

# Evaluation of Take-Home Exposure and Risk Associated with the Handling of Clothing Contaminated with Chrysotile Asbestos

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The potential for para-occupational (or take-home) exposures from contaminated clothing has been recognized for the past 60 years. To better characterize the take-home asbestos exposure pathway, a study was performed to measure the relationship between airborne chrysotile concentrations in the workplace, the contamination of work clothing, and take-home exposures and risks. The study included air sampling during two activities: (1) contamination of work clothing by airborne chrysotile (i.e., loading the clothing), and (2) handling and shaking out of the clothes. The clothes were contaminated at three different target airborne chrysotile concentrations (0–0.1 fibers per cubic centimeter [f/cc], 1–2 f/cc, and 2–4 f/cc; two events each for 31–43 minutes; six events total). Arithmetic mean concentrations for the three target loading levels were 0.01 f/cc, 1.65 f/cc, and 2.84 f/cc (National Institute of Occupational Health and Safety [NIOSH] 7402). Following the loading events, six matched 30-minute clothes-handling and shake-out events were conducted, each including 15 minutes of active handling (15-minute means; 0.014–0.097 f/cc) and 15 additional minutes of no handling (30-minute means; 0.006–0.063 f/cc). Percentages of personal clothes-handling TWAs relative to clothes-loading TWAs were calculated for event pairs to characterize exposure potential during daily versus weekly clothes-handling activity. Airborne concentrations for the clothes handler were 0.2–1.4% (eight-hour TWA or daily ratio) and 0.03–0.27% (40-hour TWA or weekly ratio) of loading TWAs. Cumulative chrysotile doses for clothes handling at airborne concentrations tested were estimated to be consistent with lifetime cumulative chrysotile doses associated with ambient air exposure (range for take-home or ambient doses: 0.00044–0.105 f/cc year).

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**KEY WORDS:** Asbestos; exposure assessment; para-occupational exposure; take-home exposure

## 1. INTRODUCTION

Asbestos has had many industrial uses over the past century because of its physical properties, including heat resistance, strength, and flexibility.<sup>(1)</sup>

During that same time period, asbestos has been a prominent inhalation hazard in many occupational environments.<sup>(2)</sup> As a result, many studies have been undertaken to measure airborne asbestos concentrations associated with the manufacture and use of asbestos-containing products in hundreds of workplace settings.<sup>(3–10)</sup> In addition to concerns about the workplace, exposure to asbestos has been reported for individuals living in the residences of workers who returned home wearing contaminated clothing. Concerns for this household population have included the potential for bystander exposures due to the presence of contaminated

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clothing in the residence, as well as the exposure potential associated with the active handling of contaminated clothing prior to laundering. The exposure of household contacts has often been called “secondary,” “para-occupational,” “domestic,” or “take-home” exposure.<sup>(11,12)</sup> Previously published studies on asbestos and take-home exposures have primarily explored the association between disease incidence and reports that asbestos was likely brought home on clothing. However, these studies have shed little light on some of the potentially important determinants of take-home exposures, including the magnitude of airborne exposure associated with handling and laundering activities of clothes contaminated in the workplace. Similarly, the nonoccupational risk associated with household exposure potential in specific scenarios has not been quantitatively characterized.

### 1.1. Background

There are six asbestos mineral fiber types currently recognized by the U.S. Occupational Safety and Health Administration (OSHA) in its regulatory exposure standards.<sup>(13)</sup> Of these six mineral types, chrysotile is in the serpentine mineral class, whereas amosite, crocidolite, tremolite, actinolite, and anthophyllite are in the amphibole mineral class.<sup>(14)</sup> These two classes of minerals differ significantly in terms of their physical properties and chemical composition, which, in turn, greatly impacts the biological characteristics of the mineral fibers. Chrysotile is a hydrated magnesium silicate that forms curly and pliable fibers, whereas amphiboles, such as crocidolite and amosite, contain iron and form inflexible rod-like fibers.<sup>(15,16)</sup> Over the past several decades, evidence has accumulated in the published literature indicating that there are significant differences in cancer potency according to asbestos mineral fiber type, with chrysotile being the least potent of the common industrial mineral types for both lung cancer and mesothelioma induction, and crocidolite being the most potent of the fiber types used commercially.<sup>(17-23)</sup> Despite these reported differences, the OSHA permissible exposure limits (PELs) for asbestos are the same for all mineral types. The PELs for asbestos are 0.1 fibers per cubic centimeter (f/cc) as an eight-hour time-weighted average (TWA) and 1.0 f/cc as a 30-minute excursion limit for fibers that meet the dimension criteria of  $\geq 5 \mu\text{m}$  in length and  $\geq 0.25 \mu\text{m}$  in width, with an aspect ratio of 3:1 or greater. These dimensions are important, because studies have shown that the asbestos fiber

disease potency for both lung cancer and mesothelioma differ substantially according to fiber length and width characteristics, with longer and thinner fibers being more potent for disease.<sup>(20,24-26)</sup>

In the context of asbestos-contaminated clothes-handling activities, it is important to properly identify the airborne fiber types because both clothing and asbestos fibers could be present in the air after shaking out contaminated clothing. Up until the 1960s, the specific measurement of airborne asbestos fiber concentrations proved difficult due to method limitations. The midget impinger was among the most common sampling methods for asbestos at that time and was most suitable for counting particles and reporting concentrations in million particles per cubic foot rather than by fibers per cubic centimeter. In addition, often the impinger collection method could only be used to sample for short periods of time rather than the total work shift, and it appeared to have a less effective collection efficiency for fibers compared to dusts.<sup>(27,28)</sup> Other historic sampling methods for asbestos reported the results in mass of asbestos per volume of air rather than fibers per volume.<sup>(29)</sup> In 1969, the U.S. Bureau of Occupational Health first published a method describing the use of a new filter counting method for industrial dusts and fibers that allowed for the characterization of the sizes of the individual airborne fibers collected.<sup>(30)</sup>

In 1971, OSHA specified the use of this type of filter membrane sampling method with phase contrast illumination for counting fibers and estimating fiber exposure.<sup>(31)</sup> The current U.S. National Institute of Occupational Health and Safety (NIOSH) Analytical Method 7400 describes essentially the same phase contrast microscopy (PCM) sampling and analytical method and provides the PCM fiber dimension counting criteria (fibers  $\geq 5 \mu\text{m}$  in length, and  $\geq 0.25 \mu\text{m}$  in width, with an aspect ratio of 3:1 or greater). Although commonly used today and available at a low cost, the PCM method still does not distinguish asbestos fibers from other fibers of similar dimension; that is, all fibers meeting the counting criteria must be included in the analytical report, whether asbestos or not. In order for asbestos-specific airborne concentrations to be evaluated, a more expensive analytical method such as transmission electron microscopy (TEM) must be used. Analysis by TEM using NIOSH Analytical Method 7402 can provide the specific concentration for the airborne asbestos fibers on a sample that meet the fiber dimension criteria for the PCM analytical method. The NIOSH 7402 TEM method

also reports airborne asbestos concentrations in PCM equivalents, or “PCME.” To calculate PCME under NIOSH 7402, the measured asbestos to nonasbestos fiber ratio is calculated using the TEM results. This ratio is then multiplied by the PCM concentration measured using the NIOSH 7400 method to obtain a PCME concentration. In other words, the PCME method combines two approaches by using both the PCM fiber concentration and the TEM asbestos to nonasbestos fiber ratio.

This combined method can be more reliable for establishing fiber concentrations than TEM alone because a much larger portion of the sample filter is analyzed using PCM. However, the PCME calculation rules call for the use of a zero value whenever no asbestos fibers are detected by TEM (regardless of the number of fibers detected using PCM). This approach could lead to an underestimate of exposure potential, particularly when large numbers of non-detectable samples are found in a data set. Therefore, in addition to the PCM and PCME results as specified under NIOSH 7400 and 7402, TEM can be used to directly measure airborne asbestos fiber concentrations (in addition to just calculating the ratio of asbestos to nonasbestos fibers) for the fiber dimensions of interest under PCM. This “TEM” approach measures airborne asbestos concentrations from a different (although smaller) portion of the sample filter and results in an asbestos concentration independent from the PCM method. For these reasons, all three of these counting methods (PCM, TEM, and PCME) can be helpful for fully characterizing airborne concentrations of asbestos, particularly when a data set contains large numbers of nondetects.

## 1.2. Studies of Asbestos and Take-Home Exposure

In the early 1960s, both Wagner and Newhouse published reports of mesothelioma cases for which the only exposure to asbestos was believed to result from nonoccupational sources.<sup>(32,33)</sup> Nearly a decade later, one of the first attempts to formally evaluate the disease potential of such nonoccupationally exposed individuals was conducted by researchers at the Mt. Sinai School of Medicine in New York.<sup>(34-38)</sup> These studies evaluated household members of workers who were occupationally exposed to amosite asbestos. The authors found four instances of pleural mesothelioma among 2,218 nonoccupationally exposed household individuals. They also reported that the prevalence of pleural abnormalities in

the nonoccupational population was correlated with the relative duration of amosite exposure, as well as time since initial exposure.<sup>(38)</sup> Since the Mt. Sinai studies, a number of additional researchers have examined disease risk in household contacts who laundered the clothing of individuals who were occupationally exposed to asbestos.<sup>(39-52)</sup>

In a meta-analysis of published epidemiological studies on the risk of pleural mesothelioma in household contacts of workers who were occupationally exposed to asbestos, Bourdes *et al.* reported a statistically significant increased relative risk of 8.1 (95% CI: 5.3, 12) for pleural mesothelioma compared to controls who did not have household asbestos exposure potential.<sup>(53)</sup> The exposures of the workers in the studies evaluated were mainly to friable asbestos and occurred primarily before 1975.<sup>(12)</sup> In addition, according to the study authors, all but one of the household studies evaluated “were conducted in areas [with] either predominant or concomitant amphibole exposure.”<sup>(53, p. 413)</sup> In 2013, another meta-analysis of take-home asbestos exposure studies reported a summary relative risk estimate for mesothelioma of 5.02 (95% CI: 2.48, 10.13).<sup>(54)</sup> Similarly, these authors indicated that this risk estimate was associated with “workers involved in occupations with a traditionally high risk of disease from exposure to asbestos (i.e., asbestos product manufacturing workers, insulators, shipyard workers, and asbestos miners).”<sup>(54, p. 5629)</sup> In addition, the authors stated that “when reported, the workers via whom the individuals were domestically exposed were nearly always exposed to amphiboles.”<sup>(54, p. 5652)</sup>

In the mid 1990s, the NIOSH completed a comprehensive review and report to the U.S. Congress on the potential for and prevalence of contamination of workers’ homes as a result of the workers’ occupational contact and transport of specific substances, including asbestos, into the home.<sup>(55)</sup> In its report, NIOSH cited indirect evidence of asbestos exposure to household members, including questionnaire responses, evidence from medical evaluations, and potential sources of asbestos in the home environment (i.e., accounting for products and building materials in the home that could contain asbestos). The NIOSH report also addressed health effect studies of individuals who had been identified as having potential household asbestos exposures, noting that six of seven case-control studies reported cases of mesothelioma among household contacts of asbestos workers. NIOSH did not attempt to look at differences in the prevalence of mesothelioma by asbestos

fiber type used in the occupational settings, other than noting that Gibbs *et al.* found “various forms of asbestos fibers in [the lungs of family members] reflecting the types of asbestos to which the working family member was exposed.”<sup>(55,56, p. 11)</sup> In addition, NIOSH did not report any air measurements associated with asbestos concentrations in homes or with activities that could generate airborne asbestos in the home.

More recently, Donovan *et al.* performed an extensive review of the published literature on take-home exposure potential for asbestos.<sup>(12)</sup> The authors reported on the occurrence of disease among household members, and also grouped their findings by industry. They noted that “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.”<sup>(12, p. 725)</sup> They stated that many of the industries associated with disease occurrence were known to use amphibole asbestos, although the individual fiber type associated with specific cases of disease was often not reported, or was reported as mixed fibers. Because of the presence of mixed asbestos fibers for many of these studies, it was not possible to discern from these results alone whether take-home risk has been more heavily associated with a certain type of fiber (i.e., amphibole fibers vs. chrysotile fibers).

### 1.3. Fiber Concentrations Associated with Asbestos-Contaminated Clothing

Three studies from the 1970s and 1980s reported measured airborne fiber concentrations associated with the presence or handling of asbestos-contaminated clothing.<sup>(57–59)</sup> Two of these studies reported sampling results using the PCM analytical method,<sup>(57,59,60)</sup> while the third study reported sampling results in mass of asbestos per volume of air.<sup>(58)</sup> Because of the sampling and analytical methods used, the results from all three of these studies are difficult to interpret. The PCM method used in the first two studies did not distinguish asbestos fibers from other fibers of similar dimension collected during sampling. In such studies, clothing fibers could have affected the air measurements. The results of the third study (in mass per volume of air) are not easily compared to the regulatory asbestos exposure standards, which use the dose-metric of f/cc in air.

More recent studies have also measured airborne asbestos concentrations associated with handling lightly contaminated clothing, all of which were evaluated during various automotive repair tasks.<sup>(61–64)</sup> For most of these studies, the reported airborne measurements of asbestos were very low or not detectable (ND) by TEM or PCME. The relatively low concentrations reported in these studies translated to even lower or ND exposures from handling work clothes worn during the various activities.<sup>(61–64)</sup> Although these studies are insightful for the individual products and scenarios evaluated, they do not specifically address the proportional relationship between workplace concentrations and the corresponding potential household concentrations from handling work clothing.

### 1.4. Purpose of This Study

Since there have been virtually no quantitative data presented in the literature to characterize the take-home risk for asbestos, it was determined that actual field data were needed to understand the plausible exposure potential associated with specific take-home scenarios, particularly in light of the epidemiological studies that show a potential for increased disease incidence in household members. It was also recognized that it would be beneficial to evaluate exposures and associated risks for individual fiber types in light of the reported fiber potency differences between chrysotile, amosite, and crocidolite. In this initial study, the focus was therefore on take-home exposure potential to chrysotile, as this fiber type was used to make many common commercial products such as brakes, gaskets, packing, roofing materials, and floor tiles.<sup>(65,66)</sup> Further, chrysotile use continued in the making of these types of encapsulated products into the 1980s, after the commercial use of amphibole asbestos declined.<sup>(67)</sup> Most of the epidemiological studies evaluating take-home risks, however, examined take-home disease incidence associated with industrial amphibole or mixed fiber use in the 1930s to 1960s.<sup>(12)</sup> These epidemiology studies are likely not relevant for characterizing the risks of take-home exposure to products that contained exclusively or predominantly chrysotile.

The purpose of this study, then, was to quantitatively characterize the relationship between different workplace airborne chrysotile concentrations and the corresponding concentrations associated with handling and shake out of chrysotile-contaminated work clothing prior to laundering. The study also

quantitatively evaluated bystander airborne concentrations for clothes handling and the risk potential associated with cumulative exposures to these same activities. By quantifying the relationship between workplace and take-home exposures at different workplace airborne concentrations and clothing contamination levels, it was anticipated that these data would allow for the improved characterization of chrysotile asbestos take-home risk potential in scenarios well beyond what has been described in the currently available published literature.

## 2. METHODS

### 2.1. Study Preparation and Setting

Prior to performing the study, a medical institutional review board (IRB) reviewed and approved the study design (Copernicus Group, Study ID #CRI1–11–208; Durham, NC, USA). The study was conducted in a sealed chamber constructed of painted plywood panels with a concrete floor that measured approximately 16 ft long  $\times$  16 ft wide  $\times$  8 ft high. The approximate air volume of the chamber when empty was 1,950 ft<sup>3</sup> (RJ Lee Group; Monroeville, PA, USA). An air filtration device (AFD) equipped with high-efficiency particulate air filters provided negative pressure in the chamber throughout the study to prevent the release of asbestos-contaminated air through chamber openings and to remove asbestos from the air within the chamber prior to exhausting to the outdoors. The AFD had two settings; it was operated on the low setting at a rate of 400–600 cubic feet per minute, or 13–19 air changes per hour (ACH), during the study events, and on the high setting at a rate of 24 ACH between study events. Thus, the high setting was used to remove asbestos from the chamber air more quickly at the conclusion of each study event prior to properly protected personnel entering the chamber. Ambient outdoor temperatures during the four days of testing, whereas temperatures within the study chamber ranged from 18.7 °C to 26.9 °C during the four days of testing, whereas temperatures within the study chamber ranged from 19.2 °C to 26.2 °C during testing. Humidity levels outside the chamber ranged from 39.3% to 82.3%, whereas levels inside the study chamber measured 47.5–72.4%. Grade 7T chrysotile asbestos was selected for use in this study. Grade 7 chrysotile was commonly used in many historical chrysotile-containing products, including floor tiles, roofing felts, coatings and components, and others.<sup>(65,66)</sup>

### 2.2. Loading Events (Simulated Workplace Environment)

Three different airborne concentration ranges were used during a total of six loading events, with the goal of generating concentrations that were broadly classified at the target loading levels of low (0–0.1 f/cc), medium (1–2 f/cc), and high (2–4 f/cc). The purpose of the loading events was to establish three target airborne concentration levels that could be considered representative of a variety of workplaces in which chrysotile asbestos was present during worker activities. The measurements collected for each of the six loading events were intended for direct comparison between the estimated loading (simulated workplace) concentrations and the corresponding airborne concentrations measured during the handling of clothes that were contaminated with chrysotile (simulated home environment). Two clothes-loading events involving different sets of clothes were conducted at each target concentration; each event lasted 31–43 minutes. For each clothes-loading event in the chamber, six dressed mannequins were placed in a circular pattern at a distance of 6 ft from a dust generator placed at the center of the chamber. Prior to each loading event, the mannequins were dressed with new, unwashed clothing. Additional information regarding the design of the loading events and the dust generation system, as well as the safety and decontamination procedures employed during the study, can be found in the Supporting Information and Fig. S1.

No study participants entered the chamber during the active clothes-loading events. Area air samples were collected throughout the chamber during each loading event, and the sampling locations remained consistent for all loading events. Simulated personal breathing zone samples were collected on the right and left lapels of each mannequin, and additional area samples were collected in the center of the room, in between the mannequins, and in the corners of the room. Area samples were collected at breathing zone height of approximately 5 ft. At the end of the loading events, to minimize disturbance of the loaded clothing prior to the clothes-handling events, one set of clothing at each loading level (low, medium, and high) was left on the mannequins and not disturbed until the clothes-handling event that immediately followed. The clothing from the remaining three loading events was carefully removed from the mannequins and placed in separate nonconductive storage boxes. The storage boxes

were sealed and saved for use during subsequent clothes-handling events.

### 2.3. Clothes-Handling and Shake-Out Events (Simulated Home Environment)

A total of six 30-minute clothes-handling and shake-out events were performed during the study. The clothing that was contaminated during each loading event was matched to a separate, subsequent clothes-handling and shake-out event so that the data for each of the six loading and shake-out event pairs could be quantitatively analyzed. As a result, the clothing from six mannequins (or six days' worth of work clothing) was shaken out during each clothes-handling event. Two handling/shake-out events were completed using the clothing from each of the three target loading levels (low, medium, and high), totaling six comparable data sets.

Each shake-out event consisted of 15 minutes of active clothes handling, followed by an additional 15 minutes of no activity, for a total of 30 minutes per event. A 15-minute active shake-out period was selected because the authors concluded that this time duration represented an upper bound of the total amount of time that a household member might handle and shake out contaminated clothing prior to laundering, particularly after collecting contaminated clothing from each day over the course of a work week. The additional 15-minute period of no activity was included to help characterize exposure potential when a household member remained in the laundry room or area for some period of time following the handling and shake out of contaminated clothing, such as to fold clean clothes, iron clothing, etc. This consideration of the airborne concentration in the chamber during the second 15 minutes in addition to the first active 15-minute period also helps to provide an upper bound of exposure potential for individuals living in small residences or working in small laundry rooms where individuals might regularly have continued to work on other activities in the same area after shaking out clothing.

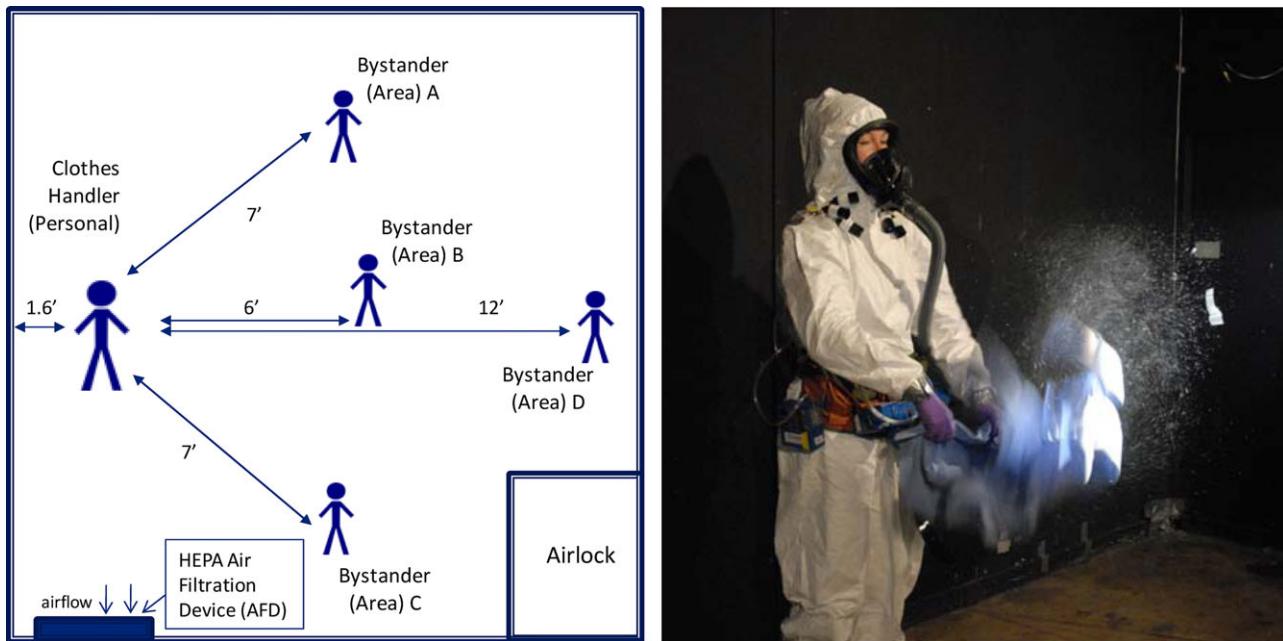
A total of six personal airborne fiber samples were collected on the clothes handler during each shake-out event. One set of right and left lapel personal samples was collected for the first 15-minute period, and another set of right and left lapel samples was collected during the second 15-minute period; a third set was collected over the full 30-minute event. Four area samples intended to reflect the ex-

posure of bystanders were also collected for the duration of each 30-minute handling and shake-out event. Bystander distances ranged from approximately 6–12 ft from the handling activities. A diagram depicting the locations of the simulated bystander locations in relation to the clothes handler has been provided in Fig. 1.

During the first 15 minutes (active clothes handling and shaking out of clothes), the clothes handler either removed the clothing from the mannequins or a nonconductive storage box (depending upon event sequence) and placed the clothing in a pile representing a laundry pile or basket. To simulate common types of clothes-handling activities prior to laundering, each individual article of clothing was picked up and shaken to dislodge visible dust or manipulated by brushing, turning inside out, and/or carrying clothing across the study chamber. During the second 15-minute period, study participants remained in the chamber but were not in physical contact with the clothing.

### 2.4. Sampling and Analytical Methods

All air samples were collected in accordance with NIOSH sampling method criteria and analyzed by PCM (NIOSH 7400) and TEM (NIOSH 7402). Samples were collected using either personal high-volume or stationary high-volume sampling pumps (Dawson/Emerson/SKC, Inc., PA, USA). The sampling pumps, along with the assembled sampling trains, were calibrated with a frictionless piston primary flow meter before and after each sample was collected (BIOS DryCal DC Lite/Defender; Prairieville, LA, USA). Area air samples during loading events were collected with high-volume sampling pumps at a rate of 5.5 liters per minute (L/min)–10 L/min. Air samples on the lapels of the mannequins during loading events, on study participants during shake-out events, and on study participants working outside the chamber were collected at airflow rates ranging from 1 L/min to 2 L/min. Samples of ambient air outside the study chamber and outdoors at the AFD exhaust were collected at airflow rates ranging from 7 L/min to 10 L/min throughout each day of the study. At least two field blanks were collected during each loading and clothes-handling event. Samples of the bulk 7T chrysotile were also analyzed to determine specific fiber characteristics. All samples were analyzed by the RJ Lee Group in Monroeville, PA, which has been accredited by the



**Fig. 1.** Depiction of chamber and testing arrangement. Photo depicts person shaking asbestos-contaminated clothing.

American Industrial Hygiene Association Laboratory Accreditation Program (AIHA-LAP, #100364).

Analytical sensitivity limits for the TEM samples (NIOSH 7402) were estimated based on the assumption that 0.5 fibers could be counted within 40 microscopic fields. For the PCM (NIOSH 7400) analytical method, the limits of detection (LODs) were calculated using a fiber density of 5.5 fibers per 100 fields analyzed. PCME concentrations were calculated by multiplying the ratio of asbestos fibers to nonasbestos fibers as measured by TEM by the corresponding measured PCM concentration for each sample.

## 2.5. Data and Statistical Analysis

Descriptive statistics were performed on the PCM, TEM, and calculated PCME concentrations of total airborne fibers and airborne chrysotile collected during both the loading and clothes-handling events. As discussed earlier, all three of these concentration metrics were used in order to fully characterize the results, since each sampling method has advantages and disadvantages. Arithmetic means and standard deviations (*SD*) were calculated for each event by measurement type (PCM, TEM, and PCME), along with standard error of the mean (*SE*) for comparison purposes. When sample results were reported as below the LOD or sensitivity limit as defined by

the NIOSH 7400 (PCM) and 7402 (TEM) methods, a value of one-half of the LOD or sensitivity limit was used in PCM and TEM calculations so as not to underestimate fiber concentrations,<sup>(68)</sup> although it should be noted that a value of zero for nondetects has also previously been used for the purposes of calculating airborne asbestos concentrations.<sup>(69)</sup> In calculating PCME, for the samples in which the TEM concentration was below the sensitivity limit, the asbestos fiber to nonasbestos fiber ratio was reported to be zero. For those samples, the corresponding PCME concentration was therefore also calculated as zero. If the PCM concentration was below the LOD for a sample but a TEM concentration and fiber ratio were reported, a PCME concentration was calculated using a value of one-half of the PCM LOD.

So as not to bias or underestimate the average fiber concentrations to be reported, particularly for events with a number of ND sample results and corresponding PCME concentrations of zero, the airborne fiber concentrations obtained using the TEM method were the primary concentration results used in the analysis. These results represent the measured airborne chrysotile fiber concentrations meeting the PCM dimension criteria ( $\geq 5 \mu\text{m}$  in length,  $\geq 0.25 \mu\text{m}$  in width, aspect ratio of 3:1 or greater), and for which a sensitivity limit could also be calculated. However, the concentrations by all three methods (PCM, TEM,

and PCME) are presented for comparison because of the differences between each method. The combined results of these three methods can be helpful to fully characterize the data, and also to help understand the differences between each of the methods. For this study, of particular interest was the relative proportion of airborne clothing fibers compared with airborne asbestos fibers during clothes-handling activities. Pair-wise comparisons were evaluated between PCM and TEM values for each loading event using PROUCL 4.0, a statistical program developed by the U.S. Environmental Protection Agency (EPA). On the basis of the data distributions for the loading events, a nonparametric means comparison test (the Gehan method) was used to evaluate statistical differences between the PCM and TEM data for the loading events. This method is useful for censored data sets with multiple reporting limits and data sets without a clear fit to a particular distribution curve (i.e., normal or lognormal).<sup>(70,71)</sup>

## 2.6. Evaluation of Lifetime Cumulative Doses and Health Risks

There are several published approaches for evaluating the nonoccupational risks of disease associated with asbestos exposures, including the U.S. EPA model and associated framework, the Hodgson and Darnton model, and the Berman and Crump model.<sup>(19,20,25,26,72,73)</sup> Although these models apply different methods to estimate cancer risk, all rely upon historical epidemiologic literature of primarily heavily exposed asbestos cohorts, which can lead to inherent challenges and uncertainties when attempting to extrapolate and estimate lower dose cancer risks. Although it is well accepted today that amphiboles are more potent than chrysotile in causing mesothelioma and lung cancer and that long fibers (e.g., >20–40  $\mu\text{m}$  in length) are more hazardous than short asbestos fibers (e.g., <10  $\mu\text{m}$  in length), the U.S. EPA asbestos risk model utilizes a linear dose-response model that does not discriminate between fiber type or dimensions. The lung cancer and mesothelioma potency factors derived in historical risk assessments were based on cohorts exposed to high cumulative doses (approximately 50–1,000 f/cc year) of mixed fibers.<sup>(72,74)</sup> It has been shown that the U.S. EPA regulatory model can over-predict the theoretical risks of mesothelioma among certain chrysotile cohorts.<sup>(23,75,76)</sup> More recently developed asbestos risk models such as Berman and

Crump take into account factors such as fiber type and fiber size. Although this model has been shown to better predict and estimate risks than the U.S. EPA model, fiber dimension analysis beyond what is represented by PCME is necessary for the application of this model and therefore precluded its use in this study.<sup>(76)</sup> As a result of the lack of consensus between regulatory approaches and the current scientific literature regarding the estimation of cancer risk for exposures measured using PCME data, nonoccupational risks for this study were evaluated using a cumulative lifetime dose metric, which allowed for a direct comparison of the results to the available epidemiological literature. Estimated cumulative chrysotile doses for clothes handling were calculated and compared with the estimated cumulative lifetime doses associated with exposure to background levels of chrysotile in ambient air for reference. These doses were then also evaluated against relevant epidemiological literature on the risk of asbestos-related disease at similar doses of asbestos.

For the purposes of the example dose calculations, an exposure duration (ED) of 25 years for a commercial/industrial worker was assumed based on the U.S. EPA's upper-bound estimate of the time a person spends working for a single employer.<sup>(77)</sup> No published data on laundry exposure factors were identified, and so the exposure frequency for clothes-handling activities was estimated to be once per week, for example, 52 days per year. This assumption was also consistent with the events conducted in our study, since each clothes handler shook out and handled six sets of work clothing per event (approximately one week's worth of work clothing). For the clothes handler, an exposure time (ET) of 0.5 hours per day was assumed in order to take into account the potential for 15 minutes of active clothes handling and shake-out time for one week's worth of clothing plus an additional 15 minutes spent in the same area where the clothes handling occurred.

A range of outdoor ambient asbestos concentrations of approximately 0.00003–0.002 f/cc has been reported for various geographical areas in the United States.<sup>(59,78–82)</sup> Although these values are reported using TEM analysis and are specific to asbestos, not all of the studies were specific to chrysotile and therefore are not directly comparable to the chrysotile dose estimates calculated in this study. For chrysotile specifically, the average ambient outdoor air concentration of chrysotile has been recently reported by U.S. EPA to be 0.00005 f/cc in a small-town

environment (chrysotile analysis conducted using the International Standards Organization [ISO] Method 10312, 1995; counts include all fibers  $\geq 0.5 \mu\text{m}$  in length, aspect ratio 5:1) (see Tables 4–6, U.S. EPA, 2009).<sup>(83)</sup> Nolan and Langer also measured an average outdoor ambient chrysotile concentration near buildings of 0.0015 f/cc (fibers  $> 5 \mu\text{m}$  in length).<sup>(81)</sup> This chrysotile-specific range of ambient concentrations (0.00005–0.0015 f/cc) was used to calculate a range of lifetime cumulative doses associated with ambient chrysotile (assuming exposures for 24 hours a day, 365 days a year, over a 70-year period).

### 3. RESULTS

A total of 12 air-monitoring events were conducted (six loading events and six shake-out events) over a five-day period. Sampling conducted to monitor personnel safety during the study showed that airborne fiber concentrations outside the chamber during all study events were ND by PCME. All clearance samples taken inside the chamber prior to the handling and shakeout events were also ND by PCME.

#### 3.1. Airborne Fiber Concentrations During the Simulated Occupational Exposure Scenarios (Loading Events)

Analysis of the fiber concentrations collected from different locations within the study chamber during the loading events (on mannequin lapels, in the chamber center, between mannequins, and in the chamber corners) indicated that the concentrations in the chamber were relatively uniform (see Supporting Information and Fig. S2 for additional information). Therefore, the arithmetic mean of all the samples collected in the chamber was used to characterize the airborne concentration for each loading event (Fig. 2). Fig. S3 depicts the distributions of PCM and TEM fiber concentrations (5th, 25th, median, arithmetic mean, 75th, and 95th percentiles) measured for each of the loading events. Table SI indicates the number of samples, number of detects, sample duration, arithmetic mean, and SD for each loading event by PCM, TEM, and PCME. The number of air samples per loading event ranged from 20 to 34 in order to obtain a robust characterization of the air in the test chamber.

#### 3.1.1. TEM Airborne Fiber Concentrations During Loading

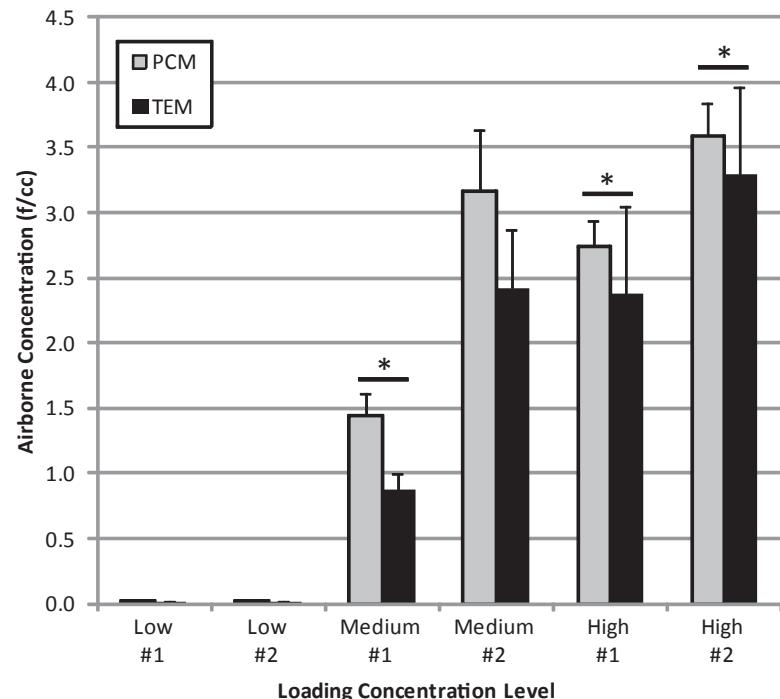
The average TEM concentrations for each loading event (Low #1, Low #2, Medium #1, Medium #2, High #1, and High #2) ranged between 0.01 f/cc and 3.30 f/cc of air. Arithmetic mean TEM concentrations for events Low #1 and #2 were 0.01 f/cc; for events Medium #1 and #2 were 0.88 f/cc and 2.42 f/cc; and for events High #1 and #2 were 2.38 f/cc and 3.30 f/cc. Events Low #1 and Low #2 had a sample detection frequency of 42% and 33%, respectively, by TEM. The mean TEM sensitivity limits (LODs) for loading events Low #1 and Low #2 were 0.006 f/cc and 0.009 f/cc.

#### 3.1.2. PCM Airborne Fiber Concentrations During Loading

The arithmetic mean PCM airborne concentrations for the six loading events ranged from 0.02 f/cc to 3.59 f/cc. Arithmetic mean PCM concentrations for events Low #1 and #2 were 0.02 f/cc; for events Medium #1 and #2 were 1.44 f/cc and 3.17 f/cc; and for events High #1 and #2 were 2.74 f/cc and 3.59 f/cc. Results indicated that the mean PCM airborne concentrations were statistically significantly higher than the mean TEM airborne concentrations for loading events Medium #1, High #1, and High #2 (Gehan test,  $p < 0.05$ ; Fig. 2). Loading events Low #1 and Low #2 both had a low sample detection frequency (21% and 6%, respectively, for PCM). The mean PCM LODs for loading events Low #1 and Low #2 were 0.037 f/cc and 0.047 f/cc, respectively.

#### 3.2. Airborne Fiber Concentrations During the Simulated Take-Home Exposure Scenarios (Clothes Handling and Shakeout Shake-Out Events)

The clothes-handling data were analyzed in two ways: first, the personal breathing zone fiber concentrations for the clothes handlers during the active 15-minute shake-out period were compared to the subsequent 15 minute period of no activity (Figs. 3A and B). Second, the personal breathing zone fiber concentrations for the clothes handler during the full 30-minute shake-out sampling period (15 minutes of clothes handling and shake out plus 15 minutes of inactivity) were compared to the 30-minute area (bystander) samples that were collected 6–12 ft away from the clothes handler (Figs. 4A and B). For the



**Fig. 2.** Arithmetic mean airborne fiber concentrations during the clothes-loading events PCM concentrations (NIOSH 7400 [gray]) and TEM concentrations (NIOSH 7402 [black]) associated with airborne fiber concentrations reported for the six clothes-loading events (low, medium, high). The asterisks indicate where the PCM concentration was statistically significantly different from the TEM concentration ( $p < 0.05$ ). The error bars indicate  $SE$ ;  $n = 20$  to 34 per event.

15-minute samples, Table SII shows the sample number ( $n = 2$  per event); the number of detected samples; average PCM, TEM, and PCME concentrations for each event; and  $SDs$ .

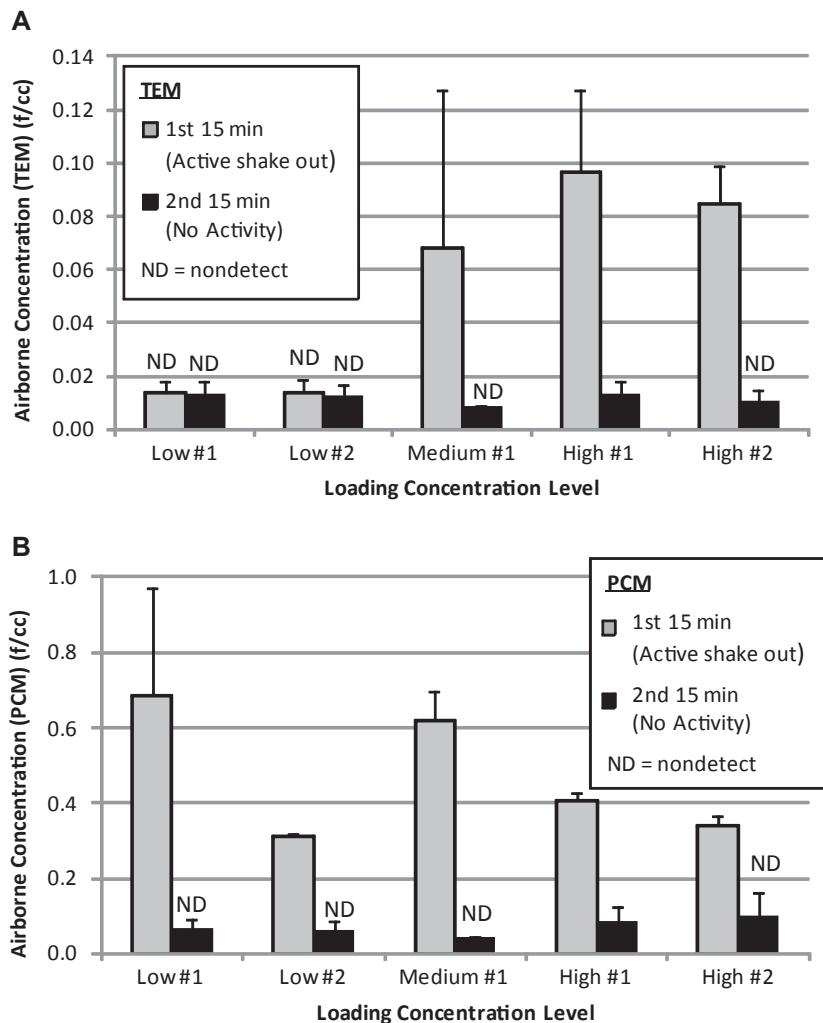
For all clothes handling and shake-out events except Medium #2, data were available for the 15-minute period of active clothes handling and shake out, as well as a second 15-minute period of no activity for comparison. For the Medium #2 shakeout event, air concentration data were only collected for the full 30-minute sampling period. For the 30-minute samples, Table SIII shows the sample number ( $n = 2$  to 4 per event); number of detected samples; average PCM, TEM, and PCME 30-minute concentrations for each event; and  $SDs$ .

### 3.2.1. TEM Airborne Fiber Concentrations During Clothes Handling and Shake Out

The arithmetic mean TEM concentrations during the 15-minute active clothes-handling period ranged from 0.014 f/cc to 0.097 f/cc for the five applicable handling and shake-out events (Fig. 3A). Arithmetic mean TEM concentrations for clothes-handling events corresponding to loading events Low #1 and #2 were 0.014 f/cc, for event Medium #1 was 0.068 f/cc, and for events High #1 and #2

were 0.097 f/cc and 0.085 f/cc. Correspondingly, the arithmetic mean TEM concentrations during the subsequent 15-minute period of inactivity immediately following ranged from 0.009 f/cc to 0.014 f/cc for the five applicable handling and shake-out events. During the 15-minute period of inactivity, the mean TEM concentrations decreased for the Medium #1, High #1, and High #2 events by 86–87% (0.068–0.097 f/cc TEM during the first 15 minutes vs. 0.009–0.014 f/cc TEM during the second 15 minutes; Fig. 3A). Across all five events (Low #1, Low #2, Medium #1, High #1, and High #2), only one chrysotile fiber was detected in all the samples collected during the second 15 minutes of inactivity, indicating that fiber concentrations decreased rapidly after active clothes handling and shake out ceased.

For the 30-minute clothes-handling samples, the arithmetic mean TEM concentrations ranged from 0.006 f/cc to 0.063 f/cc for the six handling and shake-out events (Fig. 4A). Arithmetic mean TEM concentrations for the 30-minute clothes-handling events Low #1 and #2 were 0.007 f/cc, for events Medium #1 and #2 were 0.021 f/cc and 0.006 f/cc, and for events High #1 and #2 were 0.050 f/cc and 0.063 f/cc. For events Medium #1, High #1, and High #2, the corresponding 30-minute area (bystander) TEM concentrations were 62–88% lower than the mean clothes



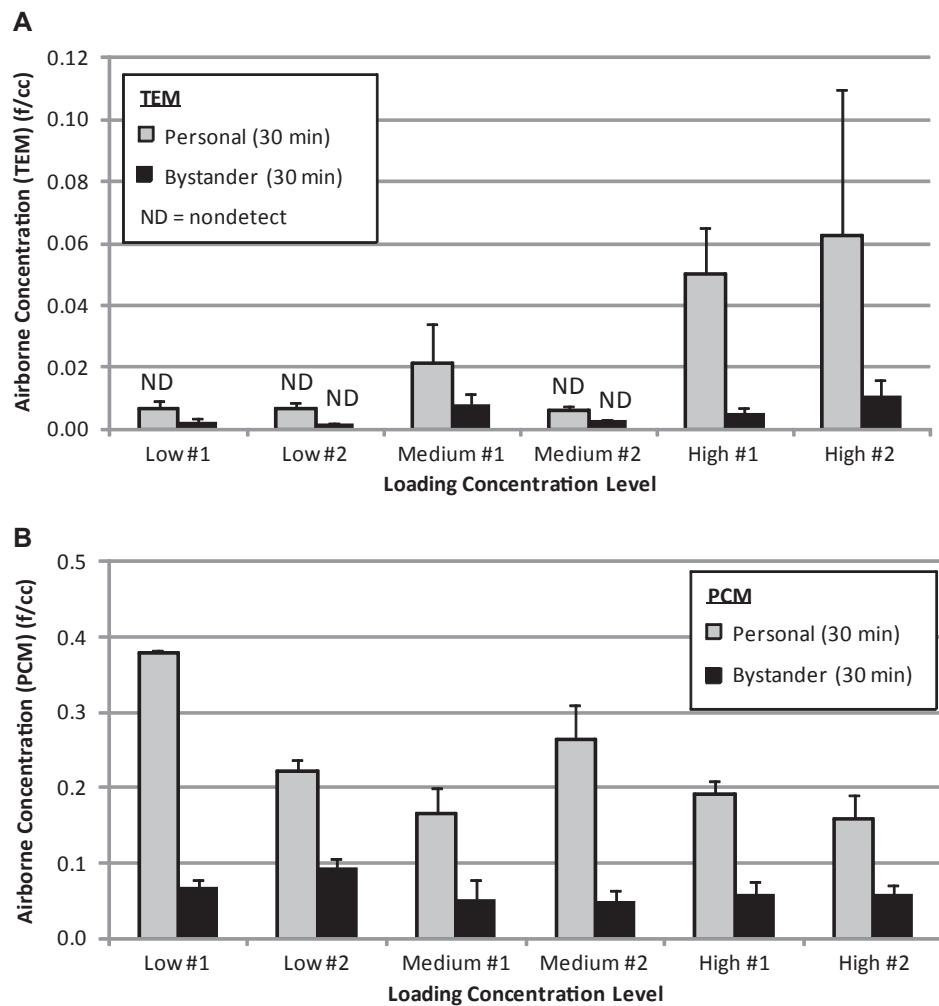
**Fig. 3.** First 15-minute and second 15-minute personal airborne fiber concentrations for clothes handling and shake-out arithmetic means of the TEM values using NIOSH 7402 (A) and PCM values using NIOSH 7400 (B). Airborne fiber concentrations measured during active simulation of personal clothes-handling activities including shake out of clothing (gray) and during 15 minutes of inactivity immediately following (black). Groups for which there were no detectable fibers on any of the sample filters are indicated (ND). Error bars indicate *SE*.

handler TEM concentrations (0.006–0.011 f/cc TEM for bystander locations vs. 0.021–0.063 f/cc TEM for personal samples; Fig. 4A). For the Low #1, Low #2, and Medium #2 events, there were too few detects to establish a meaningful comparison between personal and area (bystander) TEM measurements.

### 3.2.2. PCM Airborne Fiber Concentrations During Clothes Handling and Shake Out

Arithmetic mean PCM concentrations for the clothes handler during the 15-minute active clothes-handling period ranged from 0.312 f/cc to 0.687 f/cc (Fig. 3B). Mean PCM concentrations for events Low #1 and #2 were 0.687 and 0.312 f/cc, for event Medium #1 was 0.617 f/cc, and for events High #1 and #2 were 0.407 and 0.341 f/cc. Unlike the TEM data,

there were no trends in the PCM data by event. For example, the arithmetic mean PCM air concentration for the Low #1 event was higher than the mean PCM air concentration for the High events. Notably, during the 15-minute active clothes-handling and shake-out period, the PCM concentrations were 4- to 50-fold higher than the corresponding TEM concentrations, indicating that the majority of fibers measured by PCM were not chrysotile. For the second 15-minute period of inactivity, arithmetic mean PCM concentrations for events Low #1 and #2 were 0.068 f/cc and 0.064 f/cc, for event Medium #1 was 0.045 f/cc, and for events High #1 and #2 were 0.085 f/cc and 0.100 f/cc. Overall, the average personal breathing zone concentrations for the clothes handler as measured by PCM during the 15 minutes of active clothes handling and shake out were approximately



**Fig. 4.** Thirty-minute airborne fiber concentrations for clothes-handling and shake-out arithmetic means of the TEM values using NIOSH 7402 (A) and PCM values using NIOSH 7400 (B) for the airborne fiber concentrations from a 30-minute sampling period composed of 15 minutes of active clothes handling followed by 15 minutes of inactivity. Samples were taken in the breathing zone of the clothes handler (gray) and at locations 6–12 ft away from the clothes handler (representing bystander exposures [black]). Groups for which there were no detectable fibers on any of the sample filters are indicated (ND). The error bars indicate SE.

3- to 14-fold higher than those measured during the 15 minutes of inactivity immediately following.

For the 30-minute samples, the PCM values were 2- to 50-fold higher than the TEM values, and overall did not trend by concentration of asbestos used in the loading event, which was consistent with what was seen for the first 15-minute (active clothes handling) samples (Figs. 4A and B). For all six clothes-handling and shake-out events, the mean of the 30-minute personal PCM concentrations for the clothes handlers was two- to five-fold higher than the corresponding 30-minute area (bystander) PCM measurements (6–12 ft from the clothes handler), indicating that fiber concentrations overall

decreased with distance from the clothes handler (Fig. 4B).

### 3.3. Comparison Between Loading Events and Clothes-Handling and Shake-Out Events

For the six loading versus clothes-handling and shake-out event pairs, a trend of increasing clothes handler airborne TEM chrysotile concentrations was observed as the corresponding loading TEM concentrations increased. The mean TEM concentrations during loading events Low #1 and Low #2 were found to be significantly lower than for the other

loading events (Gehan test, Bonferroni-adjusted  $\alpha = 0.05$ ). Similarly, the mean TEM concentrations for the medium and high clothes-handling and shakeout events (0.068–0.097 f/cc) were three- to nine-fold greater than for the low events (0.014 f/cc; Fig. 3A). In contrast to the TEM results, the clothes-handling PCM results for events Low #1 and Low #2 did not tend to be lower than for the medium and high events (Figs. 3B and 4B) indicating no trend by loading concentration.

For the medium and high loading concentration levels, 30-minute personal clothes handler TEM concentrations ranged from 0.3% to 2.4% of the corresponding average loading TEM concentrations. For the calculated PCME 30-minute personal clothes handler concentrations, relative to the PCME loading concentrations, the ratios were in a very similar range of 0–2.1%. For the Low #1 and Low #2 events, a quantitative comparison between clothes-loading/shake-out event pairs was not conducted since all of the clothes handler personal breathing zone samples were nondetects, and a large fraction (42% for Low #1 and 33% for Low #2) of the loading samples were also ND for chrysotile. This ratio was not estimated for the PCM results because of the lack of a pattern between loading levels.

To account for differences in the ED between the loading and clothes-handling and shake-out events, TWA TEM chrysotile concentrations for each loading event were calculated and were then compared to the calculated TWA TEM chrysotile concentrations for the corresponding 30-minute clothes-handling and shake-out event. These conversions allowed for a direct ratio analysis of the loading and clothes-handling airborne concentrations for each event pair. The calculations were conducted using both eight-hour TWAs (for comparison to daily clothes-handling and shake-out activities) and 40-hour TWAs (for comparison to once-per-week clothes-handling and shake-out activities). The TWA concentrations were calculated assuming that, aside from the loading or clothes-handling time (approximately 30–45 minutes each), asbestos concentrations for the remainder of the day or week were ND (i.e., a value of zero was used). The eight-hour TWA was calculated for each loading and clothes-handling event using the approach described by OSHA in 1910.1000(d)(1)(i).<sup>(60)</sup> The 40-hour TWAs were calculated in an analogous manner. Once again, a comparison of the TWAs for the Low #1 and Low #2 events was not performed

because of the large number of nondetects among both the loading and clothes-handling and shake-out samples.

Overall, the eight-hour TWA airborne chrysotile concentrations for the clothes-handling and shake-out events were between 0.2% and 1.4% (0.0004–0.0040 f/cc TEM vs. 0.0976–0.5491 f/cc TEM) of the corresponding eight-hour TWA loading airborne chrysotile concentrations (Table I). When considered on a 40-hour TWA work week basis, the relative differences in concentrations (TEM) corresponded to clothes-handling and shake-out TWA concentrations that were between 0.03% and 0.27% of the corresponding 40-hour TWAs (Table II). For the calculated PCME values, when comparing the eight-hour TWA loading and shake-out concentrations, the ratios were similar to TEM (despite a number of ND concentration values that were counted as zeros) and ranged from 0% to 0.8%. Regarding the bystander exposure concentrations, the TEM eight-hour TWA chrysotile concentrations for bystanders to clothes handling were between 0.1% and 0.5% (0.0002–0.0007 f/cc TEM vs. 0.0976–0.5491 f/cc TEM) of the corresponding eight-hour TWA loading concentrations; these same ratios were between 0% and 0.2% by PCME. The 40-hour TWA concentrations for the bystanders to clothes handling and shake out were 0.01–0.1% of the corresponding 40-hour work week TWAs.

### 3.4. Results of Lifetime Cumulative Dose Calculations

Estimates of the nonoccupational cumulative chrysotile doses associated with the handling and shaking out of clothing contaminated with chrysotile at each of the measured clothes-loading levels studied, along with estimates of the comparative lifetime doses associated with chrysotile exposure in ambient air, are shown in Table III. These estimates indicate that the cumulative chrysotile doses for the clothes-handling events conducted in this study are either below or consistent with lifetime cumulative doses for ambient or background chrysotile over a 70-year lifetime. The estimated range of cumulative doses associated with the clothes-handling events was 0.00044–0.0047 f/cc year for the personal clothes handler and 0.00011–0.00059 f/cc year for bystanders to clothes-handling activity. For comparison, the estimated lifetime ambient cumulative chrysotile dose range was 0.0035–0.105 f/cc year.<sup>(81,83)</sup>

**Table I.** Comparison of Eight-Hour TWA Airborne Chrysotile Concentrations (TEM) for Loading and 30-Minute Shake-Out and Bystander Measurements

Loading Event	30-Minute Clothes-Handling Event Sampling <sup>a</sup>				Bystander <sup>b</sup> (TEM)	
	Loading Sampling (TEM)		Personal Breathing Zone (TEM)			
	Calculated Eight-Hour TWA (f/cc)	Calculated Eight-Hour TWA (f/cc)	Ratio (%) <sup>c</sup>	Calculated Eight-Hour TWA (f/cc)		
Medium #1	0.0976	0.0013	1.4	0.0005	0.5	
Medium #2	0.2520	0.0004 <sup>e</sup>	0.2 <sup>e</sup>	0.0002 <sup>e</sup>	0.1 <sup>e</sup>	
High #1	0.2869	0.0032	1.1	0.0004	0.1	
High #2	0.5491	0.0040	0.7	0.0007	0.1	

*Note:* Calculated eight-hour TWA concentrations (f/cc, TEM) for the medium and high loading events were compared to the calculated eight-hour TWA exposure of the household member who handled chrysotile-contaminated clothing. The bystander TWAs were also compared to the TWAs of the loading events. When the analytical result for an individual sample was below the sensitivity limit according to the method (NIOSH 7402), a value of one-half the sensitivity limit was used for the purposes of calculating the eight-hour TWA. These percentages can be considered as an upper bound, comparable to laundry activities conducted daily.

<sup>a</sup>Thirty minutes (15 minutes of active clothes handling immediately followed by 15 minutes of inactivity).

<sup>b</sup>Stationary sampling points 6–12 ft from the clothes handler.

<sup>c</sup>100 × (calculated eight-hour TWA associated with clothes handling/calculated eight-hour TWA associated with loading).

<sup>d</sup>100 × (calculated eight-hour TWA associated with bystander locations during clothes handling/calculated eight-hour TWA associated with loading).

<sup>e</sup>All of the clothes-handling samples and bystander samples were below the sensitivity limit for the analytical method (NIOSH 7402). For the purposes of calculating the eight-hour TWA, one-half of the sensitivity limit was used.

#### 4. DISCUSSION

This study quantitatively compared the measured airborne chrysotile concentrations in a simulated work environment with the corresponding concentrations generated during the handling of clothing (over a range of contamination concentrations). The study also evaluated the nonoccupational risks associated with cumulative chrysotile doses from handling clothing contaminated at the levels evaluated in the study.

The study results allowed for the development of a series of ratios comparing the simulated work environment (i.e., the loading event TEM air concentrations) to the simulated home environment (i.e., clothes-handling and shake-out event TEM air concentrations). These ratios are likely to be useful when evaluating the risk to a family member or household contact of an individual in a specific occupation that had known chrysotile exposure. The ratios were calculated using both eight-hour TWAs and 40-hour TWAs (TEM). The eight-hour TWA ratios and percentages are comparable to daily handling and shake out of contaminated clothing, while the 40-hour TWA ratios and percentages are comparable to weekly clothes handling. The eight-hour TWA airborne chrysotile concentrations during clothes-handling and shake-out events were consistently

between 0.2% and 1.4% of the corresponding eight-hour TWA airborne chrysotile concentrations during clothes-loading events (0.1–0.5 f/cc TEM as an eight-hour TWA for loading compared with 0.0004–0.004 f/cc TEM as an eight-hour TWA for the clothes handler). Correspondingly, the eight-hour TWA airborne chrysotile bystander concentrations at 6–12 ft away from the clothes handler were measured between 0.1% and 0.5% of the eight-hour TWA simulated workplace concentrations (TEM). For the 40-hour TWA work week ratios, personal clothes-handling and shake-out 40-hour TWA concentrations were between 0.03% and 0.27% of the 40-hour simulated work week TWAs (TEM). For the bystanders to clothes handling and shake out, 40-hour TWAs were 0.01–0.1% of the corresponding 40-hour work week TWAs (TEM).

In order to evaluate the nonoccupational risks for asbestos-related disease associated with the clothes-handling activities in the study, chrysotile doses based on cumulative exposures were calculated for each clothes-handling event, assuming 25 years of chrysotile-contaminated clothes handling for 30 minutes once a week (assumptions discussed in Section 2). These cumulative doses were then compared with a range of estimates for lifetime cumulative doses associated with exposure to chrysotile in the ambient air. The cumulative

**Table II.** Comparison of 40-Hour TWA Airborne Chrysotile Concentrations (TEM) for Loading and 30-Minute Shakeout Shake-Out and Bystander Measurements

Loading Event	30-Minute Clothes-Handling Event Sampling <sup>a</sup>				
	Loading Sampling (TEM)	Personal Breathing Zone (TEM)		Bystander <sup>b</sup> (TEM)	
		Calculated 40-Hour TWA (f/cc)	Calculated 40-Hour TWA (f/cc)	Ratio (%) <sup>c</sup>	Calculated 40-Hour TWA (f/cc)
Medium #1	0.0976	0.00027	0.27	0.00010	0.10
Medium #2	0.2520	0.00008 <sup>e</sup>	0.03 <sup>e</sup>	0.00004 <sup>e</sup>	0.01 <sup>e</sup>
High #1	0.2869	0.00065	0.23	0.00007	0.02
High #2	0.5491	0.00081	0.15	0.00015	0.03

Note: Calculated 40-hour TWA concentrations (f/cc, TEM) for the medium and high loading events were compared to the calculated 40-hour TWA exposure of the household member who handled chrysotile-contaminated clothing. The bystander TWAs were also compared to the TWAs of the loading events. When the analytical result for an individual sample was below the sensitivity limit according to the method (NIOSH 7402), a value of one-half the sensitivity limit was used for the purposes of calculating the 40-hour TWA. These percentages can be considered as a typical case, comparable to laundry activities conducted once weekly.

<sup>a</sup>Thirty minutes (15 minutes of active clothes handling immediately followed by 15 minutes of inactivity).

<sup>b</sup>Stationary sampling points 6–12 ft from the clothes handler.

<sup>c</sup>100 × (calculated 40-hour TWA associated with clothes handling/calculated 40-hour TWA associated with loading).

<sup>d</sup>100 × (calculated 40-hour TWA associated with bystander locations during clothes handling/calculated 40-hour TWA associated with loading).

<sup>e</sup>All of the clothes-handling samples and bystander samples were below the sensitivity limit for the analytical method (NIOSH 7402). For the purposes of calculating the 40-hour TWA, one-half of the sensitivity limit was used.

**Table III.** Example Estimates for Lifetime Personal and Bystander Cumulative Dose Associated with Clothes Handling and Shake Out Compared to Lifetime Cumulative Dose Associated with Ambient Outdoor Exposure to Chrysotile

Activity	Chrysotile Exposure Concentration (f/cc, TEM NIOSH 7402) <sup>a</sup>	Exposure Time (ET) (Hours/Day)	Exposure Frequency (EF) (Days/Year)	Exposure Duration (ED) (Years)	Lifetime Cumulative Dose (f/cc year) <sup>b</sup>
Ambient airborne asbestos	0.00003 <sup>c</sup>	24	365	70	0.0021
Ambient airborne chrysotile	0.00005 <sup>d</sup>	24	365	70	0.0035
Ambient airborne chrysotile	0.0015 <sup>e</sup>	24	365	70	0.105
Clothes handling—Low #1	0.007	0.5	52	25	0.00052
Clothes handling—Low #2	0.007	0.5	52	25	0.00052
Clothes handling—Medium #1	0.021	0.5	52	25	0.0016
Clothes handling—Medium #2	0.006	0.5	52	25	0.00044
Clothes handling—High #1	0.05	0.5	52	25	0.0037
Clothes handling—High #2	0.063	0.5	52	25	0.0047
Bystander—Low #1	0.003	0.5	52	18	0.00016
Bystander—Low #2	0.002	0.5	52	18	0.00011
Bystander—Medium #1	0.008	0.5	52	18	0.00043
Bystander—Medium #2	0.003	0.5	52	18	0.00016
Bystander—High #1	0.006	0.5	52	18	0.00032
Bystander—High #2	0.011	0.5	52	18	0.00059

Note: Estimates of example cumulative lifetime chrysotile doses for both clothes handlers and bystanders associated with the handling and shaking out of clothing contaminated with chrysotile, according to the assumptions provided. Also provided for comparison are estimates of the lifetime cumulative dose associated with exposure to chrysotile in ambient air.

<sup>a</sup>Except for USEPA,<sup>83</sup> which counted chrysotile fibers using ISO 10312, all fibers >0.5 μm in length.

<sup>b</sup>
$$\frac{f}{cc \cdot yr} = \frac{f}{cc} \times \frac{ET}{24} \times \frac{EF}{365} \times ED$$

<sup>c</sup>Lee and Van Orden<sup>79</sup> (f/cc, all asbestos fibers >5 μm in length).

<sup>d</sup>USEPA<sup>83</sup> (f/cc, chrysotile fibers >0.5 μm in length).

<sup>e</sup>Nolan and Langer<sup>81</sup> (f/cc, chrysotile fibers >5 μm in length).

doses for clothes handling for this study were either below or consistent with lifetime background (e.g., ambient air) chrysotile doses (0.00044–0.0047 f/cc year for clothes handling vs. 0.0035–0.105 f/cc year for ambient chrysotile exposures).<sup>(81,83)</sup> A number of published epidemiological studies have indicated that exposures to ambient asbestos concentrations (of any fiber type) are not associated with a significantly increased incidence of asbestos-related disease.<sup>(84–91)</sup> Similarly, numerous epidemiological studies have demonstrated no significantly increased incidence of mesothelioma or lung cancer in vehicle mechanics who have worked with chrysotile-containing vehicle parts such as brakes.<sup>(40,50,92–118)</sup> It has been estimated that upper-bound cumulative lifetime doses of chrysotile for vehicle mechanics, up through the 1970s, were in the range of 1.96–2.79 f/cc year.<sup>(119)</sup>

These comparative dose estimates and the corresponding epidemiological literature indicate that chrysotile workplace exposures at the airborne concentrations tested in this study (up to 0.55 f/cc as an eight-hour TWA) would not measurably increase the nonoccupational risk of asbestos-related disease above background rates for those in the home handling chrysotile-contaminated clothing under conditions similar to this study. For some historical occupational scenarios with airborne asbestos concentrations above those studied here, or for scenarios with substantial disparities in fiber potency to this study, these differences could very well be sufficient to explain the asbestos-related diseases reported in studies of persons with take-home exposure. The analyses of Bourdes *et al.*, Donovan *et al.*, and Goswami *et al.* all point to an increased risk of disease associated with take-home exposures to amosite or mixed fibers above certain cumulative lifetime doses from the clothing of certain workers such as asbestos product manufacturers, insulators, or shipyard personnel.<sup>(12,53,54)</sup>

This study also had several other interesting findings. It was observed that airborne chrysotile concentrations declined dramatically after the cessation of clothes handling, and resulted in a nondetectable concentration for the second 15-minute period of inactivity following clothes handling for all but one of the handling and shake-out events. The results of the study also demonstrated an important finding that PCM concentrations were 2- to 50-fold higher than TEM concentrations for all handling and shakeout events. This result clearly showed that many, or most, of the fibers detected by PCM were clothing fibers rather than chrysotile fibers.

Although this study systematically and carefully attempted to quantitatively address take-home exposure potential for the conditions tested, there are aspects of the study design that should be considered in future studies. First, the ventilation rate used in the chamber during the study might be higher in some cases than what could be seen in a home environment. This difference was unlikely to have influenced the measured personal breathing zone concentrations during clothes handling (e.g., in the near field within arm's length of the clothes handler), but may have influenced the results obtained for the area (bystander) sample locations and the concentrations during the 15-minute period of no activity. For the bystander locations, the level of dilution ventilation would have affected the airborne chrysotile concentrations over time. Second, the mannequins did not move during clothes-loading events, whereas a worker moves around all day long, likely causing some fibers to be released from the clothing back into the work area. The mannequin scenario may have increased the number of fibers remaining on the clothing at the end of the loading event periods and prior to clothes handling. Third, the study did not directly consider other mechanisms of fiber removal, such as contact with surfaces or vehicle transport during commuting between the workplace and home environment, which likely historically influenced the number of chrysotile fibers that would remain on a worker's clothing. And fourth, the effect of the duration of clothes-handling and shake-out activities (e.g., one to five minutes vs. 15 minutes of clothes handling) or the influence of conducting an initial clothing shake out or brush off of the clothing outside the home before taking it to the laundry area was not directly addressed. The way clothes are treated prior to handling and shake out could potentially substantially influence exposures to a clothes handler in an enclosed, indoor environment, such as was evaluated in this study.

It should be noted that, similarly, some of the variations in the magnitude of handling and shake-out air concentrations in our study could have been caused by differences in how the clothes were treated between the loading and shake-out events. For example, the clothing from the Medium #2 and High #2 loading events was carefully removed from the mannequins and placed in a nonconductive box prior to handling and shake out, whereas the clothing from the Medium #1 and High #1 loading events was left on the mannequins until handling and shake out began. The difference in average air concentrations between the Medium #1 and Medium #2 30-minute

handling and shake-out events was notable (0.021 f/cc TEM for Medium #1 vs. 0.006 f/cc TEM or ND for Medium #2) and might have been affected by the removal of some of the residual fibers on the clothing when the clothes were taken off the mannequins. However, such a difference was not seen for average concentrations measured during 30-minute handling and shake-out events for High #1 and High #2 (0.05 f/cc TEM for High #1 and 0.063 f/cc TEM for High #2). Additional studies would be desirable for quantifying the effects of the degree of contamination of the clothing due to factors such as transfer to auto seats, whether the clothes were blown off at work, whether the clothing was shaken out in the outdoors prior to bringing it in the home for washing, etc. It is expected that these activities would likely decrease the extent of asbestos contamination on the clothing that would eventually be brought into the home.

There are a few other important considerations to keep in mind regarding use of the results of this study for evaluating the potential risk for take-home exposure in specific scenarios. First, fiber release from the clothing may not always be proportional to the loading time (i.e., if the loading time were eight times longer, the corresponding airborne concentrations generated during clothes handling and shake out should not be assumed to be eight times the reported clothes-handling concentrations). There may be a limit at which no additional loading of fibers on the clothing is likely to occur. Therefore, although this limit did not appear to be reached in this study, it would not be appropriate to assume that there would be no upper limit to the amount of fibers that could be loaded onto the clothing, or at what concentration this upper limit might occur. In addition, known contamination of clothing with chrysotile may not always result in measurable chrysotile exposure potential to the clothes handler during shake out for all scenarios and concentrations. Although this study used a value of one-half of the LOD or sensitivity limit to estimate PCM and TEM exposures below the sample detection limit, so as not to underestimate the airborne concentrations, take-home exposures to chrysotile were not measurable for some of the occupational loading concentrations tested in the study. And finally, the relevance of the study data to amosite asbestos is unclear. Key differences in particle characteristics such as electrical charge, physical variations in the fibers, or surface area of fiber contact with clothing surfaces could affect the direct relationship between amosite and chrysotile workplace and

take-home exposures.<sup>(120,121)</sup> It would be helpful to conduct a similar study with amosite to remove this uncertainty.

This study provides quantitative information about how airborne chrysotile concentrations in a work environment may translate to exposures and corresponding nonoccupational risks in the home through clothes-handling and shake-out activities prior to laundering. An important observation from this study was the extent to which clothing fibers released during the handling and shake-out events contributed to the total PCM measurements. PCM measurements were up to 50-fold greater than those observed with asbestos-specific analytical techniques (e.g., TEM/PCME). In addition, there were clear trends observed for the TEM and PCME measurements (i.e., in general, as occupational loading concentrations increased, so too did the concentrations during clothes handling and shake out). This trend was not apparent with the PCM data, underscoring the contribution of clothing fibers to the PCM results, and the value of asbestos-specific analytical methods, particularly when there is a need to characterize asbestos exposures in the context of health risk. No previous historical study from the 1970s and 1980s used TEM, and therefore their results most likely overestimated exposure. In summary, the quantitative airborne chrysotile concentration measurements for the simulated occupational settings evaluated in this study and the corresponding take-home airborne concentrations associated with the handling and shake out of the clothing contaminated with chrysotile give some quantitative insight into both exposure potential and risk to household members who historically conducted such activities.

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## REFERENCES

1. Selikoff IJ, Lee DH. *Asbestos and Disease* (Table 1-3). New York: Academic Press, 1978.
2. NIOSH. Criteria for a recommended standard: Occupational exposure to asbestos. HSM 72-10267. In Series Criteria for a Recommended Standard: Occupational Exposure to Asbestos. HSM 72-10267. Washington, DC: U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1972.
3. Merewether ERA, Price CW. Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry. Part I and II. London: His Majesty's Stationery Office, 1930.
4. Fleischer WE, Viles FJ, Jr., Gade RL, Drinker P. A health survey of pipe covering operations in constructing naval vessels. *Journal of Industrial Hygiene and Toxicology*, 1946; 28:9-16.
5. Marr WT. Asbestos exposure during naval vessel overhaul. *American Industrial Hygiene Association Journal*, 1964; 25:264-268.
6. Balzer JL, Cooper WC. The work environment of insulating workers. *American Industrial Hygiene Association Journal*, 1968; 29(3):222-227.
7. Cooper WC, Balzer JL. Evaluation and control of asbestos exposure in the insulating trade. In Proceedings of the Second International Conference on the Biological Effects of Asbestos, April 21-24. Dresden, Germany, 1968.
8. Gibbs GW, Lachance M. Dust exposure in the chrysotile asbestos mines and mills of Quebec. *Archives of Environmental Health*, 1972; 24(3):189-197.
9. Nicholson WJ, Holaday DA, Heimann H. Direct and indirect occupational exposure to insulation dusts in United States shipyards. Pp. 37-47 in Proceedings of an International Symposium on Safety and Health in Ship Building and Ship Repairing; Helsinki, Finland. Occupational Safety and Health Series: 27. Geneva, Switzerland: International Labour Organization, 1972.
10. Williams PR, Phelka AD, Paustenbach DJ. A review of historical exposures to asbestos among skilled craftsmen (1940-2006). *Journal of Toxicology and Environmental Health Part B: Critical Reviews*, 2007; 10(5):319-377.
11. Zirscky J. Take-home toxin pathway. *Journal of Environmental Engineering*, 1996; 122(5):430-436.
12. Donovan EP, Donovan BL, McKinley MA, Cowan DM, Paustenbach DJ. Evaluation of take home (para-occupational) exposure to asbestos and disease: A review of the literature. *Critical Reviews in Toxicology*, 2012; 42(9):703-731.
13. OSHA. 29 CFR Parts 1910. Occupational exposure to asbestos; Final rule. *Federal Register*, 1994; 59:40964-41162. Washington, DC: U.S. Department of Labor, Occupational Safety and Health Administration (OSHA), August 10, 1994.
14. IARC. Asbestos. Pp. 11-81 in IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Asbestos, Vol. 14. Lyon, France: International Agency for Research on Cancer, 1977.
15. Shukla A, Gulumian M, Hei TK, Kamp D, Rahman Q, Mossman BT. Multiple roles of oxidants in the pathogenesis of asbestos-induced diseases. *Free Radical Biology and Medicine*, 2003; 34(9):1117-1129.
16. Veblen DR, Wylie AG. Mineralogy of amphiboles and 1:1 layer silicates. Pp. 61-137 in Gutherie GD, Mossman BT (eds). *MSA Reviews in Mineralogy*, Volume 28. Health Effects of Mineral Dusts. Washington, DC: Mineralogical Society of America, 1993.
17. Enterline PE, Henderson V. Type of asbestos and respiratory cancer in the asbestos industry. *Archives of Environmental Health*, 1973; 27(5):312-317.
18. Meurman LO, Kiviluoto R, Hakama M. Mortality and morbidity among the working population of anthophyllite asbestos miners in Finland. *British Journal of Industrial Medicine*, 1974; 31(2):105-112.
19. Hodgson JT, Darnton A. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *Annals of Occupational Hygiene*, 2000; 44(8):565-601.
20. Berman DW, Crump KS. Final Draft: Technical Support Document for a Protocol to Assess Asbestos-Related Risk, October 2003. EPA# 9345.4-06. Washington, DC: U.S. Environmental Protection Agency (EPA), Office of Solid Waste and Emergency Response Report No. EPA# 9345.4-06.
21. ERG. Report on the Expert Panel on Health Effects of Asbestos and Synthetic Vitreous Fibers: The Influence of Fiber Length Prepared for the Agency for Toxic Substances and Disease Registry (ATSDR), Division of Health Assessment and Consultation, Lexington, MA, March 17, 2003.
22. Yarborough CM. Chrysotile as a cause of mesothelioma: An assessment based on epidemiology. *Critical Reviews in Toxicology*, 2006; 36(2):165-187.
23. Gibbs GW, Berry G. Mesothelioma and asbestos. *Regulatory Toxicology and Pharmacology*, 2008; 52(1):S223-S231.
24. Hodgson JT, McElvenny DM, Darnton AJ, Price MJ, Peto J. The expected burden of mesothelioma mortality in Great Britain from 2002 to 2050. *British Journal of Cancer*, 2005; 92(3):587-593.
25. Berman DW, Crump KS. A meta-analysis of asbestos-related cancer risk that addresses fiber size and mineral type. *Critical Reviews in Toxicology*, 2008; 38(1):49-73.
26. Berman DW, Crump KS. Update of potency factors for asbestos-related lung cancer and mesothelioma. *Critical Reviews in Toxicology*, 2008; 38(1):1-47.
27. Ayer H. The proposed ACGIH mass limits for quartz: Review and evaluation. *American Industrial Hygiene Association Journal*, 1969; 30(2):117-123.
28. Lynch JR, Ayer HE. Measurement of asbestos exposure. *Journal of Occupational Medicine*, 1968; 10(1):21-24.
29. Bloomfield JJ, Dallavalle JM. The Determination and Control of Industrial Dust, Public Health Bulletin No. 217. Washington, DC: U.S. Treasury Department, 1935.
30. Bayer SG, Zumwalde RD, Brown TA. Equipment and Procedure for Mounting Millipore Filters and Counting Asbestos Fibers by Phase Contrast Microscopy. Cincinnati, OH: Bureau of Occupational Health, U.S. Dept. of Health, Education and Welfare, July 1969.
31. OSHA. Title 29-Labor, Chapter XVII, Part 1910-Occupational Safety and Health Standards, National Consensus Standards and Established Federal Standards (Introduction, Subparts A and B, and Portion of Subpart G). 36 FR 10466-10469, 10503-10506. Washington, DC: U.S. Department of Labor-Occupational Safety and Health Administration (OSHA), May 29, 1971.
32. Wagner JC, Sleggs CA, Marchand P. Diffuse pleural mesothelioma and asbestos exposure in the North Western

Cape Province. *British Journal of Industrial Medicine*, 1960; 17:260–271.

33. Newhouse ML, Thompson H. Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. *British Journal of Industrial Medicine*, 1965; 22(4): 261–269.
34. Rom W, Miller A, Anderson H, Selikoff IJ. Changes in pulmonary function in household contacts of asbestos workers. *American Review of Respiratory Disease*, 1976; 113(4):88.
35. Anderson HA, Lilis R, Daum SM, Fischbein AS, Selikoff IJ. Household-contact asbestos neoplastic risk. *Annals of the New York Academy of Sciences*, 1976; 271:311–323.
36. Anderson H, Lilis R, Daum S, Fischbein A, Selikoff IJ. Household exposure to asbestos and risk of subsequent disease. Pp. 145–156 in Lemen R, Dement J (eds). *Dusts and Disease*. Chicago, IL: Pathotox Publishers, 1979.
37. Anderson HA, Lilis R, Daum S, Selikoff IJ. Asbestosis among household contacts of asbestos factory workers. Pp. 387–399 in Selikoff IJ, Hammond EC (eds). *Health Hazards of Asbestos Exposure*. Vol. 330. New York: Annals of the New York Academy of Sciences, 1979.
38. Anderson HA. Family contact exposure. Pp. 349–362 in *Asbestos, Health and Society: Proceedings of World Symposium on Asbestos*. Montreal, Quebec, Canada: Canadian Asbestos Information Center, 1982.
39. Vianna NJ, Polan AK. Non-occupational exposure to asbestos and malignant mesothelioma in females. *Lancet*, 1978; 1(8073):1061–1063.
40. McDonald AD, McDonald JC. Malignant mesothelioma in North America. *Cancer*, 1980; 46(7):1650–1656.
41. Epler GR, Fitz Gerald MX, Gaensler EA, Carrington CB. Asbestos-related disease from household exposure. *Respiration*, 1980; 39(4):229–240.
42. Gibbs AR, Jones JS, Pooley FD, Griffiths DM, Wagner JC. Non-occupational malignant mesotheliomas. *IARC Scientific Publications*, 1989; (90):219–228.
43. Kilburn KH, Lilis R, Anderson HA, Boylen CT, Einstein HE, Johnson SJ, Warshaw R. Asbestos disease in family contacts of shipyard workers. *American Journal of Public Health*, 1985; 75(6):615–617.
44. Ashcroft T, Heppleston G. Mesothelioma and asbestos in Tyneside: A pathological and social study. Pp. 177–179 in *Pneumoconiosis: Proceedings of the International Conference 1969*, Johannesburg. Cape Town: Oxford University Press, 1970.
45. Edge JR, Choudhury SL. Malignant mesothelioma of the pleura in Barrow-in-Furness. *Thorax*, 1978; 33(1):26–30.
46. Magnani C, Terracini B, Ivaldi C, Botta M, Budel P, Mancini A, Zanetti R. A cohort study on mortality among wives of workers in the asbestos cement industry in Casale Monferrato, Italy. *British Journal of Industrial Medicine*, 1993; 50(9):779–784.
47. Reid A, Heyworth J, de Klerk N, Musk AW. The mortality of women exposed environmentally and domestically to blue asbestos at Wittenoom, Western Australia. *Occupational and Environmental Medicine*, 2008; 65(11):743–749.
48. Magnani C, Agudo A, Gonzalez CA, Andriola A, Calleja A, Chellini E, Dalmasso P, Escolar A, Hernandez S, Ivaldi C, Mirabelli D, Ramirez J, Turuguet D, Usel M, Terracini B. Multicentric study on malignant pleural mesothelioma and non-occupational exposure to asbestos. *British Journal of Cancer*, 2000; 83(1):104–111.
49. Ferrante D, Bertolotti M, Todesco A, Mirabelli D, Terracini B, Magnani C. Cancer mortality and incidence of mesothelioma in a cohort of wives of asbestos workers in Casale Monferrato, Italy. *Environmental Health Perspectives*, 2007; 115(10):1401–1405.
50. Rake C, Gilham C, Hatch J, Darnton A, Hodgson J, Peto J. Occupational, domestic and environmental mesothelioma risks in the British population: A case-control study. *British Journal of Cancer*, 2009; 100(7):1175–1183.
51. Case BW, Camus M, Richardson L, Parent M-E, Desy M, Siemiatycki J. Preliminary findings for pleural mesothelioma among women in the Quebec chrysotile mining regions. *Annals of Occupational Hygiene*, 2002; 46(1):128–131.
52. Howel D, Arblaster L, Swinburne L, Schweiger M, Renvoize E, Hatton P. Routes of asbestos exposure and the development of mesothelioma in an English region. *Occupational and Environmental Medicine*, 1997; 54(6):403–409.
53. Bourdes V, Boffetta P, Pisani P. Environmental exposure to asbestos and risk of pleural mesothelioma: Review and meta-analysis. *European Journal of Epidemiology*, 2000; 16(5):411–417.
54. Goswami E, Craven V, Dahlstrom DL, Alexander D, Mowat F. Domestic asbestos exposure: A review of epidemiologic and exposure data. *International Journal of Environmental Research and Public Health*, 2013; 10(11):5629–5670.
55. NIOSH. Report to Congress on Workers' Home Contamination Study Conducted Under the Worker's Family Protection Act (29 U.S.C. 671a). NIOSH Pub. No. 95–123. September 1995. Cincinnati, OH: National Institute for Occupational Safety and Health, 1995.
56. Gibbs AR, Griffiths DM, Pooley FD, Jones JS. Comparison of fibre types and size distributions in lung tissues of paraoccupational and occupational cases of malignant mesothelioma. *British Journal of Industrial Medicine*, 1990; 47(9):621–626.
57. Sawyer RN. Asbestos exposure in a Yale building. Analysis and resolution. *Environmental Research*, 1977; 13(1): 146–169.
58. Nicholson WJ, Rohl AN, Weisman I, Selikoff IJ. Environmental asbestos concentrations in the United States. Pp. 823–827 in Wagner JC (ed). *Biological Effects of Mineral Fibres*, Vol. 2. Lyon, France: Lyon International Agency for Research on Cancer, 1980.
59. Mangold C. The Actual Contribution of Garlock Asbestos Gasket Materials to the Occupational Exposure of Asbestos Workers. Bellevue, WA: Environmental Control Sciences, Inc., October Series, 1982.
60. OSHA. Title 29–Labor, Chapter XVIII, Part 1910–Occupational Safety and Health Standards, Emergency Standard for Exposure to Asbestos Dust. 36 FR 23207–23208. Washington, DC: U.S. Department of Labor–Occupational Safety and Health Administration (OSHA), December 7, 1971.
61. Weir FW, Tolar G, Meraz LB. Characterization of vehicular brake service personnel exposure to airborne asbestos and particulate. *Applied Occupational and Environmental Hygiene*, 2001; 16(12):1139–1146.
62. Madl AK, Scott LL, Murbach DM, Fehling KA, Finley BL, Paustenbach DJ. Exposure to chrysotile asbestos associated with unpacking and repacking boxes of automobile brake pads and shoes. *Annals of Occupational Hygiene*, 2008; 52(6):463–479.
63. Madl AK, Gaffney SH, Balzer JL, Paustenbach DJ. Airborne asbestos concentrations associated with heavy equipment brake removal. *Annals of Occupational Hygiene*, 2009; 53(8):839–857.
64. Jiang GC, Madl AK, Ingmundson KJ, Murbach DM, Fehling KA, Paustenbach DJ, Finley BL. A study of airborne chrysotile concentrations associated with handling, unpacking, and repacking boxes of automobile clutch discs. *Regulatory Toxicology and Pharmacology*, 2008; 51(1): 87–97.
65. NTP. Asbestos, CAS No. 1332–21–4. Report on Carcinogens, 11th ed. Research Triangle Park, NC: U.S. Department of

Health and Human Services (DHHS), Public Health Service, National Toxicology Program (NTP), 2005.

- 66. OSHA. Final Regulatory Impact and Regulatory Flexibility Analysis of the Revised Asbestos Standard. Report No. PB-86-221827/XAB. Washington, DC: OSHA Administration OSHA, 1986.
- 67. Virta RL. Asbestos: Geology, Mineralogy, Mining, and Uses. Open-File Report 02-149. Reston, VA: U.S. Department of the Interior, US Geological Survey, October 2002.
- 68. Ignacio J, Bullock W. A Strategy for Assessing and Managing Occupational Exposure, 3rd ed. Fairfax, VA: American Industrial Hygiene Association Press, 2006.
- 69. Oehlert GW. Statistics analysis of asbestos fibre counts. *Environmetrics*, 1995; 6:115-126.
- 70. Millard SP, Deverel SJ. Nonparametric statistical methods for comparing two sites based on data with multiple nondetect limits. *Water Resources Research*, 1988; 24(12):2087-2098.
- 71. Palachek AD, Weier DR, Gatilffe TR, Splett DM, Sullivan DK. Statistical methodology for determining contaminants of concern by comparison of background and site data with applications to operable unit 2. In *Statistical Applications*. SA-93-010. Golden, CO: EG&G Rocky Flats, Inc., 1993.
- 72. USEPA. Airborne asbestos health assessment update. EPA/600/8-84/003F. June 1986. Pp. 71-73 in *Series Airborne Asbestos Health Assessment Update*. EPA/600/8-84/003F. June 1986. Washington, DC: U.S. Environmental Protection Agency (EPA), Office of Health and Environmental Assessment, 1986.
- 73. USEPA. Framework for Investigating Asbestos-Contaminated Superfund Sites. OSWER Directive 9200.0-68. Washington, DC: U.S. Environmental Protection Agency (USEPA), Office of Solid Waste and Emergency Response, Technical Review Workgroup, Asbestos Committee, Sept. 2008.
- 74. OSHA. 29 CFR Parts 1910 and 1926: Occupational exposure to asbestos, tremolite, anthophyllite, and actinolite; final rules. *Occupational Safety and Health Administration (OSHA)*. *Federal Register*, 1986; 51(119):22612-22790.
- 75. Camus M, Siemiatycki J, Case BW, Desy M, Richardson L, Campbell S. Risk of mesothelioma among women living near chrysotile mines versus USEPA asbestos risk model: Preliminary findings. *Annals of Occupational Hygiene*, 2002; 46(1):95-98.
- 76. Berman DW. Apples to apples: The origin and magnitude of differences in asbestos cancer risk estimates derived using varying protocols. *Risk Analysis*, 2011; 31(8):1308-1326.
- 77. USEPA. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual Supplemental Guidance "Standard Default Exposure Factors," Interim Final. OSWER Directive 9285.6-03. Washington, DC: U.S. Environmental Protection Agency (USEPA), Toxics Integration Branch, Office of Emergency and Remedial Response, March 25, 1991.
- 78. Crump KS, Farrar DB. Statistical analysis of data on airborne asbestos levels collected in an EPA survey of public buildings. *Regulatory Toxicology and Pharmacology*, 1989; 10(1):51-62.
- 79. Lee RJ, Van Orden DR. Airborne asbestos in buildings. *Regulatory Toxicology and Pharmacology*, 2008; 50(2):218-225.
- 80. Mangold C. Asbestos Fibers in the Ambient Air in the Greater San Francisco Area. Bellevue, WA: Environmental Control Sciences, Inc., March Series, 1983.
- 81. Nolan RP, Langer AM. Concentration and type of asbestos fibers in air inside buildings. Pp. 39-51 in Nolan RP, Langer AM, Ross M (eds). *The Health Effects of Chrysotile Asbestos: Contribution of Science to Risk-Management Decisions*. Vol. 5. Ottawa, Canada: Canadian Mineralogist, Special Publication, 2001.
- 82. Wendlick JD. Ambient Asbestos Fiber Levels at Selected Sites in Philadelphia, Pennsylvania. November Series, Federal Way, WA, 1984.
- 83. USEPA. Summary of Outdoor Ambient Air Monitoring for Asbestos at the Libby Asbestos Site Libby, Montana (October 2006 to June 2008). Denver, CO: U.S. Environmental Protection Agency (EPA), Region 8 and SRC, February 9, 2009.
- 84. Price B, Ware A. Mesothelioma trends in the United States: An update based on surveillance, epidemiology, and end results program data for 1973 through 2003. *American Journal of Epidemiology*, 2004; 159(2):107-112.
- 85. Price B, Ware A. Mesothelioma: Risk apportionment among asbestos exposure sources. *Risk Analysis*, 2005; 25(4): 937-943.
- 86. Teta MJ, Mink PJ, Lau E, Sceurman BK, Foster ED. US mesothelioma patterns 1973-2002: Indicators of change and insights into background rates. *European Journal of Cancer Prevention*, 2008; 17(6):525-534.
- 87. Moolgavkar SH, Meza R, Turim J. Pleural and peritoneal mesotheliomas in SEER: Age effects and temporal trends, 1973-2005. *Cancer Causes Control*, 2009; 20(6):935-944.
- 88. Antman KH, Schiff PB, Pass HI. Benign and malignant mesothelioma. Pp. 1853-1878 in DeVita VT, Hellman SS, Rosenberg SA (eds). *Cancer: Principles & Practice of Oncology*, 5th ed. Vol. 2. Philadelphia: Lippincott-Raven Publishers, 1997.
- 89. McDonald JC. Health implications of environmental exposure to asbestos. *Environmental Health Perspectives*, 1985; 62:319-328.
- 90. McDonald JC, McDonald A. Mesothelioma: Is there a background? Pp. 37-45 in Jaurand M, Bignon J (eds). *The Mesothelial Cell and Mesothelioma*. New York: Marcel Decker, 1994.
- 91. Moore AJ, Parker RJ, Wiggins J. Malignant mesothelioma. *Orphanet Journal of Rare Diseases*, 2008; 3:34.
- 92. Teta MJ, Lewinsohn HC, Meigs JW, Vidone RA, Mowad LZ, Flannery JT. Mesothelioma in Connecticut, 1955-1977. Occupational and geographic associations. *Journal of Occupational Medicine*, 1983; 25(10):749-756.
- 93. Spirtas R, Keehn R, Wright W, Stark A, Beebe G, Dickson E. Mesothelioma risk related to occupational or other asbestos exposure: Preliminary results from a case-control study. *American Journal of Epidemiology*, 1985; 122(3):518.
- 94. Teschke K, Morgan MS, Checkoway H, Franklin G, Spinelli JJ, van Belle G, Weiss NS. Mesothelioma surveillance to locate sources of exposure to asbestos. *Canadian Journal of Public Health*, 1997; 88(3):163-168.
- 95. Woitowitz HJ, Rodelsperger K. Mesothelioma among car mechanics? *Annals of Occupational Hygiene*, 1994; 38(4):635-638.
- 96. Agudo A, Gonzalez CA, Bleda MJ, Ramirez J, Hernandez S, Lopez F, Calleja A, Panades R, Turuguet D, Escolar A, Beltran M, Gonzalez-Moya JE. Occupation and risk of malignant pleural mesothelioma: A case-control study in Spain. *American Journal of Industrial Medicine*, 2000; 37(2):159-168.
- 97. Hessel PA, Teta MJ, Goodman M, Lau E. Mesothelioma among brake mechanics: An expanded analysis of a case-control study. *Risk Analysis*, 2004; 24(3):547-552.
- 98. Milham S, Ossiander E. Occupational mortality in Washington State 1950-1999. Pp. 1-67, 1659-1660, 835-851, 2096-2104 in *Series Occupational Mortality in Washington State 1950-1999*. Olympia, WA: Washington State Department of Health, Office of Epidemiology, 2001.
- 99. Olsen JH, Jensen OM. Occupation and risk of cancer in Denmark. An analysis of 93,810 cancer cases, 1970-1979. *Scandinavian Journal of Work, Environment and Health*, 1987; 13(1):1-91.

100. Jarvholt B, Brisman J. Asbestos associated tumours in car mechanics. *British Journal of Industrial Medicine*, 1988; 45(9):645–646.

101. NIOSH. Letter from J.T. Walker to M.J. Teta Regarding Her Inquiry About PMRs for Auto Mechanics, with Attached Data Sheets. May 7, 2002. Cincinnati, OH: Department of Health and Human Service (DHHS), Public Health Service, National Institute for Occupational Safety and Health (NIOSH), 2002.

102. Hansen J, Meersohn A. Cancer Mortality Among Danish Employees (1970–1997) by Industry Classification [Kraeftsygelihed blandt danske lønmodtagere (1970–97) fordelt på Arbejdstilsynets 49 branchegrupper]. Kraeftens Bekæmpelse, Kobenhavn: Institut for Epidemiologisk Kraeftforskning, 2003.

103. Rolland P, Gramond C, Lacourt A, Astoul P, Chamming's S, Ducamp S, Frenay C, Galateau-Salle F, Ilg AG, Imbernon E, Le Stang N, Paireon JC, Goldberg M, Brochard P. Occupations and industries in France at high risk for pleural mesothelioma: A population-based case-control study (1998–2002). *American Journal of Industrial Medicine*, 2010; 53(12):1207–1219.

104. Rolland P, Gramond C, Berron H, Ducamp S, Imbernon E, Goldberg M, Brochard P. Pleural mesothelioma: Professions and occupational areas at risk among humans [Mesotheliome pleural: Professions et secteurs d'activité à risque chez les hommes]. Saint-Maurice, France: Institut de Veille Sanitaire, Département Santé Travail, October 2005.

105. Coggon D, Inskip H, Winter P, Pannett B. Differences in occupational mortality from pleural cancer, peritoneal cancer, and asbestosis. *Occupational and Environmental Medicine*, 1995; 52(11):775–777.

106. Hodgson JT, Peto J, Jones JR, Matthews FE. Mesothelioma mortality in Britain: Patterns by birth cohort and occupation. *Annals of Occupational Hygiene*, 1997; 41(1):129–133.

107. Goodman M, Teta MJ, Hessel PA, Garabrant DH, Craven VA, Scrafford CG, Kelsh MA. Mesothelioma and lung cancer among motor vehicle mechanics: A meta-analysis. *Annals of Occupational Hygiene*, 2004; 48(4):309–326.

108. Wong O. Malignant mesothelioma and asbestos exposure among auto mechanics: Appraisal of scientific evidence. *Regulatory Toxicology and Pharmacology*, 2001; 34(2):170–177.

109. Williams RR, Stegens NL, Goldsmith JR. Associations of cancer site and type with occupation and industry from the Third National Cancer Survey Interview. *Journal of the National Cancer Institute*, 1977; 59(4):1147–1185.

110. Lerchen ML, Wiggins CL, Samet JM. Lung cancer and occupation in New Mexico. *Journal of the National Cancer Institute*, 1987; 79(4):639–645.

111. Benhamou S, Benhamou E, Flamant R. Occupational risk factors of lung cancer in a French case-control study. *British Journal of Industrial Medicine*, 1988; 45(4):231–233.

112. Vineis P, Thomas T, Hayes RB, Blot WJ, Mason TJ, Pickle LW, Correa P, Fontham ET, Schoenberg J. Proportion of lung cancers in males, due to occupation, in different areas of the USA. *Int J Cancer*, 1988; 42(6):851–856.

113. Hrubec Z, Blair A, Rogot E, Vaught JB. Mortality Risks by Occupation Among US Veterans of Known Smoking Status. Bethesda, MD: U.S. Department of Health and Human Services, Public Health Service, National Institute of Health, 1954–1980.

114. Morabia A, Markowitz S, Garibaldi K, Wynder EL. Lung cancer and occupation: Results of a multicentre case-control study. *British Journal of Industrial Medicine*, 1992; 49(10):721–727.

115. Boillat MA, Lob M. [Risk of asbestosis in workers employed in replacing automobile brake linings]. *Schweiz Med Wochenschr*, 1973; 103(39):1354–1359.

116. Elliehausen H-J, Paur R, Rodelsperger K, Woitowitz H-J. Zum Risiko von Asbestinhaltionsfolgen bei Draft-fahrzeugmechanikern in Bremsendiensten [About the risk of asbestos inhalation in automobile mechanics in the brake service]. *Arbeitsmed Sozialmed Prevenitmed*, 1985; 20:256–261.

117. Marcus K, Jarvholt BG, Larsson S. Asbestos-associated lung effects in car mechanics. *Scandinavian Journal of Work, Environment and Health*, 1987; 13(3):252–254.

118. Nicholson WJ, Daum SM, Lorimer WV, Velez H, Lilis R, Seilikoff IJ, Miller A, Andson HA, Fischbein SA, Holstein EC, Rom WN, Rosenman K, Todaro JD, Cheng W, Li V, Tarr DT. Investigation of Health Hazards in Brake Lining Repair and Maintenance Workers Occupationally Exposed to Asbestos. Cincinnati, OH: National Institute for Occupational Safety and Health, 1979.

119. Finley BL, Richter RO, Mowat FS, Mlynarek S, Paustenbach DJ, Warmerdam JM, Sheehan PJ. Cumulative asbestos exposure for US automobile mechanics involved in brake repair (circa 1950s–2000). *Journal of Exposure Science and Environmental Epidemiology*, 2007; 17(7):644–655.

120. ATSDR. Toxicological Profile for Asbestos. Atlanta, GA: U.S. Department of Health and Human Services (DHHS), Public Health Service, Agency for Toxic Substances and Disease Registry (ATSDR), 2001.

121. Aust AE, Cook PM, Dodson RF. Morphological and chemical mechanisms of elongated mineral particle toxicities. *Journal of Toxicology and Environmental Health Part B: Critical Reviews*, 2011; 14(1–4):40–75.

## SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article at the publisher's website:

**Fig. S1.** Depiction and chamber design for clothes-loading events (simulated work environment).

**Fig. S2.** Airborne fiber measurements during clothes-loading events by sample location in the test chamber.

**Fig. S3.** Box plot distributions of the PCM (NIOSH 7400) and TEM (NIOSH 7402) airborne concentrations for each loading event.

**Table SI.** Loading Event Sample Numbers, Concentrations, and Standard Deviations (PCM, TEM, and PCME)

**Table SII.** First and Second 15-Minute Clothes-Handling and Shakeout Shake-Out Event Sample Numbers, Concentrations, and Standard Deviations (PCM, TEM, and PCME)

**Table SIII.** Thirty-Minute Clothes-Handling and Shakeout Shake-Out Event Sample Numbers, Concentrations, and Standard Deviations (PCM, TEM, and PCME)