Editorial

Commentary on 'Evaluation of take home (para-occupational) exposure to asbestos and disease: a review of the literature', Donovan *et al.*[1]

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We believe that Donovan *et al.*'s paper titled, "Evaluation of take home (para-occupational) exposure to asbestos and disease: a review of the literature" omits important information that contradicts the authors' conclusion that take home exposure to chrysotile asbestos does not cause asbestos disease.[1] The paper's stated purpose suggests a single-minded scope, scope with its conclusions indicating that the paper was meant to address issues presented in litigation: "In recent years, toxic tort litigation involving asbestos claims has reached record levels; an increasing number of these cases involve claims of para-occupational exposure."[1] Furthermore, Dr. Paustenbach, one of the authors and the President of ChemRisk, which employs all of the authors, sent a solicitation letter to potential litigation clients stating that this paper and similar papers "enhance our reputation within the scientific, governmental, and environmental health communities, as well as in the court room." [Emphasis added][2] He highlights Donovan *et al.*'s paper in this correspondence: "Please pay particular attention to the 'asbestos take home' paper. It represents a major commitment by our firm."[2] We believe Donovan *et al.*'s financial and ideological biases resulted in omission of key literature and misrepresentation of the data that they did include.

Incomplete Literature Search

While Donovan *et al.* claim to have "conducted a comprehensive search of publicly available documents that directly or indirectly discussed disease in household contacts of persons who worked with asbestos occupationally…and efforts were made to locate unpublished studies," they failed to consider literature with conclusions that differ from those of the authors.

Cohorts of para-occupational exposure from asbestos

Donovan *et al.* omit at least two important articles that discuss para-occupational exposures. First, they fail to mention a 2009 paper which discusses a cohort of women with mesothelioma and domestic/residential asbestos exposure.[3] Egilman reported a cohort of 37 women diagnosed with mesothelioma who had non-occupational exposure to asbestos that was brought home by relatives who worked in Virginia shipyards.[3] The cohort's exposed family members represented a diverse group of occupations including pipefitter, machinist, welder, electrician, and brakeworker.

Where they do address such types of asbestos exposures, Donovan *et al.* asserts that they were always "mixed fiber" exposures, yet fail to cite any reference for this key assumption. We are unaware of any study of fiber type in home exposures. Additionally, Donovan et al. state, "… based on what is known about the types of asbestos used in manufacturing or insulation work up through the 1960s, it is likely that exposures in these settings would have involved amphiboles, and potentially at high airborne concentrations." However, elsewhere in this paper, they correctly conclude that the "vast majority of asbestos used in the United States was chrysotile…"[1] In fact, chrysotile constituted 95% of all the asbestos used in the USA.[4]

Donovan *et al.* also omit a 1998 study of residents of a Canadian chrysotile-mining town.[5] Camus *et al.* reported a statistically significant increased rate of mesothelioma in community residents, some of whom were exposed to take home asbestos (seven in a population of fewer than 20 000). Estimated exposures were low: 7.8 fiber/cc-years or 2–3 fibers/cc per

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year. This result stands in sharp contrast to Donovan et al.'s suggestion "that it takes very high lifetime doses of chrysotile to increase the risk of mesothelioma,"[1] which has also been rebutted elsewhere.[6,7]

Donovan *et al.* also fail to mention a case involving the wife of a Belgian plant worker employed by Eternit, located in Kapelle-op-de-Bos, Belgium.[8] Two of the authors of Donovan *et al.*'s paper (DP and MM) reported on this plant, indicating that it primarily used chrysotile ("less than 10% of the potential asbestos exposures involved amphiboles").[9,10] However, their review failed to report that a woman married to a worker there was recently awarded compensation for her mesothelioma and subsequent death.[8] Eternit admitted, in a widely publicized case, that her disease resulted from para-occupational and environmental exposure.[8]

The authors state that they focused primarily on "para-occupational exposures to asbestos as a result of contact with a household member who had been occupationally exposed." Thus, by design they excluded neighborhood (environmental) cases of mesothelioma which are relevant to the underlying issue of asbestos potency because most of these victims experienced very low exposures. Donovan *et al.* fail to consider environmental cases, despite the fact that they asserted that "these exposures were thought to also contribute to cases of disease."[1] They thus arbitrarily omitted a relatively large body of literature which found that mesothelioma resulted from low exposures, as compared to household or bystander occupational exposures.[11–17]

Environmental exposures likely explain the many asbestos-caused mesothelioma cases that treating physicians erroneously misdiagnosed as idiopathic. For example, in one case, a physician mistakenly diagnosed a woman's mesothelioma as idiopathic until she (not the doctor) remembered that she had lived near an asbestos factory for 2 years during her childhood.[11] Asbestos has been used in the USA in thousands of products, and few physicians are aware of the extent of asbestos usage; as a result, they frequently fail to discover para-occupational exposures. For example, asbestos has been used in Cashmere Bouquet body powder and in some baby powders.[18] Published neighborhood and environmental exposure literature certainly provides evidence that "low" exposures are common, dangerous, and unrecognized.[13] Indeed, Donovan *et al.* fail to mention the environmental cases that were reported in many of their cited papers.[19–23] For example, Donovan *et al.* cite Newhouse and Thomson but fail to mention the 36 patients whom they had identified as having mesothelioma because of environmental exposures from their close proximity to a factory.[24] Additionally, Donovan *et al.* cite one home-related mesothelioma from Mirabelli, while Mirabelli in fact reported at least five, and possibly six, other mesothelioma cases related to environmental exposure outside of the mine.[25] Donovan *et al.* do not explain why they chose to omit these cases. Another citation mentions eight cases of pleural mesothelioma that resulted from environmental exposure (living near a factory), but again, Donovan *et al.* omit this information.[26] Donovan *et al.*'s decision to omit environmental exposure literature resulted in an underestimate of asbestos risk and potency.

State-of-the-Art

Donovan *et al.* claim their literature search identified the 1960s as the time period during which "the first case reports emerged indicating a possible risk of asbestos-related disease among persons who had grown up in asbestos mining or manufacturing areas, as well as among family members of workers at asbestos manufacturing or mining facilities."[1] Not only was risk of para-occupational exposure to asbestos understood in the early 1930s, but risk to family members from para-occuaptional exposure to toxic substances in general has been recognized for over a century. By the 1960s, US corporations were well aware of these risks. As Selikoff noted, "Scientific observations established hazards associated with exposure to asbestos in the manufacture and use of asbestos products, including insulation work. It remained for appropriate epidemiologic studies to provide quantitative measures of the mortality risk."[27]

Tolman's 1913 textbook commented on the risk of home exposure from toxic dusts and described the need to remove "the working-clothes before meals and before leaving the factory [so] the poison is not carried into the lunchroom or into the homes of the workers."[28] (Haddow later labeled asbestos disease as "asbestos poisoning.")[29] In 1914, Thompson noted the need for "opportunities for removing overalls so that they do not have to be worn home when impregnated, for example, with lead dust or dyes."[30] Others also noted the importance of supplying changing rooms and/or different clothes for work in dusty conditions.[31] Finally, Stewart reported asbestos bodies in neighbors of an asbestos plant in 1932, thus indicating — albeit absent any clinical disease — that para-occupational environmental exposures to asbestos had resulted in pathological changes in lung tissue.[32]

Beginning in 1930, with the publication of Merewether's classic report on asbestos disease at the Turner & Newall textile plant in Rochdale England, health professionals recognized that workers near dusty processes were at risk of contracting asbestosis.[33] In the 1940s, several authors suggested

that personnel living near asbestos factories were at risk for contracting asbestos-caused cancer.[34–36] Asbestosis requires an exposure level at least 10-fold above the current TLV that is designed to reduce — but not eliminate — asbestos-induced cancer. In 1949, Wyers identified both men and women, most of whom did not work directly with asbestos, who developed asbestosis:

> The writer has 28 cases under observation at present. The men are occupied as follows: business executive, storekeeper, painter and window cleaner, lagger (in other employ), cleaning brasses, ventilation engineer, in charge of sports and social sections, laboratory handyman, retired, small printing press operator, foreman in charge of stores, foreman in charge of rubber department, light carpenter, tally clerk (week-end relief), handyman in canteen, at home recovering from tuberculosis and one relief gateman. The women are occupied as follows: light housework, in charge of works solarium, canteen work, repairing overalls in works laundry, three doing light housework and two doing sewing duties and mending duties at home.[37]

Most of these cases appear to have resulted from para-occupational exposure in both men (such as business executive and relief gateman) and women (light housework, sewing and mending duties at home). Other textbooks specifically note para-occupational risk for asbestos work. For example, in 1959, Eckardt, the medical director of Esso, wrote, "The use of double lockers, one for street clothes and one for work clothes, seems warranted in this [asbestos] industry."[38]

Post-1960 awareness of risk and dose

After 1960, companies developed direct knowledge of asbestos take home exposures that resulted in mesotheliomas. In 1964, Thomas Mancuso, a professor at the University of Pittsburg, told a consulting actuary for Phillip Carey, an asbestos insulation manufacturer:

> It was found that the wives developed Berylliosis from laundering the workers' clothes, just as now observed with the wives of asbestos workers. Similarly just as Berylliosis was detected among residents within a ¾ mile of the plant from air pollution, so now they have found cancer (mesotheliomas) of residents living within a half mile of the asbestos plant. You recall of course the law suits that followed with Brush Beryllium Company, and the Beryllium Company of Pennsylvania…[39]

Donovan *et al.* fail to cite the 1975 testimony of the Johns-Manville Corporation to the Occupational Safety and Health Administration (OSHA) which contradicts their argument that home exposures are somehow as low as their arbitrary calculation of 0.56 f/cc-year due to laundry and shaking out clothes which relies on unsubstantiated assumptions.[1] Manville's testimony includes a discussion of take home exposure potential and risk from asbestos: "Once asbestos is carried home by the workman, it accumulates in the home, and its presence in the home is likely to be permanent…family members are getting a 24-hour a day, 7-day a week exposure relatively speaking, rather than a partial exposure… These household exposures also provide an opportunity for repetitive, high, short peak exposures due to the shaking out of work clothes. Lacking specific dust counts over the appropriate time period, any conclusion that the exposures were minimal is totally unacceptable."[40]

Donovan *et al.* excluded important information regarding the fact that there is no asbestos exposure that has been shown not to cause cancer.[41–45] Absent this information, it is impossible to declare after 1964 that unmeasured para-occupational exposure is — or was ever thought to be — benign. The case reports that we (and in some cases, they) cite also contradict their assertion. At the Biological Effects of Asbestos Conference in October of 1964, CG Addingley, research director and director of the industrial health unit of British Belting and Asbestos Ltd, stated, "We do not believe there is any safe level." John Wells, MD, of US Rubber Co. responded, "Our own conclusion, as we began seeing what was happening in our own process, was that the only safe amount of asbestos dust exposure was zero…The safe level is nil and anything above the safe level represents certain risk."[46] It is also important to note that no speaker mentioned differences in potency related to fiber type.

Donovan *et al.* omit more recent publications which conclude that there is no safe level of exposure which has been identified that cannot result in mesothelioma. This includes one of their own citations which explicitly states, "[S]ince there is no known safe level of asbestos exposure, we urge that studies of other groups of family contacts be undertaken so that additional data be available to confirm whether such limited asbestos contact is as dangerous as it appears."[47] National organizations such as National Institute for Occupational Safety and Health, OSHA, Environmental Protection Agency, American Industrial Hygiene Association, the Surgeon General, the United States Congress, the Consumer Product Safety Commission, and local state agencies (Illinois, Louisiana, Maryland, Oregon) all agree that there is no known level below which asbestos does not cause mesothelioma.[48–58] There is no scientific support for the proposition that there is a minimum dose that is required to cause mesothelioma, regardless of the fiber type. Ford Motor has actually declared a "[c]ompany policy when dealing with asbestos, a known carcinogen, in which no safe limit or threshold of exposure is

known…"[59] Ford lawyers have also paid millions of dollars to support their defense of lawsuits brought by users of Ford products containing asbestos.[60,61]

Speculations on Chrysotile Potency

Donovan *et al.* state that "based on our review, the available data do not implicate chrysotile alone as a significant cause of disease among household contacts, but we acknowledge that one cannot rule out the possibility that chronic exposures to concentrations of chrysotile that are high enough to cause asbestosis, and involve very long fibers, may increase the risk of developing mesothelioma."[1] Donovan *et al.*'s unsubstantiated contentions that short fibers are innocuous and that there is a known threshold for cancer causation may be suitable for courtroom testimony but are not scientific arguments.[62] Donovan *et al.* fail to address data from numerous publications that conclude otherwise.[3,63] They cite OSHA's 5 μm fiber length counting cut-off as evidence that shorter fibers are not toxic when in fact, the 5 μm limit is based on feasibility of testing and has nothing whatsoever to do with any fiber length risk analysis. Donovan *et al.* fail to cite any papers which indicate that long fibers are ever found in the pleura of mesothelioma victims. Furthermore, they fail to include references to the literature in which short fiber chrysotile is the only or predominant fiber found in the lung of mesothelioma patients.[3,64,67] Donovan *et al.* both assert a minority view without citing any supporting literature and ignore contrary evidence.[61–63]

Donovan *et al.* fail to acknowledge current literature by Hodgson and Darnton as well as Kanarek that confirms the carcinogenicity of chrysotile alone.[68,69] Donovan et al. also cite Gibbs (1990) and the elevated levels of crocidolite and amosite in Gibbs' lung burden analysis, yet they fail to mention data elsewhere in his paper which shows that chrysotile was the major fiber found in the para-occupational exposures.[70] In a prior paper, Gibbs found that chrysotile was the main fiber in the para-occupational exposure group (218.9 chrysotile, 31.8 crocidolite, 1.5 amosite).[71] In two cases, Gibbs reported that chrysotile was the only fiber type that was present.[71]

Donovan *et al.* do not acknowledge multiple reports of low dose cases of mesothelioma (as short as 1 day).[72] Low doses have been associated with longer latent periods.[73] Many of the papers that Donovan *et al.* do cite with regard to dose contradict their conclusion. For example, Epler states that "asbestos-related manifestations may result from brief or minimal exposure."[74] Magee found mesothelioma cases associated with levels of asbestos consistent with environmental exposures of chrysotile.[75] Without suggesting a particular fiber type, Magnani concluded, "[O]ur results suggest that non-occupational exposure to relatively low-doses of asbestos is a hazard that may contribute to the burden of mesothelioma over the next few decades."[76] In a previous paper, Magnani studied the exposures at a plant where chrysotile was the main exposure.[77,78]

Donovan *et al.* additionally fail to address the possibility of synergistic effects of combined fiber exposures.[4,79] Seidman *et al.* found a five-fold increase in mesothelioma in an amosite insulation manufacturing plant, while Ribak *et al.* found a ten-fold increase in a plant where workers were exposed to both chrysotile and amosite.[80,81]

Donovan *et al.* review the adhesion of particles in simulations of clothes handling and create a mathematical model to predict exposure. They review four studies of "concentrations in simulation studies during agitation or handling of contaminated clothing."[1] They use these studies, conjectures about historical exposures (5 mppcf), incorrect particle–fiber conversion ratios, and non-conservative (and now retracted) estimates of chrysotile potency to suggest that para-occupational exposures from normal household processes are not high enough to cause disease.[43] However, if they are correct, then they have supplied additional evidence to support the proposition that low doses can cause disease since there is irrefutable evidence that low dose environmental exposures to chrysotile have resulted in mesothelioma.[5]

Finally, Donovan *et al.* suggest that somehow there were "vast differences between the work conditions and resulting airborne concentrations of asbestos between 1930 and the 1960s and those experienced by workers after the creation of the Occupational Health and Safety Administration (OSHA) in 1970." There is no evidence that exposures dropped until companies removed asbestos from products, and this did not occur in large part until the late 1970s through the mid-1980s. It is unclear if OSHA, tort litigation, public awareness, or some combination of these influences drove this change. While we agree that the total number of exposed workers has declined, asbestos removal and remediation work still results in considerable exposure.[82] Additionally, Donovan *et al.* claim there is need for additional "fiber burden" studies to determine historical doses, but they fail to explain what hypothesis they would be testing. We know that historical home exposures can cause — and have caused — mesothelioma, yet there is no way to simulate the doses that caused these cases.

Donovan *et al.* wrote this paper to help market their services to their corporate clients seeking to undermine legitimate compensation claims that were based on "para-occupational" exposure, asserting

that "more research is needed" to quantify these exposures before mesothelioma, the signature asbestos disease, can be attributed to para-occupational exposure. The scientific community has never required dose calculation to establish cause–effect relationships for any substance nor has a clinical physician ever required or had a dose estimate prior to diagnosing any occupational or environmental disease. Furthermore, the authors conflate the narrowly defined "para-occupational exposure" with "household exposure" and then fail to cite important literature on household cases, meanwhile completely ignoring important environmental exposure literature. Donovan *et al.* reveal the true purpose of their "research" in the last sentence of the paper when they state that it can "perhaps even help to determine, on a case by case basis, whether disease is likely to be related to asbestos exposure or may have occurred spontaneously (e.g. two to three per million per year)."[1] Yet, as we have noted, some of these background cases were para-occupational. By assuming all background cases are "idiopathic," Donovan *et al.* use a circular argument to create a straw man (dose estimates on a case by case basis) in an effort to establish criteria for compensation that cannot be met.

Disclosures

One of the authors (DE) serves as an expert witness at the request of persons injured by asbestos and asbestos product manufacturing companies who have been sued in asbestos personal injury tort litigation.

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