremolite (Nelson, 2009; Berman, 2008). The regulatory *definition* of asbestos used by the Occupational Safety and Health Administration (OSHA) includes the six silicates above and "any of [them] that has been chemically treated or altered." By asbestos "*fiber*" (the regulated form), OSHA refers to a particular form of asbestos that is 5 μm or longer in length, with a length-to-diameter ratio of at least 3 to 1 (when viewed microscopically using NIOSH Analytical Method #7400 or its equivalent). OSHA has the following three standards to protect workers from exposure to asbestos in the workplace: 29 CFR 1926.1101which covers construction work (e.g. alteration, repair, renovation, and demolition of structures containing asbestos); 29 CFR 1915.1001 which covers asbestos exposure during work in shipyards; and 29 CFR 1910.1001 which applies to asbestos exposure in the general industry, such as exposure during brake and clutch repair, custodial work, and manufacture of asbestos-containing products. The OSHA workplace *Permissible Exposure Limit* is 0.1 fiber/cc of air measured as an 8-hour Time-Weighted-Average

(TWA), and with an *Excursion Limit* of 1 fiber/cc, averaged over 30 minutes. (Asbestos, 2012; OSHA, 2013; Nelson, 2009).

The potential for asbestos exposures exists virtually everywhere, including older homes (Fig. 1; from Environmental Assistance and Protection-North Carolina, 2013).

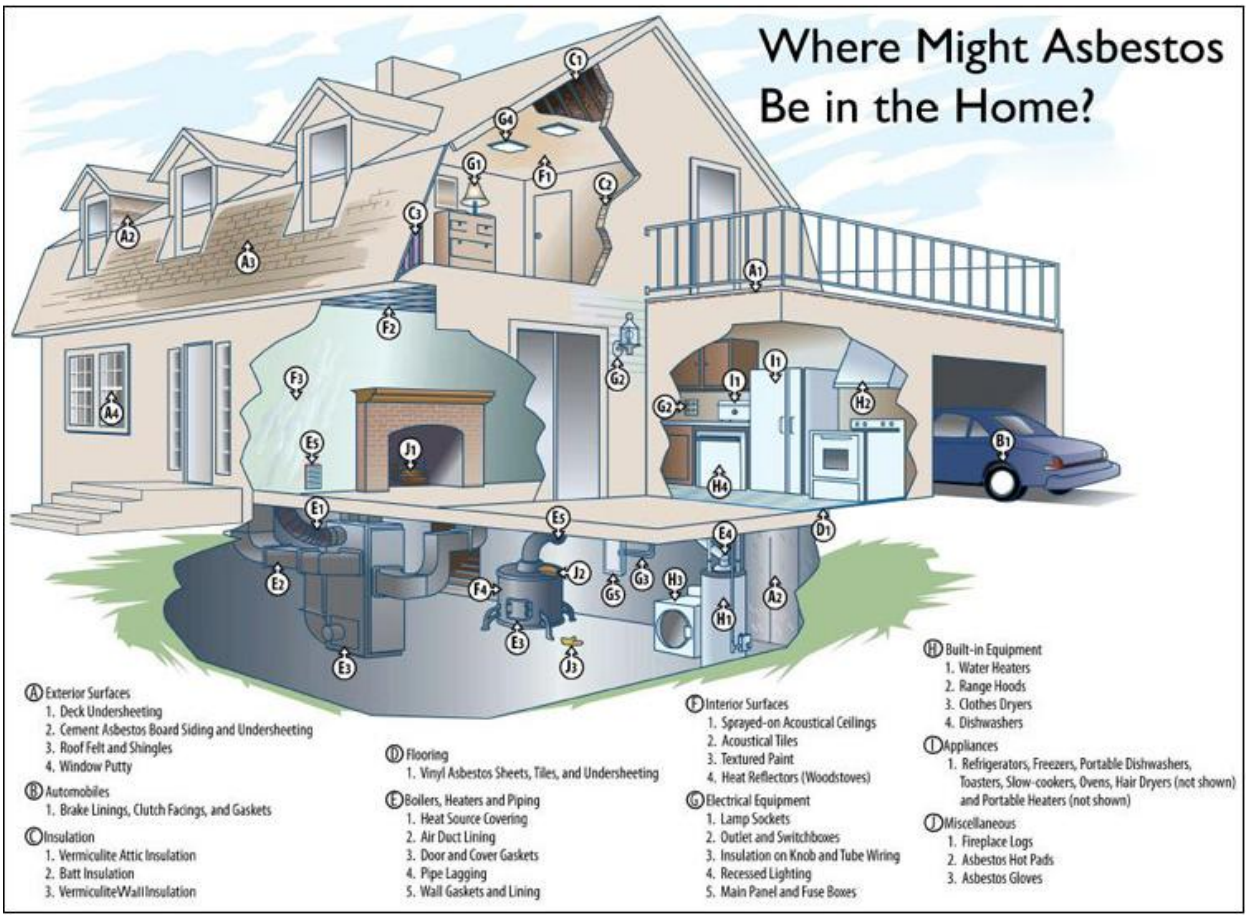

Figure 1. Potential Asbestos Sources in Homes (Environmental Assistance and Protection – North Carolina)

Even a limited sampling of the catalogs from companies that supplied research laboratories by the 1970s, shows the multitudinous variety of asbestos lab supplies, including tongs, insulation sleeves, gloves/mittens, wire gauzes, and Transite boards (Sargent-Welch, 1979; UT-Arlington, 2013).

Recently there have been concerns that exposure to asbestos outside the high exposure, insulation environment may have been high enough to lead to asbestos-related disease, specifically lung cancer and mesothelioma. Among these low-exposure occupations, we find brake mechanics and laboratory workers. A number of studies have aimed at reproducing detailed work settings in which these exposures may have occurred in the past, and where asbestos personal breathing zone (PBZ) samples have been taken. A number of the research articles report personal exposures above current regulatory levels (but still lower than historical PELs) as evaluated by Phase Contrast Microscopy (PCM). Selected studies are summarized in Table 2. For this table, when available, only Transmission Electron Microscopy, or TEM, data is included. PCM measures total fibers, and unlike TEM, cannot discriminate between asbestos and non-asbestos fibers.

Table 2. Exposure in Various Low Asbestos-exposure Jobs

Specific Job	**PBZ Sample Size (n)**	**Exposure 8-h TWA (f/cc)**	**PEL (f/cc)**	**Does it exceed PELs? (Yes/No)**	**References**	**Notes**
Light Aircraft Brake Replacement	9	<0.003	0.1	No	(Blake, 2009)	
Automobile asbestos-containing sealants & clutch replacement	3	0.0063-0.019 [TEM]		No	(Blake, 2008)	
Automobile asbestos-containing clutch repair	15	$3.75x10^{-5}$ - 0.03		No	(Cohen, 2008)	
Mitts wear in glass manufacturing	33	0.03 - 0.55		Yes	(Cherrie, 2005)	The higher concentration had “no vent.” (< 5 air exchanges/h)
Glove wear in (glassware) sterilization work	8	0.07 – 0.99		Yes	(Samimi, 1981)	“unventilated rooms”

To-date few studies have documented exposure assessments in the research lab. And the few published articles have included only one type of product, so far gloves and mitts. In order to assess potential exposure risk while performing research in labs with asbestos-containing supplies, we conducted a series of experiments in an enclosed environment such as an environmental chamber (Fig. 2); or testing included wire gauze pads (Fig. 3), gloves (Fig. 4), tongs (sleeves or socks; Fig. 5), and Transite (Fig. 6).

Figure 2. Chamber for Test 3

Figure 3. Wire Gauze Sample

Figure 1. Transite Being Drilled

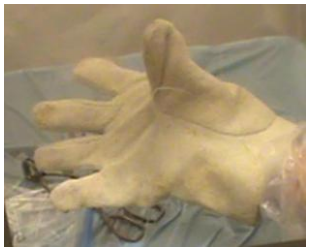

Figure 4. Glove Being Handled

Figure 6. Tong with Sleeve

Our goal was to better understand the potential asbestos exposures to which workers would have been exposed in these settings. The conditions we simulated were expected to generate exposures under "a worst case scenario," in conditions intended to be more austere than the average exposure in the simulated setting.

MATERIALS AND METHOD

For Test 1, the evaluation was conducted in a chamber with a working area measuring close to 360 ft^3. These test conditions provide a worst-case exposure scenario that hoped to overestimate the exposures compared to real-life, routine lab work. Background or baseline air sampling was performed with the ventilation off. The simulation equipment: workbench; four tripod-mounted Bunsen burners with their propane supply; four Erlenmeyer flasks; four wire gauze pads (size = 4 x4 in) and the sampling equipment.

For Test 2, the second experiment was performed in what was intended to be a standard working condition in laboratory room with a workbench with adequate ventilation for the size of the test room. Equipment used included gauze pads, ringed stand, flasks filled with water, and Bunsen burners.

In Test 3, the experiment was conducted in a chamber (dimensions = 3 x 7 ft) with an enclosed inner area inside which all tests were conducted. Each item was individually tested with cleaning of chamber between tests. Other materials employed consisted in a small working table, and depending on the test, supporting equipment: a stand with ring, Erlenmeyer flasks with water, Bunsen burner; a pair of forceps and flasks for manipulation with the test gloves on; hammer, drill, chisel, and pliers to work with the Transite.

Sampling was performed in accordance with NIOSH 7400/7402 recommendations: mixed cellulose ester membrane filters with 0.8 μm pores inside 25 μm diameter electrically conductive, extended cowl cassettes. PBZ Samples flow rates were in the 2.5-2.9 L/min range for Tests 1 and 2, and 15 L/min for Test 3. Area samples, in Tests 1 and 2, were set between 6-9.5

L/min flow rates, at 5 feet above floor level and 1.5 meters from the workbench. All PCM-intended sampling had a corresponding TEM sample obtained. All tested materials for Test 1 (Table 3), Test 2 (Table 4) and Test 3 (Table 5) had a pre-test bulk sampling performed. Tests 1 and 2 also had a post-test (post-heating) bulk sampling performed. For the 3 Tests, the bulk samples were analyzed by Polarized Light Microscopy.

For Test 1, four newly purchased, asbestos-containing gauze pads were evaluated. These pads were in a wire mesh, covered with a 3.5-inch diameter pad. Air samples were collected in the pre-test, intra-test (when heated) and post-test periods, as follows.

For the *pretesting* air sampling, 5 area samples were collected from inside the chamber and 3 from its outside, over a minimum of 120-minutes period. Six-area**,** *intratest air samples* were collected inside and 2 outside the chamber. Two PBZ samples were collected, 1 sample per person--starting with the opening of the gauze pad and covering the actual burn test, which occurred immediately after retrieving the pads from the packages; 1 set of samples stopped at 4 and the other at 6 hours after starting collection; another set stopped after a 30-min period, measuring possible excursion exposures. The 2 longer periods (4 and 6 hours) sampled air at the time just before beginning the simulation and after the manipulations and heating ceased. The Technician's (PBZ) sample (the person) was relatively mobile during the test, while the Assistant's (PBZ) sample (the person) was relatively stationary during sampling.

For Test 2, two gauze pads were placed on ring stands, and flasks filled with water were placed on them. The water boiled for 30 min, 1 hour, and 2 hours. The 30 minutes tests were run twice. Therefore, there were a total of 4 independent heating (burning) runs. The 30 min runs had a pair of PBZ samples and 3 area samples. The 1-h and 2-h runs each had one 30 min sample

(excursion) and 2 TWA samples. A total of 10 PBZ samples were collected. There was 12 area samples collected, a set of 3 per run: one on the test bench, the other flanking the bench 1.5 meters away from the bench. These were meant to quantify what would be considered bystander exposure.

For Test 3, the sampling filters were placed 18 inches above the worktable at the same length to the person's nose but right above the test material. Each sample was 30 min long. There was one *cumulative sample* that was run in parallel to all the individual 30 min tests (4.5 hour total cumulative exposure). We manipulated/tested 1 used wire gauze pad, 3 used gloves, 3 used tongs (with asbestos-containing sleeves), and 3 Transite boards. During each test, participants manipulated the item for the entire 30 minutes, and >50% of the time some more "aggressive" handling was applied. At the end, in all testing runs, there were visible fibers on the table that broke off from the test item. The Transite manipulation included drilling between 12-18 holes, cutting with saw, chiseling, and smashing it with a hammer; at the end of the Transite manipulation, there was dust spread over both horizontal and vertical surfaces (sheeting) in the chamber.

RESULTS

For the bulk sampling in Test 1, out of the 4 gauze pads, 1 specimen per pad before heating and 3 specimens per pad after the heating were cut off and sent for bulk analysis. All samples contained between 75% and 85% chrysotile asbestos, in addition to small quantities of calcium carbonate, and even smaller amounts of mineral wool (Table 3).

Table 3. Bulk Sample Results for Test 1

Pad Number	Time/Place Sampled on Pad	Asbestos % Type	Other Fibers	Particulate
1	Before study	80% Chrysotile	10% mineral wool	10% Ca Carbonate
	After Study/Center	75% Chrysotile	N/D	Binder/Filler
	After Study/Middle	75% Chrysotile	N/D	Binder/Filler
	After Study/Edge	85% Chrysotile	N/D	Binder/Filler
2	Before study	80% Chrysotile	5% Mineral Wool	15% Ca Carbonate
	After Study/Center	75% Chrysotile	N/D	Binder/Filler
	After Study/Middle	75% Chrysotile	N/D	Binder/Filler
	After Study/Edge	80% Chrysotile	N/D	Binder/Filler
3	Before study	85% Chrysotile	5% Mineral Wool	10% Ca Carbonate
	After Study/Center	75% Chrysotile	N/D	Binder/Filler
	After Study/Middle	75% Chrysotile	N/D	Binder/Filler
	After Study/Edge	80% Chrysotile	N/D	Binder/Filler
4	Before study	80% Chrysotile	10% Mineral Wool	10% Ca Carbonate
	After Study /Center	75% Chrysotile	N/D	Binder/Filler
	After Study/Middle	75% Chrysotile	N/D	Binder/Filler
	After Study/Edge	75% Chrysotile	N/D	Binder/Filler
N/D = No other fiber detected				

However, though the overall result of its heating was a decrease by 5% in asbestos contents, pads 1 and 2 had a 5% increase each in the "Edge" specimens. Again, all other pre-/post bulk analyses showed a decrease of 5% in asbestos after subjecting the pads to heating.

For the bulk sampling in Test 2, the samples were removed from each wire gauze pad, 1 specimen before (new pads) and 2 specimens after the tests (similar to Test 1). The post-heating specimens were removed from the "center" and from the "edge" of the pads. There was more than 50% decrease in tremolite asbestos contents after the burning: from 5% when new, to 2% after heating. Talc was also reported as a non-asbestos material in the composition of the pads (Table 4).

Table 4. Bulk Samples Results for Test 2

Item Analyzed		Sampling Origin	**Asbestos**	**Non-Asbestos**
New Pads		Pad 1	Tremolite 5%	N/D
		Pad 2	Tremolite 5%	N/D
Pads After Heating	Pad 1	Center	Tremolite 2%	Talc 2%
		Edge	Tremolite 2%	Talc 2%
	Pad 2	Center	Tremolite 2%	Talc 2%
		Edge	Tremolite 2%	Talc 2%
N/D = No other fiber detected				

For the bulk sampling in Test 3, the 10 samples tested (wire gauze, gloves, tongs, and Transite) were found to have a variable amount of asbestos (Table 5). All items tested had between 15% to 45% chrysotile asbestos, except for the Transite, which had only 10% chrysotile (in addition to 5% amosite). Cellulose was the most abundant non-asbestos material for all samples except from the Transite (which also contained cement). From one large Transite board, 3 smaller size boards were sectioned and were individually tested for sample runs 8-10.

Table 5. Bulk Samples Results for Test 3

Bulk Sample Designation	Sample Run Order	Item Type	Chrysotile Asbestos (unless otherwise specified)	Cellulose
14B	1	Wire Gauze Pad	15%	Non-fibrous other
3	2	Glove	40%	50%
9	3	Glove	35%	55%
5	4	Glove	30%	65%
6	5	Tong	45%	50%
7A	6	Tong	40%	55%
12A	7	Tong	35%	55%
11*	8	Transite	10% (5% Amosite)	Cement
11*	9	Transite	10% (5% Amosite)	Cement
11*	10	Transite	10% (5% Amosite)	Cement
*11--These represent the same bulk sample from which the tested boards for runs 8-10 were obtained				

Regarding the Test 1, *pretest*, background area samples, in summary, there was no asbestos in any of the 5 area samples from inside the chamber (Table 6) or the 3 environmental samples from outside the chamber (data not shown). Specifically, the TEM analysis reported the sample concentrations as below the limit of detection.

Table 6. Background Pretest Samples (Inside Chamber) for Test 1

Statistics	PCM (f/cc)	AHERA TEM (s/mm2)
Maximum	0.005	<16.00
Minimum	0.002	<16.00
Count	5	5
AHERA = Asbestos Hazard Emergency Response Act; per this standard, TEM results are reported in asbestos structures/squared millimeter		

The 6 *intratest*, area samples taken during the experiment (intratest) showed an average fiber per milliliter (f/cc) of 0.0076 by PCM and 0.0010 by TEM, with a range concentrations of 0.0069 -- 0.0085 by PCM, respectively. By TEM, there was a range of asbestos exposure concentration of 0.0017 -- <0.00091, respectively (Table 7).

Table 7. Intratest Area Samples for Test 1

Statistics	PCM (f/cc)	TEM (f/cc)
Maximum	0.0085	0.0017
Minimum	0.0069	<0.00091
Count (n)	6	6

As stated earlier, each participant (Technician and Assistant) had 2 personal sampling devices on them. One of these cassettes collected PBZ air for 4 hours inclusive of the experimental period; the other device, simultaneous with the 4 hours sample, continued to sample for an additional 2 hours (Table 8). There were no asbestos fibers found in any of the personal samples.

Table 8. Intratest PBZ Samples at 4- and 6-hour for Test 1

Sample	PCM (f/cc)	TEM (f/cc)
Technician-6 h	0.012	<0.0026
Assistant- 6 h	0.0053	<0.0026
Technician- 4 h	0.018	<0.0040
Assistant- 4 h	0.013	<0.0041
Count (n)	4	4
TEM samples were all below detection limits		

For the short-term (excursion) samples (Table 9), two sets of 30-min PBZ samples were obtained from the Technician and Assistant over 2 serial 30 minutes that covered the first 2 burn times and pad manipulations. These were intended to collect air over the expected highest exposure, attempting to capture surrogates of the OSHA excursion limit. Their range, average and standard deviation are reported. No fibers were detected in any of these personal air samples by PCM (all 3 samples were < 0.032 f/cc); therefore, no TEM analysis was performed for them.

Table 9. Lab Technician, Assistant PBZ Excursion Levels for Test 1

Statistics	PCM (f/cc)	TEM (f/cc)
Maximum (T*)	<0.032	N/A
Minimum (T)	<0.029	N/A
Count (n; T)	2	0
Maximum (A)	<0.032	N/A
Minimum (A)	<0.029	N/A
Count (n; A)	2	0
T=Technician sample; A= Assistant sample; N/A = Not analyzed by TEM		

None of the samples from Test 1 were found to have a fiber count above the current PEL of 0.1 f/cc.

In case of the exposure samples from Test 2, when analyzed using PCM, 3 of the PBZ samples (for the 2-h burn test) showed the presence of airborne fibers (TWA of 0.033, 0.0048, 0.0054 f/cc), none of which were found to have asbestos (TEM examination = No fibers). The rest of the samples (1/2-h and 1-h burn runs) were found to have no fibers or BDL for PCM (Table 10).

Table 10. PBZ Samples for Test 2

Sample Time	Sample Type	PCM (f/cc)	TEM Fiber Ratio (%)	Asbestos Adjusted PCM (f/cc)
2-h Burn Test	8-h TWA	0.033	0	0
	8-h TWA	0.048	0	0
	Excursion	0.054	0	0
1-h Burn Test	8-h TWA	<0.014	N/A	N/A
	8-h TWA	<0.014	N/A	N/A
	Excursion	<0.027	N/A	N/A
½-h Burn Test	8-h TWA	<0.030	N/A	N/A
	8-h TWA	<0.028	N/A	N/A
½-h Burn Test	8-hTWA	<0.030	N/A	N/A
	8-h TWA	<0.029	N/A	N/A
N/A=Not analyzed by TEM TWA = Time-weighted average				

For the area samples (Table 10), only 5 samples were found to have fibers by PCM: 3 of the 2-h burn tests, 1 of the 1-h tests, and 1 sample out of the ½-h test sample pair. Only 1 sample (from the 2-h tests; the Test Bench specimen) was found to have 55% asbestos among its fibers. This sample was then shown to have a TEM-adjusted PCM count of 0.029 f/cc. The other 4 area samples with or without fibers visualized by PCM had no asbestos fibers when analyzed by TEM.

Table 11. Area Airborne Fiber Concentrations for Test 2

Sample	Location	PCM (f/cc)	TEM Fiber Ratio %	TEM-Adjusted Asbestos Fiber Concentration (f/cc)
2-h Burn Test	TB*	0.042	55%	0.029
	NE* Corner	0.029	0	0
	SE* Corner	0.050	0	0
1-h Burn Test	TB	<0.014	N/A	- -
	NE Corner	<0.016	N/A	- -
	SE Corner	0.028	0	0
½-h Burn Test	TB	<0.034	N/A	- -
	NE Corner	<0.032	N/A	- -
	SE Corner	<0.032	N/A	- -
½-h Burn Test	TE	<0.030	N/A	- -
	NE Corner	<0.032	N/A	- -
	SE Corner	0.056	0	0

*TB=Test Bench; NE=North East; SE = South East; N/A = Not analyzed by TEM
"- -" = Samples that were not analyzed by TEM

Similar to Test 1, all Test 2 fiber-positive samples, whether area samples or PBZ samples, were found to have a fiber count below the PEL of 0.1 f/cc.

In Test 3, all sample exposure measurements obtained were meant to represent PBZ samples (also reported as the regulatory adjusted 8-h TWA; Table 12). Out of one wire gauze sample, three tongs (sleeves), three gloves, and three Transite samples, we had 3 samples with identifiable fibers under PCM. Of those, sample 1, the wire gauze pad had fiber quantities BDL

(<0.006 f/cc) by PCM and TEM analysis. Samples 5-7, the tongs, had exposure concentrations by PCM of 0.056 to 0.058 f/cc; tong sleeve sample 6, however, was dust overloaded. In the TEM results for the tongs, out of the 3 samples, 2 samples (#5 and 6) had an undetectable fiber load (<0.006 f/cc); sample 7 had a fiber count of 0.160 f/cc. For the Transite samples, all the PCM samples were unreadable due to being overloaded with dust; the Transite TEM samples 8 and 10 had an exposure concentration of 0.210 and 0.320 f/cc, respectively. The Transite TEM sample 9 could not be read due to being overloaded with dust. Extrapolating the 8-h TWA, assuming the exposures occurred once in an 8-h work period, are also reported. To obtain this TWA column, the values from the PCM and TEM columns were divided by 480 (minutes) and then multiplied by 30 minutes. The gloves (samples 2-4) and the composite (sample 10), were unreadable, whether by PCM or TEM, due to being dust overloaded.

Table 12. PBZ Samples for Test 3

Sample #	Sample Type	PCM (f/cc)	TEM (f/cc)	8-h TWA (PCM/TEM)
1	Wire Gauze pad	<0.006	<0.006	BDL / BDL
2	Glove	- -	- -	N/A
3	Glove	- -	- -	N/A
4	Glove	- -	- -	N/A
5	Beaker tong sleeve	0.056	<0.006	Not applicable
6	Beaker tong sleeve	- -	<0.006	N/A / BDL
7	Beaker tong sleeve	0.058	0.160	0.0036 / 0.01
8	Transite	- -	0.210	N/A / 0.013
9	Transite	- -	- -	N/A / N/A
10	Transite	- -	0.320	N/A / 0.02
Composite		- -	- -	N/A / N/A

"- -" = Samples that were overloaded with dust
N/A = samples that were *not analyzed* by TEM
BDL = Sample results below the limit of detection

DISCUSSION

Scant literature exists on the asbestos exposures from research laboratory ACM. In our lab we evaluated various ACM, including wire gauze pads, tongs (sleeves), gloves and Transite. In Tests 1 and 2, the wire gauze pads were purchased new. In Test 3, all items were previously used. All the test items for all experiments were verified to contain asbestos by bulk sampling, either for Test 1 (Table 3), Test 2 (Table 4), and Test 3 (Table 5).

In Test 1 (Table 3) and Test 2 (Table 4), we performed pre- and post-heating bulk sampling. In Test 1 (Table 3), the change in asbestos pads contents was very minimal, and mostly decreased. However, for Test 2 pads (Table 4), there was a significant decrease (>50%) in asbestos. The percent of asbestos (tremolite) in Test 2 pads was relatively low (5%) from the start, compared to >70% asbestos (chrysotile) in Test 1 pads. What caused the differences in asbestos contents in the two tests? It is difficult to tell, however one wonders if the difference is a function of asbestos fiber types having a different resistance profile to degradation by fire, or that materials with small amount of asbestos from the start tend to lose their asbestos faster. Interestingly, the Test 2 pre-heating samples were 5% tremolite without talc being reported with it. After the burning, coincidental with the 3% decrease in tremolite, now a 2% talc composition was reported. Is the appearance of talc proportional to the decrease in tremolite is difficult to ascertain. It has been observed from research with automobile engine gaskets that asbestos contents in chrysotile-containing gaskets may decrease with time due to the “baking” caused by thermal stress from heat (Blake, 2006). Whether this is a function of the gaskets being heated, or

a function of the pressure placed the gaskets by their overlying load plus heating combined, we are unable to determine.

In their research with mitts, Cherrie *et al* found that exposures to clean but well-worn gloves released more fibers than brand-new gloves, and that fiber emission decreased with increased surface soiling (Cherry, 2005). Unfortunately, in our project we had dust overload in the glove samples, and are therefore, unable to determine the possible exposures generated with these ACM. However, we did identify asbestos in these gloves by bulk sampling (Table 5).
We conducted 2 tests exclusively to evaluate wire gauze pads. Both experiments, Tests 1 and 2, showed very low airborne asbestos fibers released from these ACM. Though the pads in Test 1 and Test 2 contained different types of asbestos—chrysotile versus amosite, the exposure profile was similarly low.

For Test 1, the background environmental (area) samples, whether pre-heating (Table 6) or in the intratest heating/manipulation (Table 7), we found the samples to have asbestos exposures significantly lower than the PEL. The maximum exposure measured in the 5 environmental samples was 0.005 f/cc. The TEM results were BDL (Table 6). The 6 intratest area samples (Table 7) also showed a maximum of 0.0069 f/cc by PCM and 0.0017 f/cc by TEM, both several-fold below the PEL. The 4 PBZ samples that measured the 4h and 6h periods (Table 8) showed the largest exposure value in the Technician 4 h sample (0.018 f/cc, by PCM), and the lowest in the Assistant 6 h sample (0.0053 f/cc, by PCM). The corresponding TEM samples all showed exposures BDL (Table 8); for this reason no TWA was calculated for this set. The 2 excursion exposure samples (Table 9) showed a maximum exposure BDL (<0.032 f/cc) for both the Technician (T) and the Assistant (A). For this reason, no TEM analysis for these cassettes

was performed (Table 9). For all Test 1 exposure measurements, whether environmental, area, or personal sampling, exposures were significantly below the PEL.

For Test 2, The PBZ samples were BDL except for the 2 h burn tests, with a maximum (excursion) of 0.054 f/cc, which decreased to 0.048 and 0.003 f/cc in the TWA-equivalent samples; however, there were no asbestos fibers detected in the TEM cassettes (Table 10). The rest of the PBZs, those from the ½-h and 1-h heating runs were all BDL (Table 10). The area samples, had a 2 h exposure measurement with 0.042 f/cc by PCM, but only 55% of those were asbestos by TEM; this yielded a TEM-corrected concentration of 0.0029 f/cc; the rest of the area samples, ½ h, 1 h, and 2 h, either had no asbestos by TEM or had a BDL concentration by PCM (Table 11). In summary for Test 2, there was either an exposure significantly below the PEL or no airborne asbestos at all in the samples.

For Test 3, there was only one (used) wire gauze pad. For this pad, the exposure analysis showed exposures BDL, which is consistent with the low exposure findings with the wire gauze pads in Tests 1 and 2. Two researchers quoted earlier (Table 2) have found different magnitude of exposure to asbestos from mitts and gloves. Samimi, testing gloves in sterilization work (glassware autoclaving) found exposures to asbestos overall higher than the current PEL (ranged from 0.07 to 2.93 f/cc; Samimi, 1981). Similarly, though in general lower than Samimi's exposure levels, Cherrie *et al*, in research with (new and used) mitts, found exposures below and above our current PEL (ranged from 0.03 to 0.55 f/cc; Table 2; Cherrie, 2005). Neither Samimi's nor Cherrie's articles reported employing TEM in their sample analysis. Their results were not characterized beyond total fiber concentration; for this reason, we cannot determine if the reported exposure by these two researchers was actually from asbestos fibers, or what fraction of their samples was actually asbestos. We are also unable to determine from Cherrie's or Samimi's

articles if the mitts or gloves manipulation was equivalent or similar to our sample manipulation. Furthermore, we are not able to compare our exposures with theirs as our glove cassettes (samples 2, 3, and 4; Table 11) were overloaded with dust, rendering quantification of exposure impossible by the prescribed NIOSH methodology. The gloves we included in our experiment were old and stressed, one of them with its inner liner detached from the inside. Since we went out of our way to handle them "aggressively," we ended up generating large amounts of dust, perhaps contributing to their being overloaded with dust. For future experiments, especially if using these same gloves, either a slower pump flow rate or a shorter sampling time should be used to address this hurdle. The tong sleeves analyses (Table 11) showed relatively low exposures for two of the PCM readings, the larger exposure of the two being 0.056 f/cc. This amount, assuming a single episode of such exposure in 8 h, yields a TWA of 0.0036 f/cc. If we assume four times as much exposure (4 x 0.0036 f/cc) in an 8-h period, we still have an exposure significantly below the PEL or 0.014 f/cc. Similarly negligible exposures are reported by TEM on tong samples 5 and 6. However, in one tong sample (#7), the TEM exposure was 0.160 f/cc, approximately 63% above the current PEL of 0.1 f/cc. If we had one such exposure in an 8-h period, the TWA would be 0.01 f/cc, one order of magnitude below the current PEL. Even so we still need to keep in mind that it is possible, under very aggressive handling conditions, to generate enough airborne asbestos fibers for exposures above the PEL. The Transite board sampling (Table 11) yielded 4 unreadable (overloaded) samples (3 for PCM and 1 for TEM). Only 2 of the 3 TEM samples (#8 and 10) yielded a quantifiable exposure for these boards. These exposures ranged between 0.210 (sample 8) and 0.320 f/cc (sample 10), both approximately double and triple the PEL level, respectively. However, if this aggressive board manipulation is performed once in an 8-h period, the exposure would be below the PEL in both

cases: 0.013 f/cc for sample 8 and 0.02 f/cc for sample 10. It is unlikely that one would need to break/shape these boards more than once daily, if at all. More likely than not, the typical lab usage of Transite would occur sporadically as labs often recycle their customized Transite for different experiments. The cumulative sample, the "composite," was also overloaded with dust beyond analysis by either PCM or TEM.

In summary for Test 3, in our simulation we attempted a "worst-case-scenario" ACM manipulation. All exposures 8-h TWA concentrations were below the current PEL of 0.1 f/cc. Our 3 Tests have found that asbestos exposure from wire gauze pads is very minimal, orders of magnitude below the PEL. The exposure from one of the tongs and the Transite boards are higher, from 0.160 to 0.320 f/cc in a 30 minute sampling time. The conditions simulated by our research were intentionally more austere than the expected handling in the average lab work. But since we found asbestos in our samples, it is important to place this exposure in context. The epidemiological data that linked asbestos to asbestosis, lung cancer and mesothelioma derives from environments were airborne fibers have been significantly higher than lab work (if our findings are representative of such exposure). Williams *et al* (Table 1) reported that exposure measurements in insulation work has been much higher than 1 f/cc. From insulation work performed in Great Britain, exposures to asbestos have ranged between 68 to 579 f/cc. In the U.S. full ship insulation workers had exposures ranging from 3-20 f/cc (18 f/cc annually, between 1962-1972 at Puget Sound). These exposures are significantly higher than the ones obtained in our experiment. OSHA's first PEL was 12 f/cc in 1971; this value was reduced over the years arriving at our current PEL 0.1 f/cc (120 times lower than the first PEL; Williams *et al*, 2009). In comparison to OSHA's historical occupational exposure limits, our findings demonstrate much lower exposures in typical laboratory work. If we treat our Test 3, 30-minute

samples as excision levels, then the limit is 1 f/cc, which is multiple times higher than the highest exposure (0.320 f/c from a Transite board) we obtained (Table 12). Overall, exposures generated by our wire gauze samples and 2 of our tong samples are similar to those obtained by Blake (2008, 2009) and Cohen (2008) in their brake and clutch repair exposure determinations, significantly below the current PEL of 0.1 f/cc (Table 2).

CONCLUSION

We evaluated multiple ACM including wire gauze pads, tong sleeves, and Transite board. We found, in the case of the Transite boards and one tong sleeve manipulation that it is possible to generate at least excursion levels of airborne fibers. These levels, if sustained for an 8-h period, could exceed the current 8-h TWA PEL of 0.1 f/cc for asbestos. However, if the simulated work did not occur repetitively during the laboratory workday, it is unlikely that lab workers would have been exposed to significant levels of asbestos in these settings. A low asbestos exposure in the research lab settings would be consistent with a very low risk of asbestos-related disease if we take into account that the bulk of the epidemiological data for asbestosis and cancer comes from very high exposures to asbestos.

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