THE STATE OF SOUTH CAROLINA
In The Court of Appeals

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APPEAL FROM SPARTANBURG COUNTY
Court of Common Pleas

Jean H. Toal, Circuit Court Judge

Appellate Case No. 2017-002611
Case No. 2016-CP-42-1592

Beverly Dale Jolly and Brenda Rice Jolly,..... Respondents,

v.

General Electric Company, et al., ... Defendants,

Of whom Fisher Controls International LLC and Crosby Valve, LLC are the ..... Appellants.

RECORD ON APPEAL
VOLUME XI

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Certificate of Counsel

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STATE OF SOUTH CAROLINA COUNTY OF SPARTANBURG **BEVERLY DALE JOLLY and BRENDA RICE JOLLY,** Plaintiffs, v. **GENERAL ELECTRIC COMPANY,** et al. Defendants.	)))))))))))))	IN THE COURT OF COMMON PLEAS FOR THE SEVENTH JUDICIAL CIRCUIT **C/A No. 2016-CP-42-1592** **PLAINTIFFS' CONSOLIDATED RESPONSE TO DEFENDANTS' MOTIONS *IN LIMINE* TO EXCLUDE THE OPINION THAT "EVERY EXPOSURE" CAUSES DISEASE**

Plaintiffs Beverly Dale Jolly and Brenda Rice Jolly oppose Defendants' motions to exclude the causation testimony of Plaintiffs' expert witnesses. This is a Consolidated Response to Defendant Georgia-Pacific LLC's ("Georgia-Pacific") Motion in Limine to Preclude Plaintiffs' Experts' "No Safe Level/Every Exposure" Opinion, and Defendants Fisher Controls International LLC ("Fisher") and Crosby Valve, LLC's ("Crosby") Motion in Limine No. 1(P) to Exclude Plaintiffs' Experts from Testifying that Every Exposure Contributes to Disease. For the reasons seet forth herein, these motions should be denied.

I. INTRODUCTION

Defendants ask this Court to preclude testimony from Plaintiffs' causation experts that "each and every exposure" caused Plaintiff Dale Jolly's mesothelioma. Plaintiffs' experts are pathologist Dr. John Maddox, occupational medicine doctor Dr. Arthur Frank, and cellular biologist Dr. Arnold Brody. Contrary to Defendants' mischaracterization, Plaintiffs' experts do not hold the opinion that "any exposure" or "every exposure" or "every asbestos fiber" causes mesothelioma generally or caused Jolly's mesothelioma. They will instead testify that mesothelioma is caused by a person's cumulative asbestos exposure. Drs. Maddox and Frank, who will be testifying about specific causation, will further testify that exposures that contribute to the

cumulative dose by meeting certain well-defined criteria can be considered a substantial factor in causing the mesothelioma.

In *Garvin v. Agco Corp.*, Judge Hill rejected a challenge to the admissibility of Dr. Maddox and Dr. Brody's causation testimony. *See* **Ex. 1**, No. 2012-CP-40-6675 (S.C. Ct. Comm. Pleas Nov. 14, 2014). The Court held that the opinions of Dr. Maddox and Dr. Brody were reliable and helpful to the trier of fact. *Id.* at 21-25. The Court further determined that the opinions of these experts supported the jury's finding that the plaintiff developed mesothelioma from breathing asbestos fibers from Crane valves. *Id.* at 25. He rejected the defendant's contention that Dr. Maddox or Dr. Brody hold the opinion that every asbestos exposure contributes to cause disease, noting that "[t]he trial record does not contain any such testimony from any of Plaintiffs' experts." *Id.* at 23.

Contrary to Defendants' assertions, Plaintiffs' experts' opinions regarding the cumulative nature of asbestos diseases and the lack of any safe threshold of exposure are not "unreliable," but are generally accepted medical facts that have been established by a significant body of peer-reviewed medical and scientific literature and have been part of the asbestos litigation since its inception. As in *Garvin*, the opinions of Plaintiffs' experts are reliable and admissible expert opinion testimony. Defendants' motion should therefore be denied.

II. LEGAL STANDARDS FOR THE ADMISSIBLITY OF EXPERT TESTIMONY

The admissibility of expert testimony is governed by South Carolina Rule of Evidence 702. That rule provides that "[i]f scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise." S.C. R. Evid. 702.

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Courts evaluating the admissibility of scientific expert evidence "must find the evidence will assist the trier of fact, the expert witness is qualified, and the underlying science is reliable." *State v. Council*, 335 S.C. 1, 20, 515 S.E.2d 508 (S.C. 1999). The reliability of scientific evidence is evaluated based on several factors, including: (1) the publications and peer review of the technique; (2) prior application of the method to the type of evidence involved in the case; (3) the quality control procedures used to ensure reliability; and (4) the consistency of the method with recognized scientific laws and procedures. *State v. White*, 382 S.C. 265, 274, 676 S.E.2d 684 (S.C. 2009) (citing *Council*, 335 S.C. at 19).

Importantly, the trial court's gatekeeper role is not intended to replace the adversary system. *See Maiz v. Virani*, 253 F.3d 641, 666 (11th Cir. 2001). Rather, "cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof" are the ordinary means to attack an opposing expert. *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 596 (1993).

Courts should not exercise their gatekeeping responsibility by excluding expert testimony that falls within the range of matters on which reasonable experts can disagree. *See Milward v. Acuity Specialty Prods. Group, Inc.*, 639 F.3d 11, 22 (1st Cir. 2011). Trial courts are not "empowered 'to determine which of several competing scientific theories has the best provenance.'" *Id.* at 15 (quoting *Ruiz-Troche v. Pepsi Cola of P.R. Bottling Co.*, 161 F.3d 77, 85 (1st Cir. 1998)). While the trial court may look at the reliability of the expert's methodology, it is for the jury to determine the soundness of the facts underlying the expert's opinion and the correctness of the expert's conclusions. *See id.* 22. Moreover, the entire body of evidence relied on by the expert should be taken into consideration in evaluating the reliability of the opinion, and

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the court should refrain from an "atomistic" approach that determines that each piece of evidence is insufficient, on its own, to support the expert's conclusion. *Id.* The mere fact that an expert's methodology requires the application of scientific judgment does not render that method unreliable. *See id.* at 18.

As set forth herein, the methodology of Plaintiffs' experts is entirely reliable, and their opinions are supported by a wealth of scientific and medical evidence that scientists in their fields rely on. Defendants simply disagree with the conclusions they draw from that evidence. Such disagreement may be explored during cross-examination at trial, but does not warrant excluding the experts' conclusions under Rule 702.

IV. THE QUALIFICATIONS AND CONCLUSIONS OF PLAINTIFFS' EXPERTS.

A. The qualifications and opinions of Dr. Maddox.

Dr. John Maddox is a board-certified anatomical and clinical pathologist, currently practicing as a pathologist at Riverside Regional Medical Center in Newport News, Virginia, where he has been for over twenty years. **Ex. 2**, Maddox Curriculum Vitae; **Ex. 3**, Maddox Depo., 3/31/17, *Taylor v. 3M Company*, at 20:20-24. As a result of Dr. Maddox's active practice and consulting activities, he is intimately familiar with the medical literature regarding asbestos related diseases. **Ex. 4**, Maddox Reliance List. In his report, Dr. Maddox expressed the opinion that Jolly has malignant mesothelioma that would cause his death, and that, "[b]ased on the exposure history, the patient's cumulative asbestos exposures caused this lethal malignant pleural mesothelioma." **Ex. 5**, Maddox Report, 4/13/16, at 3.

Dr. Maddox is not of the unqualified opinion that "one fiber" causes mesothelioma or that "every exposure" causes mesothelioma. Rather, he has testified that only exposures meeting certain well-defined criteria are considered a substantial factor in causing disease:

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Q. . . . . How have you been determining whether you have an opinion as to whether somebody has an exposure that's a substantial factor in the development of disease?

A. Well, I've got six points that I've mentioned in some depositions.

Q. Okay.

A. Let me mention those real quick, and I'll be glad to discuss any of them that you want.

Q. Sure.

A. Point number one, it should be a real exposure.

Point number two, it should be significantly above normal background environmental ambient air levels.

Number three, if you can see visible dusts, well, that's fine. That's plenty high enough. Otherwise, you should be able to relate it back to something, some measurement that's been done in the literature. By measurement I'm talking about either a measurement of the patient, a measurement of other workers doing similar work, or a work practice simulation that tells you that it's above background.

Q. Okay.

A. Point number four, it should be repetitive.

Point number five, a dose-response relationship applies between the dose and the risk of developing mesothelioma.

And point number six, it should be within a reasonable latency period.

Maddox Depo., 3/31/17, at 31:17-32:23. The six points that Dr. Maddox utilizes in evaluating whether a particular exposure is a substantial factor in causing disease are derived from the Helsinki Criteria. *Id.* at 91:5-14.

By "real exposure," Dr. Maddox means the following:

There should be some kind of documentation of it. There should be a description of it by the patient or by a coworker, or it was inevitable because there is product identification in the company ledger book that says, yeah, we were using this product.

Product identification as well as identification of use of the product. That's what I mean by real.

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Id. at 38:7-15. Not all exposures satisfy this criteria. For example, someone just walking through a boiler room where gaskets are present would not have a real asbestos exposure that would contribute to cause their disease. **Ex. 41**, Maddox Depo., 6/13/17, at 99:16-100:11, 110:15-17.

The federal asbestos MDL has found that, contrary to Defendant's mischaracterization, "Dr. Maddox does not assert that a single fiber can cause mesothelioma." **Ex. 6**, *Schumacker v. Amtico*, No. 5:10-1627, 2010 U.S. Dist. LEXIS 144831, at *4 (E.D. Pa. Nov. 2, 2010). Rather, his opinion is that "cumulative low-level exposures can result in mesothelioma." *Id.* at *5. The MDL has repeatedly ruled that Dr. Maddox's causation opinions satisfy the *Daubert* admissibility standard for expert testimony. *See id.* at *8; **Ex. 7**, *Rabovsky v. Air & Liquid Sys. Corp.*, No. 10-cv-03202, 2012 U.S. Dist. LEXIS 9169, *15-*18 (E.D. Penn. Jan. 25, 2012), *affirmed by* **Ex. 8**, 2012 U.S. Dist. LEXIS 34085, at *12-*14 (E.D. Penn. Mar. 13, 2012).

In reaching his opinion that Jolly has an asbestos-caused mesothelioma, Dr. Maddox relied on a summary of Jolly's exposure history provided by Plaintiffs' counsel. Maddox Depo., 6/13/17, at 7:10-21, 23:21-25. Based on his occupational history of exposure to asbestos, and the scientific literature regarding the causation of mesothelioma, Maddox has concluded that Jolly's mesothelioma was caused by his cumulative asbestos exposure. Maddox Report at 3. At trial, he intends to give specific causation opinions in response to hypothetical questions that incorporate the exposure facts for a given product or defendant. Maddox Depo., 6/13/17, at 46:21-47:3. In Maddox's opinion, Jolly's exposures to products that meet the six criteria set forth above are substantial factors in causing his disease. *Id.* at 95:13-98:16.

B. The qualifications and opinions of Dr. Frank.

Dr. Frank has been practicing medicine since 1972, when he received his medical degree from the Mount Sinai School of Medicine. **Ex. 52**, Frank Report, 3/11/16, at 1. During this time

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and thereafter, he worked with renowned asbestos researcher Dr. Irving Selikoff regarding health concerns associated with exposure to asbestos. *Id.* From 1973 to 1975, he served two years at the Lung Cancer Branch at the National Cancer Institute at the National Institutes of Health in Bethesda, Maryland as a Commissioned Officer in the United State Public Health Service. *Id.*

Dr. Frank is board-certified in both internal medicine occupational medicine, and has a Ph.D. in biomedical sciences. *Id.* He has been on the faculty of a number of medical schools since 1977, including Mt. Sinai, the University of Kentucky College of Medicine, and the University of Texas Health Science Center. From 2002 to 2014, he was the Chairman and Professor for the Department of Environmental and Occupational Health at the Drexel School of Public Health in Philadelphia, Pennsylvania. *Id.* He continues to be a professor in the Drexel School of Public Health, as well as a Professor of Medicine in the Division of Pulmonary Medicine at Drexel. His research focuses on occupational toxins, and he teaches courses in environmental and occupational health, occupational cancers, and occupational toxicology. *Id.*

Dr. Frank has consulted with various agencies of the U.S. government on occupational and environmental health issues, including the National Institute for Occupational Safety and Health (NIOSH), the Occupational Health and Safety Administration (OSHA), and the Environmental Protection Agency (EPA). *Id.* Dr. Frank also holds a prestigious position in the Collegium Ramazzini, a very selective international organization of health care professionals that take positions on public health issues globally. *Id.*

Dr. Frank has explained that he does "not believe that exposure to a *single* asbestos fiber or a *single breath* of air containing asbestos fibers has ever caused a mesothelioma or any other asbestos related cancer or any non malignant asbestos disease." **Ex. 53**, Excerpts of Frank Affidavit, 12/20/16, at ¶ 406. The scientific literature has established, however, that even relatively

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short exposures involve exposures to very high levels of asbestos fibers. It has been determined, for example, that "a single day of exposure at the *current* OSHA legal limit of exposure results in hundreds of thousands of fibers being inhaled in a single eight hour day." *Id.* Dr. Frank has explained that "an 'exposure' is never a single fiber . . . when someone breathes visible dust from an asbestos product, there may be millions or billions of asbestos fibers present." *Id.* at ¶ 407. In light of how much exposure occurs from breathing visible dust from asbestos products, it is Dr. Frank's opinion that every time someone is exposed in this manner in an occupational, para-occupational, environmental, or domestic setting, those exposures contribute to the risk of developing mesothelioma. *Id.*

Like Plaintiffs' other experts, Dr. Frank holds that opinion that a person's cumulative asbestos exposure causes their mesothelioma. *Id.* In developing specific causation opinions, he employs the methodology set forth in the Helsinki Criteria. *Id.* at ¶ 416. Specifically:

> In determining the relative contribution of any exposures to asbestos above background levels, it is important to consider a number of factors, including: the nature of exposure, the level of exposure and the duration of exposure, whether a product gives off respirable asbestos fibers, the level of exposure, whether a person was close to or far away from the source of fiber release, how frequently the exposure took place and how long the exposure lasted, whether engineering or other methods of dust control were in place, and whether respiratory protection was used.

Id. at ¶ 415.

Dr. Frank's causation testimony is routinely accepted in courts across the country. One of the most prominent recent examples was by the Supreme Court of Pennsylvania, in *Rost v. Ford Motor Co.*, 151 A.3d 1032, 1045-46 (Pa. 2016). **Ex. 54**. There, Ford Motor Company accused Dr. Frank of holding the opinion that "each and every breath" of asbestos is a substantial factor in causing mesothelioma. *Id.* at 1041. The court rejected this mischaracterization of his opinion, noting that "Dr. Frank never indicated that a single exposure was a substantial cause of Rost's

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mesothelioma. Dr. Frank explained to the jury, in some detail, about the dose response relationship between exposure to asbestos and the possibility of contracting mesothelioma." *Id.* at 1045. The court explained that instead of offering "'each and every breath' testimony, in this case Dr. Frank relied upon a generally accepted methodology, taking into consideration exposure history, individual susceptibility, biological plausibility, and relevant scientific evidence (including epidemiological studies)." *Id.* at 1046. Further, "his testimony regarding the frequency, regularity and proximity of Rost's exposures to asbestos while at Smith Motors provided a sufficient basis to create a jury question regarding the existence of a causal connection between his exposure to asbestos-containing Ford products and the subsequent development of mesothelioma." *Id.* at 1047.

C. The qualifications and opinions of Dr. Brody.

Dr. Brody is a cell biologist who specializes in lung pathology, or the biology of disease. **Ex. 9**, Brody Depo., *Voelker v. Alfa Laval, Inc.*, 5/8/15, at 7:12-18, 8:20-23, 22:9-12. At the beginning of his career, he worked and studied with Dr. Wagner, who in 1960 published the seminal study establishing that asbestos causes mesothelioma. *Id.* at 9:22-10:19. Dr. Brody studied how asbestos causes disease with Dr. Wagner, and that topic became the focus of Dr. Brody's research for the next several decades. *Id.* at 10:15-19. He studied how asbestos causes disease for 15 years at the National Institute for Environmental Health, a federally funded laboratory, and thereafter as a professor in the departments of pathology and environmental health sciences at Tulane University. *Id.* at 11:21-13:20. At Tulane, his work was supported almost entirely by grants from the National Institutes of Health. *Id.* at 13:7-14:23. He has published 153 peer-reviewed articles, and about 110 or 120 of them are on asbestos disease. *Id.* at 14:24-16:25.

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As Defendants are aware, the purpose of Dr. Brody's testimony is to educate the jury about the manner in which asbestos causes disease on a cellular level and which types of asbestos can cause the cellular changes that can lead to disease. For a number of years, Dr. Brody has testified in courtrooms throughout the United States about the mechanical pathways that lead to the causation of asbestos-related diseases. His testimony has not changed over the years. His testimony is routinely accepted and is rarely challenged. He is one of the most universally respected and well-known expert witnesses by either the plaintiff or defense bars. In fact, in the past John Crane has hired him to give the same testimony about the causation of asbestos disease that he gives for plaintiffs. Brody Depo., 5/8/15, at 21:10-24.

Contrary to Defendants' contention, Dr. Brody does not believe that all exposures to asbestos cause mesothelioma. When asked about that in another recent case, he explained that "if you have a level of asbestos exposure above background for, you know, weeks, months, whatever, it could be a brief exposure, that my opinion is enough to cause genetic errors and start a disease process." **Ex. 10**, Brody Depo., *Bodine v. 3M Company*, 11/13/14, at 29:7-12. Dr. Brody believes that every exposure above background is part of the cumulative exposure that causes the disease, but he does not believe that isolated or minimal exposures contribute. He explained that, "multiple days in a setting where we know that there are exposures above background, that would contribute. So the walk-through, I wouldn't say contributes and it never has." Brody Depo., 11/13/14, at 35:6-10. His reference to a "walk-through" was in response to a question about whether merely walking through an automotive garage would be a causative exposure, and Dr. Brody testified that it would not be:

> Q. So if the person who has the eight weeks of exposure 30 years later develops mesothelioma, and prior to that eight weeks of exposure walks through a garage where there is brake work going on or friction work going on, I think you would tell me that contributed to his mesothelioma?

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A. I think you're putting words in my mouth. I wouldn't say that walking through a passive brake job contributes. I would never say that.

My testimony is that you have to be exposed days, weeks, typically months or more for that to be a contributing factor. So walking through doesn't mean anything to me.

Brody Depo., 11/13/14, at 33:19-34:10 (emphasis added).

Like Dr. Maddox and Dr. Frank, Dr. Brody follows the Helsinki criteria in attributing a particular mesothelioma to asbestos exposure. Brody Depo., 5/8/15, at 68:24-69:11. The Helsinki criteria, established by an international group of experts in asbestos diseases in 1997, and reaffirmed last year, allows attribution based on a documented history of asbestos exposure. *Id.* at 69:12-24.

Dr. Brody testified that all asbestos diseases, including mesothelioma, are dose-response diseases, meaning the more a person is exposed, the more likely he or she is to develop the disease. Brody Depo., 5/8/15, at 71:20-72:1. A person's cumulative dose is that amount of asbestos fibers that accumulate in the lungs over the years, and "the more that accumulates, the more likely you are to get a cancer." *Id.* at 72:14-22. Scientists have not been able to establish a "safe" level of exposure to asbestos, and there is no known threshold below which mesothelioma will not occur. *Id.* at 73:21-74:4.

Dr. Brody's testimony has not only been accepted by this Court, but also by the federal asbestos MDL in the Eastern District of Pennsylvania. **Ex. 11**, *Larson v. Bondex Int'l*, No. 09-69123, 2010 U.S. Dist. LEXIS 123090 (E.D. Penn. Nov. 15, 2010). In rejecting a challenge to the reliability of Dr. Brody's general causation opinions, Judge Eduardo Robreno determined that Dr. Brody's testimony about the manner in which asbestos causes disease was supported by "'scientific investigations on humans and correlative animal and cell studies' confirming the relationship between asbestos fibers and four asbestos-related diseases discussed," including 146

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peer-reviewed publications and fifty book chapters authored or co-authored by Dr. Brody. *Id.* at *7 (citing Magistrate Judge Angell's Memorandum Opinion). Judge Robreno therefore held that, "[g]iven the purpose of Dr. Brody's testimony, to assist the jury in understanding the relationship between exposure to asbestos fibers and disease processes generally, and the breadth of peer-reviewed publications relied on," his testimony is reliable under Federal Rule of Evidence 702 and *Daubert v. Merrell Dow Pharmaceuticals Inc.*, 509 U.S. 579 (1993); *see also* Ex. 7, *Rabovsky v. Air & Liquid Sys. Corp.*, No. 10-cv-03202, 2012 WL 252919 (ED. Penn. Jan. 25, 2012), *affirmed by* Ex. 8, 2012 WL 876752 (E.D. Penn. Mar. 13, 2012). Trial courts in Oklahoma, Iowa, and Florida, have similarly ruled recently that Dr. Brody's general causation testimony is reliable and admissible. **Ex. 12**, *Galier v. Borg-Warner Morse Tec, Inc.*, at 160 (Okla. Dist. Ct. Jan. 6, 2015); **Ex. 13**, *Kinseth v. A.Y. McDonald Industries, Inc.*, at 11-13 (Iowa Dist. Ct. Feb. 26, 2014); **Ex. 14**, *Muniz v. Advanced Stores Company*, at second page (Fl. Cir. Ct. Nov. 26, 2013).

V. ARGUMENT AND AUTHORITIES

A. Plaintiffs' experts' causation conclusions are based on reliable science and are generally accepted in the scientific community.

1. There has never been a known "safe" level of exposure to asbestos and extremely low exposures to asbestos are known to cause mesothelioma.

Plaintiffs' experts' conclusions causation opinions are well-grounded in an abundance of medical and scientific evidence and are accepted as the consensus causation opinion by the vast majority of the medical and scientific communities, including government agencies and independent experts from numerous national and international institutions. The relevance and reliability of their opinions are such that they should be permitted to assist the jury in formulating their conclusions regarding the responsibility for Jolly's mesothelioma.

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It is undisputed that mesothelioma is a signal tumor for exposure to asbestos. The connection between the agent and the result is so strong that medical science has been unable to determine a threshold or minimum level of exposure to asbestos that has not been shown to cause mesothelioma. Accordingly, while a few cases of mesothelioma do develop in the presence of only environmental or ambient levels of asbestos, 90% or more are properly attributed to exposures to asbestos in excess of background levels. Given the undeniable link between exposure to asbestos and mesothelioma, universally accepted attribution criteria acknowledge that "a history of significant occupational, domestic, or environmental exposure to asbestos will suffice for attribution." **Ex. 15**, *Consensus Report: Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution*, SCAND J WORK ENVIRON HEALTH, 23:311-6 (1997) ("Consensus Report").

In January of 1997, a group of 19 experts from 8 different countries met in Helsinki, Finland "to discuss disorders of the lung and pleura in association with asbestos and to agree upon state-of-the-art criteria for their diagnosis and attribution with respect to asbestos." These experts included pathologists, radiologists, occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists, and clinical and laboratory scientists specializing in tissue fiber analysis. Collectively, the group "published over 1000 articles on asbestos and associated disorders." In rendering criteria on the attribution of mesothelioma, the Consensus Panel considered the following generally accepted concepts regarding mesothelioma:

- The great majority of mesotheliomas are due to asbestos exposure.
- Mesothelioma can occur in cases with low asbestos exposure. However, very low background environmental exposures carry only an extremely low risk.
- About 80% of mesothelioma patients have had some occupational exposure to asbestos, and therefore a careful occupational and environmental history should be taken.

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- An occupational history of brief or low-level exposure should be considered sufficient for mesothelioma to be designated as occupationally related.
- A minimum of 10 years from the first exposure is required to attribute the mesothelioma to asbestos exposure, though in most cases the latency interval is longer (e.g., on the order of 30 to 40 years).

Consensus Report at 313. Relying on these medical facts, the Consensus Panel concluded that "a history of significant occupational, domestic, or environmental exposure to asbestos will suffice for attribution." *Id.* These conclusions were reaffirmed by these same international experts as recently as last year. **Ex. 16**, *Consensus Report: Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution 2014*, Scand J Work Environ Health, 41(1):5-15 (2014) ("Consensus Report 2014").

Significantly, the Helsinki Criteria does not require a quantitative estimate of a patient's asbestos "dose" exceeding some undefined level in order to attribute mesothelioma to a given asbestos exposure. In order for a doctor to conclude that a mesothelioma is asbestos related, there needs to be evidence (such as a patient interview or lay testimony) that the mesothelioma patient breathed asbestos fibers in an occupational, paraoccupational, domestic, or similar setting.

The opinion of Plaintiffs' experts that exposures meeting certain criteria contribute to the aggregate dose that ultimately caused Jolly's mesothelioma is premised on the Consensus Report and built on a foundation of well-established medical facts, including that there is no threshold level of exposure to asbestos above the miniscule level of asbestos in the ambient air that has been clearly shown not to cause mesothelioma, that mesothelioma is a dose-response disease, and that there is an inverse relationship between dose and latency – the higher the dose, the shorter the latency period, conversely, the lower the dose, the longer the latency period. As succinctly stated by the U.S. Environmental Protection Agency ("EPA"): "Because asbestos fibers remain in the

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body, each exposure increases the likelihood of developing an asbestos-related disease." **Ex. 17**, *Guide for Ship Scrappers: Tips for Regulatory Compliance,* p. 2-6 (Summer 2000).

In 2014, the Occupation Safety and Health Commission reiterated the same facts of medical causation: "Asbestos exposures as short as in duration as a few days have caused mesothelioma in humans. Every occupational exposure to asbestos can cause injury of disease; every occupational exposure to asbestos contributes to the risk of getting an asbestos-related disease." **Ex. 18,** OSHA Website, Safety and Health Topics: Asbestos, https://www.osha.gov/SLTC/asbestos, last visited 1/8/14. Further, "[h]eavy exposures tend to occur . . . in ship repair, particularly during the removal of asbestos materials due to renovation, repairs, or demolition." *Id.*

Numerous federal and international agencies have concluded that there is no known safe level of asbestos exposure. These include the World Health Organization ("WHO"), the International Agency for Research on Cancer ("IARC"), the EPA, The National Cancer Institute ("NCI"), the National Institutes of Occupational Safety and Health ("NIOSH"), and the Consumer Products Safety Commission ("CPSC"). For example, IARC (part of the World Health Organization) has stated that "[a]t present, it is not possible to assess whether there is a level of exposure in humans below which an increased risk of cancer would not occur." **Ex. 19**, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 14, Asbestos, § 5.2. The Agency has also noted that mesotheliomas "occur in individuals living in the neighborhood of asbestos factories and crocidolite mines and in household contacts of asbestos workers." *Id.*

The National Cancer Institute notes that "some individuals with only ***brief*** exposures have developed mesothelioma." **Ex. 20**, National Cancer Institute Mesothelioma Fact Sheet, at 2. "There is some evidence that family members and others living with asbestos workers have an

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increased risk of developing mesothelioma." *Id.*; *see also* **Ex. 21**, National Cancer Institute Asbestos Exposure Fact Sheet, at 2 ("investigators have found asbestos-related diseases in individuals with only ***brief*** exposures"). The Institute also notes that "[c]ases of mesothelioma have also been seen in individuals without occupational exposure, but who live close to asbestos mines or have been exposed to fibers carried home by family members working with asbestos." *Id.* at 3.

The National Institute for Occupational Safety and Health ("NIOSH"), part of the Center for Disease Control within the U.S. Department of Health and Human Services, has long held the view that there is no safe threshold for exposure to asbestos. As early as 1976, NIOSH had noted: "**Excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a 'safe' level of asbestos exposure.**" **Ex. 22**, NIOSH Revised Recommended Asbestos Standard, 1976, pg. 92 (emphasis added). Further, NIOSH believed that "the [exposure] standard should be set at the lowest level detectable by available analytical techniques, an approach consistent with NIOSH's most recent recommendations for other carcinogens (ie, arsenic and vinyl chloride)." *Id.* at 93.

The American Cancer Society similarly states that "there is no way to measure exactly the minimum amount of asbestos exposure that can lead to mesothelioma." **Ex. 23**, American Cancer Society: Asbestos, at 3. The World Health Organization states that "[n]o safe [exposure] level can be proposed for asbestos because a threshold is not known to exist. Exposure therefore should be kept as low as possible." **Ex. 24**, WHO Air Quality Guidelines, Chapter 6.2 Asbestos, at 11.

The absence of a known threshold of asbestos exposure for mesothelioma risk led the U.S. Consumer Product Safety Commission to ban the use of asbestos in drywall taping and spackling

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compounds in 1977. "The Commission noted that in the scientific literature there is general agreement that there is no known threshold level below which exposure to respirable free-form asbestos would be considered safe." **Ex. 25**, CPSC Ban of Consumer Patching Compounds Containing Respirable Free-Form Asbestos, 16 CFR Ch. 11 § 1304.5 (Dec. 15, 1997 ed.).

The findings of these regulatory agencies is supported by voluminous scientific literature establishing that there is no threshold below which there is no risk from exposure to asbestos. *See, e.g.*, **Ex. 26**, Landrigan et al., *The Hazards of Chrysotile Asbestos: A Critical Review*, IND HEALTH 37:271-280, 275 (1999); **Ex. 27**, Hillerdal, *Mesothelioma: Cases Associated with Non-Occupational and Low Dose Exposures*, OCCUP ENVIRON MED 56:505-513, 510 (1999).

Dr. Laura Welch and 51 other scientists, researchers and medical professionals representing "hundreds of years of experience researching, diagnosing, and treating asbestos-related disease in workers and their families," published a paper outlining the consensus of the mainstream scientific community regarding the causation of asbestos-related disease. **Ex. 28**, Welch, *Asbestos Exposure Causes Mesothelioma, But Not* ***This*** *Asbestos Exposure: An Amicus Brief to the Michigan Supreme Court*, INT J OCCUP ENVIRON HEALTH 13:318-327 (2007). These fifty-two independent experts explained that, "[t]he mainstream scientific community has long recognized and continues to recognize today that there is no 'safe' level of exposure to asbestos." *Id.* at 319. The scientists concluded that, "the consensus of the scientific community is that any occupational or para-occupational exposure to asbestos - even 'brief or low-level exposures' - must be considered causal in an individual with a mesothelioma." *Id.* at 321 (emphasis added).

As a group of 58 specialists in asbestos-related disease recently wrote, "[i]t is indeed an irrefutable scientific fact that each exposure does actually contribute to a person's total dose and to that person's risk or probability of developing mesothelioma and other cancers. This is scientific

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fact." **Ex. 29**, *Joyce Rost v. Ford Motor Company*, No. 56 EAP 2014 (Pa. April 7, 2015) (*Amicus Cureiae* signed by 58 physicians and scientists in Support of Appellees).

"Low level" and "short-term" asbestos exposures have been repeatedly shown to cause mesothelioma in the peer-reviewed scientific literature. For example, in large epidemiological study conducted in France, the authors found that, "[a] significant excess of mesothelioma was observed for levels of cumulative exposure that were probably far below the limits adopted in many industrial countries during the 1980s." **Ex. 30**, Iwatsubo, et al, *Pleural Mesothelioma: Dose-Response Relation at Low Levels of Asbestos Exposure in a French Population-based Case-Control Study*, AM J EPID 148(2):122-142 (1998). The authors documented that cumulative intermittent exposures as low as 0.5 fiber years resulted in a four-fold increase in the risk of mesothelioma with a 95% CI of 1.7-9.7. *See* id. at 7. The Iwatsubo study found that the risk of developing mesothelioma increased as the intensity of exposure increased. *Id.* For continuous exposure resulting in a cumulative dose of between 0.5 and 0.99 f/cc, the authors recorded a relative risk of 4.6 (95% CI 1.4-15.4). *Id.*

The findings of Iwatsubo were replicated by Dr. Rodelsperger and his colleagues, who observed a "distinct dose-response relationship even at levels of cumulative exposure below 1 fiber year." **Ex. 31**, Rodelsperger et al., *Asbestos and Man-Made Vitreous Fibers as Risk Factors for Diffuse Malignant Mesothelioma: Results From a German Hospital-Based Case-Control Study*, AM J INDUS MED 39: 262-275, 262 (2001). Specifically, Rodelsperger calculated an odds ratio of 7.9 (CI 2.1-30.0) for cumulative exposures of greater than 0.00 up to 0.15 f/cc-years. Rodelsperger, *supra*, at 269, Table VII.

Another French population-based case-control study conducted from 1998 to 2002, similarly found a significant dose-response relationship at relatively low cumulative dose,

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reporting an odds ratio of 2.8 (95% CI 1.7-4.7) for exposures between 0 and 0.07 fibers/ml-years. **Ex. 32**, Rolland, *Risk of pleural mesothelioma: A French population-based case-control study (1998-2002)* (Oct. 20, 2006).

A study by LaCourt, et al. recently corroborated these prior studies, finding a four-fold risk of pleural mesothelioma (CI 1.9 to 8.3) at exposures between >0 - 0.1 f/mL-year, and an 8.3 odds ratio at between >.1 - 1 f/mL years (CI 3.8 to 17.7). **Ex. 33**, A. Lacourt et al., *Occupational and non-occupational attributable risk of asbestos exposure for malignant pleural mesothelioma*, Thorax, published online, at Table 4 (Feb. 7, 2014). The authors noted that chrysotile was the most commonly used asbestos fiber type in the products used by those in the cohort, and that the sources of asbestos exposure included workers in the "shipbuilding and repair industry." *Id.* at 4.

Many of these studies are discussed and summarized by Dr. Markowitz in his recent article, *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin Respir Crit Care Med 36:334-346, 336, Table 1 (2015), **Ex. 34**. Markowitz notes that these studies "show a sharp rise in mesothelioma risk with increasing asbestos exposure. This pattern signifies that **at any level of occupational asbestos exposure, adding additional occupational exposure to asbestos increases the likelihood of developing malignant mesothelioma**." *Id.* (emphasis added). Further, Markowitz identifies three consequences of the observed exposure-response pattern documented in the epidemiological literature:

> First, for the individual who has had some occupational asbestos exposure, it is essential to avoid additional exposure to asbestos, because it adds, often dramatically, to their risk of developing malignant mesothelioma. Second, in terms of attribution of malignant mesothelioma to prior asbestos exposure, it is clear that each increment in occupational asbestos exposure contributes significantly to the development of mesothelioma in the group. For example, as shown in Table 1, in the two French and the German case–control studies [Iwatsubo, Rodelsperger, and Lacourt], adding up to 1 fiber/mL-year of exposure to the lowest category of asbestos exposure elevates the exposed persons to the next higher category of asbestos exposure and doubles or triples the risk of malignant mesothelioma. Third,

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> finding excess cancer risk at low levels of occupational exposure to asbestos supports the notion that there is no safe level of exposure to asbestos.

Id. at 336-37 (footnotes omitted).

To put these studies in perspective, by the time asbestos-contaminated dust is visible to the naked eye, there are 10-40 million particles per cubic foot of air (mppcf). **Ex. 35**, 3/15/17, Garza Report, at 12. Therefore, "at a point at which a person would have visibly seen asbestos-containing dust arising from activities with asbestos-containing products (8-40 mppcf), one would have potentially been exposed to 24 f/cc to 120 f/cc." *Id.* at 13.

Numerous studies have also found that relatively low exposures chrysotile asbestos fibers cause mesothelioma. In 1996, Drs. Smith and Wright concluded that "[c]hrysotile asbestos is a potent cause of pleural mesothelioma." **Ex. 36**, Smith and Wright, *Chrysotile Asbestos Is the Main Cause of Pleural Mesothelioma*, AM J IND MED 30:252-266, 255 (1996). The authors found that exposure to chrysotile asbestos "even at the relatively low levels expected in household exposures, can cause malignant mesothelioma." *Id.*

In 2011, a survey of the literature found at least thirty "studies and cases from all over the world, where the overwhelming evidence is that chrysotile asbestos was the primary or only asbestos exposure." **Ex. 37**, Kanarek, *Mesothelioma from Chrysotile Asbestos: Update*, ANN EPIDEMIOL 21:688-697, 695 (2011). The authors concluded that "[t]here are many well-documented cases of mesothelioma from chrysotile asbestos in case-reports and epidemiology studies from all over the world." *Id.* at 695.

A study of Lithuanians exposed to uncontaminated chrysotile asbestos from Russia found that three out of four patients with mesothelioma had cumulative exposures less than 0.01 fiber years. **Ex. 38**, R.P. Everatt et al., *Occupational Asbestos Exposure Amoung Respiratory Cancer Patients in Lithuania*, Am. J. Indus. Med., Supplement 50:455-463 (2007).

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Another recent study showing excess mesothelioma risk from chrysotile only exposure is an Egyptian study, M.T. Madkour, *Environmental exposure to asbestos and the exposure-response relationship with mesothelioma*, E. Mediterranean Health J. 15(1):25-38 (2009), **Ex. 39**. The authors found that those with environmental or occupational exposure to chrysotile asbestos had a 26 times greater risk of developing mesothelioma than those who were not exposed. *Id.* at 32. Mesothelioma was even more prevalent in the environmentally exposed group, with the overall mean asbestos fiber concentration in the air outside the asbestos plant measured at 0.38 fibers per milliliter (mL). *Id.* at 30, 33. Half of the mesotheliomas (45 out of 90) were among those exposed to less than 7 fiber years. *Id.* at 36, Table 10.

The importance of each significant exposure is highlighted by the inverse relationship between the amount of asbestos inhaled and the time period in which a mesothelioma will develop if it is going to develop. The lower the dose, the longer the latency period. *See e.g.*, **Ex. 40**, Bianchi, *Latency periods in asbestos-related mesothelioma of the pleura*, EUR J CANCER PREV 6:162-166 (1997).

In short, abundant scientific literature provides a reliable scientific basis for the causation opinions of Plaintiffs' experts. It demonstrates a strong foundation for their opinions that Jolly's cumulative asbestos exposure caused his mesothelioma, and for Dr. Maddox's and Dr. Frank's specific causation opinions that his repetitive exposures to visible dust from asbestos products would have been substantial factors in the development of his disease.

2. The experts' conclusions are widely accepted in the scientific community.

Recently, Dr. Laura Welch and 51 other scientists, researchers and medical professionals representing "hundreds of years of experience researching, diagnosing, and treating asbestos-related disease in workers and their families" as well publishing extensively in the field of asbestos

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and conducting dozens of epidemiologic and other studies into the issue of asbestos and disease,[1] published a paper outlining the consensus of the mainstream scientific community regarding asbestos and its ability to cause disease. *See* Welch, *supra.*

Plaintiffs' experts' causation opinions are entirely consistent with the consensus opinion Dr. Welch and her colleagues offer on asbestos and mesothelioma causation. Under the heading, "The Scientific Community is in Consensus that Even Brief and Low-level Exposure to Asbestos Can Cause Mesothelioma," these fifty-two independent experts explained:

> The mainstream scientific community has long recognized and continues to recognize today that there is no "safe" level of exposure to asbestos. As noted by NIOSH:
>
> > Excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold of for a "safe" level of asbestos exposure.
>
> Attempts to postulate thresholds for exposure have been dismissed as "logical nonsense."

Id. (emphasis added). Dr. Welch and her colleagues summarized some of the key studies that prove this view. *See id.* One of those, the 1998 Iwatsubo article noted above, showed "substantial excess mortality" at exposure levels below current regulatory levels. *See id.* Another study found that "all levels of probability and intensity [of exposure to asbestos] had an increased significant risk, except subjects with low probability of exposure." *Id.* at 319. The scientists accordingly explained that, "**the consensus of the scientific community is that any occupational or para-**

[1] The list of co-signers reads like a "who's who" of doctors, scientists, scholars and experts in the field of asbestos-related diseases. These fifty-two experts cover a broad range of specialties and institutions, spanning the globe from the United States to Sweden to Australia to Brazil to Scotland to Japan to Korea to Italy and on and on. It is compelling that against the crushing weight of this global community of authorities the defendant's best offering is a smattering of aberrant court opinions and one law review article written by two toxic tort defense attorneys.

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occupational exposure to asbestos - even "brief or low-level exposures" - must be considered causal in an individual with a mesothelioma." *Id.* at 321 (emphasis added).

Dr. Welch concludes this consensus paper powerfully:

> As scientists who have devoted substantial portions of our professional lives working to research, prevent, and treat asbestos-related diseases, we reject these attempts to fabricate uncertainly where none exists. Instead, we request that these courts attend to the work of thousands of experts from around the world who have concluded that asbestos, in any form, and through any occupational exposure, can and does cause disease.

Id. at 323.

B. Plaintiff's experts' causation conclusions have been widely accepted by courts across the country.

As noted above, the federal asbestos MDL has repeatedly ruled that the opinion that every exposure to asbestos above background levels contributes to cause mesothelioma satisfies *Daubert*. *See* Ex. 7, *Rabovsky v. Air & Liquid Sys. Corp.*, No. 10-cv-03202, 2012 U.S. Dist. LEXIS 9169 (E.D. Penn. Jan. 25, 2012), *affirmed by* Ex. 8, 2012 U.S. Dist. LEXIS 34085 (E.D. Penn. Mar. 13, 2012); Ex. 6, *Schumacker v. Amtico*, No. 5:10-1627, 2010 U.S. Dist. LEXIS 144831 (E.D. Pa. Nov. 2, 2010); Ex. 11, *Larson v. Bondex Int'l*, No. 09-69123, 2010 U.S. Dist. LEXIS 123090 (E.D. Penn. Nov. 15, 2010).

In *Schumacker*, the court found that Dr. Maddox had used a reliable methodology in reaching this opinion. He relied on a variety of peer-reviewed studies, including the Consensus Report, and considered the amount of fibers released from working with the products at issue. *Id.* at *5-*6. The court noted that there is a significant body of published literature regarding what level of asbestos exposure can cause mesothelioma and that there is a legitimate debate within the scientific community regarding what that threshold level is (and indeed, whether a numeric threshold is even ascertainable). *Id.* at *7. Although the plaintiff's expert took one position in that

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debate and the defendant's expert took the opposite position, that does not make the plaintiff's expert's opinion unreliable or inadmissible. *Id.* at *7-*8. Rather, the defendant's criticisms of the opinions were simply "fodder for cross-examination, and do not preclude the admissibility of [the] opinion." *Id.* at *7; *see also Rabovksky*, 2012 U.S. Dist. LEXIS 34085, at *12-*14; *Rabovsky*, 2012 U.S. Dist. LEXIS 9169, at *15-*18.

Similarly, the Middle District of Florida recently upheld Dr. Maddox's causation opinions as reliable and admissible under *Daubert*. **Ex. 42,** *Dugas v. 3M Co.*, No. 3:14-CV-1096-J-39JBT, 2016 WL 7246096, at *1 (M.D. Fla. Jan. 11, 2016). The court found that, consistent with the scientific literature and the requirements of the Eleventh Circuit, "Dr. Maddox considered the nature of Mr. Dugas's asbestos exposure, including its duration, concentration, distance from the source of fiber release, frequency, and ventilation." *Id.* at *3. The court concluded that Dr. Maddox reached his opinions "through scientifically sound principles as supported by numerous studies, many of which are peer-reviewed and of the type an expert in the field would review in formulating an opinion," and that he reliably applied the science to the facts of the case to reach his opinion that the defendants' asbestos products were causes of the plaintiff's mesothelioma. *Id.*

Just last month, the Eleventh Circuit upheld the same opinions expressed by Dr. Maddox in this case. In *Bobo v. Tennessee Valley Auth.*, 855 F.3d 1294 (11th Cir. Apr. 26, 2017), the court noted that expert testimony that "there is no evidence that there is a threshold level of exposure below which there is zero risk of mesothelioma," and that "all 'significant' exposures to asbestos 'contribute to cause mesothelioma,'" is not the same thing as saying that each and every exposure is causative. *Id.* The expert's causation opinion was admissible because it was based on the exposure facts in the case and was supported by scientific literature, including the Helsinki Criteria. *Id.*

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Contrary to Defendants' argument here, Judge Robreno of the federal asbestos MDL has found that a "cumulative exposure" opinion is not the same as the "each and every exposure" opinion. The court agreed with the plaintiff that the "cumulative exposure" opinion is different "in substance and by definition" from the "any exposure" opinion. **Ex. 43**, *Mortimer v. A.O. Smith Corp.*, 2015 WL 12533103, at *8 (E.D. Pa. Oct. 23, 2015). The court therefore rejected the defendant's argument that the experts' opinions were inadmissible on grounds that they are the same as the "any exposure" opinion. *Id.*

This case law generally reflects the general acceptance of the medical fact that asbestos diseases are cumulative dose diseases with documented exposures above background levels constituting a substantial contributing cause. As far back as 1973, the Fifth Circuit Court of Appeals observed that it has been "established that the effect of exposure to asbestos dust is cumulative, that is, each exposure may result in an additional and separate injury." *Borel*, 493 F.2d at 1094. Many cases from many other jurisdictions are in accord, and have admitted expert testimony to this effect. *See* **Ex. 44**, *Osterhout v. Crane Co.*, 2016 U.S. Dist. LEXIS 35724, at *57-70 (N.D.N.Y. Mar. 21, 2016); **Ex. 45**, *Davis v. Honeywell Int'l Inc.*, 245 Cal. App. 4th 477, 487, 199 Cal. Rptr. 3d 583, 591 (2016), *review denied* (May 25, 2016); **Ex. 46**, *Walashek v. Air Liquid Sys. Corp.*, No. 14CV1567 BTM(BGS), 2016 WL 614030, at *7 (S.D. Cal. Feb. 16, 2016); **Ex. 47**, *Relyea v. Borg Warner Corp.*, 2015 U.S. Dist. LEXIS 126895 (S.D.N.Y. Sept. 22, 2015); **Ex. 48**, *Kochera v. GE*, 2015 U.S. Dist. LEXIS 125726, at *11 (S.D. Ill. Sept. 21, 2015); **Ex. 49**, *Utech v. Asbestos Corp. Ltd.*, No. CV-14-4977-R (C.D. Cal. July 20, 2015); **Ex. 1**, *Garvin v. Agco Corp.*, No. 2012-CP-40-6675 (S.C. Ct. Comm. Pleas Nov. 14, 2014), at 21-25; **Ex. 50**, *Quirin v. Lorillard Tobacco Co.*, 2014 U.S. Dist. LEXIS 26218, at *9-*10 (N.D. Ill. Feb. 28, 2014); **Ex. 51**, *Lipson v. ON Marine Servs. Co.*, LLC, 2013 U.S. Dist. LEXIS 175317, at *10 (W.D. Wash. Dec. 13, 2013);

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John Crane, Inc., v. Linkus, 988 A.2d 511, 523-24 (Md. App. 2010); *Jones v. John Crane, Inc.*, 35 Cal. Rptr. 3d 144, 151 (Cal. App. 2005); *AC & S, Inc. v. Abate*, 710 A.2d 944, 988 (Md. App. 1998), *abrogated on other grounds by John Crane, Inc. v. Scribner*, 800 A.2d 727 (2002); *John Crane, Inc. v. Wommack,* 489 S.E.2d 527, 541 (Ga. Ct. App. 1997); *Mavroudis v. Pittsburgh-Corning Corp.*, 935 P.2d 684, 689 (Wash. 1997); *Kurak v. A.P. Green Refractories Co.*, 689 A.2d 757, 761 (N.J. Super. App. Div. 1997).

C. The cases relied on by Defendants do not support the exclusion of Plaintiffs' expert causation testimony.

Despite the fact that Plaintiffs' experts' opinions are consistent with the consensus view on the causation of asbestos-related disease, and the type of causation opinions they have expressed have repeatedly been recognized as reliable and admissible expert testimony, Defendants have offered critiques based on a few cases that have excluded or criticized similar causation testimony.

Defendants cite the opinions in *Butler v. Union Carbide Corp.* and *Yates v. Ford Motor Company* which faulted the plaintiff's expert's testimony because the court thought that there had not been adequate testing to establish a threshold exposure level for mesothelioma. *Yates* and other cases cited by Defendants misinterpret the experts' reliance on the scientific literature showing that there is no known safe level of exposure to asbestos. Oddly, this is taken as an absence of data to support the experts' opinions. *See Smith v. Ford Motor Co.*, No. 2:08-cv-630, 2013 U.S. Dist. LEXIS 7861, at *7 (D. Utah Jan. 18, 2013).

The fallacy with this reasoning is identified in a recent decision by the Seventh Circuit, *Schultz v. Akzo Nobel Paints, LLC*, 721 F.3d 426 (7th Cir. 2013). In this benzene exposure case, the defendant attacked the plaintiff's expert's opinion because he stated that there was no known threshold below which benzene exposure does not cause acute myeloid leukemia ("AML"), but

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had had also identified the exposure level at which studies had found an elevated risk of AML. *Id.* at 432. The court explained that there is no inconsistency in these two positions: "The first says, in essence, that scientific studies confirm the danger of exposure to more than 10 ppm-years of benzene. The second says that no one is sure whether 10 ppm-years is the floor for risk, or 5 ppm-years, or 1 ppm-year, or nothing." *Id.* The Seventh Circuit disagreed with the trial court's conclusion that the expert had found the amount of exposure to be "irrelevant," given that the expert's "unambiguous conclusion that [the plaintiff] had been exposed to a level of benzene that has been shown in studies to be a 'very toxic and dangerous level.'" *Id.* The record in this case is equivalent to that found sufficient to support the expert's opinion in *Schultz* because, as noted above, Plaintiffs' experts rely on Jolly's history of exposure and epidemiological studies finding that low cumulative occupational asbestos exposures increase the risk of mesothelioma several-fold.

Defendants also contend that the "each and every exposure" theory does not satisfy the substantial factor causation test. Besides the fact that Plaintiffs' experts have not offered this opinion, this is another argument that suffers from flawed and simplistic reasoning. The asbestos MDL rejected this argument in the *Rabovsky* case, finding that the admissibility of the expert's opinion is not really related to the sufficiency of the evidence question. 2012 U.S. Dist. LEXIS 34085, at *12-*13. This is because an asbestos plaintiff can rely on multiple types of evidence to meet her burden of proving causation, with her expert's opinion being only one part of her case. As noted above, this Court's gatekeeping function does not involve examining the correctness of the expert's conclusions, but instead focuses on the reliability of the expert's methodology. In fact, expert evidence "does not warrant exclusion simply because it fails to establish the causal link to a specified degree of probability." *Ambrosini v. LaBarraque*, 101 F.3d 129, 135 (D.C. Cir.

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1996). Rather, "[t]he dispositive question is whether the testimony will assist the trier of fact to understand the evidence or to determine a fact in issue, not whether the testimony satisfies the plaintiff's burden on the ultimate issue at trial." *Id.*

Finally, Defendants contends that Plaintiffs' expert testimony will not be helpful to the jury because it is insufficient to meet the substantial factor causation standard. Defendants ignore that asbestos plaintiffs can rely on multiple types of evidence to meet their burden of proving causation, with their expert's opinion being only one part of their case. The Pennsylvania Supreme Court recently upheld the admissibility of the plaintiff's expert causation opinions, and found them to be entirely compatible with the substantial factor causation standard:

> We must agree with the Rosts that Ford has confused or conflated the "irrefutable scientific fact" that every exposure cumulatively contributes to the total dose (which in turn increases the likelihood of disease), with the legal question under Pennsylvania law as to whether particular exposures to asbestos are "substantial factors" in causing the disease. It was certainly not this Court's intention, in either Gregg or Betz, to preclude expert witnesses from informing juries about certain fundamental scientific facts necessary to a clear understanding of the causation process for mesothelioma, even if those facts do not themselves establish legal (substantial factor) causation.

Rost v. Ford Motor Co., 151 A.3d 1032, 1045-46 (Pa. 2016).

In evaluating the relevance of Plaintiffs' experts' causation testimony, this evidence must be considered in the context of the record as a whole. Their testimony undoubtedly has the tendency to make the causation facts more probable. They offer expertise regarding the manner in which asbestos exposure causes disease and the levels of exposure found sufficient in the literature to cause mesothelioma. At trial, they intend to evaluate Jolly's exposures to Defendants' products under defined criteria derived from the Consensus Report. Combined with the evidence of Jolly's frequent, regular, and proximate exposure to asbestos from Defendants' products, the causation opinions of Dr. Maddox, Dr. Frank, and Dr. Brody will certainly aid the jury.

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Contrary to Defendants' contentions, the opinions of Dr. Maddox, Dr. Frank, and Dr. Brody have a reliable foundation in the factual and scientific evidence. As the Court found in *Garvin*, these opinions are reliable, helpful to the trier of fact, and would support a jury finding that exposure to Defendants' asbestos products caused the plaintiff's mesothelioma. Plaintiffs therefore respectfully request that the Court deny Defendants' motions to exclude the causation testimony of Plaintiffs' experts.

Respectfully submitted,

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July 13, 2017
Dallas, Texas.

CERTIFICATE OF SERVICE

I hereby certify that a true and correct copy of the above and foregoing instrument was e-filed with the Spartanburg County court's e-filing system, and thereby served electronically on all counsel of record, this 13th day of July, 2017.

/s/ Jonathan M. Holder
Jonathan M. Holder
Attorney for Plaintiffs

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EXHIBIT 15

Consensus report

Scand J Work Environ Health 1997;23:311—6

Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution

The International Expert Meeting on Asbestos, Asbestosis, and Cancer was convened in Helsinki on 20—22 January 1997 to discuss disorders of the lung and pleura in association with asbestos and to agree upon state-of-the-art criteria for their diagnosis and attribution with respect to asbestos. The group decided to name this document *The Helsinki Criteria*.

The requirement for diagnostic criteria was perceived in part because of new developments in diagnostic methods, with better identification of asbestos-related disorders. Such developments enhance awareness of health hazards imposed by asbestos, lead to practical prevention and appropriate compensation, and also provide an opportunity to carry out international comparisons. They also provide possible models for the risk assessment of other mineral dusts.

The meeting was attended by 19 participants from 8 countries not producing asbestos. The chairmen were Professor Douglas W Henderson (Flinders Medical Centre, Australia) and Professor Jorma Rantanen (Finnish Institute of Occupational Health, Finland). The group was a multidisciplinary gathering of pathologists, radiologists, occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists, and clinical and laboratory scientists specializing in tissue fiber analysis. Collectively, the group has published over 1000 articles on asbestos and associated disorders. This document is based on a more comprehensive report providing scientific evidence for the conclusions and recommendations (*People and Work Research Reports*, no 14, Finnish Institute of Occupational Health, Helsinki, 1997).

The meeting was scientifically supported by leading institutions in the field of asbestos research, and it was funded by the Ministry of Social Affairs and Health and the Finnish Work Environment Fund.

General considerations

Occupational exposures to asbestos dust have been widespread in all industrial countries and continue as a consequence of "in-place" materials. In detailed interviews about 20% to 40% of adult men report some past occupations and jobs that may have entailed asbestos exposure at work. In Western Europe, North America, Japan, and Australia the use of asbestos peaked in the 1970s, and currently about 10 000 mesotheliomas and 20 000 asbestos-induced lung cancers are estimated to occur annually in the population of approximately 800 million people.

In general, reliable work histories provide the most practical and useful measure of occupational asbestos exposure. Using structured questionnaires and checklists, trained interviewers can identify persons who have a work history compatible with significant asbestos exposure. Dust measurements can be used in the estimation of past fiber levels at typical workplaces and in the use of asbestos-containing materials. A cumulative fiber dose, as expressed in fiber-years per cubic centimeter, is an important parameter of asbestos exposure.

The clinical diagnosis of asbestos-related diseases is based on a detailed interview of the patient and occupational data on asbestos exposure and appropriate latency, signs and symptoms, radiological and lung physiology findings, and selected cytological, histological and other laboratory studies. Histopathological confirmation is required for suspected asbestos-related malignancies and for the resolution of differential diagnoses. A multidisciplinary approach is suggested for the evaluation of problem cases.

The chest radiograph is the basic tool for identifying asbestos-related diseases such as asbestosis, pleural abnormalities, lung cancer, and mesothelioma. The limitation of the chest radiograph in the detection of asbestosis and asbestos-associated pleural abnormalities is widely recognized. Computed tomography (CT) and high resolution computed tomography (HRCT) can facilitate the detection of asbestosis and asbestos-related pleural abnormalities, as well as asbestos-related malignancies; they are not recommended as a screening tool but may be invaluable for individual clinical evaluation and research purposes. Examples are the detection of pleural abnormalities in suspected cases of asbestosis and the detection of parenchymal disease obscured on the chest film and also use as an aid to differential diagnosis. As new imaging techniques such as digital radiography are evolving, standard images and interpretations must be developed. The place of other imaging techniques (ultrasound, magnetic resonance imaging, gallium scanning, ventilation-perfusion studies, positron-emission tomography) has yet to be established, and they are not currently recommended for the clinical diagnosis of asbestos-related disorders.

Analysis of lung tissue for asbestos fibers and asbestos bodies can provide data to supplement the occupa-

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ional history. For clinical purposes, the following guidelines are recommended to identify persons with a high probability of exposure to asbestos dust at work: over 0.1 million amphibole fibers (>5 µm) per gram of dry lung tissue *or* over 1 million amphibole fibers (>1 µm) per gram of dry lung tissue as measured by electron microscopy in a qualified laboratory *or* over 1000 asbestos bodies per gram of dry tissue (100 asbestos bodies per gram of wet tissue) *or* over 1 asbestos body per milliliter of bronchoalveolar lavage fluid, as measured by light microscopy in a qualified laboratory.

Each laboratory should establish its own reference values. The median values for occupationally exposed populations should be substantially above the reference values. Efforts to standardize analytical methods for fiber burden analyses by different laboratories are recommended.

Asbestosis

Asbestosis is defined as diffuse interstitial fibrosis of the lung as a consequence of exposure to asbestos dust. Neither the clinical features nor the architectual tissue abnormalities sufficiently differ from those of other causes of interstitial fibrosis to allow confident diagnosis without a history of significant exposure to asbestos dust in the past or the detection of asbestos fibers or bodies in the lung tissue greatly in excess of that commonly seen in the general population. Symptoms of asbestosis include dyspnea and cough. Common findings are inspiratory basilar crackles and, less commonly, clubbing of the fingers. Functional disturbances can include gas exchange abnormalities, a restrictive pattern, and obstructive features due to small airway disease.

Asbestosis is generally associated with relatively high exposure levels with radiological signs of parenchymal fibrosis. However, it is possible that mild fibrosis may occur at lower exposure levels, and the radiological criteria need not always be fulfilled in cases of histologically detectable parenchymal fibrosis. The recognition of asbestosis by chest radiography is best guided by standardized methods such as the classification of the International Labour Organisation (ILO) and its modifications. Standard films must always be used. For research and screening purposes, radiological findings of small opacities, grade 1/0, are usually regarded as an early stage of asbestosis. Inspiratory basilar rales, restrictive impairment, small airway obstruction, and gas exchange disturbances in pulmonary function are considered valuable information for clinical diagnosis, for occupational health practice, and for attribution purposes. HRCT can confirm radiological findings of asbestosis and show early changes not seen on chest X rays, but should be performed only in selected cases.

Smoking effects should be considered in the evaluation of early asbestosis, lung function tests, and respiratory symptoms.

A histological diagnosis of asbestosis requires the identification of diffuse interstitial fibrosis in well inflated lung tissue remote from a lung cancer or other mass lesion, *plus* the presence of either 2 or more asbestos bodies in tissue with a section area of 1 cm^2 or a count of uncoated asbestos fibers that falls into the range recorded for asbestosis by the same laboratory.

In order to achieve reasonable comparability between different studies, a standardized system for the histological diagnosis and grading of asbestosis is required. The Roggli-Pratt modification of the CAP-NIOSH system is recommended as a reasonably simple and reproducible scheme for this purpose.

There is evidence that rare cases of asbestosis occur without significant numbers of asbestos bodies. These cases are recognizable — and distinguishable from idiopathic pulmonary fibrosis — only by analysis of the uncoated fiber burden. Rare cases of asbestosis in relation to the inhalation of pure chrysotile can occur, with a prolonged interval between the last exposure and the diagnosis and few or no detectable asbestos bodies and a low fiber burden. The existence of such cases is speculative and, if the diagnosis can be made, it must be done from other compelling clinical or radiological grounds combined with exposure data.

Fibro-inflammatory patterns other than conventional asbestosis have also been described for workers with occupational exposure to asbestos, including a pattern resembling desquamative interstitial pneumonia (DIP), the occurrence of granulomatous inflammation, a picture that resembles lymphocytic interstitial pneumonia, and organizing pneumonia with bronchiolitis obliterans. Although the DIP-like picture with asbestos bodies is probably asbestos-related, the other patterns have not yet been shown to be so related.

Pleural disorders

Asbestos-related pleural abnormalities are divided into pleural plaques, mainly involving the parietal pleura, sometimes with calcification, and diffuse pleural thickening, which is a collective name for pleural reactions involving mainly the visceral pleura. These include benign asbestos-related pleural effusion, blunted costophrenic angle, crow's feet or pleuroparenchymal fibrous strands, and rounded atelectasis. Avoidance of the term "pleural asbestosis" is recommended. Pleural plaques are usually asymptomatic, and without clinically important findings.

The specificity of pleural plaques according to the ILO 1980 Classification of Radiographs of Pneumoconioses is low unless the plaques are radiographically well

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defined. The most common differential diagnosis is subpleural fat. Radiographic findings are reliable for the diagnosis of asbestos-related pleural plaques when they are characteristic (eg, bilateral circumscribed plaques, bilateral calcification, diaphragmatic plaques).

Pleural plaques represent circumscribed areas of fibrous thickening, typically of the parietal pleura, due to the deposition of paucicellular collagenous tissue with a laminar or basket-weave pattern; they may or may not calcify. In regions where plaques are not endemic, 80—90% of the plaques that are radiologically well defined are attributable to occupational asbestos exposure. The presence of pleural plaques may justify follow-up among occupationally exposed groups.

Diffuse pleural fibrosis designates noncircumscribed fibrous thickening of variable cellularity, which usually affects the parietal, but mainly the visceral, layers. In the setting of occupational asbestos exposure, such diffuse fibrosis is probably a result of benign asbestos pleuritis with effusion. It may or may not be associated with rounded atelectasis. Diffuse pleural thickening can be associated with mild, or rarely moderate or severe, restrictive pulmonary function defects.

Low exposures from work-related, household, and natural sources may induce pleural plaques. For diffuse pleural thickening, higher exposure levels may be required.

Mesothelioma

Malignant mesothelioma affecting any serosal membrane may be induced by asbestos inhalation. The histological, immunohistochemical and ultrastructural markers for the diagnosis of mesothelioma are well established. Expert opinion should be sought on atypical cases; or on those in which the diagnosis is uncertain because of discordant findings or in which the amount of material available is insufficient for definite diagnosis. Mesothelioma is frequently presented with pleural effusion, dyspnea, and chest pain.

With the exception of certain histological types of mesothelioma that are benign or of uncertain or borderline malignant potential (eg, multicystic mesothelioma, benign papillary mesothelioma), all types of malignant mesothelioma can be induced by asbestos, with the amphiboles showing greater carcinogenic potency than chrysotile.

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

The question is unresolved of whether or not a case of mesothelioma for which the lung fiber count falls within the range recorded for unexposed urban dwellers is related to asbestos. More information is needed regarding the interpretation of fiber burdens in the pleura or samples of tumor tissue before these measures can be used for the purposes of attribution.

The following points need to be considered in the assessment of occupational etiology:

- The great majority of mesotheliomas are due to asbestos exposure.
- Mesothelioma can occur in cases with low asbestos exposure. However, very low background environmental exposures carry only an extremely low risk.
- About 80% of mesothelioma patients have had some occupational exposure to asbestos, and therefore a careful occupational and environmental history should be taken.
- An occupational history of brief or low-level exposure should be considered sufficient for mesothelioma to be designated as occupationally related.
- A minimum of 10 years from the first exposure is required to attribute the mesothelioma to asbestos exposure, though in most cases the latency interval is longer (eg, on the order of 30 to 40 years).
- Smoking has no influence on the risk of mesothelioma.

Lung cancer

All 4 major histological types (squamous, adeno-, large-cell and small-cell carcinoma) can be related to asbestos. The histological type of a lung cancer and its anatomic location (central or peripheral, upper lobe versus lower lobe) are of no significant value in deciding whether or not an individual lung cancer is attributable to asbestos. Clinical signs and symptoms of asbestos-related cancer do not differ from those of lung cancer of other causes.

As examples, 1 year of heavy exposure (eg, manufacture of asbestos products, asbestos spraying, insulation work with asbestos materials, demolition of old buildings) or 5—10 years of moderate exposure (eg, construction, shipbuilding) may increase the lung cancer risk 2-fold or more. In some circumstances of extremely high asbestos exposure, a 2-fold risk of lung cancer can be achieved with exposure of less than 1 year.

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The relative risk of lung cancer is estimated to increase 0.5—4% for each fiber per cubic centimeter per year (fiber-years) of cumulative exposure. With the use of the upper boundary of this range, a cumulative exposure of 25 fiber-years is estimated to increase the risk of lung cancer 2-fold. Clinical cases of asbestosis may occur at comparable cumulative exposures.

A 2-fold risk of lung cancer is related to retained fiber levels of 2 million amphibole fibers (>5 μm) per gram of dry lung tissue or 5 million amphibole fibers (>1 μm) per gram of dry lung tissue. This lung fiber concentration is approximately equal to 5000 to 15 000 asbestos bodies per gram of dry tissue, or 5 to 15 asbestos bodies per milliliter of bronchoalveolar lavage fluid. When asbestos body concentrations are less than 10 000 asbestos bodies per gram of dry tissue, electron microscopic fiber analyses are recommended.

Chrysotile fibers do not accumulate within lung tissue to the same extent as amphiboles because of faster clearance rates; therefore, occupational histories (fiber-years of exposure) are probably a better indicator of lung cancer risk from chrysotile than fiber burden analysis is.

A lung fiber burden within the range recorded for asbestosis in the same laboratory should be assigned a significance similar to that of asbestosis. For a patient with lung cancer and a fiber count that falls within the range recorded for unexposed urban dwellers, the relationship of the tumor to amphibole asbestos is doubtful at most.

Estimates of the relative risk for asbestos-associated lung cancer are based on different-sized populations. Because of the high incidence of lung cancer in the general population, it is not possible to prove in precise deterministic terms that asbestos is the causative factor for an *individual* patient, even when asbestosis is present. However, attribution of causation requires *reasonable* medical certainty on a probability basis that the agent (asbestos) has caused or contributed materially to the disease. The likelihood that asbestos exposure has made a substantial contribution increases when the exposure increases. Cumulative exposure, on a probability basis, should thus be considered the main criterion for the attribution of a substantial contribution by asbestos to lung cancer risk. For example, relative risk is roughly doubled for cohorts exposed to asbestos fibers at a cumulative exposure of 25 fiber-years or with an equivalent occupational history, at which level asbestosis may or may not be present or detectable. Heavy exposure, in the absence of radiologically diagnosed asbestosis, is sufficient to increase the risk of lung cancer. Cumulative exposures below 25 fiber-years are also associated with an increased risk of lung cancer, but to a less extent.

The presence of asbestosis is an indicator of high exposure. Asbestosis may also contribute some additional risk of lung cancer beyond that conferred by asbestos exposure alone. Asbestosis diagnosed clinically, radiologically (including HRCT), or histologically can be used to attribute a substantial causal or contributory role to asbestos for an associated lung cancer.

Pleural plaques are an indicator of exposure to asbestos fibers. Because pleural plaques may be associated with low levels of asbestos exposure, the attribution of lung cancer to asbestos exposure must be supported by an occupational history of substantial asbestos exposure or measures of asbestos fiber burden. Bilateral diffuse pleural thickening is often associated with moderate or heavy exposures, as seen in cases with asbestosis, and should be considered accordingly in terms of attribution.

A minimum lag-time of 10 years from the first asbestos exposure is required to attribute the lung cancer to asbestos.

Not all exposure criteria need to be fulfilled for the purposes of attribution. For example, the following can be considered: (i) significant occupational exposure history with low fiber burdens (eg. long exposure to chrysotile and long lag-time between the end of exposure and mineralogical analysis) and (ii) high fiber counts in lung or broncholavage fluid with an uncertain history or without long-term duration (short exposures can be very intense).

At very low levels of asbestos exposure, the risk of lung cancer appears to be undetectably low.

Although tobacco smoking affects the total lung cancer risk, this effect does not detract from the risk of lung cancer attributable to asbestos exposure. No attempt has been made in this report to apportion the relative contributions of asbestos exposure and tobacco smoking.

Prevention and screening

Screening of asbestos-exposed populations can be carried out for practical and scientific purposes. There are 4 goals of screening: (i) to identify high risk groups, (ii) to target preventive actions, (iii) to discover occupational diseases, and (iv) to develop improved tools for treatment, rehabilitation and prevention. Screening should aim to prevent asbestos-related diseases and therefore lead to gained healthy years of life among the screened or among those in similar risk situations. The benefits to the individual person should be viewed cautiously. The substantial morbidity and mortality related to asbestos exposure argue for continued efforts to increase the preventive power of screening.

Any screening for purely scientific purposes requires appropriate methods and criteria (eg. low cost and high predictive value). Before a screening program is initiated, the ethical, financial, and legislative aspects need to be considered. These aspects may include patient notification, data protection, allocation of costs, and follow-up of identified abnormalities. In addition, provision should

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be made for epidemiologic analyses, quality control, primary and secondary prevention, and the assessment of program effectiveness.

As tools for screening, questionnaires and personal interviews should include items related to asbestos exposure, smoking, and other contributing factors. Questionnaires should preferably be validated for smoking habits and occupational histories. When possible, questionnaires should be applied nationally to permit epidemiologic analysis of the results.

Chest X-ray examinations can include frontal and lateral roentgenograms. Appropriate lung function tests can measure respiratory flow volumes and rates. In spirometry, attention should be given to careful calibration, acceptable performance efforts, and reproducibility.

The prevention strategies of asbestos-related diseases can be based on the identification of exposure sources and exposed people. There are 3 main targets for prevention: (i) an individual worker, (ii) a selected group of workers, and (iii) the work environment. At the level of the individual worker, the tools for prevention include health education and the introduction of safe work practices, the avoidance of tobacco smoking, and careful follow-up of health by surveillance. The group level methods are in part the same as at the individual level (ie, health information, education, and recommendations including the use of respiratory protective equipment).

The work environment is the most important target for preventive measures, starting from avoiding the use of asbestos, carefully controlling dust emissions using wet techniques, and controlling passive smoking at the workplace. Many countries have prohibited the use of asbestos, but there are still substantial amounts of asbestos in consumer products and in buildings that can expose workers in repair and removal work. Some countries have permitted asbestos work only under special authorization, training, and protective measures.

From the knowledge on potential exposures to asbestos, high-risk populations can be identified among persons exposed 10 or more years ago. The availability of registers — union, workers' compensation, and employment records — can be explored for this purpose.

Subjects can be assigned to subgroups for intervention or screening as defined by their risk (eg, the current risk of lung cancer and risk projected to given time windows in the future). Criteria for inclusion in each intervention or screening group should be established in the study protocol. Subsequently, the members of each subgroup can serve as separate targets for group-based and individual intervention programs.

Protocols for intervention should be designed in such a way that they serve each subject and subgroup optimally in terms of promoting individual health and the early detection of asbestos-related diseases. Data on these subgroups can also form a basis for more specific studies of disease outcome or various biomarkers. Identified abnormalities should be followed by the best clinical and occupational practices.

Research needs

There are several issues that still require clarification and further study. The following list of recommendations and future directions is not intended to be exhaustive.

- Improvement in the assessment and quantification of exposure to asbestos, to include specific worker groups, with collation of data and the development of an international standardized protocol for the assessment of exposure.
- Further analysis of job-exposure data and further studies on asbestos fiber burdens in tissue in relation to various asbestos-related disorders.
- Studies on chrysotile fiber burdens in lung tissue relative to the risk of lung cancer (also to include experimental investigations).
- Lung cancer relative to the lung tissue burdens of mineral fibers other than asbestos (eg, refractory ceramic fibers and zeolites).
- Improvement of the ILO system for the radiological diagnosis and categorization of pleural abnormalities.
- Development of a standardized system for the reporting of HRCT scans of asbestos-related disorders, analogous to the ILO system.
- Studies on the specificity of lesions of the pleura visualized by CT as markers of asbestos exposure and studies on the prognosis of diffuse pleural abnormalities.
- Improvement in ultrasound imaging of the pleura.
- Development of new digital imaging techniques for the investigation of asbestos-related diseases.
- Standardization of the approach to lung crepitations with the use of special auditory devices.
- Investigation of mesothelioma as a potential outcome of exposure to mineral fibers other than asbestos — such as refractory ceramic fibers — to include experimental studies and a series of mesothelioma patients without exposure to asbestos or erionite, supported by lung tissue fiber analysis.
- Multicenter studies on biomarkers for the detection of early asbestos diseases and the assessment of the response to new treatment modalities.
- Investigation of asbestos-associated tumors other than lung cancer and mesothelioma (eg, laryngeal carcinoma and renal carcinoma).
- Further studies on the effectiveness of screening programs.

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ty of Washington, United States), John M Dement (Duke University Medical Center, United States), Paul De Vuyst (Cliniques Universitaires de Bruxelles, Hopital Erasme, Belgium), Gunnar Hillerdal (Karolinska Hospital, Sweden), Matti S Huuskonen (Finnish Institute of Occupational Health, Finland), Leena Kivisaari (Helsinki University Central Hospital, Finland), Yukinori Kusaka (Fukui Medical School, Japan), Aarne Lahdensuo (Tampere University Hospital, Finland), Sverre Langård (The National Hospital, Norway), Gunnar Mowe (Department of Social Insurance Medicine, University of Oslo, Norway), Toshiteru Okubo (University of Occupational and Environmental Health, Japan), John E Parker (National Institute for Occupational Safety and Health, United States), Victor L Roggli (Duke University Medical Center, United States), Klaus Rödelsperger (Justus-Liebig University, Germany), Joachim Rösler (Justus-Liebig University, Germany), Antti Tossavainen (Finnish Institute of Occupational Health, Finland), Hans-Joachim Woitowitz (Justus-Liebig University, Germany).

Reprint requests to Dr Antti Tossavainen, Department of Industrial Hygiene and Toxicology, Finnish Institute of Occupational Health, Topeliuksenkatu 41 a A, FIN-00250 Helsinki, Finland (free of charge).

The reprint plus a copy of the conclusions and recommendations (People and Work Research Reports, no 14) can be obtained from the Finnish Institute of Occupational Health, Sirvi Lehtinen, Topeliuksenkatu 41 a A, FIN-00250 Helsinki, Finland, for a price of FIM 80.00 + postage.

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EXHIBIT 16

Scand J Work Environ Health. 2015;41(1):5–15. doi:10.5271/sjweh.3462

Asbestos, asbestosis, and cancer, the Helsinki criteria for diagnosis and attribution 2014: recommendations

Introduction

Although the use of asbestos has been banned in several industrialized countries, many workers continue to be exposed in asbestos repair and removal work, and asbestos is still widely used in various newly industrialized, rapidly developing countries. According to the most recent World Health Organization (WHO) estimates, more than 107 000 people die each year from asbestos-related lung cancer, mesothelioma, and asbestosis resulting from exposure at work (1). The asbestos epidemic is far from over.

The expert meeting on "asbestos, asbestosis, and cancer" was convened in Helsinki in 1997 (2) and consisted of 19 participants from eight countries. This meeting had the goal to "discuss disorders in association with asbestos and agree upon state of the art criteria for diagnosis and attribution with respect to asbestos". In addition, questions concerning the surveillance of asbestos-exposed workers were discussed. The resulting consensus report was titled "Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution" (in the current report a shorter name "Helsinki criteria" or just "criteria" will be used). A follow-up expert meeting on new advances in radiology and screening of asbestos-related diseases was organized in 2000 in Helsinki (3).

This report summarizes the results of a project, organized by the Finnish Institute of Occupational Health (FIOH), to update the 1997 and 2000 Helsinki criteria documents in view of the new advances in research. It presents the conclusions of a meeting on 10–13 February 2014 in Espoo, Finland, among an international group of experts working to update the criteria.

Recommendations from the Helsinki criteria of 1997

In the following, recommendations from the Helsinki criteria document of 1997 (and for radiology 2000) are quoted together with commentary by the authors.

General considerations

"In general, reliable work histories provide the most practical and useful measure of occupational asbestos exposure". "Using structured questionnaires and checklists, trained interviewers can identify persons who have a work history compatible with significant asbestos exposure." "A cumulative fibre dose expressed as fibre-years per cubic centimetre, is an important parametre of asbestos exposure."

"Analysis of lung tissue for asbestos fibres and asbestos bodies can provide data to supplement the occupational history."

"For clinical purposes, the following guidelines are recommended to identify persons with a high probability of exposure to asbestos dust:

- over 0.1 million amphibole fibres (>5 μm) per gram of dry lung tissue or
- over 1 million amphibole fibres (>1 μm) per gram of dry tissue as measured by electron microscopy in a qualified laboratory or
- over 1000 asbestos bodies per gram of dry tissue (100 asbestos bodies per gram of wet tissue) or
- over 1 asbestos body per millilitre of bronchoalveolar lavage fluid as measured by light microscopy in a qualified laboratory.

Each laboratory should establish its own reference values."

Asbestosis

"Asbestosis is defined as diffuse interstitial fibrosis of the lung as a consequence of exposure to asbestos dust." It is noted that neither clinical nor histological features of asbestosis differ sufficiently from those of other causes of interstitial fibrosis to allow confident diagnosis without a history of asbestos exposure or the detection of increased levels of asbestos bodies or asbestos fibres in lung tissue.

"Asbestosis is generally associated with relatively high exposure levels." It is however noted that mild fibrosis may occur at lower exposure levels and that histologically detectable fibrosis can occur in situations when radiological criteria are not fulfilled.

"A histological diagnosis of asbestosis requires the identification of diffuse interstitial fibrosis" in technically sound lung samples "plus the presence of either 2

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or more asbestos bodies in tissue with a section area of 1 cm^2 or a count of uncoated asbestos fibres that falls in the range recorded for asbestosis by the same laboratory." It is also noted there is evidence that, in rare cases, asbestosis can occur without the presence of asbestos bodies and that such cases are only recognizable by uncoated fibre burden. In addition, it is noted there may be cases of chrysotile-induced asbestosis without increased levels of asbestos bodies or increased fibre burden, however, this is considered speculative.

"In order to achieve reasonable comparability between different studies a standardized system for the histological diagnosis and grading of asbestosis is required. The Roggli-Pratt modification of CAP-NIOSH system is recommended."

Pleural disorders

"Pleural plaques represent circumscribed areas of fibrous thickening typically of the parietal pleura." "In regions where plaques are not endemic, 80–90% of the plaques that are radiologically well-defined are attributable to occupational asbestos exposure."

"Diffuse pleural fibrosis designates non-circumscribed fibrous thickening of variable cellularity" involving mainly the visceral pleura. It "is probably the result of benign asbestos pleuritis with effusion. Diffuse pleural thickening can be associated with rounded atelectasis, it can be associated with mild or rarely moderate or severe restrictive pulmonary defects."

"Low exposures" from various sources "may induce pleural plaques. For diffuse pleural thickening higher exposure levels may be required".

Mesothelioma

"With the exception of certain histological types of mesothelioma that are benign or of an uncertain malignant potential (eg, multicystic mesothelioma, benign papillary mesothelioma), all types of malignant mesothelioma can be induced by asbestos with the amphiboles showing greater carcinogenic potency than chrysotile."

On requirements for and documentation of asbestos exposure to attribute causation:

- "A lung fibre count exceeding the background range for the laboratory in question." This is different and lower from the threshold value for persons with a high probability of exposure to asbestos dust at work mentioned earlier in the 'general considerations' paragraph.
- "or the presence of radiographic or pathological evidence of asbestos related tissue injury (e.g. asbestosis or pleural plaques)
- "or histopathological evidence of abnormal asbestos content (e.g. asbestos bodies in histological sections of lung) should be sufficient to relate a case of mesothelioma to asbestos exposure on a probability basis.

In the absence of such markers a history of significant occupational, domestic or environmental exposure will suffice for attribution. There is evidence that peritoneal mesotheliomas are associated with higher levels of asbestos exposure than pleural mesotheliomas."

"The following points need to be considered in the assessment of occupational aetiology:

- "The great majority of mesotheliomas are due to asbestos exposure;
- "Mesotheliomas can occur in cases with low asbestos exposure. However, very low background environmental exposures carry only an extremely low risk;
- "About 80% of mesothelioma patients have had some occupational exposure to asbestos, and therefore a careful occupational and environmental history should be taken;
- "Even a brief or low-level exposure should be considered sufficient for mesothelioma to be designated as occupationally related;
- "A minimum of 10 years from the first exposure is required to attribute the mesothelioma to asbestos exposure, though in most cases the latency interval is longer (eg, in the order of 30–40 years); and
- "Smoking has no influence on the risk of mesothelioma."

Lung cancer

Concerning types of lung cancer associated with asbestos exposure, "all four major histological types (squamous, adeno, large-cell and small cell) can be related to asbestos". In addition, location of the tumor in the lungs is not considered important in determining attributability.

"Attribution of causation requires a reasonable medical certainty on a probability basis that the agent (asbestos) has caused or contributed materially to the disease." Noting that increased exposure to asbestos also increases the risk, "cumulative exposure, on a probability basis should thus be considered the main criterion for the attribution of a substantial contribution by asbestos to lung cancer risk."

Using an estimate of 4% increase of risk for each fibres per cubic centimetre per year (fibre year) of cumulative

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exposure: "A cumulative exposure of 25 fibre-years is estimated to increase the risk of lung cancer 2-fold, clinical cases of asbestosis may occur at comparable cumulative exposures."

"A 2 fold risk of lung cancer is related to retained fibre levels of 2 million amphibole fibres (>5 μm) per gram of dry lung tissue or 5 million amphibole fibres (>1 μm) per gram of dry lung tissue. This lung fibre burden is approximately equal to 5000–15 000 asbestos bodies per gram of dry lung tissue, or 5–15 asbestos bodies per millilitre of bronchoalveolar lavage fluid." However, elsewhere in the consensus report, it is noted that each laboratory should establish its own reference values due to the lack of standardization of these methods.

"Occupational histories (fibre years of exposure) are probably a better indicator of lung cancer risk from chrysotile than fibre burden analysis" because of the higher clearance rates for chrysotile. "The presence of asbestosis is an indicator of high exposure. Asbestosis may also contribute some additional risk of lung cancer beyond that conferred by asbestos."

"Pleural plaques are indicators of exposure to asbestos fibres. Because pleural plaques may be associated with low levels of exposure, the attribution of lung cancer to asbestos must be supported by an occupational history of substantial asbestos exposure or measures of asbestos fibre burden," ie, pleural plaques alone are insufficient for the attribution of lung cancer to asbestos.

"Bilateral diffuse pleural thickening is often associated with moderate or heavy exposure, as seen in cases of asbestosis and should be considered accordingly in terms of attribution," ie, this type of pleural thickening is considered to be "close" to asbestosis in terms of exposure.

"A minimum lag time of ten years from the first asbestos exposure is required to attribute the lung cancer to asbestos."

"Not all exposure criteria need to be fulfilled for the purposes of attribution." For example, high fibre or asbestos-body counts with an uncertain work history should be considered.

"Although tobacco smoking affects the total lung cancer risk, this effect does not detract from the risk of lung cancer attributable to asbestos exposure. No attempt has been made in this report to apportion the relative contributions of asbestos exposure and tobacco smoking."

Radiology and screening, including follow-up meeting in 2000

For asbestosis diagnosis, the finding of International Labor Organization (ILO) profusion 1/0 was regarded as an early stage asbestosis and high-resolution computed tomography (HRCT) was recommended to be used as a diagnostic aid in selected cases in 1997.

In 2000, the technical requirements for asbestos HRCT were defined and the need for an international classification system for occupational HRCT findings led to a cooperative process between several investigators.

The issue of screening asbestos exposed workers for lung cancer remained open: research activity and the follow-up of ongoing investigations were encouraged.

Recommendations

To evaluate the need to update the Helsinki criteria, FIOH sent a questionnaire to selected international experts. In the questionnaire, we inquired about various aspects of the criteria, policies regarding the surveillance of asbestos-exposed workers, and experts' opinion on the need for and focus of updates to the criteria. The answers indicated a considerable variation of opinions although some general trends could be seen. The questionnaires, the possible updates, and the need for an update were discussed at an initial meeting of international experts and FIOH staff on 2 December 2011. After these discussions the following subject areas were selected for updating:

- CT screening for asbestos-related lung cancer;
- Follow-up of asbestos-exposed workers and diagnosis of non-malignant asbestos diseases;
- New asbestos-related disease entities; and
- Pathology and biomarkers.

For each subject area, a work-group prepared a review published separately. (4) Based on the reviews, recommendations for each subject area were made.

Area 1. CT screening for asbestos-related lung cancer.

It has been shown in the US national lung screening trial (NLST) (5) that lung cancer screening with low-dose computed tomography (LDCT) reduces both lung cancer mortality and over-all mortality among current and former smokers. Consequently, a growing number of organizations have issued recommendations to screen current or former (quit within 15 years) smokers in slightly varying age groups with LDCT (6–10). Some also endorse beginning of screening at age 50 years among adults with a 20-pack-year smoking history if they also have an additional risk factor, such as asbestos exposure, or if

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there is an additional cumulative risk of developing lung cancer of ≥5% in the next five years (10, 11)

There are screening/surveillance programs of asbestos workers based on chest X-rays in some countries (12). This typically is a medicolegal program, and there is little scientific evidence that it is an effective strategy for reducing lung cancer mortality. There is increasing evidence from adults with significant smoking history that screening with LDCT reduces lung cancer mortality, but the generalizability to individuals at similar high risk based on asbestos exposure with or without smoking history is uncertain. Thus, we have two overarching recommendations for data gathering to evaluate these assumptions.

First, existing studies should be assessed for the potential of pooling to verify the generalizability of the LDCT random-controlled trial (RCT) results to asbestos-exposed high-risk populations. Second, since these updated Helsinki criteria recommendations are based on inferential evidence and modeling, the introduction of lung screening among asbestos-exposed workers should be accompanied by standardized data collection in order to acquire the necessary evidence to validate and refine these recommendations. Also, we strongly recommend the establishment of an international multicenter research project that could acquire these data for pooling, so that the specific effects of LDCT screening among asbestos-exposed workers can be measured and contribute to health policy. We believe the following study designs allow for the simultaneous enrollment of asbestos-exposed workers into a screening program, while also collecting minimum, but essential data. In settings:

(a) where LDCT is offered to current and former smokers at high risk, offer screening to high-risk workers with asbestos exposure with or without smoking history if the lung cancer risk is similar to the risk in high-risk smokers as defined by the NLST entry criteria and compare their outcomes with screened individuals at high risk based only on smoking history;
(b) that will demand evidence from RCT of asbestos-exposed workers, conduct national or international pooled RCT;
(c) where LDCT screening is available for asbestos exposed workers but it is not offered to (a) current and former smokers at high risk in organized screening programs or (b) in RCT of asbestos-exposed workers, follow standardized protocols from any one of the LDCT RCT to implement screening for asbestos-exposed workers who meet entry criteria based on asbestos exposure with or without tobacco exposure history [as defined in (a)] and monitor process and disease outcomes in comparison with respective RCT data for adults at risk for lung cancer based on smoking history alone.

In each of the examples above, the benefits, harms, and economic issues of LCDT screening should be studied. At this time, there is scant direct evidence on issues related to risk estimation and LDCT screening in workers at high risk for lung cancer due to asbestos exposure with or without a history of smoking. Based on the lung cancer LDCT screening studies, the dose–response risk of lung cancer in asbestos-exposed workers, and the established relationship of interaction of asbestos exposure and smoking, we recommend the following groups for LDCT screening:

- workers with any asbestos exposure and a smoking history equal to the entry criteria of the NLST study; and
- workers with asbestos exposure with or without a smoking history, which alone or together would yield an estimated risk level of lung cancer equal to that in the entry criteria of the NLST study.

Much work remains to be done related to risk estimation for lung cancer screening eligibility, especially the interplay between age, smoking history, other exposures to tobacco smoke, and other risk factors such as occupational history or genetic predisposition. In addition, the optimum screening interval should be further studied in forthcoming pilot trials with probably both annual and biennial arms included. Evidence may also be gained through modeling of existing materials, especially the NLST material.

Ideally, the responsibility for screening should be nationally or regionally organized so that preferably a single unit/institute would be in charge of the entire process. This includes the organization of screening, quality control, the collection and analysis of all data on benefits and complications as well as the evaluation of economic issues. The organizer may be the national occupational health institute, and it should have sufficient expertise on epidemiology, pulmonology, radiology and other relevant sciences. The participating units should be included in the most feasible manner nationally and educated in a sufficient manner in order to achieve high quality screening and follow-up and data collection. International collaboration is a high priority.

Area 2. Follow-up of asbestos-exposed workers and diagnosis of non-malignant asbestos diseases

Follow up of asbestos-exposed workers. The follow-up of asbestos-exposed workers has a long tradition from a preventive point of view and is mandatory in many countries. The recommended follow-up routines – including both active and retired workers – differ among countries. Exposure history, disease history, pulmonary function and the chest radiograph have traditionally

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been the dominant methods. Computer tomography (CT) has during recent years been identified as useful in diagnosis and is emergent in screening of asbestos-related diseases.

The traditional method for medical screening of asbestos-exposed workers has been the conventional chest radiograph, with standardized interpretation using the ILO system for classification of radiographs for pneumoconiosis (13). Radiographic surveillance as a part of periodical health examination (eg, every 3–5 years) of asbestos-exposed workers is still commonly practiced in developed countries. There is little specific scientific evidence of the health benefits of such surveillance. The potential benefits of the timely detection of non-malignant asbestos-related diseases includes the reduction in current asbestos exposure, an incentive to cease smoking, the encouragement of immunization and early treatment of respiratory infections, and increased health knowledge obtained by the participants. Those potential benefits must be weighed against the harm incurred as a result of the radiation dose received as part of radiographic surveillance.

The national recommendations to date have principally focused on non-malignant asbestos-related disease but may be developed at present to facilitate early detection of asbestos-related lung cancer by the use of CT. With regard to the use of CT for the early detection of lung cancer, a possible follow-up schema of asbestos-exposed workers is presented in the recommendations from the Area 1 group.

With regard to the follow-up of non-malignant effects, the traditional guidelines vary widely between countries and are only to a limited degree based on scientific evidence. We still recommend that asbestos-exposed workers should be offered a medicolegal surveillance according to national regulations or compensation rules. These surveillance activities should when possible be organized as national programs and utilized for research.

A general follow-up schema of asbestos-exposed workers should be stratified according to the intensity, latency, and duration of exposure. In general, reliable work histories provide the most practical and useful measure of occupational asbestos exposure. High priority should be given to the high-risk groups, including retired workers.

Both radiographic and functional pulmonary deterioration (worsening) may occur long after asbestos exposure. Follow-up is necessary especially if it is relevant from a compensation point of view. We propose that the follow-up of highly asbestos-exposed workers should be continued for up to 30 years after the cessation of exposure.

In accordance with previous Helsinki criteria 1997 recommendations, we still recommend the use of spirometry together with questionnaires on past or current exposure, and current symptoms as a reference check-up for all asbestos-exposed workers. The questionnaire should provide detailed information on past (and current) asbestos exposure and smoking habits, specifically age of initiation, cigarettes per day, and date of cessation for former smokers. Especially if the follow-up schema is stratified according to the quantity of estimated exposure, it is recommended to calculate or approximate worker's exposure as fibre years if possible.

For clinical and medicolegal purposes, regular follow-up with spirometry, based on the national practice and legal requirements, is useful. A reasonable periodic interval is every 3–5 years, dependent on past exposure level, time since cessation of exposure, and age. Measurements of carbon monoxide diffusion capacity might be used at baseline and in patients with documented asbestosis, but not for repeated screening purposes. It is important to advise all asbestos-exposed workers to contact their physician if they develop symptoms or signs of respiratory disease.

Biomarkers for follow-up and diagnostics. More studies are needed to assess if the markers of inflammation could be used to predict the risk of developing asbestosis in asbestos-exposed workers.

Immunization of asbestosis patients. We recommend influenza and pneumococcal vaccination to patients with asbestosis, even if there are no vaccination trials available (12).

Diagnosis of non-malignant asbestos diseases (asbestosis and pleural thickening)

The use of CT imaging in diagnosis of asbestos-related diseases may be useful when:

- a borderline finding of lung fibrosis (ILO 0/1-1/0) is detected;
- there is a discrepancy between lung function finding of restriction and radiographs interpreted as normal;
- widespread pleural changes severely hamper the radiographic visibility of lung parenchyma.

CT imaging should be done using state-of-the-art multislice scanner technology and high resolution reconstruction algorithms. Exposure to ionizing radiation should be kept as low as possible.

The international expert meeting in Helsinki in 2000 recommended that a common, international classification scheme for pulmonary and pleural abnormalities detected on CT scans of asbestos-exposed workers should be established for the early identification of both malignant and non-malignant asbestos diseases, comparable to the 1980 ILO international classification

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of radiographs for pneumoconiosis (13). Such a common classification system is described in a monograph (14) and has been adopted by many countries. This classification has now been further developed to cover all types of occupational and environmental diseases (ICOERD) (15). Some countries such as France, have developed a nationally based classification system. For international comparison of studies of asbestos-exposed groups, we recommend use of the ICOERD classification.

Minimum criteria for diagnosis of asbestosis in CT. It is well known that small irregular opacities and pleural changes occur in the general population without work-related asbestos exposure. It is also well known that CT-detected lung fibrosis can be seen also in those with unlikely asbestos exposure and shows a distinct, although limited increase with age. The occurrence of limited lung fibrosis in the general population makes the definition of a threshold value for asbestosis important. We therefore recommend that the sum grade of ≥2–3 bilateral irregular opacities in lower zones according to the reference film or bilateral honeycombing (sum grade ≥2) would be sufficient to represent fibrosis according to the ICOERD system. In histopathology, bronchiolar wall fibrosis has been associated with asbestos exposure and other exposures including smoking (16). Subpleural curvilinear lines or dots in HRCT are findings of bronchiolar fibrosis.

Minimal pleural thickening. Very small pleural thickening is common and hard to separate from naturally thicker than average pleura, intercostal muscles, subpleural scars, adhesions etc ... in CT. According to the ICOERD, all visible pleural thickening is recorded: differential diagnosis of etiology depends upon disease and occupational history.

Area 3. New asbestos-related disease entities

Asbestos-related cancers. The legal standard of "more likely than not", equivalent to a relative risk (RR) of 2, has been used in many countries as a threshold for attribution of causation of disease in individuals by hazardous exposures and was used as a threshold in the 1997 Helsinki criteria. However, the choice of a threshold for RR related to a hazardous exposure is based on social, economic, and political considerations, which vary between countries. In some situations, exposures associated with RR of as little as 1.1 have been viewed as making a material contribution to causation. We recommend that the threshold RR used for individual attribution should be no greater than 2 and can be set at lower levels. Whatever the threshold, it should be recognized that asbestos can contribute to causation of disease in exposed populations at even lower levels. To provide flexible guidance for setting threshold levels for individual causation, we have determined the relationship between RR for each reviewed new cancer entity and RR for lung cancer, using data from cohort studies that evaluated RR for both. The exposure needed to reach a threshold RR for the new entity cancer can then be determined based on that relationship. In our evaluation, this provided the most practical approach to providing criteria for individual attribution for these cancer entities.

Laryngeal cancer. Based on cohort studies evaluating standardized incidence ratios, the RR for laryngeal cancer in an asbestos-exposed cohort is somewhat less than the RR for lung cancer. The estimated RR based on cohort studies for laryngeal cancer would reach 2 under conditions where the RR for lung cancer in an exposed population was 2.8 (17). Standardized mortality ratios for the two cancer entities do not correlate well, perhaps in part because laryngeal cancer is less likely to result in mortality if appropriately treated and thus might be more variably captured on death certificates. Under conditions of asbestos exposure associated with an RR for lung cancer of 2, the estimated RR for laryngeal cancer is 1.6, with an estimated attributable fraction (AF) in the exposed population for asbestos-causation, of about 37%.

The IARC has concluded (18) there is sufficient evidence for asbestos-causation of laryngeal cancer in humans, and the weight of litreature published after the IARC evaluation remains consistent with this evaluation. Thus, laryngeal cancer should be viewed as a disease that is caused by asbestos.

Ovarian cancer. The RR for ovarian cancer was slightly higher than the RR for lung cancer in an asbestos-exposed cohort (17). The estimated RR for ovarian cancer would reach 2 under conditions where the RR for lung cancer in an exposed population was about 1.7. Under conditions of asbestos exposure associated with a RR for lung cancer of 2, the estimated RR for ovarian cancer is 2.2, with an estimated AF in the exposed population for asbestos-causation, of about 54%.

The IARC has concluded that there is sufficient evidence for asbestos-causation of ovarian cancer in humans (18). The weight of the litreature remains consistent with the IARC's recent evaluation. Thus, ovarian cancer should be viewed as a disease that is caused by asbestos. Peritoneal mesothelioma should be considered in the differential diagnosis of women presenting with possible ovarian cancer who have been exposed to asbestos, since the two conditions can present similarly and be confused. Additional research is needed to document the specific histopathological types of ovarian cancer that are caused by asbestos exposure.

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Colorectal cancer. Few studies have determined risk estimates separately for colon and rectal cancer. Some data suggest that asbestos affects the risk of cancer of the colon, especially the proximal (right side) colon. The IARC's analysis of the relationship shows that in an asbestos-exposed cohort, the RR for colorectal cancer is much less than the RR for lung cancer (18). The estimated RR for colorectal cancer would reach 2 under conditions where the RR for lung cancer in an exposed population was about 5.2. Based on the IARC's analysis, under conditions of asbestos exposure associated with an RR for lung cancer of 2, the estimated RR for colorectal cancer is 1.1, with an estimated AF in the exposed population for asbestos-causation of colorectal cancer, of about 9%.

The IARC has concluded that there is limited evidence in human epidemiology studies for an association between asbestos exposure and colorectal cancer in humans (18). The reports published since the IARC evaluation are not definitive. Recognizing that the available evidence is relatively strong, the weight of litreature remains consistent with the IARC's recent evaluation, and colorectal cancer cannot currently be viewed with certainty as a disease that is caused by asbestos.

Stomach cancer. In an asbestos-exposed cohort, the RR for stomach cancer is much less than the RR for lung cancer. The estimated RR for stomach cancer would reach 2 under conditions where the RR for lung cancer in an exposed population was approximately 4 (18). Under conditions of asbestos exposure associated with a RR for lung cancer of 2, the estimated RR for stomach cancer is 1.2, with an estimated AF in the asbestos-exposed population for asbestos-causation, of about 17%.

IARC has concluded (18) that there is limited evidence in human epidemiology studies for an association between exposure to asbestos and stomach cancer in humans. Overall, most cohort studies and the various meta-analyzes provide consistent evidence of an increased risk of stomach cancer associated with asbestos exposure. Risk estimates tend to be higher in cohorts where heavy exposure to asbestos occurs and with long follow-up periods. There is also evidence that increasing exposure increases this risk. There are few case–control studies and these give less consistent results. The weight of the litreature remains consistent with the IARC's recent evaluation. Thus, stomach cancer cannot currently be viewed with certainty as a disease that is caused by asbestos.

Ventilatory impairment and chronic airway obstruction. Available studies indicate that the pattern of spirometric impairment associated with asbestos exposure is primarily restrictive, with reduced forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), and relatively preserved FEV_1/FVC ratio (17). Studies have also suggested that a component of airways obstruction can also be present, in particular small airways obstruction, although in the absence of other hazardous co-exposures such as tobacco smoke, obstructive changes are generally of lesser prominence and more difficult to demonstrate. Statistically significant spirometric impairment can be demonstrated in populations with sufficient exposure to cause pleural thickening or asbestosis, even among those without radiographic changes. However, the prevalence of clinically significant spirometric impairment in individuals without asbestos-related radiographic changes is unclear. The magnitude of impairment is considerably greater in the presence of diffuse pleural thickening (DPT) and/or asbestosis than if no asbestos-related radiographic changes are present. Also, some studies have shown that localized pleural thickening (LPT) can be associated with spirometric impairment. The magnitude of impairment increases with age and with smoking.

Findings of asbestos-induced pleuroparenchymal disease and emphysema have been reported to explain only about half the variance in total lung capacity (TLC), FVC, and carbon monoxide diffusing capacity (DLCO) among those exposed to asbestos with or without tobacco smoking. Therefore, more remains to be learned about the determinants of lung function impairment in asbestos-exposed individuals, particularly those without radiographic evidence of asbestos-induced parenchymal or pleural disease. In addition, more needs to be learned about how to attribute impairment in lung function to various potential causes when more than one potential cause is present.

Restrictive or mixed obstructive/restrictive patterns of ventilatory impairment associated with reduction of FEV_1 below the lower limit of normal can be considered asbestos-caused if there has been asbestos exposure and radiographic pleural or parenchymal findings consistent with asbestos exposure are present. Ventilatory impairment of this type in the absence of asbestos-related radiographic changes cannot be viewed as asbestos-caused. Purely obstructive ventilatory impairment associated with reduction of FEV_1 below the lower limit of normal cannot be viewed as being caused by asbestos.

Retroperitoneal fibrosis. Retroperitoneal fibrosis (RPF) is a rare disease featuring the proliferation of fibrous tissue in the retroperitoneal compartment of the body containing the kidneys, urinary tract, aorta, and various other structures. Evidence for causation of RPF by asbestos is suggested by a limited body of litreature including case reports, two case series and one case control study (17) Epidemiological evidence alone is insufficient to permit a firm conclusion about causation of RPF by asbestos. Still, taking the available case series into account, it is reasonable to conclude that

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RPF can be caused by asbestos. Those with sufficient exposure to develop asbestos-related pleural pathology are at increased risk. There is limited information about intensity of exposures required to cause RPF, relative potencies of different asbestos fibre types, etc.

RPF occurring in an individual with asbestos-related pleural and/or parenchymal radiographic findings can be viewed as caused by asbestos. RPF occurring in an individual with evidence of asbestos exposure, but without asbestos-related radiologic findings, can be viewed as caused by asbestos *if* other risk factors are not identified. All RPF patients should be evaluated for a history of asbestos exposure along with other risk factors.

Area 4. Pathology and biomarkers

Lung cancer types attributed to asbestos exposure. The 1997 Helsinki criteria mentions four major types of lung cancer that are associated with asbestos exposure (squamous, adeno, large-cell and small cell carcinoma). The current classification (19) mentions two additional types: sarcomatoid and adenosquamous carcinoma. Any of these six major histological categories may be considered to occur as a consequence of asbestos exposure (20).

Asbestosis: histological criteria. In the original 1997 Helsinki criteria, the Roggli-Pratt modification of the College of American Pathologists-National Institute of Occupational Safety and Health (CAP-NIOSH) classification for asbestosis was used. Recently, a new classification has emerged (16). In the new classification, bronchial fibrosis is designated "asbestos airways disease". The updated Helsinki criteria concerning the attributability of fibrotic conditions of the lungs to asbestos are applicable to both asbestosis as defined by the new classification and asbestos airways disease. The exposure levels for asbestos airways disease remain to be determined. A fibre analysis by an experienced laboratory is recommended in cases with idiopathic pulmonary fibrosis with a usual interstitial pneumonia (UIP) pattern, in which pleural plaques are present and/or some asbestos bodies are present in histological sections (but fewer than 2 AB/cm^2), or where compelling asbestos exposure history exists. In such cases, a fibre-burden level comparable to those seen in that laboratory in other asbestosis cases favors a diagnosis of asbestosis.

Biomarkers for the histopathological diagnosis of malignant mesothelioma. We recommended that in mesotheliomas with an epithelioid component, at least two positive (mesothelial) and two negative (carcinomatous) markers be used for making a histopathological diagnosis of malignant mesothelioma. Because the usage of these markers has not yet been standardized, it is recommended that each laboratory performing immunohistochemical studies determine which positive and negative markers best fit its needs. It is further recommended that markers used should have ≥80% sensitivity and specificity.

In the case of pleural tumors the main differential diagnosis concerns pulmonary carcinomas. For peritoneal mesotheliomas, pulmonary carcinomas are much less likely to be in the differential diagnosis. The selection of negative markers should reflect this. It is also recommended that for peritoneal malignancies in women, stains for estrogen (ER) and progesterone (PR) receptors be added to the panel.

The diagnostic markers used in epithelioid mesotheliomas are far less useful in sarcomatoid mesotheliomas. Cytokeratin expression is a useful marker in the differentiation of desmoplastic mesothelioma from fibrotic processes (through demonstration of invasion) as well as differentiation of sarcomatoid mesothelioma from sarcomas. This marker is not useful in separating sarcomatoid mesotheliomas from sarcomatoid lung cancers.

It should be noted that clinical correlation with the gross distribution of the tumor is critical for diagnosis of malignant mesothelioma, and none of the immunohistochemical markers are entirely specific for that diagnosis. There are no generally accepted immunohistochemical markers for distinction between benign and malignant mesothelial proliferations.

Biomarkers for screening and early diagnosis of mesothelioma. Blood-borne biomarkers would arguably be ideal for screening purposes. Several markers have been studied with some success; however, currently none are sufficiently sensitive and specific for diagnostic or screening purposes.

A combination of biomarkers in pleural fluid is potentially useful for early diagnosis. A combination of CCL2, galectin-3, and soluble mesothelin-related peptides (SMRP) may discriminate between malignant mesothelioma patients and patients with metastatic adenocarcinoma or nonmalignant pleural effusion (21). Similarly, fluorescent in-situ hybridization (FISH) for homozygous deletion of p16 (INK2A) is a promising technique for distinguishing benign and malignant mesothelial pleural proliferations (22, 23).

Some of these markers may be useful in the treatment of malignancies as a follow-up tool and might help in early clinical diagnosis. A major unresolved question is whether early detection will improve treatment outcome. At this point, no specific recommendations can be made regarding these biomarkers for screening or other purposes.

Markers for attribution to asbestos exposure in lung cancer. The observed asbestos-related molecular alterations in lung cancer are consistent with the ability of asbestos fibres to induce DNA damage and chromosomal

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Table 1. Comparison of the 1997 Helsinki criteria and the 2014 update. [BAL=broncho alveolar lavage; CAP-NIOSH= College of American Pathologists-National Institute of Occupational Safety and Health ; CT=computer tomography; HRCT=high-resolution computer tomography; ICOERD=international classification of occupational and environmental disease; ILO=International Labor Organization; LDCT=low-density computer tomography; WHO=World Health Organization.]

Item	Helsinki criteria 1997	Update 2014
General considerations	Guidelines for identifying asbestos-exposed persons with structured interview and fibers from tissue and BAL specimen given. Guidelines for the diagnostics of asbestosis, pleural disorders, mesothelioma and lung cancer given.	Update concentrates on: • screening for asbestos-related lung cancer; • follow-up of asbestos-exposed workers and diagnosis of non-malignant asbestos diseases; • new asbestos-related disease entities; and • pathology and biomarkers.
Asbestos-related non-malignant diseases	Roggli-Pratt modification of the CAP-NIOSH classification of asbestosis recommended. Radiology: small opacities with ILO grade 1/0 in radiographs regarded as early stage asbestosis, HRCT in selected cases. Development of standardized reporting of HRCT scans recommended.	New histology classification of asbestosis (16) is adapted. Criteria for the use of CT imaging in the diagnostics of asbestos related diseases presented. Recommendation to use the international ICOERD CT classification in international studies. Retroperitoneal fibrosis described as a new entity due to asbestos exposure (under certain conditions).
Asbestos-related malignant diseases		
Lung cancer	Four types of lung cancer associated with asbestos exposure defined. Cumulative exposure of 25 fiber-years increases the lung cancer risk 2-fold. Risk estimates also related to tissue fiber levels and asbestos bodies in BAL fluid.	The current classification (WHO 1999) includes two additional types of lung cancer (sarcomatoid and adenosquamous). These are included as types of lung malignancies that may occur as a consequence of asbestos exposure.
Mesothelioma	Histopathological diagnosis discussed.	Additional recommendations for histopathological diagnosis given for epithelioid and sarcomatoid mesotheliomas, separate recommendations for peritoneal mesotheliomas.
Other malignancies	Discussed as research needs	Laryngeal and ovarian cancers are included as cancers that may occur as a consequence of asbestos exposure. Guidelines for attribution given.
Surveillance and screening	Possibilities for primary and secondary prevention (screening) discussed. Scientific studies on screening recommended. Technical requirements for HRCT described (Helsinki conference in 2000). Several research topics suggested.	Medicolegal surveillance (including spirometry) recommended according to the national regulation stratified according to the intensity, latency, and duration of exposure. Vaccination against influenza and pneumococcus recommended for asbestosis patients. LDCT screening recommended for asbestos-exposed workers under certain circumstances (see text for details). The importance of obtaining standardized data in an international setting is stressed.

abnormalities. A combination of three chromosome abnormalities (2p16, 9p33.1, and 19p13) gave a clear dose–response between pulmonary fibre count and either allelic imbalance or copy number alteration or both in at least two of the regions, with a very high specificity when the three regions were combined (24). Additional international multicenter studies with standardized methodology for molecular assays and exposure assessment are necessary before these biomarkers can be applied to support causal attribution in individual cases.

Epilogue

The updating process was carried out in association with the International Conference of Monitoring and Surveillance of Asbestos-Related Diseases, 10–13 February, Espoo, Finland. The conference was organized by the FIOH with ICOH as a co-organizer. The updates are based on the preparatory work of the four pre-meeting work groups. Their work will be published as reviews and is available on the FIOH website (4, 12, 17, 20, 25). In addition, the conference also made a declaration emphasizing the importance of primary prevention, regulation, health surveillance and diagnosis, and registration as well as research and collaboration on asbestos exposure and disease (26).

Much of the original criteria (2) remain unchanged although the Helsinki Criteria Update (HCU) 2014 group made some additions in specific issues, these can be seen in table 1 which summarizes the main points of the 1997 Helsinki criteria and its 2014 updates.

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**Present at update meeting in Helsinki 10–13 February 2014.*

Acknowledgment

At the beginning of the meeting for updating the criteria, declarations of interest information were requested and obtained from all the participants. Most had nothing to declare with the following exceptions: Murray Finkelstein has provided expert opinion as a consultant in American asbestos litigation; Thomas Kraus has provided expert opinion in medico-legal cases involving asbestos-related diseases; Steven Markowitz has provided expert opinion in medico-legal cases involving asbestos-related diseases and LDCT scanning for lung cancer screening; Victor L Roggli has had his travel expenses paid to attend and present at Defense Research Institute and Perrin asbestos conferences, and he has consulted as an expert witness for plaintiff and defense firms in asbestos litigation; Leslie Stayner has provided expert opinion as a consultant in medico-legal cases of workers who have developed mesothelioma from occupational exposure to asbestos; Kurt Straif is a member of the IARC Asbest Study team. The study is funded by the Ministry of Health of the Russian Federation, approved by the IARC Ethics Committee and monitored by an independent Scientific Advisory Board; Axel Wannag has worked as an occupational physician for the Norwegian asbestos cement industry in the past (closed 1981).

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EXHIBIT 17

A GUIDE FOR SHIP SCRAPPERS: TIPS FOR REGULATORY COMPLIANCE

SUMMER 2000

Prepared for:

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Federal Facilities Enforcement Office
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NOTICE

This document provides guidance to assist regulated entities to understand their obligations under environmental laws; however, for a complete understanding of all legal requirements, you must refer to applicable federal and state statutes and regulations. This guide is a compliance assistance tool only, and it neither changes nor replaces any applicable legal requirements, nor does it create any rights or benefits for anyone. This guide also describes in a summary fashion the roles and activities of federal agencies; however, the guidance does not limit their otherwise lawful prerogatives, and the agencies may act at variance with it, based on specific circumstances. This guidance may be revised without prior notice. Mention of trade names or commercial products in this document, or in associated references, does not constitute an endorsement or recommendation for use.

RECOMMENDATIONS FOR USING THE GUIDE

We recommend that users organize this guide in 3-ring binders. Each separate stand-alone section can then be removed from the binder, copied, and easily posted or handed out to workers undertaking specific ship scrapping operations. Each section can also be used in training workers about the best practices for specific ship scrapping operations. Additionally, Appendix C, which is a series of summaries of inspector highlights, can be used to review important regulatory requirements for each process. Users may want to laminate copies of the summaries for each worker or to post the summaries near the job site as reminders of regulations and best practices. It would be helpful to have someone translate the information if your workers are more familiar with a language other than English.

SURVEY REQUEST !!!!!!!!!!!!

You are invited to share your opinions and thoughts about this document. Please complete the survey questionnaire–A Guide for Ship Scrappers Survey. It is located on the U.S. EPA Web Site at: http://www.epa.gov/oeca/fedfac/fflex.html.

PREFACE

This guide is intended to provide site supervisors at ship scrapping facilities with an overview of the most pertinent environmental and worker health and safety requirements to assist them in ensuring compliance at their facilities. The guide is structured by specific processes (e.g., asbestos removal, metal cutting, fuel and oil removal) that occur in ship scrapping operations. Taking a process-specific approach allows the guide to be a more manageable and useful reference tool for key ship scrapping facility personnel. Ship scrappers can review key environmental, safety, and health requirements for each process. References of where to find the requirements in the *Code of Federal Regulations* have been provided throughout the guide, and readers are encouraged to review these regulations in detail. Where possible, helpful shadow and check boxes have been provided to emphasize guidance or tips.

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2. ASBESTOS REMOVAL AND DISPOSAL

During ship scrapping activities, the removal and disposal of asbestos is a primary environmental concern, as well as a health and safety concern for your workers. The following sections present background information on asbestos, discuss the effects of asbestos exposure, and describe some of the regulatory requirements with which your facility must comply.

2.1 INFORMATION ABOUT ASBESTOS

This section provides background information on asbestos, including what it is, where it can be found on ships, how exposure can occur, and the dangers of exposure.

What is asbestos?

"Asbestos" refers to a group of minerals that occur naturally as masses of long silky fibers. There are three main types of asbestos fibers:

- Chrysotile fibers (white asbestos) are fine, silky flexible white fibers. They are pliable and cylindrical, and arranged in bundles. This was the most commonly used asbestos in the United States.
- Amosite fibers (brown asbestos) are straight, brittle fibers that are light grey to pale brown. This was the most commonly used asbestos in thermal system insulation.
- Crocidolite fibers (blue asbestos) are straight blue fibers that are like tiny needles.

There are three other types of asbestos fibers: anthopylite, tremolite, and actinolite. Unlike most minerals, which turn into dust particles when crushed,

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asbestos breaks up into fine fibers that are too small to be seen by the human eye.

Individual asbestos fibers are often mixed with a material that binds them together, forming what is commonly called asbestos-containing material (ACM). There are two kinds of ACM: friable and non-friable.

- **Friable ACM** is any material containing more than 1% asbestos that, when dry, may be crumbled, pulverized, or reduced to powder by hand pressure.

- **Non-friable ACM** is any material containing more than 1% asbestos that, when dry, cannot be crumbled, pulverized, or reduced to powder by hand pressure. Non-friable ACM is divided into two categories.

  - **Category I** non-friable ACM includes asbestos-containing resilient floor coverings, packings, and gaskets.

  - **Category II** non-friable ACM includes all other non-friable ACM that is not included in Category I.

What is presumed asbestos containing material (PACM)? Thermal system insulation and surfacing material found in buildings, vessels, and vessel sections constructed no later than 1980 may be considered PACM.

Why has asbestos been so widely used?

Asbestos was widely used in construction and industry due to its unique properties, and because there were few other available substances that combined the same qualities. Asbestos is resistant to abrasion and corrosion, inert to acid and alkaline solutions, and stable at high temperatures. It is strong yet flexible, non-combustible, conducts electricity poorly, and is an effective thermal insulator.

Where is asbestos found on a ship?

Asbestos is found on ships in many types of materials, including, but not limited to:

- Bulkhead and pipe thermal insulation
- Bulkhead fire shields/fireproofing
- Uptake space insulation
- Exhaust duct insulation
- Electrical cable materials
- Brake linings
- Floor tiles and deck underlay
- Steam, water, and vent flange gaskets
- Adhesives and adhesive-like glues (e.g., mastics) and fillers
- Sound damping
- Molded plastic products (e.g., switch handles, clutch facings)
- Sealing putty
- Packing in shafts and valves
- Packing in electrical bulkhead penetrations
- Asbestos arc chutes in circuit breakers
- Pipe hanger inserts
- Weld shop protectors and burn covers, blankets, and any fire fighting clothing or equipment
- Any other type of thermal insulating material

Caution!! ACM may be found <u>underneath</u> materials that do not contain asbestos.

Status of the Asbestos Ban

There is a rather common misunderstanding about the status of the EPA 1989 ban on asbestos-containing products or uses. Two years after EPA's ban, the U.S. Fifth Circuit Court of Appeals vacated much of EPA's rule in 1991 leaving only six asbestos-containing product categories (including corrugated paper, rollboard, commercial paper, specialty paper, flooring felt, and new uses of asbestos) still subject to the asbestos ban, In addition, several uses of ACM products remained banned, including the sprayed-on application of ACM (>1% asbestos) and the installation of certain types of asbestos-containing insulation. **Besides the products and uses listed above, EPA has no existing bans on other asbestos containing products or uses.** EPA does not track the manufacture, processing or distribution in commerce of asbestos-containing products. For further information, contact the TSCA Assistance Information Service at 202-554-1404, call your EPA Regional Asbestos Coordinator (see *Section 9. Resources*), or access http://www.epa.gov/asbestos and go to the "Helpful Information" button.

What are the four classes of asbestos work?

The Occupational Safety and Health Administration (OSHA) standard for asbestos specifies four classes of asbestos activities [29 CFR 1915.1001(b)]. These are:

- "Class I" asbestos work means activities involving the removal of thermal system insulation (TSI) and sprayed-on or troweled-on or otherwise applied surfacing ACM or PACM.
- "Class II" asbestos work means activities involving the removal of ACM which is neither TSI or surfacing ACM. This includes, but is not limited to, the removal of asbestos-containing wallboard, floor tile, and construction mastics.
- "Class III" asbestos work means repair and maintenance operations where ACM (including TSI and surfacing ACM and PACM) is likely to be disturbed.
- "Class IV" asbestos work means maintenance and custodial activities during which employees contact, but so as not disturb ACM or PACM, and activities to clean up dust, waste, and debris resulting from Class I, II, and III activities.

How can exposure to asbestos occur?

As a site supervisor, you should be aware that you and your workers can be exposed to asbestos in several ways. When ACM is deteriorated, crushed, or otherwise disturbed,

asbestos fibers break up into very fine fibers and are released to the environment by either dispersing in the air, floating on water or accumulating on the ground. Exposure to asbestos can occur by:

- **Occupational exposure**: Workers may be exposed to asbestos if working at facilities, including ships, which contain asbestos. Because asbestos fibers are small and light, they can be suspended in the air for long periods and possibly inhaled by those working in these areas. Airborne asbestos fibers are small, odorless, and tasteless. They range in size from 0.1 to 10 microns in length (a human hair is about 50 microns in diameter). The amount of asbestos a worker is exposed to will vary according to: (1) the concentration of fibers in the air; (2) duration of exposure; (3) the worker's breathing rate (workers doing manual labor breath faster); (4) weather conditions; and (5) the protective devices the worker wears. It is estimated that between 1940 and 1980, 27 million Americans had significant occupational exposure to asbestos. People may also ingest asbestos if they eat in areas where there are asbestos fibers in the air.

  During ship scrapping, the most significant asbestos concerns for workers arise when removing asbestos-bearing thermal insulation; handling of circuit breakers, cable, cable penetrations; and removing floor tiles (from asbestos in the mastic and in the tile). Additional concerns can arise from handling and removing gaskets with piping and electrical systems, as well as molded plastic parts.

- **Paraoccupational exposure**: Workers' families may inhale asbestos fibers released by their clothes that have been in contact with ACM.

- **Neighborhood exposure:** People who live or work near asbestos- related operations may inhale asbestos fibers that have been released into the air by these operations.

What are the effects of exposure to asbestos?

While scientists have not been able to determine a "safe" or threshold level for exposure to airborne asbestos, EPA, OSHA, and the National Institute for Occupational Safety and Health (NIOSH) believe there is no known safe level of asbestos exposure.

Preventing exposure. Using controls to prevent asbestos exposure is vital to protecting the health of workers.

In short, some people exposed to asbestos develop asbestos-related health problems; some do not. Some known diseases caused from asbestos exposure include: (1) asbestosis (scarring of

the lungs resulting in loss of lung function that often progresses to disability and to death), and (2) cancer, such as mesothelioma (cancer affecting the membranes lining the lungs and abdomen), lung cancer, or cancers of the esophagus, stomach, colon, and rectum.

If inhaled, asbestos fibers can easily penetrate body tissues, and may be deposited and retained in the airways and lung tissue. Because asbestos fibers remain in the body, each exposure increases the likelihood of developing an asbestos-related disease. Asbestos-related diseases may not appear until years after exposure. Ingesting asbestos may be harmful, but the consequences of this type of exposure have not been clearly documented. Note: The risks of asbestos exposure are multiplied 10-fold or more if a worker smokes.

2.2 WHO REGULATES ASBESTOS REMOVAL AND DISPOSAL?

Asbestos regulations are important to the ship scrapping industry because many ships being scrapped contain significant amounts of ACM. During ship scrapping activities, ACM must be properly removed and disposed of. Therefore, being aware of and complying with all applicable regulations for asbestos removal and disposal is important for your ship scrapping operation. The process of removing and disposing of ACM is subject to various federal, state, and local environmental and safety and health requirements.

- **EPA.** EPA is responsible for developing and enforcing regulations necessary to protect human health and the environment. Asbestos is regulated by EPA under two laws: (1) the Clean Air Act (CAA), under the Asbestos National Emissions Standards for Hazardous Air Pollutants (NESHAP), and (2) the Toxic Substances Control Act (TSCA). Some of the requirements for asbestos removal and disposal under these laws include inspections; notifications; supervisor training; and the proper removal, transport and disposal of asbestos.

  Specifically, the Asbestos NESHAP [40 CFR 61 Subpart M] is intended to minimize the release of asbestos fibers during demolition and renovation activities (including ship scrapping) through work practices. EPA has delegated authority to inspect and enforce the asbestos NESHAP regulations to most states. Where the program has been delegated, the state agency may have requirements that are more stringent than the federal requirements. The asbestos NESHAP requirements will be discussed in more detail in the following sections.

- **OSHA.** OSHA is responsible for the health and safety of workers who may be exposed to asbestos in the work place. OSHA regulations covering asbestos exposure set a maximum exposure limit and include provisions for engineering controls and

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Asbestos

What is asbestos?

Asbestos is the name given to a group of naturally occurring minerals that are resistant to heat and corrosion. Asbestos has been used in products, such as insulation for pipes (steam lines for example), floor tiles, building materials, and in vehicle brakes and clutches. Asbestos includes the mineral fibers chrysotile, amosite, crocidolite, tremolite, anthophyllite, actinolite and any of these materials that have been chemically treated or altered. Heavy exposures tend to occur in the construction industry and in ship repair, particularly during the removal of asbestos materials due to renovation, repairs, or demolition. Workers are also likely to be exposed during the manufacture of asbestos products (such as textiles, friction products, insulation, and other building materials) and during automotive brake and clutch repair work.

What are the hazards of asbestos?

Asbestos is well recognized as a health hazard and its use is now highly regulated by both OSHA and EPA. Asbestos fibers associated with these health risks are too small to be seen with the naked eye. Breathing asbestos fibers can cause a buildup of scar-like tissue in the lungs called asbestosis and result in loss of lung function that often progresses to disability and death. Asbestos also causes cancer of the lung and other diseases such as mesothelioma of the pleura which is a fatal malignant tumor of the membrane lining the cavity of the lung or stomach. Epidemiologic evidence has increasingly shown that all asbestos fiber types, including the most commonly used form of asbestos, chrysotile, causes mesothelioma in humans.[1,2,3]

What can be done to reduce the hazards of asbestos?

Worker exposure to asbestos hazards are addressed in specific OSHA standards for the construction industry, general industry and shipyard employment sectors. These standards reduce the risk to workers by requiring that employers provide personal exposure monitoring to assess the risk and hazard awareness training for operations where there is any potential exposure to asbestos. Airborne levels of asbestos are never to exceed legal worker exposure limits. There is no "safe" level of asbestos exposure for any type of asbestos fiber.[4,5] Asbestos exposures as short in duration as a few days have caused mesothelioma in humans.[4,5,6,7] Every occupational exposure to asbestos can cause injury of disease; every occupational exposure to asbestos contributes to the risk of getting an asbestos related disease.[8] Where there is exposure, employers are required to further protect workers by establishing regulated areas, controlling certain work practices and instituting engineering controls to reduce the airborne levels. The employer is required to ensure exposure is reduced by using administrative controls and provide for the wearing of personal protective equipment. Medical monitoring of workers is also required when legal limits and exposure times are exceeded.

How can OSHA Help?

OSHA has developed this webpage to provide workers and employers useful, up-to-date information on asbestos. For other valuable worker protection information, such as Workers' Rights, Employer Responsibilities and other services OSHA offers, read OSHA's Workers page.

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- Hazards
- Evaluating and Controlling Exposure
- Construction
- OSHA Standards
- Other Resources
- Page History

Page last reviewed: 03/28/2013

Highlights

- Self-Inspection Checklist. OSHA. Use this checklist to determine compliance to the asbestos standard.

Other Related Topics

- Carcinogens
- Hazardous and Toxic Substances
- Hazardous Waste

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EXHIBIT 19

WORLD HEALTH ORGANIZATION

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER

IARC Monographs on the Evaluation of Carcinogenic Risks to Humans

Volume 14
Asbestos

Summary of Data Reported and Evaluation

Asbestos

Last updated: 26 March 1998

ASBESTOS

VOL.: 14 (1977)

5. Summary of Data Reported and Evaluation

5.1 Animal data

All commercial forms of asbestos tested are carcinogenic in mice, rats, hamsters and rabbits. In mice, mesotheliomas were induced following intraperitoneal injection of asbestos fibres. In rats, various types of asbestos produced lung carcinomas and mesotheliomas following their inhalation and mesotheliomas and sarcomas following their intrapleural or intraperitoneal injection. The oral administration of asbestos filter material to rats also resulted in an increased incidence of tumours. Mesotheliomas were produced in hamsters and rabbits after intrapleural injection of a number of types of asbestos fibre.

The size and shape of the fibres influence the incidence of tumours; fibres less than 0.5 μm in diameter are more active in producing tumours. Glass fibres and nemalite of a similar size can also produce mesotheliomas following their intrapleural or intraperitoneal injection in rats.

5.2 Human data

In humans, occupational exposure to chrysotile, amosite, anthophyllite and mixed fibres containing crocidolite has resulted in a high incidence of lung cancer; a predominantly tremolitic material mixed with anthophyllite and small amounts of chrysotile has also caused an increased incidence of lung cancer. Many pleural and peritoneal mesotheliomas have been observed after occupational exposure to crocidolite, amosite and chrysotile. An excess risk of gastrointestinal tract cancers has been demonstrated in groups exposed occupationally to amosite, chrysotile or mixed fibres containing crocidolite. An excess of cancers of the larynx was also observed in exposed workers. Mesotheliomas also occur in individuals living in the neighbourhood of asbestos factories and crocidolite mines and in household contacts of asbestos workers.

Occupational exposure to asbestos may occur during the mining of fibrous minerals, as well as of minerals embodied in rocks, which may contain asbestiform fibres as a contaminant.

Both cigarette smoking and occupational exposure to asbestos fibres independently increase lung cancer incidence, but when they are present together they act in a multiplicative fashion.

The general population may also be exposed to asbestos fibres in air, beverages, drinking-water, food and pharmaceutical and dental preparations and by consumer use of asbestos-containing products. The presence of asbestos and asbestiform minerals from natural sources in the environment, other than mines or quarries, has only recently shown itself to be a further potential problem.

At present, it is not possible to assess whether there is a level of exposure in humans below which an increased risk of cancer would not occur.

Previous evaluation: Vol. 2 (1973)

Subsequent evaluation: Suppl. 7 (1987)

Last updated: 26 March 1998

EXHIBIT 20

NATIONAL CANCER INSTITUTE
FactSheet

Reviewed: 05/13/2002

Mesothelioma: Questions and Answers

Mesothelioma is a rare form of cancer in which malignant (cancerous) cells are found in the mesothelium, a protective sac that covers most of the body's internal organs. Most people who develop mesothelioma have worked on jobs where they inhaled asbestos particles.

1. What is the mesothelium?

   The mesothelium is a membrane that covers and protects most of the internal organs of the body. It is composed of two layers of cells: One layer immediately surrounds the organ; the other forms a sac around it. The mesothelium produces a lubricating fluid that is released between these layers, allowing moving organs (such as the beating heart and the expanding and contracting lungs) to glide easily against adjacent structures.

   The mesothelium has different names, depending on its location in the body. The peritoneum is the mesothelial tissue that covers most of the organs in the abdominal cavity. The pleura is the membrane that surrounds the lungs and lines the wall of the chest cavity. The pericardium covers and protects the heart. The mesothelial tissue surrounding the male internal reproductive organs is called the tunica vaginalis testis. The tunica serosa uteri covers the internal reproductive organs in women.

2. What is mesothelioma?

   Mesothelioma (cancer of the mesothelium) is a disease in which cells of the mesothelium become abnormal and divide without control or order. They can invade and damage nearby tissues and organs. Cancer cells can also metastasize (spread) from their original site to other parts of the body. Most cases of mesothelioma begin in the pleura or peritoneum.

3. How common is mesothelioma?

   Although reported incidence rates have increased in the past 20 years, mesothelioma is still a relatively rare cancer. About 2,000 new cases of mesothelioma are diagnosed in the United States each year. Mesothelioma occurs more often in men than in women and risk increases with age, but this disease can appear in either men or women at any age.

4. What are the risk factors for mesothelioma?

   Working with asbestos is the major risk factor for mesothelioma. A history of asbestos exposure at work is reported in about 70 percent to 80 percent of all cases. However, mesothelioma has been reported in some individuals without any known exposure to asbestos.

   Asbestos is the name of a group of minerals that occur naturally as masses of strong, flexible fibers that can be separated into thin threads and woven. Asbestos has been widely used in many industrial products, including cement, brake linings, roof shingles, flooring products, textiles, and insulation. If tiny asbestos particles float in the air, especially during the manufacturing process, they may be inhaled or swallowed, and can cause serious health problems. In addition to mesothelioma, exposure to asbestos increases the risk of lung cancer, asbestosis (a noncancerous, chronic lung ailment), and other cancers, such as those of the larynx and kidney.

   Smoking does not appear to increase the risk of mesothelioma. However, the combination of smoking and asbestos exposure significantly increases a person's risk of developing cancer of the air passageways in the lung.

5. Who is at increased risk for developing mesothelioma?

   Asbestos has been mined and used commercially since the late 1800s. Its use greatly increased

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during World War II. Since the early 1940s, millions of American workers have been exposed to asbestos dust. Initially, the risks associated with asbestos exposure were not known. However, an increased risk of developing mesothelioma was later found among shipyard workers, people who work in asbestos mines and mills, producers of asbestos products, workers in the heating and construction industries, and other tradespeople. Today, the U.S. Occupational Safety and Health Administration (OSHA) sets limits for acceptable levels of asbestos exposure in the workplace. People who work with asbestos wear personal protective equipment to lower their risk of exposure.

The risk of asbestos-related disease increases with heavier exposure to asbestos and longer exposure time. However, some individuals with only brief exposures have developed mesothelioma. On the other hand, not all workers who are heavily exposed develop asbestos-related diseases.

There is some evidence that family members and others living with asbestos workers have an increased risk of developing mesothelioma, and possibly other asbestos-related diseases. This risk may be the result of exposure to asbestos dust brought home on the clothing and hair of asbestos workers. To reduce the chance of exposing family members to asbestos fibers, asbestos workers are usually required to shower and change their clothing before leaving the workplace.

6. What are the symptoms of mesothelioma?

Symptoms of mesothelioma may not appear until 30 to 50 years after exposure to asbestos. Shortness of breath and pain in the chest due to an accumulation of fluid in the pleura are often symptoms of pleural mesothelioma. Symptoms of peritoneal mesothelioma include weight loss and abdominal pain and swelling due to a buildup of fluid in the abdomen. Other symptoms of peritoneal mesothelioma may include bowel obstruction, blood clotting abnormalities, anemia, and fever. If the cancer has spread beyond the mesothelium to other parts of the body, symptoms may include pain, trouble swallowing, or swelling of the neck or face.

These symptoms may be caused by mesothelioma or by other, less serious conditions. It is important to see a doctor about any of these symptoms. Only a doctor can make a diagnosis.

7. How is mesothelioma diagnosed?

Diagnosing mesothelioma is often difficult, because the symptoms are similar to those of a number of other conditions. Diagnosis begins with a review of the patient's medical history, including any history of asbestos exposure. A complete physical examination may be performed, including x-rays of the chest or abdomen and lung function tests. A CT (or CAT) scan or an MRI may also be useful. A CT scan is a series of detailed pictures of areas inside the body created by a computer linked to an x-ray machine. In an MRI, a powerful magnet linked to a computer is used to make detailed pictures of areas inside the body. These pictures are viewed on a monitor and can also be printed.

A biopsy is needed to confirm a diagnosis of mesothelioma. In a biopsy, a surgeon or a medical oncologist (a doctor who specializes in diagnosing and treating cancer) removes a sample of tissue for examination under a microscope by a pathologist. A biopsy may be done in different ways, depending on where the abnormal area is located. If the cancer is in the chest, the doctor may perform a thoracoscopy. In this procedure, the doctor makes a small cut through the chest wall and puts a thin, lighted tube called a thoracoscope into the chest between two ribs. Thoracoscopy allows the doctor to look inside the chest and obtain tissue samples. If the cancer is in the abdomen, the doctor may perform a peritoneoscopy. To obtain tissue for examination, the doctor makes a small opening in the abdomen and inserts a special instrument called a peritoneoscope into the abdominal cavity. If these procedures do not yield enough tissue, more extensive diagnostic surgery may be necessary.

If the diagnosis is mesothelioma, the doctor will want to learn the stage (or extent) of the disease. Staging involves more tests in a careful attempt to find out whether the cancer has spread and, if so, to which parts of the body. Knowing the stage of the disease helps the doctor plan treatment.

Mesothelioma is described as localized if the cancer is found only on the membrane surface where it originated. It is classified as advanced if it has spread beyond the original membrane surface to other parts of the body, such as the lymph nodes, lungs, chest wall, or abdominal organs.

8. How is mesothelioma treated?

Treatment for mesothelioma depends on the location of the cancer, the stage of the disease, and the patient's age and general health. Standard treatment options include surgery, radiation therapy, and chemotherapy. Sometimes, these treatments are combined.

- **Surgery** is a common treatment for mesothelioma. The doctor may remove part of the lining of the chest or abdomen and some of the tissue around it. For cancer of the pleura (pleural mesothelioma), a lung may be removed in an operation called a pneumonectomy. Sometimes part of the diaphragm, the muscle below the lungs that helps with breathing, is also removed.

- **Radiation therapy**, also called radiotherapy, involves the use of high-energy rays to kill cancer cells and shrink tumors. Radiation therapy affects the cancer cells only in the treated area. The radiation may come from a machine (external radiation) or from putting materials that produce radiation through thin plastic tubes into the area where the cancer cells are found (internal radiation therapy).

- **Chemotherapy** is the use of anticancer drugs to kill cancer cells throughout the body. Most drugs used to treat mesothelioma are given by injection into a vein (intravenous, or IV). Doctors are also studying the effectiveness of putting chemotherapy directly into the chest or abdomen (intracavitary chemotherapy).

To relieve symptoms and control pain, the doctor may use a needle or a thin tube to drain fluid that has built up in the chest or abdomen. The procedure for removing fluid from the chest is called thoracentesis. Removal of fluid from the abdomen is called paracentesis. Drugs may be given through a tube in the chest to prevent more fluid from accumulating. Radiation therapy and surgery may also be helpful in relieving symptoms.

9. Are new treatments for mesothelioma being studied?

Yes. Because mesothelioma is very hard to control, the National Cancer Institute (NCI) is sponsoring clinical trials (research studies with people) that are designed to find new treatments and better ways to use current treatments. Before any new treatment can be recommended for general use, doctors conduct clinical trials to find out whether the treatment is safe for patients and effective against the disease. Participation in clinical trials is an important treatment option for many patients with mesothelioma.

People interested in taking part in a clinical trial should talk with their doctor. Information about clinical trials is available from the Cancer Information Service (CIS) (see below) at 1–800–4–CANCER. Information specialists at the CIS use PDQ®, NCI's cancer information database, to identify and provide detailed information about specific ongoing clinical trials. Patients also have the option of searching for clinical trials on their own. The clinical trials page on the NCI's Cancer.gov Web site, located at http://www.cancer.gov/clinicaltrials on the Internet, provides general information about clinical trials and links to PDQ.

People considering clinical trials may be interested in the NCI booklet *Taking Part in Cancer Treatment Research Studies*. This booklet describes how research studies are carried out and explains their possible benefits and risks. The booklet is available by calling the CIS, or from the NCI Publications Locator Web site at http://www.cancer.gov/publications on the Internet.

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Related NCI materials and Web pages:

- National Cancer Institute Fact Sheet 3.21, *Asbestos Exposure: Questions and Answers* (http://www.cancer.gov/cancertopics/factsheet/Risk/asbestos)
- *Clinical Trials Home Page* (http://www.cancer.gov/clinicaltrials)
- *Malignant Mesothelioma Home Page* (http://www.cancer.gov/cancertopics/types/malignantmesothelioma)
- *Taking Part in Cancer Treatment Research Studies* (http://www.cancer.gov/clinicaltrials/Taking-Part-in-Cancer-Treatment-Research-Studies)

For more help, contact:

NCI's Cancer Information Service
Telephone (toll-free): 1–800–4–CANCER (1–800–422–6237)
TTY (toll-free): 1–800–332–8615
LiveHelp® online chat: https://cissecure.nci.nih.gov/livehelp/welcome.asp

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EXHIBIT 21

NATIONAL CANCER INSTITUTE FactSheet

Reviewed: 02/01/2007

Asbestos Exposure: Questions and Answers

Key Points

- Asbestos is the name given to a group of minerals that occur naturally in the environment as bundles of fibers (see Question 1).
- Exposure to asbestos may increase the risk of asbestosis, lung cancer, mesothelioma, other cancers, and other nonmalignant lung and pleural disorders (see Question 3).
- Smokers who are also exposed to asbestos have a greatly increased risk of lung cancer (see Question 6).
- Individuals who have been exposed (or suspect they have been exposed) to asbestos fibers on the job, through the environment, or at home via a family contact should inform their physician of their exposure history and any symptoms (see Question 7).
- Government agencies can provide additional information on asbestos exposure (see Questions 8, 9, and 11).

1. What is asbestos?

Asbestos is the name given to a group of minerals that occur naturally in the environment as bundles of fibers and can be separated into thin, durable threads. These fibers are resistant to heat, fire, and chemicals and do not conduct electricity. For these reasons, asbestos has been widely used in many industries.

There are two subgroups of asbestos: chrysotile, which has curly fibers and is in the serpentine family of minerals; and amphibole asbestos, which has straight, needle-like fibers and includes actinolite, tremolite, anthophyllite, crocidolite, and amosite asbestos. Chrysotile asbestos is the form that has been used predominantly in commercial applications worldwide (1, 2).

2. How is asbestos used?

Asbestos was mined and used commercially in North America beginning in the late 1800s. Its use increased greatly during World War II (3, 4). Since then, asbestos has been used in many industries. For example, the building and construction industry has used it for strengthening cement and plastics as well as for insulation, roofing, fireproofing, and sound absorption. The shipbuilding industry has used asbestos to insulate boilers, steampipes, and hot water pipes. The automotive industry uses asbestos in vehicle brakeshoes and clutch pads. Asbestos has also been used in ceiling and floor tile; paints, coatings, and adhesives; and plastics. In addition, asbestos has been found in vermiculite-containing consumer garden products and some talc-containing crayons.

In the late 1970s, the U.S. Consumer Product Safety Commission (CPSC) banned the use of asbestos in wallboard patching compounds and gas fireplaces because the asbestos fibers in these products could be released into the environment during use. Additionally, in 1979, manufacturers of electric hairdryers voluntarily stopped using asbestos in their products. In 1989, the U.S. Environmental Protection Agency (EPA) banned all new uses of asbestos; uses established prior to 1989 are still allowed. The EPA also established regulations that require school systems to inspect for damaged asbestos and to eliminate or reduce the exposure to occupants by removing the asbestos or encasing it (2).

In June 2000, the CPSC concluded that the risk of children's exposure to asbestos fibers in crayons was extremely low (1). However, the U.S. manufacturers of these crayons agreed to eliminate talc from their products. In August 2000, the EPA responded to reports it received about the adverse human health effects associated with exposure to asbestos-contaminated vermiculite by conducting a series of tests to evaluate the extent of the risk. The EPA investigation concluded that the potential exposure to asbestos from some vermiculite products poses only a minimal health risk to consumers. The EPA recommended that consumers reduce the low risk associated with the occasional use of vermiculite during gardening activities by limiting the amount of dust produced during use. Specifically, the EPA suggested that consumers use vermiculite outdoors or in a well-ventilated area; keep vermiculite damp while using it; avoid bringing dust from vermiculite use into the home on clothing; and use premixed

potting soil, which is less likely to generate dust.

The regulations described above and other actions, coupled with widespread public concern about the health hazards of asbestos, have resulted in a significant annual decline in U.S. use of asbestos. Domestic consumption of asbestos amounted to about 803,000 metric tons in 1973, but it had dropped to about 2,400 metric tons by 2005 (3, 5).

3. What are the health hazards of exposure to asbestos?

People may be exposed to asbestos in their workplace, their communities, or their homes. If products containing asbestos are disturbed, tiny asbestos fibers are released into the air. When asbestos fibers are breathed in, they may get trapped in the lungs and remain there for a long time. Over time, these fibers can accumulate and cause scarring and inflammation, which can affect breathing and lead to serious health problems (6).

Asbestos has been classified as a known human carcinogen (a substance that causes cancer) by the U.S. Department of Health and Human Services, the EPA, and the International Agency for Research on Cancer (2, 3, 7, 8). Studies have shown that exposure to asbestos may increase the risk of lung cancer and mesothelioma (a relatively rare cancer of the thin membranes that line the chest and abdomen). Although rare, mesothelioma is the most common form of cancer associated with asbestos exposure. In addition to lung cancer and mesothelioma, some studies have suggested an association between asbestos exposure and gastrointestinal and colorectal cancers, as well as an elevated risk for cancers of the throat, kidney, esophagus, and gallbladder (3, 4). However, the evidence is inconclusive.

Asbestos exposure may also increase the risk of asbestosis (a chronic lung disease that can cause shortness of breath, coughing, and permanent lung damage) and other nonmalignant lung and pleural disorders, including pleural plaques (changes in the membrane surrounding the lung), pleural thickening, and pleural effusions (abnormal collections of fluid between the thin layers of tissue lining the lung and the wall of the chest cavity). Although pleural plaques are not precursors to lung cancer, evidence suggests that people with pleural disease caused by asbestos exposure may be at increased risk for lung cancer (9).

4. Who is at risk for an asbestos-related disease?

Everyone is exposed to asbestos at some time during their life. Low levels of asbestos are present in the air, water, and soil. However, most people do not become ill from their exposure. People who become ill from asbestos are usually those who are exposed to it on a regular basis, most often in a job where they work directly with the material or through substantial environmental contact.

Since the early 1940s, millions of American workers have been exposed to asbestos. Health hazards from asbestos fibers have been recognized in workers exposed in shipbuilding trades, asbestos mining and milling, manufacturing of asbestos textiles and other asbestos products, insulation work in the construction and building trades, and a variety of other trades. Demolition workers, drywall removers, asbestos removal workers, firefighters, and automobile workers also may be exposed to asbestos fibers. However, recent studies do not support an increased risk of lung cancer or mesothelioma among automobile mechanics exposed to asbestos through brake repair (10). As a result of Government regulations and improved work practices, today's workers (those without previous exposure) are likely to face smaller risks than did those exposed in the past.

Those involved in the rescue, recovery, and cleanup at the site of the September 11, 2001, attacks on the World Trade Center (WTC) in New York City are another group at risk of developing an asbestos-related disease. Because asbestos was used in the construction of the North Tower of the WTC, when the building was attacked, hundreds of tons of asbestos were released into the atmosphere. Those at greatest risk include firefighters, police officers, paramedics, construction workers, and volunteers who worked in the rubble at Ground Zero. Others at risk include residents in close proximity to the WTC towers and those who attended schools nearby. These populations will need to be followed to determine the long-term health consequences of their exposure (11).

One study found that nearly 70 percent of WTC rescue and recovery workers suffered new or worsened respiratory symptoms while performing work at the WTC site. The study describes the results of the WTC Worker and Volunteer Medical Screening Program, which was established to identify and characterize possible WTC-related health effects in responders. The study found that about 28 percent of those tested had abnormal lung function tests, and 61 percent of those without previous health problems developed respiratory symptoms (12). However, it is important to note that these symptoms may be related to exposure to debris components other than asbestos.

Although it is clear that health risks from asbestos exposure increase with heavier exposure and longer exposure time, investigators have found asbestos-related diseases in individuals with only brief exposures. Generally, those who develop asbestos-related diseases show no signs of illness for a long time after their first exposure. It can take from 10 to 40 years or more for symptoms of an asbestos-related condition to appear (2).

There is some evidence that family members of workers heavily exposed to asbestos face an increased risk of developing mesothelioma. This risk is thought to result from exposure to asbestos fibers brought into the home on the shoes, clothing, skin, and hair of workers. To decrease these exposures, Federal

law regulates work practices to limit the possibility of asbestos being brought home in this way. Some employees may be required to shower and change their clothes before they leave work, store their street clothes in a separate area of the workplace, or wash their work clothes at home separately from other clothes (2).

Cases of mesothelioma have also been seen in individuals without occupational exposure, but who live close to asbestos mines or have been exposed to fibers carried home by family members working with asbestos (6).

5. What factors affect the risk of developing an asbestos-related disease?

Several factors can help to determine how asbestos exposure affects an individual, including (2, 6):

- Dose (how much asbestos an individual was exposed to).
- Duration (how long an individual was exposed).
- Size, shape, and chemical makeup of asbestos fibers.
- Source of exposure.
- Individual risk factors, such as smoking and pre-existing lung disease.

Although all forms of asbestos are considered hazardous, different types of asbestos fibers may be associated with different health risks. For example, results of several studies suggest that amphibole forms of asbestos may be more harmful than chrysotile, particularly for mesothelioma risk, because they tend to stay in the lungs for a longer period of time (1, 2). All common commercial types of asbestos have been associated with lung cancer (13).

6. How does smoking affect risk?

Many studies have shown that the combination of smoking and asbestos exposure is particularly hazardous. Smokers who are also exposed to asbestos have a risk of developing lung cancer that is greater than the individual risks from asbestos and smoking added together (3, 6). There is evidence that quitting smoking will reduce the risk of lung cancer among asbestos-exposed workers (4). Smoking combined with asbestos exposure does not appear to increase the risk of mesothelioma (9). However, people who were exposed to asbestos on the job at any time during their life or who suspect they may have been exposed should not smoke.

7. How are asbestos-related diseases detected?

Individuals who have been exposed (or suspect they have been exposed) to asbestos fibers on the job, through the environment, or at home via a family contact should inform their doctor of their exposure history, whether or not they experience any symptoms. The symptoms of asbestos-related diseases may not become apparent for many decades after exposure. It is particularly important to check with a doctor if any of the following symptoms develop (6):

- Shortness of breath, wheezing, or hoarseness.
- A persistent cough that gets worse over time.
- Blood in the sputum (fluid) coughed up from the lungs.
- Pain or tightening in the chest.
- Difficulty swallowing.
- Swelling of the neck or face.
- Loss of appetite.
- Weight loss.
- Fatigue or anemia.

A thorough physical examination, including a chest x-ray and lung function tests, may be recommended. The chest x-ray is currently the most common tool used to detect asbestos-related diseases. However, it is important to note that chest x-rays cannot detect asbestos fibers in the lungs, but they can help identify any early signs of lung disease resulting from asbestos exposure (2).

Studies have shown that computed tomography (CT) (a series of detailed pictures of areas inside the body taken from different angles; the pictures are created by a computer linked to an x-ray machine) may be more effective than conventional chest x-rays at detecting asbestos-related lung abnormalities in individuals who have been exposed to asbestos (14).

A lung biopsy, which detects microscopic asbestos fibers in pieces of lung tissue removed by surgery, is the most reliable test to confirm the presence of asbestos-related abnormalities. A bronchoscopy is a less invasive test than a biopsy and detects asbestos fibers in material that is rinsed out of the lungs. It is important to note that these tests cannot determine how much asbestos an individual may have been exposed to or whether disease will develop (14). Asbestos fibers can also be detected in urine, mucus, or feces, but these tests are not reliable for determining how much asbestos may be in an individual's lungs (2).

8. How can workers protect themselves from asbestos exposure?

The Occupational Safety and Health Administration (OSHA) is a component of the U.S. Department of Labor (DOL) and is the Federal agency responsible for health and safety regulations in maritime, construction, manufacturing, and service workplaces. OSHA established regulations dealing with asbestos exposure on the job, specifically construction work, shipyards, and general industry, that employers are required to follow. In addition, the Mine Safety and Health Administration (MSHA), another component of the DOL, enforces regulations related to mine safety. Workers should use all protective equipment provided by their employers and follow recommended work practices and safety procedures. For example, National Institute for Occupational Safety and Health (NIOSH)-approved respirators that fit properly should be worn by workers when required.

Workers who are concerned about asbestos exposure in the workplace should discuss the situation with other employees, their employee health and safety representative, and their employers. If necessary, OSHA can provide more information or make an inspection. Regional offices of OSHA are listed in the "United States Government" section of a telephone directory's blue pages (under "Department of Labor"). Regional offices can also be found at http://www.osha.gov/html/RAmap.html on the Internet.

More information about asbestos is available on the OSHA Asbestos Web page, which has links to information about asbestos in the workplace, including what OSHA standards apply, the hazards of asbestos, evaluating asbestos exposure, and controls used to protect workers. This page is available at http://www.osha.gov/SLTC/asbestos/index.html on the Internet. OSHA's national office can be contacted at:

Organization:	**Office of Public Affairs** **Occupational Safety and Health Administration** **U.S. Department of Labor**
Address:	Room N-3649 200 Constitution Avenue, NW. Washington, DC 20210
Telephone:	202-693-1999 1-800-321-6742 (1-800-321-OSHA)
TTY (for deaf or hard of hearing callers):	1-877-889-5627
Internet Web site:	http://www.osha.gov/as/opa/worker/index.html (workers' page)

Mine workers can contact MSHA at:

Organization:	**Office of Public Affairs** **Mine Safety and Health Administration** **U.S. Department of Labor**
Address:	21st Floor 1100 Wilson Boulevard Arlington, VA 22209
Telephone:	202-693-9400 1-800-746-1554
Internet Web site:	http://www.msha.gov http://www.msha.gov/codeaphone/codeaphonenew.htm (National Hazard Reporting Page)

The National Institute for Occupational Safety and Health (NIOSH), part of the Centers for Disease Control and Prevention, is another Federal agency that is concerned with asbestos exposure in the workplace. NIOSH conducts asbestos-related research, evaluates work sites for possible health hazards, and makes exposure control recommendations. In addition, NIOSH distributes publications on the health effects of asbestos exposure and can suggest additional sources of information. NIOSH can be contacted at:

Organization:	**Education and Information Division** **Information Resources Branch** **National Institute for Occupational Safety and Health**
Address:	Robert A. Taft Laboratories 4676 Columbia Parkway Cincinnati, OH 45226
Telephone:	1-800-356-4674 (1-800-35-NIOSH)
E-mail:	eidtechinfo@cdc.gov
Internet Web site:	http://www.cdc.gov/niosh

9. What programs are available to help individuals with asbestos-related diseases?

Some people with asbestos-related illness may be eligible for Medicare coverage. Information about benefits is available from Medicare's Regional Offices, located in 10 major cities across the United States and serving specific geographic areas. The Regional Offices serve as the agency's initial point of contact for beneficiaries, health care providers, state and local governments, and the general public. Contact information for each Regional Office can be found at http://www.cms.hhs.gov/RegionalOffices on the Internet. General information about Medicare is available by calling toll-free 1-800-633-4227 (1-800-MEDICARE) or visiting http://www.medicare.gov on the Internet.

People with occupational asbestos-related diseases also may qualify for financial help, including medical payments, under state workers' compensation laws. Because eligibility requirements vary from state to state, workers employed by private companies or by state and local government agencies should contact their state workers' compensation board. Contact information for state workers' compensation officials may be found in the blue pages of a local telephone directory or at http://www.dol.gov/esa/regs/compliance/owcp/wc.htm on the Internet.

If exposure occurred during employment with a Federal agency, medical expenses and other compensation may be covered by the Federal Employees' Compensation Program, which is administered by the DOL, Employment Standards Administration's Office of Workers' Compensation Programs. This program provides workers' compensation benefits to Federal (civilian) employees for employment-related injuries and diseases. Benefits include wage replacement, payment for medical care, and, where necessary, medical and vocational rehabilitation assistance in returning to work. Benefits may also be provided to dependents if the injury or disease causes the employee's death. The program has 12 district offices nationwide.

In addition, the Longshore and Harbor Workers' Compensation Program provides benefits to longshoremen, harbor workers, other maritime workers, and other classes of private industry workers who are injured during the course of employment or suffer from diseases caused or worsened by conditions of employment. Information about eligibility and how to file a claim for benefits under either of these programs is available from:

Organization:	**Office of Workers' Compensation Programs** **Employment Standards Administration** **U.S. Department of Labor**
Address:	Frances Perkins Building 200 Constitution Avenue, NW. Washington, DC 20210
Telephone:	1–866–692–7487 (1–866–OWCPIVR) 202–693–0040 (Federal Employees' Compensation Program) 202–693–0038 (Longshore and Harbor Workers' Compensation Program)
E-mail:	OWCP-Public@dol.gov
Internet Web site:	http://www.dol.gov/esa/owcp_org.htm

Eligible veterans may receive health care at a Department of Veterans Affairs (VA) Medical Center for an asbestos-related disease. Veterans can receive treatment for service-connected and nonservice-connected medical conditions. Information about eligibility and benefits is available from the VA Health Benefits Service Center at 1–877–222–8387 (1–877–222–VETS) or on the VA Web site at http://www1.va.gov/health on the Internet.

10. Is there legislation to help victims of asbestos-related health diseases?

No Federal legislation has been enacted yet to compensate victims of asbestos-related diseases or to protect people from asbestos exposure. However, in 2005, the Fairness in Asbestos Injury Resolution (FAIR) Act (Senate bill 852) was introduced into Congress (15). This bill would create a national trust fund to compensate victims suffering from asbestos-related diseases. The proposed trust fund would be administered by the DOL, outside of the courts, through a claims process whereby all those with certain medical symptoms and evidence of asbestos-related disease would be compensated. Funding for the trust would come from insurance companies and companies that mined, manufactured, and sold asbestos or asbestos products. Under the bill, individuals affected by asbestos exposure would no longer be able to pursue awards for damages in any Federal or state court.

11. What other organizations offer information related to asbestos exposure?

The organizations listed below can provide more information about asbestos exposure.

The **Agency for Toxic Substances and Disease Registry (ATSDR)** is the principal Federal agency responsible for evaluating the human health effects of exposure to hazardous substances. The agency works in close collaboration with local, state, and other Federal agencies, with tribal governments, and with communities and local health care providers to help prevent or reduce harmful human health effects from exposure to hazardous substances. The ATSDR provides information about asbestos and where to find occupational and environmental health clinics. The ATSDR can be contacted at:

Organization:	**Agency for Toxic Substances and Disease Registry**
Address:	1825 Century Boulevard Atlanta, GA 30345
Telephone:	1–800–232–4636 (1–800–CDC–INFO)
TTY:	1-888-232-6348
E-mail:	cdcinfo@cdc.gov
Internet Web site:	http://www.atsdr.cdc.gov

The **U.S. Environmental Protection Agency (EPA)** regulates the general public's exposure to

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asbestos in buildings, drinking water, and the environment. The EPA offers a Toxic Substances Control Act (TSCA) Hotline and an Asbestos Ombudsman. The TSCA Hotline provides technical assistance and information about asbestos programs implemented under the TSCA, which include the Asbestos School Hazard Abatement Act and the Asbestos Hazard Emergency Response Act. The Asbestos Ombudsman focuses on asbestos in schools and handles questions and complaints. Both the TSCA Hotline and the Asbestos Ombudsman can provide publications on a number of topics, particularly on controlling asbestos exposure in schools and other buildings. The Ombudsman operates a toll-free hotline for small businesses, trade associations, and others seeking free, confidential help.

The EPA Web site includes a list of EPA regional and state asbestos contacts at http://www.epa.gov/asbestos/pubs/regioncontact.html on the Internet. In addition, EPA's Asbestos and Vermiculite home page provides information about asbestos and its health effects and links to asbestos resources, including suggestions for homeowners who suspect asbestos in their homes, and laws and regulations applicable to asbestos. This page can be found at http://www.epa.gov/asbestos/ on the Internet. Questions may be directed to:

Organization:	**U.S. Environmental Protection Agency**
	EPA West Building
	National Program Chemicals Division
Address:	Mail Code 7404T
	1200 Pennsylvania Avenue, NW.
	Washington, DC 20460
TSCA Hotline:	202–554–1404
TTY:	202–554–0551
Asbestos Ombudsman:	1–800–368–5888
E-mail:	tsca-hotline@epa.gov
Internet Web site:	http://www.epa.gov/asbestos/

Another EPA resource that may be of interest is the brochure titled *Current Best Practices for Preventing Asbestos Exposure Among Brake and Clutch Repair Workers*. Released in April 2007, this brochure includes work practices for both automotive professionals and home mechanics that may be used to avoid asbestos exposure. It also summarizes existing OSHA regulatory requirements for professional auto mechanics. The brochure supersedes the EPA's existing *Guidance for Preventing Asbestos Disease Among Auto Mechanics*, which is known as the "Gold Book." The brochure can be found at http://www.epa.gov/asbestos/pubs/brakesbrochure.html on the Internet.

The **U.S. Consumer Product Safety Commission (CPSC)** is responsible for protecting the public from unreasonable risks of serious injury or death from more than 15,000 types of consumer products, including asbestos, under the agency's jurisdiction. The CPSC maintains a toll-free 24-hour hotline where callers can obtain product safety and other agency information and report unsafe products. In addition, CPSC publications provide guidelines for repairing and removing asbestos, and general information about asbestos in the home. CPSC can be contacted at:

Organization:	**Office of Information and Public Affairs**
	U.S. Consumer Product Safety Commission
Address:	4330 East West Highway
	Bethesda, MD 20814
Telephone:	1–800–638–2772
TTY (for deaf or hard of hearing callers):	1–800–638–8270
E-mail:	info@cpsc.gov
Internet Web site:	http://www.cpsc.gov

Individuals can also contact their local or state health department with questions or concerns about asbestos.

Information about cancer is available by calling the National Cancer Institute's (NCI) Cancer Information Service (CIS) at 1–800–4–CANCER (1–800–422–6237) or by visiting the NCI's Web site at http://www.cancer.gov on the Internet. The NCI is a component of the National Institutes of Health. Information about quitting smoking, including smoking cessation counseling, is available by calling the NCI's Smoking Quitline toll-free at 1–800–44U–QUIT (1–800–448–7848) or visiting the NCI's smoking cessation Web site at http://www.smokefree.gov on the Internet.

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Related Resources

Publications (available at http://www.cancer.gov/publications)

- National Cancer Institute Fact Sheet 6.36, *Mesothelioma: Questions and Answers*
- *What You Need To Know About™ Cancer of the Larynx*
- *What You Need To Know About™ Kidney Cancer*
- *What You Need To Know About™ Lung Cancer*

National Cancer Institute (NCI) Resources

Cancer Information Service (toll-free)
Telephone: 1–800–4–CANCER (1–800–422–6237)
TTY: 1–800–332–8615

Online
NCI's Web site: http://www.cancer.gov
LiveHelp, NCI's live online assistance:
https://cissecure.ncl.nih.gov/livehelp/welcome.asp

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EXHIBIT 23

Prevention and Early Detection

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Asbestos

What Is Asbestos?

Asbestos is a group of naturally occurring fibrous minerals. These minerals, found in soil and rocks in many parts of the world, are made of magnesium, silicon, and other elements. Some asbestos fibers are curly (or "serpentine"). The most common asbestos in industrial use, known as chrysotile, or white asbestos, has curly fibers. Other asbestos fibers are straight and needle-like (or "amphibole"). These include amosite, crocidolite, tremolite, actinolite, and anthophyllite. Asbestos fibers are resistant to heat and many chemicals. As a result, asbestos has been used as an insulating material since ancient times. Since the industrial revolution, asbestos was used to insulate factories, schools, homes, and ships, to make automobile brake and clutch parts, roofing shingles, ceiling and floor tiles, cement, and textiles, and hundreds of other products.

During the first half of the 1900s, growing evidence demonstrated that asbestos is a health hazard causing scarring of the lungs. In the early 1900s, exposure to asbestos dust in the workplace was not controlled. Beginning in England in the 1930s, steps were taken to protect workers in the asbestos industry by installing ventilation and exhaust systems. However, in the huge shipbuilding effort during World War II, large numbers of workers were exposed to high levels of asbestos. As asbestos-related cancers became better recognized in the second half of the twentieth century, additional measures were taken to reduce exposure, and exposure standards were established. In addition to more careful handling, there has been a dramatic decrease in the import and use of asbestos since the mid-1960s, and alternative insulating materials have been developed. As a result, asbestos exposure has dropped dramatically in the United States. However, exposure continues in some other countries. And in this country, there is still a potential for exposure from asbestos that remains in place in older buildings, water pipes, and other settings.

How Are People Exposed to Asbestos?

People are exposed to asbestos mainly through inhaling fibers in the air they breathe. This may occur when mining and processing asbestos, producing asbestos-containing products, or installing asbestos insulation. It may also occur when older asbestos-containing materials begin to break down. In any of these situations, asbestos fibers tend to create a dust composed of tiny particles that can float in the air. In addition, asbestos can enter the body through ingestion. This may occur when people consume contaminated food or liquids (such as water that flows through asbestos cement pipes). It may also occur when people cough up asbestos they have inhaled, then swallow their saliva.

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Many people are exposed to very low levels of naturally occurring asbestos in outdoor air as a result of erosion of asbestos-bearing rocks. The potential for such exposure is higher in areas where rocks have higher asbestos content. After the collapse of the World Trade Center in 2001, there was concern about the presence of asbestos in the dust. Recommendations were made for testing and cleaning of residences in Lower Manhattan to minimize any health risks from such exposures.

However, the people with the heaviest exposure were those who worked in asbestos industries, such as shipbuilding and insulating. Many of these people recall working in thick clouds of asbestos dust, day after day.

Family members of asbestos workers are also potentially exposed to higher levels of asbestos because the fibers are carried home on the workers' clothing, and can then be inhaled by others in the household. Removing asbestos from homes and other buildings can cause some exposure, although modern asbestos abatement workers are trained to use appropriate protective equipment to minimize exposure.

Exposure to asbestos-containing building material is also a concern, particularly in older buildings. If building materials like insulation and ceiling and floor tiles begin to decompose over time, asbestos fibers can be found in indoor air and may pose a threat to human health (EPA). There is no risk to human health if the asbestos is bonded into intact finished products, such as walls and tiles. As long as the material is not damaged or disturbed, for example by drilling or remodeling, there is no way for the fibers to be released into the air and inhaled. Maintenance workers who sweep up and dispose of the asbestos dust or handle damaged asbestos-containing building materials are often exposed to higher levels than other occupants of these buildings.

Asbestos may be detected in the water supply as well as in the air. It may be released into the water through several sources, such as erosion or natural deposits, corrosion from asbestos-cement pipes, and the break down of roofing materials containing asbestos that are then transported into sewers.

Although use of asbestos has declined in many developed countries, its use continues to present a hazard in other parts of the world. More than 70% of the world's asbestos production is used in Eastern Europe, Latin America, and Asia.

Does Asbestos Cause Cancer?

Lung Cancer

Inhalation of asbestos fibers has been proven to cause lung cancer. The connection between asbestos exposure and lung cancer was noted as early as 1925, and confirmed by many epidemiologic studies of asbestos-exposed workers over the next 70 years. All four main types of commercially used asbestos, chrysotile, amosite, anthophyllite, and mixtures containing crocidolite, are associated with an increased risk of lung cancer. An increase in lung cancer has also been observed after exposure to minerals containing tremolite and actinolite, and to tremolitic material mixed with anthophyllite and small amounts of chrysotile (IARC-Supp 17).

Approximately 1 in 7 people who suffer from asbestosis, a lung disease

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caused by prolonged high exposure, eventually develops lung cancer. The higher the exposure to asbestos, the higher the risk of lung cancer. Smoking acts together with asbestos to greatly increase the risk of lung cancer.; asbestos workers who smoke face a much higher risk than asbestos workers who do not smoke. Evidence suggests that asbestos-exposed workers who quit smoking can reduce their risk of developing lung cancer by as much as 50% within 5 years of quitting (NCI).

Mesothelioma

Mesothelioma is a rare form of cancer that affects the thin membranes lining the abdomen and chest. Mesothelioma is closely linked with asbestos; most cases of mesothelioma result from direct occupational asbestos exposure.

However, mesotheliomas have been observed not only among workers who are occupationally exposed to crocidolite, amosite, and chrysotile, but also among their family members and people living in the neighborhoods surrounding asbestos factories and mines (IARC). It is estimated that one third of the mesothelioma cases in the United States may be due to non-occupational exposure such as these (IARC). Although scientists know that the risk of developing mesothelioma increases with the amount of asbestos exposure, there is no way to measure exactly the minimum amount of asbestos exposure that can lead to mesothelioma. However, mesothelioma is very rare in the general population of the United States.

Unlike lung cancer, mesothelioma risk is not increased among smokers.

Other Types of Cancer

There is evidence that asbestos also causes other types of cancer, although the evidence is not as strong as for lung cancer and mesothelioma. Cancer of the larynx is one such cancer, although it has been difficult to separate the contributory role of asbestos from the strong effect of smoking. Many studies have also found an increase in stomach and colon cancer among workers in the asbestos industry, although this association is still debated.

What Do Epidemiologic Studies Show?

As explained above, much of the evidence about asbestos-associated cancers comes from epidemiologic studies.

What Do Animal Studies Show?

Tests on several different species, using several different methods of exposure, have confirmed that asbestos causes cancer in animals. Inhalation exposure of asbestos has been tested in rats, intrapleural administration (injection into the chest cavity) in rats and hamsters; intraperitoneal (injection into the abdominal cavity) injection in mice, rats, and hamsters; and ingestion in rats and hamsters (IARC). All commercial forms of asbestos have produced tumors in animals. The size and shape of the asbestos fibers influence the incidence of tumors; smaller fibers – less than 0.5 micrometers (um) in diameter (1 micrometer, or "micron", is 1 millionth of a meter) – seem more hazardous, perhaps because they are more likely to reach the deepest parts of the lungs (IARC).

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A series of lifetime feeding studies have been done to determine if oral exposure to asbestos increases the risk of cancer. These studies reported that short-range (98% of fibers are shorter than 10 um) asbestos fibers did not significantly increase the tumor incidence in rats; however, the incidence of benign tumors of the large intestines in male rats increased when the animals were exposed to intermediate-range (65% of fibers are longer than 10 um) asbestos fibers (EPA).

What Do the Expert Agencies Say?

Based on animal and human evidence like the examples above, expert agencies have evaluated the cancer causing nature of asbestos.

The *National Toxicology Program* evaluates exposures that may be carcinogenic (cause cancer). Exposures that are thought to be carcinogenic are included in the Reports on Carcinogens, published every 2 years. Each exposure is assigned to 1 of 2 categories: "known to be human carcinogens," and "reasonably anticipated to be human carcinogens." The first category includes substances for which human studies (epidemiology studies and/or experimental studies) provide "sufficient evidence" of carcinogenicity in humans. The second category includes substances for which there is limited evidence of carcinogenicity in humans and/or sufficient evidence of carcinogenicity in experimental animals.

Using this scheme, the National Toxicology Program classifies asbestos as a known human carcinogen.

The *International Agency for Research on Cancer (IARC* also evaluates exposures that may be carcinogenic. IARC classifies exposures into one of four categories:

- Group 1 exposures are those "known to be carcinogenic to humans," usually based on "sufficient" human evidence, but sometimes based on "sufficient" evidence in experimental animals and "strong" human evidence.
- Group 2 exposures are divided into two categories. Group 2A ("probably carcinogenic to humans") has stronger evidence, and Group 2B ("possibly carcinogenic to humans") has weaker evidence.
- Group 3 exposures are not considered classifiable, because available evidence is limited or inadequate.
- Group 4 exposures are "probably not carcinogenic to humans," based on evidence suggesting lack of carcinogenicity in humans and in experimental animals.

IARC rated asbestos a known human carcinogen (Group 1).

The **Environmental Protection Agency,** through its Integrated Risk Information System, uses a classification scheme very similar to that of IARC. It classifies exposures into 1 of 5 categories:

- (A) Human carcinogen
- (B) Probable human carcinogen

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- (C) Possible human carcinogen
- (D) Not classifiable as to human carcinogenicity
- (E) Evidence of noncarcinogenicity for humans

EPA classified asbestos as a human carcinogen.

Does Asbestos Cause Any Other Health Problems?

The major health problem caused by asbestos exposure, aside from cancer, is asbestosis. This is a scarring (or "fibrotic") disease of the lungs that develops when a person breathes high levels of asbestos over time. The principal symptoms of asbestosis are shortness of breath and cough. Asbestosis is a serious disease that can result in disability or death. It is most commonly found among asbestos workers. Asbestos can also result in scar-like tissue in the membranes around the lung, called pleural thickening, skin lesions like warts, and immunological effects.

What Should I Do if I've Been Exposed to Asbestos?

If you have been exposed to asbestos, it is important to assess the amount of your exposure. If you were exposed only very briefly, or only at very low levels, your risk of a resulting disease is minimal. However, it you were exposed at high levels, you may be at increased risk of the diseases discussed above. You can protect your health in several ways:

- If you are a smoker, it is essential that you stop smoking.
- Get regular health checkups from a doctor experienced with asbestos-related diseases. People with heavy asbestos exposure often have periodic chest x-rays and lung function tests.
- It may be advisable for you to receive vaccines against flu and pneumonia. Discuss this with your physician.
- Get prompt medical attention for any respiratory illness.

How Can I Avoid Exposure to Asbestos?

You should also avoid any future exposure to asbestos. If there is a possibility of on-the-job exposure, like renovating old buildings for example, then you should use all protective equipment, work practices, and safety procedures designed for working around asbestos (NCI). If you live in an older home, there may be asbestos-containing insulation or other materials. A knowledgeable expert can check your home to determine if there is any asbestos and if it poses any risk of exposure. This may involve testing the air for asbestos levels. You may then decide to have the asbestos removed from your home. You should hire a qualified contractor to perform this job to avoid contaminating your home further or causing any exposure to the workers. You should not attempt do remove asbestos-containing material yourself.

What's the Bottom Line?

Asbestos is well recognized as a carcinogen. It causes lung cancer, mesothelioma, and other cancers. The people at highest risk are those with very heavy exposure, usually over many years on the job. Smoking acts together with asbestos to greatly increase the risk of lung cancer. While asbestos use is much less common now than it was years ago, there is still a potential for exposure in older buildings and products.

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Other Resources Addressing Asbestos

Agency for Toxic Substances and Disease Registry (ATSDR). ToxFAQs for Asbestos.
Internet Address: www.atsdr.cdc.gov/tfacts61.html
Telephone: 1-888-422-8737

Environmental Protection Agency (EPA)
Internet Address: www.epa.gov/asbestos/
Telephone: 202-260-2090

International Agency for Research on Cancer (IARC)
Asbestos, Vol 14 at
www-cie.iarc.fr/htdocs/indexes/vol14index.html
Asbestos, Supplement 7, at
www-cie.iarc.fr/htdocs/monographs/suppl7/asbestos.html

National Cancer Institute (NCI). Asbestos Exposure: Questions and Answers
Internet Address: www.cancer.gov/cancertopics/factsheet/Risk/asbestos
Telephone: 1-800-422-6237

National Institutes of Health, National Institute of Environmental Health Sciences, National Toxicology Program. Eleventh report on carcinogens.
Internet Address: ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s016asbe.pdf

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EXHIBIT 24

WHO

Chapter 6.2 Asbestos

General Description

The term "asbestos" designates a group of naturally occurring fibrous serpentine or amphibole minerals that have extraordinary tensile strength, conduct heat poorly and are relatively resistant to chemical attack. The principal varieties of asbestos are chrysotile, a serpentine mineral, and crocidolite, amosite, anthophyllite, tremolite and actinolite, all of which are amphiboles.[a]

Chrysotile fibres consist of aggregates of long, thin, flexible fibrils that resemble scrolls or cylinders of uniform chemical composition. Although chrysotile is a reasonably well defined mineral, the five amphibole asbestiform fibres have such variable chemical compositions and physical properties that positive identification is sometimes troublesome.

The macroscopic asbestos fibres are actually bundles of thinner fibres made up of fibrils which, in the case of chrysotile, have a diameter of 20-25 nm. Each macroscopic fibre is highly anisotropic and tends to decompose into its thinner constituents under industrial handling or from weathering, giving rise to a fibrous, partially respirable aerosol.

Sources

Natural sources are important, because asbestos minerals are widely spread throughout the earth's crust and are not restricted to the few mineable deposits. In particular, chrysotile is present in most serpentine rock formations. Emissions are due to natural weathering and can be enhanced by man's activities, such as quarrying or street building. Very little, however, is known about the amounts emitted from natural sources.

Man-made emissions originate from activities in the following categories:

(a) mining and milling

(b) manufacture of products

(c) construction activities

(d) transport and use of asbestos-containing products

(e) disposal.

There has been a steep rise in the production and use of asbestos in the last 100 years. Asbestos consumption has levelled off in recent years *(1)* to about 4 million tonnes (1983) and, because of the relative decline in crocidolite and amosite usage *(1)* the figure represents essentially chrysotile production (in 1982 only about 5% of the total asbestos produced was in the form of amphibole asbestos (crocidolite and amosite) *(2))*.

In many industrialized countries most of the asbestos used was in the building sector (70-90% in several western European countries) *(2)*, and the demand for asbestos in these

[a] Man-made mineral fibres and other natural mineral fibres such as fibrous zeolites, wollastonite, attapulgite and sepiolite will not be discussed here.

countries had already reached a constant level before the health effects of asbestos were widely debated. Because of its specific technical properties, asbestos has found an extremely large variety of applications (in about 3000 different products). In the future, legislative restrictions and the success in finding substitutes for asbestos in fibre-cement, brake-linings, insulation and many other applications will most probably lead to declining asbestos consumption in the above-mentioned countries.

Asbestos emissions occur during processing. When air filtration is used, the dust emissions from processing can be kept below 100 g per tonne *(3)*.

Rain acidity (from carbon dioxide and air pollutants) is known to corrode asbestos-cement sheets, constituting a further source of emission *(4)*. Brake-linings in cars are another source in the urban environment. Only a few measurements have been made of the contribution made by these sources to fibre emissions in urban air *(5,6)*.

Indoor asbestos fibre concentrations can be considerably higher than outdoor concentrations *(7)*. Indoor asbestos dust originates from insulation material sprayed on steelwork or ceilings (such material may become highly friable after some years), asbestos plasters, low-weight insulation plates, etc. *(8,9)*. Sometimes such materials have been used in direct or close contact with air-conditioning equipment. Even though some of these materials, such as spray asbestos, are no longer used, they are still found in many public buildings. Until the mid-1970s electric storage-heaters and some other electrical household equipment contained asbestos. One of the main forms of use of asbestos is as asbestos-cement; in this case the release of fibres to the general environment is minimized, since the fibres are essentially "locked" in the cement matrix. Asbestos cement products, therefore, do not usually pose problems for indoor air quality.

Factors such as renovation and repair, maintenance, external vibrations and vandalism can considerably increase the emission of asbestos dust from existing indoor sources *(10,11)*. Increased emission is also possible as a result of changes in temperature and reduction of humidity.

Unlike levels of asbestos fibres in ambient air, fibre concentrations in the air of indoor environments can always be related to their source, thus offering the possibility of modifying or removing the source of emission. Because people in temperate climates live indoors most of the time, the most relevant source of inhalation exposure will often be asbestos fibre concentrations in buildings.

Occurrence in air

Asbestos fibres of respirable size form part of a range of fibrous aerosols in the lower atmosphere. Other fibres include man-made mineral fibres, fibrous silica and aluminium oxide, fibrous gypsum and, in some geographical areas, fibrous zeolite, attapulgite, sepiolite and wollastonite *(1)*.

Once emitted into the atmosphere, asbestos fibres may travel considerable distances owing to their aerodynamic properties. Because no chemical breakdown of the fibres occurs, washout by rain or snow is the only cleaning mechanism.

Asbestos fibres normally constitute only a relatively small fraction of the total fibrous aerosol in ambient air *(6,12)*. The biologically more important so-called "critical" fibres are those equal to or longer than 5 μm and having diameters up to 3 μm *(1,10,12)* with an aspect ratio equal to or greater than 3 : 1.

Although asbestos fibres can be readily detected and monitored in occupational situations by using phase-contrast optical microscopy, their assessment in the environment calls for an integrated method capable of microchemical analysis of single fibres, measurement of fibre length and diameter, and counts of fibre numbers in given air samples. Electron microscopy is the only method which can detect and identify asbestos fibres among the very wide range

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of other fibrous and nonfibrous particles, of greatly varying toxic potency, in the ambient air. Used in conjunction with each other, these methods allow the identification of samples. In order positively to identify asbestos fibres in environmental ambient air it is necessary to use selected-area electron diffraction and/or energy-dispersive X-ray diffraction analysers attached to an electron microscope. Such instrumentation is costly and highly-trained personnel are required in order to obtain reliable results. Although integrated electron microscopy methods have been developed during the last 15 years *(13)*, they are still not in widespread use. Therefore, all data measured by these methods are, strictly speaking, representative only for the location tested and the time interval chosen. However, if one compares various sets of such data and restricts quantification to orders of magnitude, the following concentration pattern emerges:[b]

Rural areas (remote from asbestos emission sources):

- below 100 F/m^3 *(6,10)*

Urban areas:
- general levels may vary from below 100 to 1000 F/m^3 *(6,12)*

Near various emission sources the following figures have been measured as yearly averages *(6,12,14)*:

- downwind from an asbestos-cement plant at 300 m: 2200 F/m^3; at 700m: 800 F/m^3; at 1000 m: 600 F/m^3 *(6)*;
- at a street crossing with heavy traffic, 900 F/m^3 *(14)*;
- on an express-way, up to 3300 F/m^3 *(9)*

Indoor air:
- in buildings without specific asbestos sources, concentrations are generally below 1000 F/m^3 *(12)*;
- in buildings with friable asbestos, concentrations vary irregularly; usually less than 1000 F*/m^3 are found, but in some cases exposure reaches 10 000 F*/m^3 *(9)*, where F* = fibres counted with an optical microscope.

Occupational levels are orders of magnitude higher than those found in the environment, with values from 10^5 F*/m^3 to more than 10^8 F*/m^3 *(12,15)*, but are now being reduced to below 2×10^6 F*/m^3 in most countries and to $(0.2\text{-}0.5) \times 10^6$ F*/m^3 in some.

[b] *Note.* If not otherwise stated, concentrations in the following text are given as numbers of critical asbestos fibres per m^3, i.e. all fibres of length L> 5 μm, diameter D < 3 μm and aspect ratio L : D > 3 : 1 measured by electron microscopy methods. All concentrations are expressed as fibres per m^3 air, although concentrations in terms of fibres per litre and fibres per ml are often reported. Some studies have been carried out where results have been expressed as ng or μg per m^3 air. Because only results such as fibre number concentrations are considered to be directly relevant to an index of exposure with regard to possible health implications *(1,12)* and factors in attempts to convert mass to number concentrations are so variable *(12)*, mass concentrations are not considered here.

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Routes of Exposure

Air

Various subgroups of the population are exposed to different fibre concentrations for varying lengths of time. It is usually assumed that the risks of any two persons are roughly comparable - other factors being equal - if their accumulated fibre burdens are the same. The fibre burden is the accumulated number of critical fibres (F) = fibre concentration (F/m^3) × number of years of exposure × air volume inhaled each year at place of exposure (m^3/year). In this context it is important to note that not all fibres inhaled are deposited and available for retention in the body. However, it is assumed that the fraction of fibres exhaled is more or less the same during all inhalation exposures. Assuming a breathing rate of 10 m^3 for an 8-hour working day and 200 working days per year, a worker inhales 2000 m^3 per year during working hours, while total inhalation for the general population is 7300 m^3 per year *(10)*.

Table 1 gives orders of magnitude of lifetime fibre burdens in various population subgroups, thus indicating the relative importance of different types of inhalation exposure *(10,16)*. Values for indoor air exposure have not been included because only a few are available and details are insufficient. Only for the USA has a calculation of indoor concentration been made in terms of average fibre concentration. The median values range from 400 F/m^3 *(10)* to 500 F/m^3 *(12)*. If these estimates were correct, they would result in a lifetime fibre burden of up to 2×10^8 F. In this case outdoor exposure would be of minor importance to most of the US population.

There can also be more irregular exposure of the general public, with peak concentrations at specific sites. Table 1 shows that such exposure for 1% of a lifetime (0.24 hours per day) could contribute significantly to the total accumulated burden of fibres. Peak exposures in people passing building sites have been reported by several authors. These exposures occurred when asbestos-cement sheets were being cut *(16)* or spray insulation was being carried out *(17)*; in these cases no dust suppression measures were applied. Peak exposures may also occur in para-occupational situations (e.g. household activities *(18)*).

Table 1. Lifetime fibre burdens typical for industrial countries

Population[a] (%)	Subgroup	Fibre concentration (F/m^3)[b]	Exposure time (years)	Inhaled volume (m^3/year)	Accumulated critical fibres (F > 5 μm)
70	Urban population (moderate exposure)	30	70	7300	$\sim 1.5 \times 10^7$
25	Rural population	10	70	7300	$10^5 - 10^6$
5	Urban population (high exposure)	200	70	7300	$\sim 10^8$
1-2	General construction workers	$10^3 - 10^5$ [c]	50	2000	$10^8 - 10^{10}$ [c]
0.1	Asbestos workers	$10^5 - 10^6$ [c]	50	2000	$10^{10} - 10^{11}$ [c]
	Irregular exposure (example)	10^4	0.7	7300	5×10^7

[a] These percentages reflect an assumed population distribution.
[b] F >5 μm.
[c] Fibre count with optical microscope.

Drinking-water and food

Drinking-water and food may contain asbestos fibres from natural sources (e.g. rock) and man-made sources (e.g. asbestos-cement pipes). The total fibre content (fibres of all lengths) in drinking-water can vary from 10^4 F/litre to more than 10^8 F/litre *(3,4,19,20)*.

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Relative significance of different routes of exposure
Inhalation is by far the most important route of exposure of humans to critical fibres, the amount of uptake by ingestion being questionable and, at the least, significantly lower.

Kinetics

Health effects due to asbestos exposure in the occupational environment have been clearly associated with inhalation. The relevance of the oral intake of asbestos fibres for human health is unclear; in any case, ingestion is far less important than inhalation. For this reason, only the deposition, retention and clearance of fibres from the human lungs are described here.

Deposition
Fibres with a diameter greater than 3 μm are not respirable *(10)*. Symmetrical fibres have a lower probability of deposition in the upper airways than nonsymmetrical fibres such as the "curly" chrysotile. A considerable number of fibres are deposited in the upper ciliated airways, where fibres can be removed by cleaning mechanisms *(10)*. Chrysotile fibres can split into fibrils and undergo partial dissolution within the lungs. The amphiboles do not subdivide into fibrils of smaller diameter or break up by length. They are much less soluble in lung fluids, and they have long residence times in the lungs *(10)*.

Clearance
Several mechanisms are involved in the clearing of fibres from their site of deposition, i.e. mucociliary clearance, translocation of alveolar macrophages containing small fibres, and uptake by epithelial cells lining the airways *(17)*. Overall, the clearing mechanisms are very effective (95-98%), although in the alveolar regions some fibres can remain. It should be noted, however, that one of the most effective clearance mechanisms (mucociliary clearance) is impaired by smoking *(1)*. Deposited fibres less than 5-10 μm long may often be engulfed by a single macrophage and thereafter be translocated; fibres longer than this are more difficult to clear. Long fibres may, however, become chemically dissolved or even mechanically broken down in the lung tissue. Durability seems to be greatest for amphiboles, less for chrysotiles *(9,12,21,22)*; chrysotile can, in fact, split readily into fibrils and undergo partial dissolution in lung fluids *(1)*.

The number of fibres retained in the lungs has been investigated very intensively in post-mortem lung material. All sorts of fibres known to exist in the atmospheric aerosol, including asbestos, have been found. The number of asbestos fibres per cm^3 of lung sample is related to past exposure, the ratio of lung burdens for asbestos workers and controls being roughly the same as that of their respective exposures *(10,12)*.

Health Effects

There is an intensive and well documented series of case studies and epidemiological observations which link past occupational exposure to asbestos with asbestosis, lung cancer and mesothelioma *(1,3,9,10,12,23)*. Another series of studies has mainly examined subgroups of the population in particular exposure situations, mostly indoor exposure or para-occupational exposure (see above).

Asbestosis is a slowly developing fibrosis of the lung caused by the inhalation of high concentrations of asbestos dust and/or long exposure *(24)*. Its severity depends both on the

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length of time since onset of exposure and on the intensity of the latter *(23)*. Although nonmalignant in itself, advanced asbestosis is often associated with lung cancer, especially among smokers. Hypoxia with cor pulmonale does occur in severe cases, while mild forms may not necessarily be associated with marked disablement *(10)*.

Lung cancer (bronchial carcinoma) is the most frequent kind of cancer in the male population (accounting for about 10% of all male deaths in many industrialized countries) and is clearly related to external factors such as smoking, ionizing radiation (e.g. radon) and occupational exposures to certain substances, including asbestos inhalation, in the latter case even without co-existing asbestosis *(9)*. Many studies have shown that smokers have a higher risk of developing lung cancer than nonsmokers when exposed to asbestos *(23)*.

Mesothelioma is a malignant tumour of the pleura or peritoneum *(9,23)*. It is a rare type of cancer (less than 0.04% of all deaths in the general population of the USA) *(10)*. A higher incidence of mesothelioma has nearly always been related to the inhalation of mineral fibres, and in the majority of cases to occupational asbestos exposure *(9)*. Causing few symptoms initially, mesothelioma is incurable when diagnosed *(10)*.

Effects on experimental animals and *in vitro* test systems

Experimental toxicology has developed models to establish the relative carcinogenic potency of different types and sizes of fibres and to examine possible interactive effects with other airborne pollutants *(3)*. This approach has proved helpful in the interpretation of epidemiological studies. Besides inhalation studies, ingestion and implantation studies have also been carried out; these have additionally been used to explore the association between asbestos and other forms of cancer in man, e.g. cancers of the gastrointestinal tract, which are epidemiologically less well established *(9,23)*.

Species vary in their response to asbestos. Effects in rats seem to resemble those in man more closely than those in other animals *(9)*; if not otherwise stated, the results reported refer to this species.

Inhalation

Lung cancer, mesothelioma and asbestosis have been observed in several studies regarding various asbestos and other fibres *(10)*. In three inhalation experiments with 10 mg chrysotile per m^3 over a period of 1 year, at least 20% of exposed rats developed lung tumours *(25-27)*.

Intrapleural and intraperitoneal injection

The direct injection of a specific amount of dust with a given particle-size distribution into the pleura or the peritoneum makes it possible to study carcinogenic potency as a function of fibre type and shape without interference from variable factors such as deposition, clearance and disintegration, which are difficult to control in inhalation experiments.

The injection of asbestos produced mesotheliomas, the resulting incidence being highly fibre-specific and sensitive *(3,9)*. Intraperitoneal injection of 1 mg chrysotile (UICC Canada) or 0.3 mg Actinolite resulted in a tumour rate of about 90% and 80%, respectively *(28)*. Experiments show that fibre length and diameter are the most important factors in causing mesothelioma. As shown by Pott in his graph of the carcinogenicity factor *(29)*, fibres shorter than 5 μm and thicker than 2 μm elicit little response, while those longer than 10 μm and thinner than 0.5 μm yield the highest response. Nonasbestos fibres of the same critical dimensions also caused mesothelioma in animals, while experiments with fibres of noncritical dimensions are negative, irrespective of fibre category *(29)*. This finding is important considering the presence of many other fibre types in ambient air.

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Ingestion
In several studies, rats and other laboratory animals were exposed to asbestos in diet or drinking-water. Only a single - usually very high - dose was applied in each investigation. The results are inconclusive, if not negative *(1,20)*.

Effects on humans

Asbestosis
While early stages of asbestosis are still observed at asbestos-cement plants under modern regulations *(24)*, asbestosis has never been observed in relation to nonoccupational asbestos exposure. It is therefore concluded that environmental concentrations of asbestos are not sufficient to induce asbestosis *(1,9)*.

Mesothelioma
Without doubt, exposure to all kinds of asbestos is closely linked to mesothelioma. Several epidemiological studies indicate that amphibole asbestos is more potent in inducing mesothelioma than chrysotile *(1)*, which seems to contradict the findings from intraperitoneal injection experiments *(9)*. The contradiction is, however, to a great extent resolved by the observation that technical processes involving amphiboles produce dust with a higher proportion of long, thin - i.e. more dangerous - fibres *(9)*. Furthermore, amphibole fibres are more resistant in the body and have a lower lung clearance than chrysotile *(9)*.

The time elapsing between first exposure to asbestos and the clinical manifestation of tumours ranges from 20 to 50 years for mesothelioma in the populations of workers studied *(23)*. Dose-response relationships are derived from retrospective epidemiological studies of exposure data relating to situations several decades ago. Clearly these exposure data can only be approximations *(9,15)*. Increased incidence rates seen in nonoccupationally exposed people living in the same household as asbestos workers or in the vicinity of strong asbestos emission sources have been attributed to this exposure *(30)*.

Lung cancer
Clarifying the relationship between asbestos exposure and lung cancer is a much more complicated task. Both the synergism of smoking and asbestos inhalation and the high level of lung cancer in the general population (background exposure) must be taken into account. The synergistic effect can be described by a multiplicative model *(31)* which is used below in connection with the extrapolation of lung cancer risk. The risk of lung cancer seems to rise consistently from mining and milling operations, through branches such as asbestos-cement production, to asbestos textile and insulation work, according to the increasing portion of more dangerously shaped fibres in these processes (see Table 2).

Other cancers
Recent evaluations *(9,10,20,23)* of epidemiological occupational studies provide little evidence for the induction of other cancers, including gastrointestinal cancers, although recently it has been suggested that laryngeal cancer may be related to heavy occupational exposure to asbestos *(23)*. Clearly the risk, if any, for the general population from "other" cancers is very small *(9,20,45)*.

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Table 2. Increase in the relative risk of lung cancer, as shown by different studies

K_L per 100 F*year/ml	Type of activity	Reference
0.04	mining and milling	*(33)*
0.045	mining and milling	*(34)*
0.06	friction material	*(35)*
0.1	factory processes	*(36,37)*
(M) 0.4-1.1	factory processes	*(38)* [b]
(F) 2.7 [a]	factory processes	
0.2	asbestos-cement	*(39)*
0.07	textiles (before 1951)	*(40)*
0.8 [a]	textiles (after 1950)	
6(M) 1.6 [a]	textiles	*(41)*
1.6	textiles	*(42)* [c]
1.1	insulation products	*(43)* [b]
1.5	insulation	*(44)* [b]

[a] Fewer than 10 cases of lung cancer expected (i.e. small cohort).
[b] Inadequate knowledge of actual fibre concentrations.
[c] Same factory as in *(41)*, but larger cohort.

Source: based on a table by Liddell *(32)*.

Evaluation of Human Health Risks

Exposure

Actual indoor and outdoor concentrations in air range from below one hundred to several thousand fibres per m^3.

Health risk evaluation

On the basis of the evidence from both experimental and epidemiological studies, it is clear that asbestos inhalation can cause asbestosis, lung cancer and mesothelioma. The evidence that ingested asbestos causes gastrointestinal or other cancers is insufficient. Furthermore, the carcinogenic properties of asbestos are most probably due to its fibre geometry and remarkable integrity; other fibres with the same characteristics may also be carcinogenic.

Current environmental concentrations of asbestos are not considered a hazard with respect to asbestosis. However, a risk of mesothelioma and lung cancer from the current concentrations cannot be excluded.

A WHO Task Group recently expressed reservations about the reliability of risk assessment models applied to asbestos risk. Its members suggested that such models can only be used to obtain a broad approximation of the lung cancer risk of environmental exposures to asbestos and "that any number generated will carry a variation over many orders of magnitude". The same was found to be true for estimates of the risk of mesothelioma. The same document stated: "In the general population the risks of mesothelioma and lung cancer attributable to asbestos cannot be quantified reliably and probably are undetectably low." *(1)*.

The following estimates of risk are based on the relatively large amount of evidence from epidemiological studies concerning occupational exposure. Data from these studies have been

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conservatively extrapolated to the much lower concentrations found in the general environment. Although there is evidence that chrysotile is less potent than amphiboles, as a precaution chrysotile has been attributed the same risk in these estimates.

Mesothelioma

A formula by which the excess incidence of mesothelioma can be approximated has been derived by Peto *(46)*. Fibre concentration, duration of exposure and time since first exposure are parameters incorporated in this model, which assumes a linear dose-response relationship. Peto verified this model from data on an urban population exposed for its whole life and on workers exposed for many decades. In both cases, duration of exposure is assumed to be equal or close to time since first exposure. The data show that the incidence of mesothelioma is proportional to the fibre concentration to which the workers were exposed and to time$^{3.5}$ since first exposure for both workers and the general population. Starting from this relationship, one may calculate the risk of lifetime exposure to environmental concentrations from the incidence of mesothelioma in occupational populations exposed to much higher concentrations, but for a shorter time.

Apart from incomplete knowledge about the true workplace exposure, a further complication arises from the fact that workplace concentrations were measured by means of an optical microscope, counting only fibres longer than 5 μm and thicker than, say, 0.5 μm.[c]

Several studies have been performed to calculate the risk of mesothelioma resulting from nonoccupational exposure to asbestos. Lifetime exposure to 100 F*/m^3 has been estimated by various authors to carry differing degrees of mesothelioma risk (see Table 3).

Table 3. Estimates of mesothelioma risk resulting from lifetime exposure to asbestos

Risk of mesothelioma from 100 F*/m^3	Values in original publication (risk for fibre concentration indicated)	Reference
1.0×10^{-5}	1.0×10^{-4} for 1000 F*/m^3	*(45)*
$\sim 2.0 \times 10^{-5}$	1.0×10^{-4} for (130-800) F*/m^3	*(47)*
$\sim 3.9 \times 10^{-5}$	1.56×10^{-4} for 400 F*/m^3	*(10,48)*
$\sim 2.4 \times 10^{-5}$	2.75×10^{-3} (females) } for 0.01 F/ml 1.92×10^{-3} (males)	*(17)*

The risk estimates in Table 3 differ by a factor of 4. A "best" estimate may be 2×10^{-5} for 100 F*/m^3.

An independent check of this risk estimate can be made by calculating the incidence of mesothelioma in the general population, based on a hypothetical average asbestos exposure 30-40 years ago *(49)*. If the latter had been 200-500 F*/m^3 (corresponding to about 400-1000 F/m^3 as measured today), the resulting lifetime risk of mesothelioma would be (4-10) $\times 10^{-5}$. With the average United States death rate of 9000 $\times 10^{-6}$ per year, this would give 0.4-0.9 mesothelioma cases each year per million persons from past environmental asbestos exposure. The reported mesothelioma incidence in the USA ranges from 1.4×10^{-6} per year

[c] In this chapter all fibre concentrations based on optical microscopy are marked F*/m^3 and risk estimates will be based on F*/m^3. If concentrations measured by optical microscope are to be compared with environmental fibre concentrations measured by a scanning electron microscope, a conversion factor has to be used: 2 F/m^3 = 1 F*/m^3.

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to 2.5×10^{-6} per year according to various authors *(10, 49)*. Thus, the calculated risk figures would account for only part of the observed incidence. However, other factors which may account for this discrepancy must be considered.

- Uncertainties in the risk extrapolations result from the lack of reliable exposure data in the cohort studies, errors in the medical reports, and necessary simplifications in the extrapolation model itself *(17)*. Furthermore, the amount of past ambient exposure can only be an educated guess.

- The incidence of nonoccupational mesotheliomas is calculated from the difference between the total of observed cases and the number of those probably related to occupational exposure. Neither of these two figures is exactly known. Moreover, the influence of other environmental factors in the generation of mesothelioma is unknown.

In the light of these uncertainties, the result obtained by using the risk estimate can be considered to be in relatively good agreement with the annual mesothelioma death rate based on national statistical data.

Lung cancer

Unlike mesothelioma, lung cancer is one of the most common forms of cancer. As several exogenous noxious agents can be etiologically responsible for bronchial carcinoma, the extrapolation of risk and comparison between different studies is considerably complicated. In many epidemiological studies, in particular, the crucial effect of smoking has not been properly taken into account. Differentiation of the observed risks according to smoking habits has been carried out, however, in the cohort of North American insulation workers studied by Hammond et al. *(31)*.

This study suggests that the relative risk at a given time is approximately proportional to the cumulative amount of fine asbestos dust received up to this point, for both smokers and nonsmokers. The risks for non-asbestos-exposed nonsmokers and smokers must therefore be multiplied by a factor which increases in proportion to the cumulative exposure.

The dose-response relationship in the case of asbestos-induced lung cancer can be described by the following equation *(17)*.

$$I_L \text{ (age, smoking, fibre dose)} = I^{\circ}_L \text{ (age, smoking)} [1 + K_L \times C_f \times d]$$

This equation could also be written as:

$$K_L = [(I_L / I^{\circ}_L) - 1]/C_f \times d = \text{(relative risk - 1)/(cumulative exposure)}$$

where:

K_L = a proportionality constant, which is a measure of the carcinogenic potency of asbestos

C_f = fibre concentration

d = duration of exposure, in years

I_L = lung cancer incidence, observed or projected, in a population exposed to asbestos concentration C_f during time d

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I°_L = lung cancer incidence expected in a group without asbestos exposure but with the same age and smoking habits (this factor includes age dependence).

There are several studies which allow the calculation of K_L. Liddell *(32, 50)* has done this in an interesting and consistent manner. The results are given in Table 2.

Taking the data in Table 2 as a basis, a reasonable estimate for K_L is 1.0 per 100 F*years/ml. For a given asbestos exposure, the risk for smokers is about 10 times that for nonsmokers *(31)*. In extrapolating from workers to the general public, a factor of 4 for correction of exposure time has to be applied to K_L.

The incidence of lung cancer in the general population exposed to 100 F*/m^3 is calculated as follows:

$$I_L = I^{\circ}_L\,(1 + 4 \times 0.01 \times 10^{-4}\ F^*/ml \times 50\ years)$$

or

$$I_L = I^{\circ}_L\,(1 + 2 \times 10^{-4}\ F^*/ml)$$

The extra risk is I_L - I°_L. Values for I°_L are about 0.1 for male workers and 0.01 for male nonsmokers *(10)*.

Lifetime exposure to 100 F*/m^3 (lifetime assumed to be 50 years since, in a lifetime of 70 years, the first 20 years without smoking probably do not make a large contribution) is therefore estimated as follows.

Status	Risk of lung cancer per 100 000	Range (using the highest and lowest values of K_L from Table 2)
Smokers	2.0	0.08-3.2
Nonsmokers	0.2	0.008-0.32

This risk estimate can be compared, when adjusted to 100 F*/m^3, with estimates for male smokers made by other authors or groups:

Breslow (National Research Council) *(48)*: 7.3×10^{-5};

Schneiderman et al. *(47)*: $(14\text{-}1.4) \times 10^{-5}$;

US Environmental Protection Agency *(17)*: 2.3×10^{-5}.

A fibre concentration of 100 F*/m^3 (about 200 F/m^3 as seen by scanning electron microscope) thus gives a total risk of $(2 + 2) \times 10^{-5}$ for smokers or 2.2×10^{-5} for nonsmokers.

Guidelines

Asbestos is a proven human carcinogen (IARC Group 1). No safe level can be proposed for asbestos because a threshold is not known to exist. Exposure therefore should be kept as low as possible.

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Several authors and working groups have produced estimates indicating that, with a lifetime exposure to 1000 F/m^3 (0.0005 F*/ml or 500 F*/m^3, optically measured) in a population of whom 30% are smokers, the excess risk due to lung cancer would be in the order of 10^{-6} - 10^{-5}. For the same lifetime exposure, the mesothelioma risk for the general population would be in the range of 10^{-5} - 10^{-4}. These ranges are proposed with a view to providing adequate health protection, but their validity is difficult to judge. An attempt to calculate a "best" estimate for the lung cancer and mesothelioma risk is described above.

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EXHIBIT 25

U.S. Consumer Product
Safety Commission · Washington, D.C. 20207

NEWS RELEASE

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FOR RELEASE
THURSDAY
APRIL 28, 1977

CPSC BANS USE OF ASBESTOS IN CERTAIN CONSUMER PRODUCTS

WASHINGTON, D.C. (Apr. 28) -- The U.S. Consumer Product Safety Commission (CPSC) today announced plans to ban consumer patching compounds containing asbestos or asbestiform tremolitic talc as well as artificial fireplace logs and ashes containing asbestos.

The Commission's action today was in response to petitions before it. According to the Commission, asbestos can be a cancer-causing substance and has been shown to be harmful to humans if inhaled.

The Natural Resources Defense Council, a public interest group, joined by Consumers Union, asked CPSC to ban the use of asbestos in consumer patching (spackli compounds. The Commission also granted (in part) a petition from the Health Researc Group regarding a ban of asbestiform tremolitic talc in spackling compounds because of the cancer risk from free-form asbestos fibers.

Patching compound sold to consumers is either premixed or in dry powdered form for mixing with water. It is applied to sheet rock either to fill a hole or mask a seam and, when dry, is sanded smooth. Asbestos fibers may be released into the air during mixing and/or sanding.

Rachel Scott, a consumer, and the Environmental Defense Fund petitioned CPSC to ban asbestos used as ashes for gas fireplaces. This product is intended to simulate flowing ashes.

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The Commission has directed its technical staff to provide, within the next few days, recommendations for consumer disposal of these three products. Additionally, the staff will be gathering information for the Commissioners on other consumer products containing asbestos along with recommendations for possible regulatory action.

The Commission expects to determine which legal route to take in banning these three products in the next few days.

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EXHIBIT 26

Industrial Health 1999, 37, 271–280

REVIEW ARTICLE

The Hazards of Chrysotile Asbestos: A Critical Review

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Abstract: Chrysotile, or "white", asbestos is the dominant form of asbestos in international commerce today. It accounts for 99% of current world asbestos production of 2 million tonnes. Chrysotile is an extremely hazardous material. Clinical and epidemiologic studies have established incontrovertibly that chrysotile causes cancer of the lung, malignant mesothelioma of the pleura and peritoneum, cancer of the larynx and certain gastrointestinal cancers. Chrysotile also causes asbestosis, a progressive fibrous disease of the lungs. Risk of these diseases increases with cumulative lifetime exposure to chrysotile and rises also with increasing time interval (latency) since first exposure. Comparative analyses have established that chrysotile is 2 to 4 times less potent than crocidolite asbestos in its ability to cause malignant mesothelioma, but of equal potency of causation of lung cancer. The International Agency for Research on Cancer of the World Health Organization has declared chrysotile asbestos a proven human carcinogen. Sales of chrysotile asbestos have virtually ended in Western Europe and North America, because of widespread recognition of its health hazards. However, asbestos sales remain strong in Japan, across Asia and in developing nations worldwide. The claim has been made that chrysotile asbestos can be used "safely" under "certain conditions" in those nations. That claim is not accurate. The Collegium Ramazzini, an international learned society in environmental and occupational medicine, has called for an immediate worldwide ban on all sales and uses of all forms of asbestos, including chrysotile. The rationale for this ban is threefold: (1) that safer substitute materials are readily available, (2) that "controlled" use of asbestos is not possible, and (3) that the health risks of asbestos are not acceptable in either the industrialized or the newly industrializing nations.

Key words: Chrysotile, Asbestos-related diseases, Epidemiology, Risk evaluation, Human carinogenicity, Malignant mesothelioma, Lung cancer

Introduction

Chrysotile asbestos is an important cause of human illness and death[1]. Clinical and epidemiologic studies have established beyond all reasonable doubt that chrysotile asbestos causes cancer of the lung, malignant mesothelioma of the pleura and peritoneum, cancer of the larynx and certain gastrointestinal cancers[2]. Chrysotile also causes asbestosis, a progressive fibrotic disease of the lungs. The risk of these diseases increases with cumulative exposure to chrysotile and also with time since first exposure. Chrysotile asbestos has been declared a proven human carcinogen by the US Environmental Protection Agency (EPA)[3], by the US Occupational Safety and Health Administration (OSHA)[4],

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and by the International Agency for Research on Cancer of the World Health Organization[5]. The total number of deaths in the United States that will eventually be caused by exposure to asbestos, over 95% of it chrysotile asbestos, is estimated to exceed 400,000[6].

Background

Asbestos is a term given to a group of minerals that crystallize in a fibrous habitat. Four fibrous minerals have commercial importance—chrysotile, amosite, crocidolite, and anthophyllite. Of these, chrysotile is the most important, accounting for more than 95% of current world production. The various minerals differ chemically and structurally, but all readily separate into extremely fine fibers, most of which are thinner than 1 μm in diameter. As such, they can easily become airborne during manufacturing processes or by abrasion or disturbance of asbestos-containing materials. Their thinness allows them to be readily inspired and carried into the lower regions of the human lung, where they can become lodged and cause damage.

The modern history of asbestos disease dates from the beginning of the twentieth century, when two cases of asbestotic lung scarring were briefly described in asbestos textile workers. The pulmonary disease resulting from such scarring was well described in subsequent publications and the term *asbestosis* applied to it in 1924. An association of bronchogenic carcinoma with asbestos was first suggested in the 1930s. Mesothelioma, a malignancy of the lining of the chest or abdomen, was clearly associated with asbestos in 1960.

Despite publication over the past four decades of a voluminous literature on the causal association between asbestos and disease in different populations, and despite extensive experimental demonstration of the carcinogenicity of all forms of asbestos in laboratory animals, there currently exists an extensive and often acrimonious debate concerning the carcinogenicity of chrysotile asbestos. Because of the continuing extensive sale and use of asbestos in South and Central America, Asia, and Africa and the continued widespread presence of asbestos materials in buildings in the United States and Europe, it is of vital importance that this unnecessary debate be brought to a swift conclusion.

This review considers epidemiologic studies of the health effects of asbestos in populations exposed to commercial chrysotile and to mixtures of chrysotile and other forms of asbestos. We show that evidence for the human carcinogenicity of chrysotile asbestos is incontrovertible. Claims have been made that chrysotile asbestos can be used safely. These claims are not supported by valid data.

The Spectrum of Asbestos Diseases

A seminal study that established much of our current knowledge of the spectrum of asbestos diseases was conducted by Selikoff and Seidman[1] (Table 1). It followed 17,800 heavily exposed asbestos insulation workers for 20 years and demonstrated the wide range of malignancies caused by asbestos. Of these malignant diseases, bronchogenic carcinoma and mesothelioma were the two most important, accounting for more than 90% of the increased cancer risk in this population. Nearly 24% of the deaths in this group of workers were due to bronchogenic carcinoma and 9% to mesothelioma. Lesser degrees of excess cancer mortality were seen at other sites including the esophagus, stomach, colon and rectum, larynx, pharynx and buccal cavity, gall bladder and bile ducts, and kidney. Overall, among insulators whose asbestos exposure began prior to 1968, 30.9% of deaths were excess cancer deaths resulting from one of the above malignancies, additionally another 8.6% of deaths in this population were due to asbestosis.

Although the mortality experience of this unusual group is extraordinary, the insulators were not alone either in their exposure to asbestos or in their risk of asbestos-associated cancer. In 1982 our group made estimates of the expected mortality in the United States from past occupational exposures to asbestos[2]. Considering labor turnover since 1940, it was estimated that approximately 700 asbestos-related deaths occur each year among workers directly installing or removing insulation. Additionally, a much larger population of construction workers was also exposed to asbestos, albeit to lower levels. Because of the much greater number of construction workers, many more asbestos-related deaths are taking place among them. Thus it was estimated that approximately 7000 deaths are occurring each year in current or former shipyard and construction workers and in workers exposed to asbestos through maintaining insulation materials in plants and powerhouses. We estimated that the total mortality in the United States from the asbestos epidemic will exceed 400,000 deaths. Over 95% of the asbestos responsible for this exposure is chrysotile.

Many of the workers who are now dying of asbestos-related disease were exposed to asbestos in concentrations in excess of 10 fibers/ml of air. The current US standard, promulgated in 1994, is 100 times lower, 0.1 fiber/ml. Additionally, procedural standards now require enclosures and the use of air-supplied respirators in circumstances in which release

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Table 1. Observed and expected deaths among 17,800 asbestos insulation workers, United States, 1967-1986*

Underlying cause of death	Observed, (n)	Expected, (n)	Standardized mortality ratio (SMR)
Total deaths, all sites	4951	3453.5	143
Total cancer, all sites	2295	761.4	301
Lung cancer	1168	268.7	435
Pleural mesothelioma	173	0	∞
Peritoneal mesothelioma	285	0	∞
Larynx	18	10.6	170
Oropharynx	48	22	218
Kidney	37	18.9	196
Pancreas	54	39.5	137
Esophagus	30	17.8	168
Stomach	38	29.4	129
Colon and Rectum	121	88.5	137
Gall bladder and bile ducts	14	5.4	261
Noninfectious pulmonary disease	507	144.8	350
Asbestosis	427	0	∞
All other causes	2149	2547.3	84

*From Selikoff and Seidman[1]; with permission. +Based on all available information (autopsy, surgical, clinical, and death certificate).

of asbestos fibers is likely to occur, such as during removal of in-place asbestos. These standards were not in place in years past. If US regulations are followed, asbestos-related disease from future exposures will be sharply reduced. However, workers in other regions of the world, particularly in the developing nations in South America, Asia, and Africa, are not similarly protected. In some of these nations, chrysotile asbestos is not officially recognized as hazardous and controls on its use are often minimal.

Lung cancer

It is widely accepted that asbestos fibers, including chrysotile fibers, increase the existing risk of developing lung cancer in proportion to the cumulative exposure that occurred up to a time 10 years prior to evaluation[3]. The relationship can be expressed formally by the equation $I = I_o (1 + K_L . f . d_{(t-10)})$, where I is the lung cancer incidence or mortality observed in a study population; I_o is the age- and calendar year-specific lung cancer incidence or mortality observed expected in the absence of asbestos exposure (ideally, I and I_o would explicitly consider smoking habits of each study individual); f is the intensity of asbestos exposure to fibers longer than 5 μm; d is the duration of exposure in years up to a period 10 years prior to evaluation; and K_L is a proportionality constant that is a measure of the carcinogenic potency of the asbestos exposure. K_L represents the fractional increase in lung cancer incidence or mortality that occurs from a 1-year exposure to 1 fiber/ml. The relative risk of lung cancer, I/I_o, is independent of age and depends only on exposure characteristics.

Information is available that allows exposure-response relationships between asbestos and lung cancer to be considered in various exposure scenarios. Table 2 summarizes estimates of the percentage increase in lung cancer risk associated with a 1-year exposure to 1 fiber/ml that have been made by individual researchers or by national review groups[3]. There are obviously substantial uncertainties in estimates of K_L. This uncertainty arises first, because only limited data exist on workplace asbestos exposures prior to the mid 1960s. Also when available, the data were usually expressed in particle counts, rather than in fiber counts. The relationship between particle and fiber counts is highly uncertain. When early data were unavailable, estimates of exposure have had to be made, based on later fiber counts and on information on changes in control procedures and exposure conditions over time. These adjustments too, are uncertain. Finally, uncertainties exist in the expected lung cancer risks, which depend on smoking habits and, perhaps, on exposures to lung carcinogens other than asbestos. Despite all these uncertainties, many groups have made estimates

Table 2. Risks of lung cancer and mesothelioma in workers exposed to various asbestos minerals

Asbestos exposure and location	Type of asbestos	Percentage increase in lung cancer for a 1-y exposure to 1 f/ml of asbestos*
Textile manufacturing (South Carolina)	100% Chrysotile	2.6
Textile manufacturing (Rochdale, UK)	98% Chrysotile 2% Crocidolite	1
Textile manufacturing (Pennsylvania)	98% Chrysotile 1% Crocidolite 1% Amosite	1.4
Amosite insulation manufacturing (New Jersey)	100% Amosite	4.3
Insulation application (United States)	60% Chrysotile 40% Amosite	0.8
Asbestos products (United States)	80% Chrysotile 15% Amosite 5% Crocidolite	0.5
Asbestos cement products manufacturing (Louisiana and Ontario)	89% Chrysotile 10% Crocidolite 1% Amosite	1.8
Crocidolite mining	100% Crocidolite	1
Chrysotile mining	100% Chrysotile	0.1

*Details of the calculation of these unit exposure risks are described by the Environmental Protection Agency[3]. **Chrysotile as mined in Canada is contaminated by a small amount of tremolite (generally less than 1%). This contaminant is carried over into fiber used in different industries. The percentages in the column refer to the percentage of commercially sold fiber, including the contamination. ***The percentages of the various fibers used in the plants studied were not given. The estimates are based on published product compositions.

of risk parameters. The results (Table 2) indicate remarkable consistency within an industry and even across industries. The risk of lung cancer per cumulative fiber exposure is very similar for all exposure circumstances except chrysotile mining and milling. Risks in the three chrysotile textile manufacturing studies ranged from 1% to 2.6% per 1-year exposure to 1 fiber/ml. These risks are not statistically different from one another and cannot be attributed to the small use of commercial amphibole asbestos in two of the textile plants. Indeed, the highest lung cancer risk was found in the textile plant that used no commercial amphiboles.

Among the remaining nonmining studies, the percentage increase in lung cancer for each year of exposure to 1 fiber/ml ranged from 0.5% to more than 4%, irrespective of the type of fibers used in the production process. The only exceptions were two studies of friction product manufacturing and one of asbestos cement production. In each of these three studies, severe uncertainties limit the validity of the lower risks reported. All remaining risks, involving substantial amphibole exposure, are similar to those of predominantly chrysotile exposures, within the statistical uncertainties of the data. Even a pure crocidolite exposure in mining demonstrated an increased risk of only 1% for each year of exposure to 1 fiber/ml[7].

Although studies of chrysotile mining and milling demonstrate an excess risk of lung cancer, the risk is more than 10 times lower than that seen in studies of asbestos production workers exposed only to chrysotile or to 97% to 98% pure chrysotile. The origin of this lower risk in the miners and millers is not fully understood. Part of the difference may reflect the different fiber size distributions between the mining and milling operations and the textile plants and other production facilities. Fibers are presumably clumped together and are larger in mining and milling, but more fragmented and smaller in the user industries.

In summary, the available data on workers employed in the production of asbestos products strongly indicate a lung cancer risk for chrysotile similar to that seen in workers exposed to amosite and crocidolite asbestos. The best estimate of lifetime lung cancer risk for worker exposure to chrysotile in the using industries (as opposed to the mining and milling industries), is an increase of 1% in lung cancer risk for each year of exposure to 1 fiber/ml ($K_L = 0.01$). Higher fiber exposure circumstances or longer periods of

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Table 3. Ratio of mesothelioma to adjusted excess lung cancer according to type of asbestos exposure**

Type of exposure	Studies (n)	Mesothelioma/excess lung cancer	
		Pleural cases	All cases
Chrysotile	8	0.13	0.14
Predominantly chrysotile	6	0.24	0.48
Amosite	2	0.13	0.22
Predominantly crocidolite	6	0.47	0.61
Anthophyllite	1	0	0
Talc (tremolite)	2	0	0.08
Mixed exposures	16	0.19	0.4

*From Environmental Protection Agency[7]. **Adjusted to the US male cancer rates in 1970.

exposure would give directly proportional higher risks. In certain textile operations, the unit exposure risk appears to be as much as three times higher than this lower limit. No data are available to indicate the presence of a threshold below which there is no risk from exposure to any asbestos mineral.

Malignant mesothelioma

The risk of mesothelioma by asbestos fiber type can be analyzed in several ways. First, because the risk of lung cancer is very similar across all exposures to all fiber types, excluding mining and milling of chrysotile, one can use the excess number of lung cancers as a measure of cumulative fiber exposure. With comparable follow-up periods, the ratio of the number of mesotheliomas to excess lung cancer is then a measure of the relative risk of mesothelioma at a given level of fiber exposure. Were mesotheliomas produced only by amphiboles, one would expect large differences in the mesothelioma: lung cancer ratio between populations with pure chrysotile exposure and those with extensive amphibole exposure. Table 3 summarizes this ratio by fiber type usage for more than 40 studies.

The ratio of mesothelioma to lung cancer is seen in this analysis to be the same, within statistical uncertainty, for exposures to 100% chrysotile, 97%+ chrysotile, 100% amosite, and mixtures of chrysotile, amosite, and crocidolite. Only 100% crocidolite exposures appear to have a greater ratio, about two to four times above that of predominantly chrysotile exposures. This relatively small difference in the potential for crocidolite to produce mesotheliomas cannot explain the high mesothelioma risk seen in chrysotile exposures that are accompanied by only a very small exposure to crocidolite. Canadian chrysotile that is amphibole-free

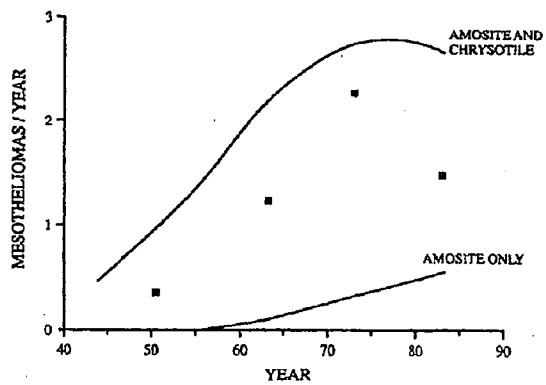

Fig. 1. Estimated and observed cases of mesothelioma/year. 1943–1987 for insulators exposed to amosite alone and amosite/chrysotile. Estimated numbers (smooth curves) are from models of asbestos cancer risk. The solid square represents the number of mesothelioma cases actually observed.

still is asociated with mesothelioma[8]. These data strongly suggest therefore that much of the mesothelioma risk in populations with predominantly chrysotile exposures is due to chrysotile[2].

The mortality risk of mesothelioma from exposure to asbestos can be described by a widely accepted mathematical model (Fig. 1). In this model there is a very low risk of mesothelioma for approximately 20 years from first exposure to asbestos. Thereafter, however, the risk rises very rapidly such that in long-term asbestos-exposed populations mesothelioma may actually become the dominant cancer risk.

Chronologic information is available from two groups of

insulation workers that allows consideration of the contribution of chrysotile to mesothelioma risk. The essence of this analysis is a comparison of the time-course of appearance of mesotheliomas with the time-course of use of (1) chrysotile and (2) other types of asbestos. The analysis takes advantage of the fact that prior to 1935 insulation workers in the United States were exposed only to chrysotile. From 1935 until 1940 they were exposed mainly to chrysotile and only occasionally to amosite. Regular exposure to mixtures of chrysotile and amosite began only after 1940. Because of the strong dependence of mesothelioma risk to time elapsed since exposure, exposures to amosite would not therefore be expected to contribute substantially to a mesothelioma risk in this workforce before the mid-1950s. Observations of mortality from mesothelioma prior to that time would relate directly to chrysotile.

The analysis was undertaken in the 632 members of the New York and New Jersey locals of the Insulator's Union who were members on January 1, 1943[2]. The majority of the group were first employed prior to 1923; for two individuals first employment as an insulator began in 1888. Follow-up of the group has continued from 1943 until the present. Through 1992, all but 40 of the 632 were deceased.

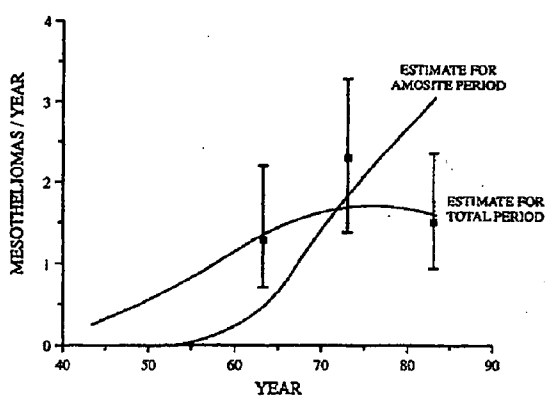

Fig. 2. Estimated and observed cases of mesothelioma/year. 1943–1987. Estimated mesothelioma risk (cases/year) adjusted to yield 56 deaths. The solid squares represent the number of mesothelioma cases actually observed.

The model given above for mesothelioma risk and for estimates of insulation workers' exposure was used to calculate the expected numbers of mesotheliomas for the period from 1943 to 1987 among this cohort. This calculation was done for two periods of time. In one calculation, only exposures subsequent to 1935 were considered ("amosite period") in the other, all asbestos exposures to individuals in the group were considered, from their first exposure to chrysotile asbestos until termination of employment or death ("total period"). The numbers of mesothelioma deaths observed were compared to the number that would be expected if amosite were the sole cause of the disease.

Figure 2 shows the results of these calculations. The principal finding is that the time course of mesothelioma risk is totally incompatible with an exposure pattern that begins only in the late 1930s. Indeed, the 95% confidence limits on three of the four data points do not intercept the distribution that would be expected, were amosite exposure responsible for mesothelioma in this population. Barring undiscovered exposures to amphiboles prior to 1935, these data present strong evidence that chrysotile is the substantial, indeed the dominant contributor to the mesothelioma risk experienced by this group of insulation workers[2].

The study by Selikoff *et al.*[10] of the entire union membership of US and Canadian insulators also strongly indicates the existence of a mesothelioma risk associated with exposure to chrysotile. Follow-up for this study began in 1967, 30 years after the earliest incorporation of amosite by insulation manufacturers into their products. However, the risk of mesothelioma rises steeply with time from first exposure for a period of at least 50 years. Were only amosite contributing to risk, this pattern would not be expected. The pattern relates clearly to chrysotile.

One can also estimate risk of mesothelioma due to chrysotile through direct calculations in mixed-exposure circumstances (Table 4). These analyses show that risk of

Table 4. Risks of mesothelioma in workers exposed to various asbestos minerals

Asbestos exposure and location	Type of asbestos	Risk coefficient
Textile production (Rochdale, UK)	Chrysotile	1×10^{-8}
Insulation workers (United States)	Chrysotile and Amosite	1.5×10^{-8}
Factory workers (Paterson, NJ)	Amosite	3.2×10^{-8}
Miners and millers	Crocidolite	13.4×10^{-8}
Cement workers (Ontario, Canada)	Chrysotile and crocidolite	12×10^{-8}

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mesothelioma/fiber exposure, as measured by K_M, is virtually the same for exposures to 98% chrysotile + 2% crocidolite, 60% chrysotile + 40% amosite, and 100% amosite. The value of K_M from the study of Canadian cement workers is higher than the chrysotile-amosite exposures, as was a value of K_L in the same group of workers. The value for a pure crocidolite exposure, as calculated by de Klerk and Armstrong[7] for the mining population of Australia, is about 10 times greater. As with the values of K_L in Table 1, K_M is not definite because of uncertainties of exposures in the early exposure years of the groups under study and from uncertainties of small numbers. Indeed, from a consideration of the ratios of mesothelioma to excess lung cancer, the mesothelioma potency of crocidolite would appear to be only two to four times greater than chrysotile or amosite mesothelioma potency.

The problem of mesothelioma from exposure to chrysotile continues to the present time. Peto *et al.*[11] predict that deaths from mesothelioma among men in Western Europe will increase from just over 5,000 in 1998 to about 9,000 by the year 2018. In Western Europe alone, past asbestos exposure will cause a quarter of a million deaths from mesothelioma over the next 35 years. The number of lung cancer deaths in Western Europe over the next 35 years[10]. In Sweden, Jarvholm has reported that the number of deaths caused each year by malignant mesothelioma is greater than the number of deaths caused in that country by all workplace injuries.

Current World Uses of Asbestos[13]

Asbestos usage worldwide is extensive, but there have been dramatic changes in the production and use of asbestos in recent years. Table 5 gives representative production data for the past three decades[14]; these data have substantial uncertainties because of limited information on Russian production. The data clearly indicate a rising world production until the late 1970's. After that time a steady decline ensued. Between 1963 and the middle 1970's amphibole mineral constituted from 5% to 7% of the total asbestos production. In subsequent years, the percentage drop in amphibole usage was considerably greater than that for chrysotile. For example, at peak production South Africa mined 269,000 tons of amphibole in 1978, but less than 47,000 in 1991, of which 20,000 was crocidolite. Amosite production ceased in 1992.

Currently over 95% of all asbestos used worldwide is chrysotile, with crocidolite being used only for very specialized purposes. Table 6 shows the 17 top chrysotile consuming nations in 1994[15]. France and other European nations have since banned asbestos from use. The current primary users of chrysotile are countries in Asia and Central/South America. Substantial use also continues in the Middle East and Eastern Europe. While there has been an overall decline in chrysotile asbestos usage, a decline has not occurred in all countries. The decline has been substantial in Western Europe and the United States, but an increase has taken place in Asian nations. Some of these changes with over time can be seen in the data on annual imports and production of asbestos in selected nations (Table 7)[14].

Parallel with the dramatic changes in the global distribution of asbestos, there have occurred changes in its uses.

Table 5. World production of asbestos

Year	Production (tonnes)
1963	2,922,000
1973	4,614,000
1978	5,159,000
1983	4,276,000
1988	4,323,000
1993	2,650,000
1994	2,410,000
1995	2,308,300 (a)
1996	2,140,000 (a)

(a) Chrysotile only.

Table 6. Principal chrysotile consuming countries in 1994

Nation	Annual usage (tonnes)
Commonwealth of Independent States	700,000
China	220,000
Japan	195,000
Brazil	190,000
Thailand	164,000
India	123,000
South Korea	85,000
Iran	65,000
France	44,000
Indonesia	43,000
Mexico	38,000
Columbia	30,000
Spain	29,000
USA	29,000
Turkey	25,000
Malaysia	21,000
South Africa	20,000
Total	2,021,000

Table 7. Annual imports and production of asbestos, in tonnes, in selected countries, by year

Country	1970	1975	1980	1985	1994 (b)
Japan	311,274	261,841	309,305	264,619	195,000
Thailand	21,271	43,024	58,756	75,516	164,000
India	56,008	63,240	99,010	123,000	
South Korea	35,292	56,960	36,787	57,143	85,000
Taiwan	6,589	13,363	31,247	24,519	
Mexico	40,460	60,981		54,871	38,000
France	151,846	138,637	127,123	68,827	44,000
United States	660,000	538,000	162,000	29,000	

(b) Chrysotile only; from reference (15).

Previously, asbestos was used in a wide variety of products. Now, however, asbestos use is largely concentrated in relatively few products, which vary from nation to nation. Worldwide, the dominant use of asbestos is in cement products. For example, in Japan 93% of all asbestos is used in various fire-retardant wallboards and 3.6% in friction products. In developing nations, such as those in Southeast Asia, asbestos-cement pipes are of importance for water systems. South Korea, on the other hand, has extensive asbestos textile and friction product industries and ships the finished products to Japan, West Europe and the United States[16].

There are also differences in national responses to the health hazards of asbestos. Scandinavian and other Western European nations have prohibited all new uses of asbestos. In the United States the permissible exposure level is 0.1 f/ml. In contrast, Japan and South Korea each have a standard of 2 f/ml for chrysotile; amosite and crocidolite use are prohibited in Japan, but lower standards apply in South Korea for amosite (0.5 f/ml) and crocidolite (0.2 f/ml). Generally, developing nations have permissible exposure levels greater than 1 f/ml.

At the international level, the International Labour Organization[17] has published a Recommendation Concerning Safety in the use of Asbestos in which they advocate the banning of new uses of crocidolite and urge controls on the use of other asbestos minerals. They particularly advocate the use of substitute materials and alternative technologies.

With the use of asbestos being predominantly in cement products, a good opportunity for control of workplace and environmental exposures exists. During normal use of such products there is limited release of fibers, and during installation exposures can be well controlled with the use of appropriate dust collectors. However, this may not always be done and workplace monitoring by regulatory agencies is important. Uncontrolled sawing can produce concentrations in excess of ten f/ml.

Conclusion: The Need for an International Ban on Chrysotile Asbestos

Chrysotile is the dominant form of asbestos in use today around the world. Evidence is incontrovertible that chrysotile asbestos is a potent cause of both lung cancer and malignant mesothelioma[3-5]. Data to support this case derive from more than 40 studies of different exposure circumstances. Chrysotile is shown to be a powerful carcinogen when the time course of risk is considered in mixed fiber exposures. Chrysotile is shown also to be a potent human carcinogen in direct calculations of risk. All available data suggest that chrysotile dominates the risk in those circumstances in which it is the principal fiber used. The risk of chrysotile in producing mesothelioma is similar to that of amosite on a per fiber exposure basis. Crocidolite would appear to have a potential two to four times greater to produce mesothelioma for equal exposure than chrysotile[2].

The Collegium Ramazzini, an independent international scholarly society in occupational medicine has called for an immediate international ban on the mining and use of chrysotile and all other forms of asbestos. We strongly support that call.

An immediate international ban on the mining and use of chrysotile and all other forms of asbestos is necessary because the risks cannot be controlled by technology or by regulation of work practices. The strictest occupational exposure limits in the world for chrysotile asbestos (0.1 f/ml) are estimated to be associated with lifetime risks of 2.3/1,000 for lung cancer and 0.8/1,000 for asbestosis[18]. These exposure limits

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can be technically achieved in the United States and in a few other highly industrialized countries, but the residual risks still are too high to be acceptable. In newly industrializing countries engaged in mining, manufacturing, and construction, asbestos exposures are often much higher, and the potential for epidemics of asbestos disease is greatly increased[18, 19].

Scientists and policy makers in these countries should be wary of claims that asbestos can be used "safely". Those claims are not supported by data. Moreover, even the best workplace controls cannot prevent occupational and environmental exposures to products in use or to waste asbestos. Environmental exposure from the continued use of asbestos is also a serious and continuing problem. A recent study of women residing in communities in Canadian chrysotile mining areas found a seven-fold increase in the mortality rate from pleural cancer[21]. Large quantities of asbestos remain as a legacy of past construction practices in many thousands of schools, homes and commercial buildings in developed countries, and are now accumulating in thousands of communities in developing countries.

An immediate international ban on mining and use of all forms of asbestos is necessary because country-by-country actions have shifted rather than eliminated the health risks of asbestos. The asbestos industry has a powerful influence over public policy in many countries. In the United States, the asbestos industry succeeded in 1991 in overturning the EPA's recommended ban and phase-out of asbestos by a technical ruling in the courts. Canada, Russia, and other asbestos-exporting countries have developed major markets in the newly industrializing nations. Conditions of current asbestos use in developing countries now resemble those that existed in the industrialized countries before the dangers of asbestos were widely recognized.

The grave health hazards of asbestos are entirely preventable. The health risks of asbestos exposure are not acceptable in either industrially developed or newly industrializing nations. Moreover, suitable, safe substitutes for asbestos are available. An immediate worldwide ban on the production and use of asbestos is long overdue, fully justified and absolutely necessary.

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EXHIBIT 27

. OF ALABAMA

REVIEW

Mesothelioma: cases associated with non-occupational and low dose exposures

Gunnar Hillerdal

Abstract

***Objectives*—To estimate the importance of low dose exposure to asbestos on the risk of mesothelioma.**

***Methods*—A review of the literature.**

***Results and conclusions*—There is no evidence of a threshold level below which there is no risk of mesothelioma. Low level exposure more often than not contains peak concentrations which can be very high for short periods. There might exist a background level of mesothelioma occurring in the abscence of exposure ot asbestos, but there is no proof of this and this "natural level" is probably much lower than the 1–2/million/year which has been often cited.**

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Keywords: low exposure; asbestos; mesothelioma

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Mesothelioma is an incurable disease which is almost exclusively due to inhalation of asbestos fibres. Asbestos has been extensively used in industry and construction in the 20th century, especially during and after the second world war, and even if the mineral is no longer used in most rich western countries the total world production remains high. There is a worldwide pollution with asbestos, as indicated by the finding of the mineral in samples of Greenland ice[1] and on the Yorkshire Moors,[2] and every citizen in the world has been exposed to some extent. Consequently, asbestos fibres can be found in most lungs at necropsy.[3] It is thus understandable that there is concern about the risk of mesothelioma for the general population.

However, it should be remembered that mesothelioma is a rare disease with incidence in industrialised countries ranging from 1 to 5/million/year among women and values for men 5–10 times higher (see table 3). Even in cohorts with a very heavy exposure to asbestos most people will die from other causes. In people with certified asbestosis—that is, with a heavy exposure—up to 10% will develop mesothelioma; among insulators in the United States and Canada, also a heavily exposed group, 9.3% of the deaths have been due to this disease; and in amphibole miners in South Africa or Australia, this figure is 2–4% (table 1). Clearly, with exposure concentrations several magnitudes lower, as occurs in the general population, the risk is very small, often impossible to measure.

A discussion of the risks from low exposure must include the dose-response curve; the existence or non-existence of a threshold, and thus a background concentration; and should try to define low exposure and estimate to what degree that really means a low concentration. From conflicting findings and opinions attempts must be made to make a meaningful conclusion.

The different types of asbestos seem to differ considerably in their ability to cause mesotheliomas. Chrysotile is considered by many authors to be a weak carcinogen in humans,[11] whereas the two amphiboles crocidolite and tremolite are much more dangerous according to many studies.[12] The third of the more important amphiboles, anthophyllite, was long considered not to cause mesothelioma, but such tumours have now been reported although the risk seems to be small.[13] There is, none the less, a minority opinion that chrysotile is in fact responsible for most of the pleural mesotheliomas in society[14] or should at least be considered to carry the same risk.[15] This discussion, however, falls outside the present review and is not important for the conclusions drawn here.

Table 1 Proportionate mortality from mesothelioma in various cohorts

Cohort	*Deaths (n)*	*Deaths due to mesothelioma (%)*	*Reference*
Amphibole miners	1225	2	4
Crocidolite miners	118	4	5
		6	6
Insulation workers	4951	9	7
Patients with asbestosis	283	9	8
	40	10	9
	59	10	10

Definition and diagnosis of mesothelioma

Mesotheliomas are, by definition, tumours that arise from mesothelial cells and can thus arise from any body cavity: the pleura, the peritoneum, the pericardial sac, and even the tunica vaginalis testis. Pleural mesotheliomas are the most common and pathologically the best defined ones. Pleural mesotheliomas have a male to female rate of about five to one, whereas for peritoneal tumours this ratio is 1.5 to 1.[16] Thus, either the aetiology is different, the

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Table 2 Possible risk factors and mediators of risk of mesothelioma (other than asbestos)

Factors	*Comments*	*References*
Erionite	Very high incidence of mesothelioma environmental exposure in Turkey	19
Chronic inflammation	Pleural scars (tuberculosis, pleurisy, therapeutic pneumothorax)	20
Radiation	Single cases after Thorotrast injection or radiotherapy; causality not proved	21 22
	One case in atomic bomb survivor	23
Beryllium	Two doubtful cases described	18
Vegetable fibres	No proof in humans	
Hereditary factors	Familial cases (explained by common asbestos exposure?)	24–26
	Correlation with parental cancer	27
Immunological factors	Rapidly progressive cases in patients with HIV infection	28 29
Dietary factors	Provitamin A, β-carotene may decrease the risk	30 31
Viruses	Mesotheliomas in animals	
	Simian virus 40 DNA sequences reported in mesotheliomas	32

diagnosis is more difficult in women, or women exposed to asbestos are more likely to develop a peritoneal than a pleural mesothelioma. In fact, one type of mesothelioma occurring almost exclusively in women is the so called multicystic mesothelioma. This tumour has a better prognosis, is more sensitive to cytostatic drugs, and seems to have no connection with exposure to asbestos.[17]

The pathological diagnosis of mesotheliomas requires experience, and confusion can occur with both benign pleural lesions and with metastatic pleural diseases.[18] Many countries now have "mesothelioma panels", which has meant a great improvement in the diagnosis. In most national cancer registries, most likely both overdiagnosis and underdiagnosis occur.

Suggested causes of mesothelioma other than mineral fibres

Apart from mineral fibres, radiation (for example, through the contrast medium Thorotrast) has been suggested to cause human mesothelioma. However, in a large retrospective study of more than 250 000 women treated for mammary carcinoma, a quarter of them initially with radiotherapy, no association between radiation and mesothelioma could be found.[22] As can be seen from table 2, other causes or contributing factors have also been suggested.

Viruses can cause mesotheliomas in animals, but this has not been described in humans. DNA sequences associated with Simian virus 40 (SV 40) transforming factors have been reported in a high proportion of mesotheliomas from some countries.[32] This suggests that there is a connection between SV40, which was a contaminant of live polio vaccines in 1959–61, and later development of mesothelioma. Thus, SV 40 might be a cofactor to asbestos in some patients with mesothelioma, but the results have not been confirmed and are still disputed.

In summary, then, as far as is known today, factors other than mineral fibres can only explain a very small proportion of mesotheliomas, and can for practical purposes be disregarded. Thus, a malignant mesothelioma can be regarded either as caused by asbestos or belonging to a normal background level—that is a spontaneously occurring tumour. The relative imnportance of these two factors has been debated and will be further explored in this review.

Incidence of mesothelioma

There is a large variation in the incidence of mesothelioma in different countries and in most places a steadily rising number of cases with time. In table 3, the incidence or mortality from mesotheliomas in different countries at various times can be seen. As mortality for practical puprposes is the same as the incidence for this disease, both figures have been used in the table. Some of the differences between the countries are probably due to diagnostic difficulties, but most of the variations can be explained by the use of asbestos in the particular society some decades earlier.

Dose-response and latency time

Most researchers agree that there is a positive dose-response curve for mesothelioma—the heavier the exposure to asbestos, the greater the risk. This is found in cohort studies as well as in analyses of amphibole asbestos fibres in the lungs.[45–48] It was realised early that time since first exposure was of great importance, and therefore the "cubic residence-time model" was suggested by Doll and Peto in their report in 1985[49]:

$$I(T) = c \times F \times (T4-(T-D)4$$

Where, I (T)=incidence at the time T after exposure; c=a constant depending on the process, F=intensity of exposure, and D=duration of exposure. This equation has been used in many studies with an acceptable fit for normal occupational exposure concentrations . F in the equation is the total exposure—a combination of fibre concentrations and exposure time—usually measured in fibre-years. (1 fibre-year = a mean of 1 fibre/ml air for 1 working year). Thus, the dose-response curve is supposed to be linear, but the result is heavily influenced by the time factor.

Unfortunately, the exact value of F is often uncertain, even in well defined highly exposed cohorts. The equation is thus rarely useful especially with low doses, in which F is usually a crude guess only. As can be seen from table 1, even with heavy exposure only up to 10% of a cohort will die from mesothelioma—so the formula is not applicable in these cases either. With very heavy exposure, most patients will die from pulmonary insufficiency due to asbestosis before there has been sufficient time to develop a mesothelioma.

Even more troublesome is the time factor T, which quickly becomes very important in this equation. If the equation is correct, the risk

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Table 3 Incidence or mortality of mesothelioma in various countries and areas over time (/ million inhabitants / year)

Country or area	*Year*	*Male*	*Female*	*Reference*
United States	1968–81	2.1	0.8	33
North America	1972	2.8	0.7	34
Nantes-Saint-Nazaire, France	1956–74	5.2	0.2	35
Texas	1976–80	5.8	2.1	36
Selected cities, United States	1970s	4.4–11.1	1.2–3.8	37
United States	1986	7–13	1–2	38
Barcelona, Spain	1983–90	8.3	4.7	39
Great Britain	1968–71	8.4	2.3	40
Finland	1990–94	10	2.9	41
Great Britain	1972–76	12.6	2.8	40
Nantes-Saint-Nazaire, France	1975–84	17.2	0.8	35
United Kingdom	1983	17.5	3.2	16
Denmark	1978–80	14.7	7.0	42
Nantes-Saint-Nazaire, France	1985–92	19.4	4.0	35
Great Britain	1968–71	20.7	4.3	40
Australia	1982–88	28.3	3.3	43
Great Britain	1982–86	30.5	4.9	40
Great Britain	1987–91	44.0	6.4	40
Australia	1994	49.9	4.8	44

would increase steeply with time, making early childhood exposure of great importance.[50] However, there are clear indications that mineral fibres clear from the lung, albeit with different half lifes for the different types of asbestos. Chrysotile has the shortest half life, and crocidolite is generally accepted to have the longest. The half life of crocidolite has been estimated to 7–8 years.[51] This clearing of fibres would, at least theoretically, tend to actually decrease the risk of mesothelioma and other diseases with time.

In conclusion, the value of the cubic residence-time formula is in practice low and it should not be used for extrapolations, at least not at the extreme ends of exposure.

The latency time varies in different cohorts, and is dependent on how long a cohort is followed up. In 370 necropsy cases from Italy, latency time could be calculated in 312.[52] Latency time was also dependent on exposure, varying from 29.6 years for insulators (with the highest exposure) to 51.7 in women with domestic exposure.

The threshold value and the background concentration

There have been strong arguments for the existence of a threshold value (a minimal exposure required for development of a mesothelioma).[53–55] In most studies, several patients with mesothelioma do not report any occupational or other exposure to asbestos,[33 56–60] and thus there seems to be a small spontaneous basal or background incidence of the tumour. In a large study from England, consisting of 185 cases and 159 controls who were very carefully interviewed, 5% of the cases (and 27% of the controls) seemed not to have any kind of exposure to asbestos.[59] This included domestic and even residential exposure. However, it is of course possible that some of these background cases might in fact be due to occupational, domestic, or even environmental exposure, unknown to (or forgotten by) the patients themselves.

There are authors who claim that the presumed background level must be very low, and retrospective searches for the tumour in the medical literature yield no convincing cases of mesothelioma before 1946,[61] although such negative evidence is of questionable value. McDonald and McDonald, in a recent review, estimated the background level to be 1–2/ million/year; they came to this figure by extrapolating backwards from epidemiological studies from various countries.[62]

Malignant mesothelioma can occur in children,[63] and such cases can be considered as proof of non-asbestos (spontaneous) aetiology, as the latency time with necessity must be very short in these cases. Asbestos fibres have, however, been reported in the lungs of children, even in stillborn ones,[64 65] showing that asbestos fibres spread in the human body and even penetrate through the placenta. Even if a latency time of only a few years is extremely rare in mesothelioma related to asbestos,[49] it can occur. There are some published examples of latency times of only 5 years.[66]

Mesotheliomas also occur in animals, from baboons[67] and domestic dogs[68–70] to fish.[71] Dogs are exposed environmentally to asbestos just like their human masters, which might explain some of the tumours,[68 69 72] but in fish it would be difficult to blame asbestos for the tumour. Thus, as in other animals, there is probably a background level of spontaneous mesotheliomas in humans.

Levels of exposure

Although many authors write about low level exposure to asbestos, there is rarely a definition of this term. In fact, in many articles low level exposure seems to be synonymous to non-occupational exposure, which, as described later, is certainly not true in many cases. Occupational as well as non-occupational exposure can be anything from very heavy to very low.

Occupational exposure to asbestos

It must be realised that occupational exposure to asbestos occurs or has occurred not only in the "classic" industries, such as asbestos mines and factories, shipyards, insulating business, asbestos cement industry, building and construction etc, but also in very many other occupations and trades. Examples are pulp and paper industry,[73] oil refineries,[74] electrical industry,[75] jewellery workers,[76] sugar refineries,[77] and cigarette filter workers.[78] Seamen and fishermen can have been exposed to asbestos used as insulation in their boats. In the reprocessed textile industry, bags heavily contaminated with asbestos could be reused for various other purposes, for instance covering heaps of rags; in an Italian investigation of such an industry, mesotheliomas and lung cancer were found to be fairly common among rag sorters.[79]

Given the extensive use of the mineral, many people have been occupationally exposed to asbestos. This exposure can have been only brief but perhaps intense during that short period. In many or most instances the workers have no idea of the exposure and it can be impossible or almost impossible to elucidate it. Also, the level of exposure is often very difficult to estimate, should the information be available.

Non-occupational exposure to asbestos

DOMESTIC EXPOSURE

The family of an asbestos worker could be exposed to considerable amounts of asbestos brought home on his working clothes, which all too often it was the duty of the wife or daughter to clean. Pleural thickening, calcifications, and pulmonary fibrosis have been described in such cases, as well as mesotheliomas.[80–82]

AIR POLLUTION FROM ASBESTOS MINES, FACTORIES, DOCK YARDS, ETC

Asbestos mines used to be great environmental nuisances. They could be seen from a distance because of the dust cloud. Asbestos fibres can spread over a distance many kilometres from a mine, and the tailings from the mines were used for paving roads, parking areas, playgrounds, etc. In the crocidolite mining areas in South Africa, the incidence of mesotheliomas was increased to at least 10 times that expected even among women, proving a non-occupational exposure. However, whether some of this exposure was domestic (see later) is not clear.[83]

Another example is the formerly active crocidolite mine at Wittenoom, Western Australia. At least 5000 people lived in the township of Wittenoom without working in the mines, and in 1993 27 cases of mesothelioma had occurred among these people.[6 48 84] It has been estimated that 1.1% of child residents and 1.9 % of the female residents of Wittenoom have died or will die from mesothelioma, whereas among the workforce this figure would be 6%.[6]

Around the chrysotile mines in Quebec, Canada, there have been at least 53 occupational mesotheliomas from the mines, nine domestic, and two with general environmental exposure only.[85] In the nearby towns, the lungs of residents who have never worked in the mines have a fibre concentration which is 10 times higher than that of the average Canadian.[86]

Mesotheliomas have also been reported from the surroundings of asbestos factories[87] and in people living near dockyards. However, the risk from residential exposure is probably low. In a large study from England, this factor accounted for only 3% of all the identified cases.[54]

CONSUMER GOODS

Asbestos has been introduced into various goods used by the public. Examples are wall paints and spackling and jointing compounds. Another is a hand held hair drier, of which 13 million were sold in the United States and which has been described as a "small asbestos spray gun".[88] In commercially produced Kent filter cigarettes, crocidolite was used in the filters from 1952–6. The sale of these cigarettes was 11.7 billion, and in the commercials the health effects of this filter was emphasised.[78 89]

Once asbestos is introduced into a home, it will spread to all rooms and is almost impossible to remove even with a vaccum cleaner. It will easily be disturbed into the air from the slightest movement, and sedimentation is very slow.[90]

URBAN AIR POLLUTION

The air in cities contains a very low concentration of asbestos. Near to construction or demolition work the concentrations are higher. It has been suggested that the air of London during the blitz was heavy with asbestos. Asbestos is also released on braking, and close to a motorway the fibre concentrations in the air will be higher than the background, but still well below the industrial threshold values. A risk not often appreciated is in dumps. In the vicinity of a waste disposal site the concentration of asbestos fibres can be 10–1000 times above the background concentration.[91]

Drinking water can contain asbestos or asbestiform fibres from natural sources or pollution. The use of asbestos cement pipes can cause a considerable number of fibres in the drinking water. Small amounts of asbestos can be found in some wines, beers, liquors, and other beverages, probably deriving from the filtration process. Fibres that are ingested will to some extent also enter the blood stream as seen from animal experiments.[92 93] In humans, there are few reports. However, when amphibole fibres were found in drinking water from Lake Superior, a very small portion could be traced in urine,[94] and when chrysotile occurred in the drinking water, very small amounts were again found in urine.[95] However, most ingested mineral fibres will never be absorbed but will be cleared in the normal way, and the harmful effects of asbestos in drinking water or drinks is probably minuscule (and much smaller than the risk of drinking the alcohol).

Even if ingested asbestos is not dangerous, it has to be realised that it is only a very small portion of the tap water that we actually drink. Most of it is used for other purposes. Once the water has dried after washing, cleaning, taking showers etc the asbestos fibres will spread in the air. Such amounts are small but not insignificant and will add to the normal background exposure.[96]

ASBESTOS IN PLACE = BUILDINGS CONTAINING ASBESTOS

A mixture containing asbestos was popular after the second world war for spraying on ceilings and walls for insulation and decoration. This was used well into the 1960s. There is now a public danger because of the plaster falling down from natural wear and tear, vandalism, or "artistic" carving in schools, etc. In many industrial buildings, asbestos was also used for spraying on the underside of the roof, and with natural wear and tear (and for instance birds building nests!)[97] there is now release of fibres to the surroundings. Asbestos was also extensively used in walls or around plumbing for insulation purposes, in cement to strengthen it, or just as a cheap filler.

In a modern city, asbestos can often be found in many places: in the cellar, where steam pipes are insulated; in storage or laundry rooms; in air conditioning sets; in theatres, museums, restaurants, etc.[98] Whenever there is damage to any construction or machine that contains asbestos, the possibilities of exposure, sometimes even fairly high, is there.

Table 4 Reported mesothelioma cases after exposure to "asbestos in place"

Occupation or exposure	*Comments*	*Reference*
School teachers	9/487 patients with mesothelioma	99
	1 case each	100–103
	4 cases	104
Attended school		102
Office clerk		102
Female office worker		105
Asbestos insulation at home	6/262 patients with mesothelioma	106

It has been claimed that up to 1000 premature deaths from lung cancer or mesothelioma will occur in the future among school children from schools where asbestos was used in the walls[50]—calculations which, however, had to be built on extrapolations and assumptions. Several case reports have been published on patients with mesothelioma, in which the only exposure to asbestos that was reported was "in place" (table 4). From various cohorts with such exposure, significant increases in radiological findings from the lungs—such as pleural plaques—have also been reported, indicating exposure ot asbestos, but these results are not undisputed and there is a probable overdiagnosis, as control groups are missing.[107]

ENVIRONMENTAL MESOTHELIOMAS FROM LOCAL DEPOSITS OF FIBROUS MINERALS

"Endemic pleural plaques" were first described from Finland and since then many such findings have been reported. In these areas, there are small local pockets of asbestos which sometimes have been quarried, often for generations, for some local use. The most common use is whitewashing of houses with tremolite, which has resulted in an extremely high incidence of mesothelioma in some villages (table 5). When the exposure is due to whitewashing of the houses the risk will disappear when this procedure is stopped, but due to the long latency time this will take many decades.[122]

A non-asbestos fibre, the zeolite erionite, has been found in some Turkish villages. Roads, buildings, etc, can contain this fibre in small amounts. Erionite is even more dangerous than crocidolite and the incidence of mesotheliomas in these unfortunate villages is extremely high.

Endemic plaques are of interest also in other countries, as many people born in these places and living there in their childhood and youth now have moved to other places, taking with them not only the plaques but also the risk of mesothelioma.[131]

Concentrations of exposure

OCCUPATIONAL CONCENTRATIONS

The concentration of exposure which the first workers exposed to asbestos have experienced can only be guessed. Estimated or recreated values from the past suggest fibre concentrations from 25 up to occasional values of 1000–2000 fibres/ml. With his own recalculations, Harries in 1970 estimated the fibre concentrations in the dockyard in 1951 as follows: sprayed asbestos insulation 171–322 fibres/cc; stripping asbestos 334; sweeping 353; adjacent passage 83; and in the passageway to the shower 25. Bagging debris 564; pipe lagging 194–200; removal of pipe lagging 171. Snap samples showed values to up to 1000–2000 fibres/cc.[132] These values are similar to the ones published by McMillan in 1983, who recreated values from the past: engine room 88; delagging in boilers room 171; bagging debris 353 f/ml.[133]

Measurements from working places in the 1960s often showed peak doses of 20 fibres/ml and much less in more recent years. Where asbestos is still used, many countries have adopted a concentration of 1–2 fibres/ml as the upper legal concentration of exposure. These figures should be compared with the few available non-occupational measurements (table 6). A problem with the legal concentration is that most asbestos use today occurs in developing countries, many of which have adopted standards which they cannot enforce. As a result, actual exposures may be much higher than the standard in these countries.

NON-OCCUPATIONAL CONCENTRATIONS

The fibre concentrations in domestic exposure might in fact be as high as in occupational exposure. Brushing clothes might give peaks of ≥100 fibres/ml.[53] Ordinary vacuum cleaning is not effective in removing asbestos fibres, which

Table 5 Local deposits of mineral fibres (asbestos or erionite), occurrence of plaques, and of malignant mesothelioma

Country or area	*Type of fibre*	*Plaques (% of investigated inhabitants)*	*Mesothelioma risk (×10⁶/y)*	*Comment*	*Reference*
Afghanistan	Tremolite	—	—	Case report only	108
Austria	Tremolite	5.3	Not increased	Vineyard and field workers	109 110
Bulgaria	Anthophyllite	2.8 women	Not increased	Tobacco growers	111 112
	Tremolite	5.6 men			
Corsica	Tremolite	41 (>50 y)	High	General pollution	113 114
Cyprus	Tremolite		High	General pollution	115
Czechoslovakia	Unknown	2.7–6.6	—	Farmers	116
Finland	Anthophyllite	6.5–9.0	Not increased		117 118
Greece:					
Metsovo	Tremolite	46.9	280	White washing houses	119–121
			140 (1985–94)		122
SW Aridea, Macedonia		24.2 (>40 y)	High		123
New Caledonia ×2			8.3*	White washing houses	124
			300†		125
South Africa	Amosite crocidolite	2.5–6.6	High	Population around mine	4
Turkey	Tremolite	1.2–25	High	White washing houses	126–128
	Erionite	65		Farmers	19, 128, 129
USSR	Unknown	Locally high			130

*12 cases/145 000 inhabitants/10 y.
†Occurrence in local area.

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Table 6 Fibre concentrations in air and lungs with non-occupational exposure to asbestos

Type of exposure	*Fibres in air (f/ml)*	*Fibers in lungs (fx10⁶/g dry tissue)*	*Reference*
Domestic exposure (paraoccupational)		5.3–319.5	11
		0.0049	102
Near asbestos mines, factories, etc:			
In Wittenoom:			48
When mine operating	0.5		
After closure of mine	0.01–0.21		
Farmers near mine in Canada		1.2–26.8	134
Local asbestos findings:			
Greece:			123
In the yard of an abandoned house	0.01		
In a newly whitewashed room	0,02–17.9		
Corsica	*	21±11	114
New Caledonia:			
While sweeping floor	78		124, 125
Road dust clouds	0.06–0.67		
Asbestos in place:			
Teacher's aid		0.0043	102
Female office worker		31 (TEM)	103

*39 ng/m³ (100 times higher than controls). TEM = transmission electron microscopy.

can remain for years in the house and be airborne again whenever disturbed. Thus, domestic exposure is not low exposure.

Environmental concentrations in the villages where whitewashing occurs, low values are reported when there is no disturbance, but in a newly whitewashed room and while sweeping floors, the concentrations can be quite considerable (table 6).

With asbestos in place, as long as the asbestos is undisturbed the concentrations in the air are very low (zero or hardly measurable: ≤0.001 fibres/ml), but once deterioration takes place values can go up to 15 fibres/ml, and when being removed there can be even higher values. In schools in the United States, the mean concentration was estimated to be 0.003 fibres/ml, and in federal buildings 0.006.[135]

Thus, with so called non-occupational exposure, the typical exposure is a low or very low, almost unmeasurable, background concentration, but occasional high exposure when there is a disturbance of some kind. It follows, firstly, that retrospective estimation of cumulative exposure from history alone is an impossibility in most cases; but secondly, and perhaps more importantly, that any person living or working in (or even temporarily visiting) buildings where asbestos has been used in construction or otherwise might well have been exposed to high concentrations of airborne asbestos fibres once or many times in their lives, and in most instances unknowingly. This includes most of us!

A better way of estimating lifelong exposure might be analysis of fibres in the lungs, but as already mentioned fibres do clear from the lungs. How big the differences in clearance are between people is unknown. Thus, the correlation between lifetime cumulative exposure and fibre concentrations in the lungs is not excellent, but the findings from the lungs probably give a better estimation of exposure than even a careful retrospective analysis of the patient's history, at least in low grade exposure. In most studies, there is a clear dose-response relation between exposure and the number of fibres in the lungs.[11 12 46 48 51]

Fibres in the lungs of patients with non-occupational mesothelioma

As has been mentioned, asbestos fibres can be found in the lungs of the general population without any known exposure. In Germany, the upper normal limit was estimated to be 300 000 fibres/g dry lung. In a large study of 324 malignant mesotheliomas, from which 46 lung samples were available, it was found that even at a fibre concentration of 100 000–200 000 fibres/g, there was a fivefold increased risk of mesothelioma, which was significant.[136] In this study (which has unfortunately only been published in German) as in many others, the mean number of fibres in the lungs of patients with malignant mesothelioma is much higher than the normal values, but there are usually patients with values that lie within the normal level.[25 137–141]

Discussion

Any asbestos fibre found in a lung must have been inhaled. As far as is known, no truly unexposed group can be found in the world. There is no proof of a threshold value—that is, a minimal lower limit below which asbestos fibres cannot cause the tumour—and thus it is plausible that even such low exposure can cause mesothelioma (even if the risk is extremely low). Patients with mesothelioma whose lungs show fibre concentrations within the normal range cannot be dismissed as background cases,—that is, not due to asbestos. The only way to prove such a hypothesis would be to compare the incidence of mesothelioma in a group with such background exposure with the incidence in a truly non-exposed group. This is not possible, as no such group can be found.

It is nevertheless possible that there is a background level of mesothelioma,—that is, that the tumour can occur even in the complete absence of asbestos (or erionite) fibres. However, the data reviewed here indicate that if so, this background level must be very low—probably much <1 case/million people/year. This figure comes from studies of industrialised countries, where background exposure to asbestos is unavoidable. What the true figure is can only be guessed.

What, then, are the consequences for the public health? From the studies of non-occupational exposures it seems probable that the occasional high level exposure situations are the ones that are most important. Although the background, hardly measurable, concentrations of fibres in the air cannot be completely dismissed, the cumulative risk of these exposures is probably minor—and what is more, there is no way to reduce these concentrations. It is the high concentration situations which should be avoided. By knowing where asbestos occurs, such risks could be identified. Any source of pollution by asbestos which releases significant amounts of fibres should be eliminated as soon as it is discovered, using correct equipment and techniques. Correct techniques are also necessary whenever rebuilding or tearing down of structures containing asbestos to avoid asbestos pollution of the environment. If

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the asbestos is well contained and not disturbed, it is usually better to leave it in place. In many instances, encapsulation is also better than removal.[142]

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EXHIBIT 28

Asbestos Exposure Causes Mesothelioma, But Not *This* Asbestos Exposure:

An Amicus Brief to the Michigan Supreme Court

LAURA S. WELCH, MD*

Manufacturers of asbestos brakes, supported by many manufacturing and insurance industry *amicus curie*, requested the Michigan Supreme Court to dismiss testimony of an expert regarding the ability of asbestos dust from brakes to cause mesothelioma as "junk science." Scientists are concerned with the sweeping and unequivocal claims that any conclusion that asbestos from brakes caused a signature asbestos-related disease in a particular person must be "junk science." The manufacturers' sweeping pronouncements are what veer from accepted, reliable mainstream scientific methods and conclusions. This article outlines the evidence supporting the conclusion that asbestos from brakes can and does cause mesothelioma, and describes the defendants' attempts to fabricate doubt about this conclusion. *Key words*: asbestos; brakes; chrysotile; mechanic; occupation; epidemiology; mesothelioma.

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Companies that made and sold asbestos-containing brakes asked the Supreme Court of Michigan to effectively rule that it is impossible to contract asbestos-related diseases as a result of exposure to asbestos from asbestos brakes.† As physicians and scientists, we are concerned about the epidemic of asbestos disease that continues to cause the deaths of thousands of workers each year in the United States. The signers of this paper represent hundreds of years of experience researching, diagnosing, and treating asbestos-related diseases in workers and their families. We have published extensively in this field for more than 30 years and have conducted dozens of epidemiologic and other studies into the issues of asbestos and disease. Many of us have testified before legislative and regulatory bodies regarding asbestos and disease and in court proceedings at the request of individuals suffering from mesothelioma and other asbestos-related diseases.

We make no claim to know the "correct" answer to disease causation in the specific case under review in the court. Our concern is with the sweeping and unequivocal claim that any conclusion that asbestos from brakes has caused a signature asbestos-related disease in a particular person must be "junk science." We find that sweeping pronouncement itself is what veers from accepted, reliable mainstream scientific methods and conclusions.

Ample Evidence Supports the Conclusion That Asbestos from Brakes Can and Does Cause Mesothelioma

Chrysotile causes cancer, including mesothelioma. "There is general agreement among scientists and health agencies . . . [e]xposure to any asbestos type (i.e., serpentine [chrysotile] or amphibole) can increase the likelihood of lung cancer, mesothelioma, and nonmalignant lung and pleural disorders."[1]

Many other reviews support this conclusion, such as those from the American Conference of Governmental Industrial Hygienists,[2] the American Thoracic Society,[3] the Environmental Protection Agency,[4] the International Agency for Research on Cancer,[5] the National Toxicology Program,[6] the Occupational Safety and Health Administration,[7] the Consumer Products Safety Commission (CPSC),[8] the World Health Organization,[9–11] and the World Trade Organization.[12] This scientific consensus is also reflected in the Consensus Report of the 1997 Helsinki Conference,[13] and publications from the American Cancer Society[14] and the National Cancer Institute of the National Institutes of Health.[15]

Thorough scientific inquiry requires consideration of all available information. Accordingly, in reaching the conclusion that chrysotile asbestos causes mesothelioma, scientists properly consider numerous accepted sources of scientific data, including epidemiologic studies of all varieties, case reports and series of case reports, controlled animal experiments, and toxicologic studies.[1,16–23]

Asbestos industry arguments to the contrary have not been supported over time. Chrysotile asbestos mining companies and manufacturers have argued for more than 30 years either that their products do not cause disease or that there is insufficient evidence to reach a reliable conclusion. Numerous scientific articles and criticisms have specifically exposed the artificial uncertainty cre-

*Dr. Welch is joined by 51 other signers onto this communication; see end of document.

†*Chapin v. DaimlerChrysler Corporation et al.*, Case No. 133178, Supreme Court of Michigan.

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ated by the proponents of chrysotile asbestos, and their position has been repeatedly and consistently rejected by the mainstream scientific and regulatory communities.[18–20,24–26]

Like many scientists, we are concerned with the development and expansion of "doubt science."[27,28] A centerpiece of the "doubt science" model is the assertion that whatever piece of evidence supports the position of the industry in question (or whatever piece of evidence might be as yet undetermined) is *the* critical piece of evidence, to the exclusion of all others. While we acknowledge that industry-sponsored research can and does often provide valuable scientific insight and developments, the efforts of the tobacco and asbestos industries to deny their products cause cancer have become a paradigm for "doubt science."

In this regard, we are cognizant of the fact that the primary articles upon which the asbestos brake manufacturers rely in this matter were paid for by Ford, General Motors, Chrysler and other asbestos brake manufacturers. Publications by Hessel,[29] Goodman,[30] and Paustenbach[31,32] were all expressly funded by Ford, General Motors, and Chrysler. Furthermore, the paper by Hessel et al. appeared in a journal funded by the Ford Motor Company and a subsidiary of General Motors. Wong[33] has been reported to have undisclosed origins as an expert witness report for a brake manufacturer.[34] Laden acknowledges funding by a law firm that is "national asbestos counsel" for another asbestos brake manufacturer.[35]

The Scientific Community is in Consensus that Even Brief and Low-level Exposure to Asbestos Can Cause Mesothelioma

The mainstream scientific community has long recognized and continues to recognize today that there is no "safe" level of exposure to asbestos.[12,13] As noted by NIOSH:

> Excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a "safe" level of asbestos exposure.[36]

Attempts to postulate thresholds for exposure have been dismissed as "logical nonsense."[37]

The lack of a defined "safe" level for exposure to asbestos has been supported by subsequent research. For example, a large French study recently concluded that substantial excess mortality occurs at exposure levels below current regulatory levels.[38] A recent study examining the relationship between historical asbestos use and disease rates further supports the conclusion that a linear dose–response relationship exists between exposure to asbestos and disease and that no "safe" level of exposure exists.[39]

One of the main studies upon which the asbestos brake manufacturers rely[40] similarly concluded that *all* levels of occupational exposure to asbestos increase the risk of mesothelioma:

> Compared to those who never worked or who were never exposed, all levels of probability and intensity [of exposure to asbestos] had an increased significant risk, except subjects with low probability of exposure. For exposure classified as "sure" the OR was 13.2.

Application of this study to the current case under review would result in his placement in the "sure" exposure category, and consequently he would be over 13 times more likely to contract mesothelioma than unexposed individuals. Despite this, the asbestos brake manufacturers assert that the Agudo study proves that no person can ever get mesothelioma from asbestos brakes. That argument is unsound and contrary to the consensus of the scientific community that there is no demonstrable threshold of exposure to asbestos below which adverse health effects do not occur. Accordingly, "an occupational history of brief or low-level exposure should be considered sufficient for mesothelioma to be designated occupationally related" to asbestos exposure.[13]

Mesothelioma Is a Signature Malignancy for Asbestos Exposure

There is no debate that asbestos causes mesothelioma, and that the great majority of mesotheliomas are demonstrably caused by asbestos.[41] Some mesotheliomas are never able to be individually linked to asbestos exposure, and the scientific community has defined these cases as "idiopathic" because information regarding asbestos exposure is unavailable.

However, we know that many individuals do not know that they have been exposed to asbestos.[42] Many more die before being interviewed regarding potential exposures, forcing researchers to make assumptions about exposure based upon information from next of kin, job titles, or death certificates; these sources often fail to reflect all jobs and exposures.[43,44] Many epidemiologic studies assess occupational exposure but not para-occupational or environmental exposure, because only occupational information is available from existing records. The fact that a percentage of mesotheliomas are labeled "idiopathic" does not, however, support the conclusion that there are large numbers of spontaneous (i.e., non–asbestos-related) mesotheliomas. To the contrary, a large study of numerous sources of information failed to demonstrate evidence for "spontaneous" mesotheliomas,[45] and a detailed review of mesothelioma cases in Australia found that over 90% had either a history of exposure or substantial asbestos in lung tissue.[42]

The asbestos brake manufacturers attempt, without support, to recast the definition of "idiopathic." First,

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in an attempt to undercut the indisputable link between asbestos and mesothelioma, they suggest that "idiopathic" mesotheliomas are not caused by asbestos rather than accepting that these are cases where individual exposure has not been identified. Second, they attempt to place mesotheliomas with demonstrable occupational exposures to asbestos—specifically asbestos from brakes—in the "idiopathic" category. There is no scientific support for either position.

Mesothelioma is a signature tumor for asbestos exposure. Individuals with known occupational exposures to asbestos cannot be recast into the "idiopathic" or "unknown exposure" category. When confronted with an individual who has a demonstrated mesothelioma and demonstrated occupational exposure to asbestos, the mainstream scientific community recognizes that the cause of that mesothelioma is the asbestos exposure of the individual even if that exposure was "brief or low-level."

Because Mesothelioma Is a Signature Malignancy with Essentially One Cause—Asbestos—the Scientific Community Has Long Considered Individual Cases of Mesothelioma to Be Sentinel Events

It is not necessary to have an epidemiologic study of a specific occupation to be able to conclude that an individual's exposure to a toxic substance in that occupation can be a cause of disease. To the contrary, as noted by Dr. Lemen,

> Specific occupations do not need to be studied nor do epidemiological studies need to be performed to show risk of disease before prevention actions are taken or causal connections concluded. To wait for epidemiology studies of each occupational group is not warranted but has been taken by many in the medico-legal profession as the only way to prove causation by occupation. Such misconceived thinking has been very harmful to the future prevention of asbestos-related diseases.[46]

This is particularly so when examining mesothelioma. Repeated studies have shown that all levels of exposure increase the risk of mesothelioma.[38,40] Moreover, unlike many other cancers, for which there are multiple, well-documented causal factors, mesothelioma is overwhelmingly caused by asbestos. As noted by one of the studies upon which the asbestos brake manufacturers rely:

> Mesothelioma is a rare cancer with one major etiologic exposure, therefore surveillance using each case as a sentinel event might seem more reasonable for this disease than for cancers with multifactoral causation.[47]

In 1983 Rutstein developed a list of sentinel health events (SHE-O) that are occupationally related.[48] Mesothelioma as a sentinel disease for asbestos exposure was on the initial list of SHE-O, and all subsequent revisions. In fact, the worldwide acceptance of mesothelioma as an asbestos-related cancer began with the case series published by Wagner in 1960.[49]

When examining the question of causation of sentinel diseases such as mesothelioma, the scientific community recognizes that case reports and case series reports are useful and valid tools.

> Case series are particularly informative in situations where there are identified occurrences of very rare conditions for which there are few, if any, established causal factors. . . . In fact, recognition of even a small number of cases of the "sentinel" diseases—such as liver angiosarcoma and malignant mesothelioma, which is strongly related to asbestos exposure.[50]

The scientific community has concluded that, for sentinel diseases such as mesothelioma, case series reports can be sufficient by themselves to allow reliable conclusions to be drawn regarding causation. Again, as noted by Checkoway:

> Case series reports can be virtually conclusive in their own right when the health outcome is a very rare disease or an uncommon manifestation of a relatively common condition.[50]

We do not suggest that such conclusions are indisputable or inviolate; scientific knowledge rarely is. The relevant question is whether reliable and scientifically justifiable conclusions can be drawn based upon such information, when considered in connection with all other available evidence. They can. In fact, proper application of the scientific method requires consideration of all forms of available evidence.

Accepted Method for Evaluating Disease Causation in an Individual: Generally and as Applied to Asbestos Exposure and Mesothelioma

Examining the question of causation of disease in an individual generally involves four questions: 1) was the individual exposed to a toxic agent 2) does the agent cause the disease present in the individual; 3) was the individual exposed to this substance at a level where disease has occurred in other settings; and 4) have other competing explanations for the disease been excluded?

There is no reasonable dispute regarding Question 2—asbestos causes mesothelioma. Additionally, there are no well-accepted competing explanations regarding mesothelioma that must be excluded, resolving Question 4. As a result, when considering the issue of causation of a mesothelioma, once an occupational or para-occupational exposure to asbestos has been established (Question 1), the sole question remaining for

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examination is whether the exposure or set of exposures of that individual is similar to exposures that have been documented to cause mesothelioma in others—Question 3.

The mainstream scientific community is in consensus regarding the resolution of Question 3. As discussed above, there is no safe level of exposure to asbestos. Even exposure at current regulatory levels results in excess mesotheliomas.[7,38] Accordingly, the consensus of the scientific community is that any occupational or para-occupational exposure to asbestos—even "brief or low-level exposures"—must be considered causal in an individual with a mesothelioma.

The Claim of the Asbestos Brake Manufacturers That the Studies upon Which They Rely Trump All Other Scientific Knowledge Is Scientifically Unsupportable.

The asbestos brake manufacturers cite a number of epidemiologic studies as proof that asbestos from brakes cannot cause mesothelioma. The manufacturers claim the fact that these studies did not detect a statistically significant increased risk of mesothelioma in the occupational groups studied is conclusive proof that no person can ever contract disease from working with asbestos brakes.

That claim is simply not scientifically supportable. We need not examine here the individual shortcomings of the studies relied upon by the asbestos brake manufacturers. Others have done so cogently and in detail.[16,34] While our rejection of the asbestos brake manufacturers' sweeping claim is supported by these critiques, the fundamental scientific failing of their claim is not based on the obvious limitations of the individual studies. There is a difference between a truly negative result and a non-positive result. A true negative study must be large, sensitive, and contain accurate exposure data. Even then, the study will be negative only with respect to the exposure level studied. Far from proving that no person can ever get sick from asbestos dust released by brakes, the best that can be said for the studies is that they are inconclusive. Instead, such a claim is based on the scientifically unsupportable proposition that one study, or group of studies, trumps all other evidence, no matter how extensive and well-documented that evidence is. Additional discussion of the implication of "negative" epidemiologic studies may be found elsewhere.[51–54]

As noted above, examination of the question of whether a substance is capable of causing disease requires consideration of all scientific disciplines and all available evidence. This is particularly true when asserting that exposure can *not* cause an effect.

> The conclusion that some exposure is devoid of harmful effect (e.g. a certain chemical is not carcinogenic) must be based on a synthesis of the whole available literature: it can never rely on one single study. Hence, all the scientific evidence (i.e. theoretical experimental, and epidemiologic) that exists must be combined.[55]

Substantial insight into this issue is provided by the industry consultants hired by Ford, General Motors, and Chrysler. When hired to represent the auto industry in asbestos-brake litigation, the industry consultants assert that their cited epidemiologic studies trump all other evidence, and conclusively refute the claim that asbestos from brakes can cause mesothelioma.[29,30] Conversely, when hired by the power industry to provide testimony regarding epidemiologic studies that were damaging to that industry, Dr. Hessel rejected this same position:

> Because of such recognized limitations, epidemiology studies by themselves generally do not provide sufficient basis to support conclusions about causation. That is why the assessment of health risk must rely on data from toxicological studies in animals, studies in human cells and tissues and experimental clinical studies.[56]

The opportunistic rejection of whatever evidence exists contrary to the position of the industry being defended is a hallmark of "doubt science." We disagree with both extremes. Epidemiologic evidence may, in cases, be sufficient to make reasoned and well-founded judgments regarding causation after consideration of other available evidence, even if evidence from one or more other scientific disciplines is absent. Conversely, consideration of other scientific evidence may allow reasoned conclusions regarding causation in the absence of positive epidemiologic studies regarding a specific population.

It is unscientific for the asbestos brake manufacturers to assert that their chosen epidemiologic studies trump all other evidence, just as it was unscientific for the tobacco industry to claim that lack of understanding of the mechanism by which tobacco causes cancer made it impossible to conclude that cigarettes cause cancer. Proper application of the scientific method requires that all available evidence be considered when examining issues of causation.

Evidence Supporting the Conclusion That Asbestos from Brakes Can and Does Cause Disease, Including Mesothelioma

The danger of asbestos in brakes has been recognized for decades. The hazard from exposure to asbestos in friction products has been known and accepted for over 70 years. In 1948, General Motors' chief industrial hygienist published regarding the hazards created when manipulating asbestos brake materials in the factory.[57] By 1958, the danger of exposure to asbestos dust from brakes was sufficiently well documented that it was

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included in the American Industrial Hygiene Association's Hygienic Guide series.[58] Additionally, mesotheliomas have been documented repeatedly in workers at friction-product factories.[34,59–62]

Today, the asbestos brake manufacturers assert that this danger is confined to the friction-product manufacturing facility. However, there is no scientific justification for asserting that dust from an asbestos brake can cause disease when the brake is ground in a factory but cannot cause disease when that same brake is ground in a garage.

Mechanics who work with asbestos brakes without dust-control measures are exposed to asbestos. Numerous studies have demonstrated that mechanics who worked with asbestos-containing brakes without dust-control measures were exposed to asbestos dust. This is particularly true when the mechanic grinds, files, or sands the new asbestos brake and uses compressed air or dry brushing to clean out wear dust from old asbestos brakes.[63–66] Both the EPA and OSHA have issued guidance to reduce the risk of disease from asbestos exposure during brake work.[7,67,68] OSHA requires the use of dust controls when employees work with asbestos-containing brakes and clutches (for specific details see appendix F of the standard).[7] The EPA has adopted these standards for municipal employees in jurisdictions not governed by state asbestos-control plans.[67] Other regulatory agencies have similarly issued guidance to mechanics to reduce exposures to asbestos from brakes and clutches.[69–72]

Equally important, it has been proven that use of effective dust-control measures can lower exposure levels during work with asbestos brakes.[73,74] Accordingly, to provide a reliable basis for the conclusion that asbestos from brakes can never cause disease, a study of brake mechanics would ascertain whether individuals considered "exposed" to asbestos brakes used dust-control practices. None of the studies relied upon by the asbestos brake manufacturers contains such information.

Studies have shown increased incidences of non-malignant asbestos-related diseases among mechanics known to have performed work with asbestos-containing brakes. Excessive non-malignant disease in mechanics occurs in individuals known to have worked with asbestos-containing brakes.[65,75] It is universally accepted that the amount of asbestos exposure needed to cause asbestosis is greater than the amount needed to cause mesothelioma. Accordingly, studies demonstrating excess asbestosis in asbestos brake-repair workers demonstrate that these workers were historically exposed to quantities of asbestos far in excess of that needed to cause mesothelioma.

Proper scientific inquiry cannot ignore the hundreds of reported cases of mesothelioma in mechanics. We reject the contention of the asbestos brake manufacturers that the scientist must close his or her eyes and refuse to consider case reports or case series of mesothelioma in mechanics, irrespective of how many cases are reported. This contention flies in the face of sound scientific reasoning, which *requires* thoughtful consideration of *all* available evidence.

As discussed above, the consideration of case reports is even more critical when examining rare, sentinel diseases such as mesothelioma because of the great difficulty in conducting epidemiologic studies with sufficient power to reliably detect increases in disease.[53,54] Hundreds of cases of mesothelioma in mechanics have been reported in the medical literature, including dozens of cases in the studies relied upon by the asbestos brake manufacturers.[16]

The precise number of cases is not important for purposes of our discussion, nor is the possibility that some cases may have involved exposures to asbestos from sources other than brakes. The important point is that proper scientific inquiry not only can consider these reports, but, in fact, must consider them. Contrary to the suggestion of the asbestos brake manufacturers, these cases cannot be cavalierly dismissed as "unscientific" or "insufficient to support conclusions regarding causation." When considering the important question of whether working with asbestos-containing brakes can cause incurable, inevitably terminal diseases, such as mesothelioma, case series must be considered and evaluated, along with all other available evidence.

There is nothing novel regarding the use of Sir Austin Bradford Hill's viewpoints to arrive at the conclusion that asbestos from brakes can cause disease. Application of his viewpoints has been an accepted and valid method for examination of questions of causation for decades and remains so today.[76] His own wise words are worth repeating:

> Here then are nine different viewpoints from all of which we should study association before we cry causation. What I do not believe—and this has been suggested—that we can usefully lay down some hard-and-fast rules of evidence that must be obeyed before we can accept cause and effect. None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a sine qua non. What they can do, with greater or less strength, is to help us make up our minds on the fundamental question—is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?

Contrary to the all-or-nothing position of the asbestos brake manufacturers, there is no single scientific discipline or type of study that takes precedence over others. Thoughtful scientific inquiry requires consideration of all evidence when making determinations regarding causation.

CONCLUSION

Asbestos causes mesothelioma. Mechanics are exposed to asbestos dust during the servicing and replacement

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of brakes. While the asbestos brake manufacturers claim that the average amount of asbestos released from brake repair work is comparatively low, there is no reasonable dispute that exposure levels were higher when mechanics routinely ground, filed, and sanded brakes and used compressed air to blow out brake wear debris, and did this work without dust control. It is those historic higher exposures that caused disease appearing now. The scientific community is in consensus that brief and low-level exposures to asbestos can cause mesothelioma. The scientific literature contains hundreds of cases of mesothelioma among brake mechanics; and epidemiologic studies of mechanics known to have performed repair work on asbestos-containing brakes have demonstrated increased levels of nonmalignant diseases.

This combination of evidence, and the vast amount of additional scientific information regarding asbestos and mesothelioma, provides more than sufficient evidence to allow someone to conclude within a reasonable degree of scientific certainty that a mesothelioma in a mechanic who worked with asbestos-containing brakes was caused by that asbestos exposure.

Since 2000, Ford, General Motors, and Chrysler have paid over $30,000,000 to hire consultants for the purpose of generating the very papers they rely upon, and for testifying regarding those papers in Courts.† One of the main industry experts has acknowledged that the papers were conceived and authored for the purpose of buttressing testimony in court cases involving mechanics suffering from mesothelioma.‡

The same expert also acknowledged that this business model is a pattern he has also followed with dioxin, benzene, hexavalent chromium, beryllium, formaldehyde, and glycol ethers. Recent revelations regarding undisclosed involvement of the employer of these experts in connection with publication of a paper favorable to the chromium industry have been well publicized and led to the retraction of that paper.[77,78] It is in no way surprising that the experts and papers financed by these manufacturers conclude that asbestos in brakes can never cause mesothelioma. To the contrary, the exoneration of the sponsoring industry is the *expected* conclusion of doubt science. Despite the best efforts of the asbestos brake manufacturers and their hired experts to fabricate scientific uncertainty where none exists, the mainstream scientific community and regulatory communities have considered the available evidence and concluded that the danger to mechanics from asbestos in brakes is real.

Apparently, the asbestos brake manufacturers hope that these arguments can be used to sway the Supreme Court of Michigan and other courts. As scientists who have devoted substantial portions of our professional lives working to research, prevent, and treat asbestos-related diseases, we reject these attempts to fabricate uncertainty where none exists. Instead, we request that these courts attend to the work of thousands of experts from around the world who have concluded that asbestos, in any form, and through any occupational exposure, can and does cause disease.

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†Ford, General Motors, and Chrysler have admitted in litigation that, since 2000, they have paid over $30,000,000 to these experts. See, Ford and General Motors, Answers to Interrogatories, *Unden v. General Motors*, Case No. 05:6311, Circuit Court for Hillsborough County, Florida, and Chrysler IRS Form 1099s produced in litigation.

‡Deposition of Dennis Paustenbach, July 1, 2005, *Mallia v. Bennett Auto et al.*, Case Number 04-16236 CA 42, Circuit Court in and for Dade County, Florida.

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EXHIBIT 29

IN THE SUPREME COURT OF PENNSYLVANIA

NO. 56 EAP 2014

JOYCE ROST EXECUTRIX OF THE ESTATE OF RICHARD ROST & JOYCE ROST,

Plaintiffs-Appellees,

v.

FORD MOTOR COMPANY,

Defendants-Appellants

BRIEF OF MUGE AKPINAR-ELCI, MD, MPH, PROF. DR. MED. XAVER BAUR, CARLOS BEDROSSIAN, MD, PHD(HON), FIAC, EULA BINGHAM, PHD, YV BONNIER-VIGER, MD, MSC, MM, CMSQ, FRCPC, JAMES BROPHY, PHD, MASSIMILIANO BUGIANI, MD, BARRY CASTLEMAN, SCD , RICHARD CLAPP, DSC, MPH, DARIO CONSONNI, MD PHD, EMILIE COUNIL, MOHAMED AQIEL DALVIE, BSC, HONS, MSC, PHD, JOHN M. DEMENT, PH.D., CIH, TONY FLETCHER, PHD, PROFESSOR BICE FUBINI, THOMAS H. GASSERT, MD, MSC, DAVID F. GOLDSMITH, MSPH, PHD, MICHAEL GOCHFELD, MD, PHD, LENNART HADELL, MD, PHD, JAMES HUFF, PHD, PETER F. INFANTE, DDS, DRPH, MOHAMED F. JEEBHAY MBCHB, MPH (OCC MED), PHD, T. K. JOSHI, FRCS, FFOM, MARGARET KEITH, PHD, JOHN R. KEYSERLINGK, MD, MSC, FRCS(C), FACS, KAPIL KHATTER, MD, CCFP, MES, SHIRA KRAMER, MHS, PHD, PHILIP J. LANDRIGAN, MD, MSC, FAAP, BRUCE LANPHEAR, MD, MPH, RICHARD A. LEMEN, PH.D., MSPH, CHARLES LEVENSTEIN, PHD, MS, ABBY LIPPMAN, PHD, GERALD MARKOWITZ, PHD, DARIO MIRABELLI, MD, SIGURD MIKKELSEN, MD, DRMEDSCI, CELESTE MONFORTON, DRPH, MPH, RAMA C. NAIR, BSC, MSTAT, MSC, PHD, FACE, L. CHRISTINE OLIVER, MD, MPH, MS, DAVID OZONOFF, MD, MPH, DOMYUNG PAEK, MD, MSC, SCD, SMITA PAKHALÉ, MD, FRCPC, MSC , ROLF PETERSEN, PHD, BETH ROSENBERG, SCD, MPH, KENNETH ROSENMAN MD, FACE, FACPM, DAVID ROSNER, PHD, CRAIG SLATIN, SC.D., MPH, MICHAEL SILVERSTEIN, MD, MPH, COLIN L. SOSKOLNE, PHD ,LESLIE THOMAS STAYNER, PHD, KEN TAKAHASHI, MD, PHD, MPH, DANIEL THAU TEITELBAUM, MD, BENEDETTO TERRACINI, ANNIE THÉBAUD-MONY, FERNAND TURCOTTE, MD, MPH, FRCPC, ANDREW WATTERSON, PHD, CFIOSH, DAVID H. WEGMAN, MD, MSC, LAURA S. WELCH, MD, PROF. EM. DR. MED. HANS-JOACHIM WOITOWITZ AS *AMICI CURIAE* IN SUPPORT OF APPELLEES

Appeal From the Judgment of the Superior Court of Pennsylvania Entered on May 19, 2014, at No. 404 EDA 2012, Affirming the Judgment Entered December 28, 2011, in the Court of Common Pleas, Civil Division, Philadelphia County at No. 1978 September Term, 2010

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NO. 56 EAP 2014

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Plaintiffs-Appellees,

v.

FORD MOTOR COMPANY,

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STATEMENT OF SUBJECT MATTER AND JURISDICTION

Amici adopt Appellees Statement of Subject Matter and Jurisdiction.

STATEMENT OF THE ORDER IN QUESTION

Amici adopt Appellees Statement of the Order in Question.

STATEMENT OF THE SCOPE AND STANDARD OF REVIEW

Amici adopt Appellees Statement of the Scope and Standard of Review

STATEMENT OF THE QUESTION INVOLVED

Amici adopt Appellees Statement of the Question Involved.

STATEMENT OF THE CASE AND FACTS

Amici adopt Appellees Statement of the Case and Facts.

STATEMENT OF INTEREST

Our[1] understanding as to the nature of the issue before this Court is whether, based upon a review of all the evidence in the record, there was sufficient data to support the jury's finding that exposure to Ford's asbestos products was a substantial contributing factor in the development of Mr. Rost's mesothelioma. Ford and manufacturers of asbestos brakes contend that because Dr. Frank testified that every exposure contributes to the dose, that there can be no admissible evidence of causation.

[1] A list of the Amici and their affiliations is attached as Exhibit A. Their titles and affiliations are given for identification purposes only.

We, as physicians and scientists, remain concerned about the ongoing epidemic of asbestos disease that causes several thousand preventable deaths each year in the United States alone. The signers of this paper collectively possess hundreds of years of experience researching, diagnosing, and treating asbestos-related diseases in workers and their families. We have published extensively in this field for more than 40 years and have conducted dozens of epidemiological and other studies into the issues of asbestos and disease. Many of us have testified before legislative and regulatory bodies regarding asbestos and disease and in court proceedings at the request of individuals suffering from mesothelioma and other asbestos-related diseases.[2]

Scientists are concerned because it is indeed an irrefutable scientific fact that each exposure does actually contribute to a person's total dose and to that person's risk or probability of developing mesothelioma and other cancers. This is scientific fact. Each individual fiber itself may not necessarily be a significant cause of the mesothelioma in question, but each does necessarily contribute to a person's total dose. Therefore, if this Court were to state that Dr. Frank's concurrence with this axiomatic scientific principle so infects the record that there

[2] This paper is being signed by 58 physicians and scientists who reviewed the document, support the contents and asked to have their name listed. The signers have received no compensation for their participation in this paper.

can be no causation, this holding will have the absurd result of allowing any asbestos defendant to escape liability merely by laying the "trap" of posing the question: "Does every exposure contribute to the dose," a question with which no credible expert could disagree.

Second, scientists are concerned that Ford and the manufacturers of asbestos brakes ask this Court to ignore the fact that Dr. Frank employed the generally accepted scientific methodology of reviewing the diagnosis, medical and occupational history, individual susceptibility, biological plausibility, and relevant case reports, case series, and epidemiological studies in order to testify as to whether Mr. Rost's exposure to asbestos from Ford's brakes and clutches were substantial factors contributing to Mr. Rost's disease. To require more than this would defy long standing generally accepted scientific principles of etiology.

SUMMARY OF ARGUMENT

There is a clear difference between the undeniable scientific fact that each exposure contributes to the total dose, versus stating that "one-fiber" or some infinitesimally small exposure to asbestos is a significant or appreciable contribution to causation. While the "one fiber" or "one hit" scientific concept may indeed be true on some theoretical plane, that is not what happens in the real world of asbestos exposures. Diverting attention from an expert testifying about

real world significant exposures by claiming that the case is based on the theory that any exposure/single fiber is a substantial contributing factor is, at best, a clear misunderstanding of the actual testimony, and at worst, intellectual dishonesty and an attempt to deceive this Court. Cumulative exposure (e.g. every exposure contributes to the dose, cumulatively) is a corner-stone of medicine and science; what makes for a significant exposure is a different topic altogether.

It is axiomatic that every exposure to asbestos, while not necessarily a substantial contributing factor, contributes to the total dose. An expert's concurrence with this scientific fact should not "infect" his testimony such that his causation opinion is negated, especially where, as here, the expert employed the generally accepted method of a multi-faceted review of the diagnosis, medical and occupational history, individual susceptibility, biological plausibility, and relevant case and epidemiological studies.

ARGUMENT

I. IT IS A SCIENTIFIC FACT THAT EACH EXPOSURE CONTRIBUTES TO THE TOTAL DOSE.

It is an irrefutable scientific fact that each exposure, while not necessarily a significant cause of the mesothelioma in question, necessarily contributes to a person's total dose. Specifically, there is a clear difference between the undeniable scientific fact that each exposure contributes to the total dose, versus stating that

"one-fiber" or some infinitesimally small exposure to asbestos is a significant or appreciable contribution to causation.

While the "one fiber" or "one hit" scientific concept may indeed be true on some theoretical plane,[3] that is not what happens in the real world of asbestos exposures. Diverting attention from an expert testifying about real world significant exposures by claiming that the case is based on the theory that any exposure/single fiber is a substantial contributing factor is, at best, a clear misunderstanding of the actual testimony, and at worst, intellectual dishonesty and an attempt to deceive this Court. Cumulative exposure (e.g. every exposure contributes to the dose, cumulatively) is a corner-stone of medicine and science; ***what makes for a significant exposure is a different topic altogether.***

It is our understanding that the legal test employed in this and some other jurisdictions already requires that the Plaintiff present evidence of the frequency, proximity and regularity of exposure to the defendant's products. Given this requirement, and given the reality that occupational and para-occupational

[3] The "one hit" hypothesis for cancer genesis is that each molecule of a cancer causing substance has a very tiny but not zero chance of inducing via mutation one fully cancerous cell that leads to a cancerous tumor. It is one possible explanation on how cancers might form and would provide some additional biological explanation for how the low dose cancers caused by radiation or asbestos occur. It may or may not ultimately be proven to be true, but it is a separate issue from determining what constitutes a significant exposure. Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology, in* Reference Manual on Scientific Evidence, 642 (Federal Judicial Center, 3rd ed. 2011).

exposures to asbestos involve billions or trillions of fibers[4], the suggestion that an expert who testifies that frequent, regular, and proximate exposure to an asbestos product is a substantial contributing factor is relying on the "any exposure/single fiber" theory is without basis in fact or logic.

II. A MULTI-FACETED DIAGNOSTIC ANALYSIS IS THE GENERALLY ACCEPTED METHOD FOR ASSIGNING SUBSTANTIAL FACTOR CAUSATION.

Ford's and the industry advocates' erroneous position is that the "only" method used by Dr. Frank was that "any exposure/single fiber" is a substantial contributing factor. On the contrary, Dr. Frank used a multi-faceted generally accepted methodology studying exposure history, individual susceptibility, biological plausibility, and relevant case reports and epidemiological studies. As set forth below, this is a widely accepted scientific methodology for ascribing substantial factor causation, and should be viewed as sufficient evidence of causation in this case.

1. Occupational history.

Using work histories to identify significantly harmful exposures is at least as ancient as the field of occupational medicine itself, as evidenced by this advice from the father of occupational medicine, Bernardino Ramazzini, in 1713:

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[4] Richard A. Lemen, *Asbestos in Brakes: Exposure and Risk of Disease,* 45 Am. J. Ind. Med. 229 (2004).

> When a doctor arrives to attend some patient of the working class...let him condescend to sit down...if not on a gilded chair...on a three-legged stool... He should question the patient carefully...So says Hippocrates in his work 'Affections.' I may venture to add one more question: *What occupation does he follow?* [5]

Qualitative history of exposure has also been the driving force behind the discovery and development of knowledge regarding the dangers of asbestos. The seminal 1960 Wagner study is a case in point.[6] Thirty-three (33) individual cases of mesotheliomas were reported from a South African asbestos mining town. Thirty-two (32) of those cases had a known qualitative history of exposure to asbestos. There were no exposure measurements, nor epidemiological/statistical analysis. Almost half of the cases were not from miners or first-hand exposure to asbestos but rather were from environmental and household exposures. Despite limitations that many industry advocates would now proclaim fatal to ascribing causation, many scientists believe that this study established that asbestos causes the very rare disease mesothelioma, and that environmental and household

[5] Bernardino Ramazzini, *De Morbis Artificum Diatriba* (1713), Trans. by W.C. Wright in A.L. Birmingham, *Classics of Medicine Library* (1983), *in* Medicine in Quotations: Views of Health and Disease through the Ages, 276 (Edward J. Huth & T.J. Murray, eds., 2nd ed. 2006).

[6] J.C. Wagner, et al., *Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province*, 17 Brit. J. Indust. Med. 260 (1960).

exposures alone were adequate: again, all based on exposure history alone.[7] Indeed, in the seminal 1965 paper on causation, *The Environment and Disease: Association or Causation?*, Sir Austin Bradford Hill emphasized the importance of exposure history used in conjunction with existing literature to determine a causal link.[8]

Internationally respected asbestos disease specialists reached a scientific and medical consensus, originally in 1997[9] and reaffirmed in 2014,[10] on the requirements for experts to attribute a given mesothelioma to asbestos exposure. The original 1997 committee consisted of 19 participants of various disciplines from eight (8) countries not producing asbestos, with over 1000 articles published between them. The resulting "Helsinki Consensus" agreed with the traditional generally accepted approach in occupational medicine that "a history of significant occupational, domestic or environmental exposure will suffice for attribution."

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[7] The first cohort study on mesothelioma was published in 1963. This landmark study provided to epidemiologists and other public health professionals proof of a statistical association. Thomas F. Mancuso, et al., *Methodology in Industrial Health Studies: The Cohort Approach, with Special Reference to an Asbestos Company,* 6 Arch. Env. Health 210 (1963).

[8] Sir Austin Bradford Hill, *The Environment and Disease: Association or Causation?* Proceedings of the Royal Society of Medicine 295 (1965).

[9] A. Tossavainen, et al., *Consensus Report: Asbestos, Asbestosis, and Cancer: The Helsinki Criteria for Diagnosis and Attribution*, 23(4) Scand. J. Work Environ. Health 311 (1997).

[10] Henrik Wolff, et al., *Consensus Report: Asbestos, Asbestosis, and cancer, the Helsinki Criteria for Diagnosis and Attribution 2014: Recommendations*, 41(1) Scand. J. Work Environ. Health 5 (2015).

The Helsinki Consensus also stated that the following points need to be considered in determining attribution of a given mesothelioma to asbestos exposures:

- "The great majority of mesotheliomas are due to asbestos exposure;
- "Mesotheliomas can occur in cases with low asbestos exposure.
- "However, very low background environmental exposures carry only an extremely low risk;
- "About 80% of mesothelioma patients have had some occupational exposure to asbestos, and therefore a careful occupational and environmental history should be taken;
- "Even brief or low-level exposure should be considered sufficient for mesothelioma to be designated as occupationally related.
- "A minimum of 10 years from the first exposure is required to attribute the mesothelioma to asbestos exposure, though in most cases the latency interval is longer (e.g., in the order of 30-40 years)

These generally accepted principles serve as the basic framework for occupational medicine and public health professionals when they are making determinations of both whether the total cumulative exposure was capable of causing mesothelioma, and whether some subset of the total exposure was a significant or appreciable exposure.

From the medical and scientific perspective, no bright-line quantitative tests can ever be applied in such causation evaluations. To require quantification where it is almost always impossible and unnecessary to do so, would be a public health travesty. That is, this would have the effect of creating an impossible burden of proof, and no claim would be able to meet the impossible standard: "only rarely are

humans exposed to environmental chemicals in a manner that permits a quantitative determination of adverse outcomes...Human exposure occurs most frequently in occupational settings where workers are exposed to industrial chemicals such as lead or asbestos; however, even under these circumstances, it is usually difficult, if not impossible, to quantify the amount of exposure."[11]

Perfect and complete exposure data is virtually never available. But using professional judgment based on the available evidence is generally accepted in the medical and scientific community.[12]

2. Individual Susceptibility.

One other important factor to consider in what constitutes a significant exposure is the concept of individual susceptibility. The great weight of evidence suggests that there are widely varying levels of susceptibility to asbestos, much as there is with tobacco and lung cancer. For example, even with very high exposures such as those experienced by insulation workers, less than 10% of the insulators developed mesothelioma.[13] On the other hand, just low level chrysotile exposure via home contamination, in the absence of occupational exposures, has been shown to induce high incidences of mesotheliomas in family groups that also have a

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[11] Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology, in* Reference Manual on Scientific Evidence, 639-640 (Federal Judicial Center, 3rd ed. 2011).

[12] Hill, *supra.*

[13] J. Ribak, et al., *Malignant Mesothelioma in a Cohort of Asbestos Insulation Workers: Clinical Presentation, Diagnosis, and Causes of Death*, 45 Brit. J. Ind. Med. 182 (1988).

germ-line BAP1 mutation.[14] Additionally, a diagnosis of mesothelioma combined with a Helsinki Consensus sufficient exposure history, is certainly proof of the body's defenses being overwhelmed and defeated. The capacity of the body's defense is certainly influenced heavily if not defined by individual susceptibility.

3. Biological Plausibility.

The emerging consensus among physicians and scientists is that most forms of cancer develop in a multistage process. This process typically involves a number of mutations, and these mutations must follow in some particular sequence for the cell(s) in question to become a fully cancerous cell which develops into a tumor:

> [It is]...now recognized that asbestos fibers themselves are carcinogenic, mainly by indirect mechanisms, and that malignant transformation is a multi-stage process...Direct effects are related to the physical interaction of fibers with target cells or by the generation of free radicals and reactive oxygen species (ROS) at the surface of fibers. Indirect effects are related to an inflammatory response to fibers, including the generation of factors, such as ROS and cytokines as a consequence of attempted but incomplete phagocytosis of fibers by macrophages ("frustrated phagocytosis"). There is now substantial scientific evidence for the indirect model, as discussed in several reviews.[15]

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[14] Joseph R. Testa, et al., *Germline BAP1 Mutations Predispose to Malignant Mesothelioma*, 43(10) Nature Genetics (2011).

[15] *See* generally Samuel P. Hammar, et al., *Neoplasms of the Pleura, in* 2 Dail and Hammar's Pulmonary Pathology Volume II: Neoplastic Lung Disease (Joseph F. Tomashefski, Jr., et al., eds., 3rd ed. 2008) and specifically pp. 587-588 and 589-599 generally.

Although, as in most areas of medicine, there is much to be learned, scientists have identified "established mechanistic events" in the development of asbestos-induced mesothelioma. These include: impaired fiber clearance leading to macrophage activation, inflammation, generation of reactive oxygen and nitrogen species, tissue injury, genotoxicity, aneuploidy and polyploidy, epigenetic alteration, activation of signaling pathways, and resistance to apoptosis.[16]

Cumulative exposure is merely an extension of the ancient concept of dose-response: "...'the dose makes the poison' is the oldest maxim in the field."[17] "Toxicologists generally posit two main dose-response curves: those that have a "threshold" and those that do not...The second general type of a dose-response curve is one that is considered to have no threshold. The most important example for toxic torts is that of cancer. The underlying cause of many cancers is a persistent genetic mutation allowing the unbridled growth of a cell which then results in a clone of cancer cells".[18] [19]

[16] International Agency for Research on Cancer (IARC), *Special Report: Policy: A review of human carcinogens - Part C: metals, arsenic, dusts, and fibres*, 10 The Lancet, 453 (2009).

[17] Bernard D. Goldstein, *Toxic Torts: The Devil is in the Dose,* 16(2) J.L. & Pol'y. 551 (2008). Bernard D. Goldstein is Professor of Environmental and Occupational Health and former Dean of the University of Pittsburg Graduate School of Public Health.

[18] *Id.* at 554-555.

Research to date confirms the findings of case reports and epidemiological studies: that total body burden (cumulative exposure) has a significant effect on the carcinogenic process and that all forms of commercial asbestos are genotoxic and mutagenic (a dose-response curve without a threshold).

4. Case Reports and Sentinel Events/Signature Diseases

Case reports, especially when dealing with rare diseases that are considered sentinel events/signature diseases for a given exposure, are a powerful form of evidence and one that researchers take into consideration along with cell and animal studies, autopsy studies, as well as epidemiology studies. Such case reports and series also support the attribution position taken in the Helsinki Consensus.

Mesothelioma is a signature malignancy for asbestos exposure. This has been generally accepted in the scientific community based on studies conducted decades ago.[20] [21] [22] [23] Mesothelioma was listed as a sentinel health event of

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[19] Also see Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology, in* Reference Manual on Scientific Evidence, 669-670 (Federal Judicial Center, 3rd ed. 2011): "For agents that produce effects other than through mutations, it is assumed that there is some level that is incapable of causing harm... If the level of exposure was below this no observable effect, or threshold, level, a relationship between the exposure and disease cannot be established ... *This analysis, however, is not applied to substances that exert toxicity by causing mutations leading to cancer.*" (emphasis added).

[20] Wagner, *supra.*

[21] Irving J. Selikoff, *Opening Remarks,* 132 Ann. N.Y. Acad. Sci. 7 (1965).

[22] Margaret R. Becklake, Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice, 114 Am. Rev. Resp. Disease 187 (1976).

occupational origin in the U.S. over thirty years ago.[24] The agent responsible was identified as asbestos and the industry/occupation of concern was "Asbestos industries and utilizers."[25] In the United States, there are no other proven major causes of malignant mesothelioma.

Regarding the specific usefulness of case reports in looking at asbestos related cancer, Phillip Enterline, M.D., in a report funded by and generated for the industry group Asbestos Information Association, stated:

> Among the things I gained from this research was a deep respect for the clinicians and pathologists who contributed to the early literature. They showed remarkable insight into the meaning of the observations, and the logic of their conclusions was far more reasoned than I had expected. I became far less sure that the sharp distinction made by many writers between epidemiological as contrasted with clinical-pathological observations served a useful purpose. Even a single case report is a kind of epidemiological observation, since the basis of such reports is usually a feeling, unexpressed, that the case is somehow aberrant for a human population.[26]

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[23] World Health Organization (WHO), International Agency for Research on Cancer (IARC), *IARC Monographs on the Evaluation of the Carcinogenic Risks for Humans, Chemical and Industrial Processes Associated with Cancer in Humans: IARC Monographs, Volumes 1 to 20,* Supp. 1 (1979); 100C World Health Organization, International Agency for Research on Cancer, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, A Review of Human Carcinogens, Part C: Arsenic, Metals, Fibres, and Dusts* 1-40, 219-309 (2012).

[24] David D. Rutstein, et al., *Sentinel Health Events (Occupational): A Basis for Physician Recognition and Public Health Surveillance*, 73(9) Am. J. Public Health 1054 (1983).

[25] *Id.* at 1056

[26] Philip E. Enterline, *Asbestos and Cancer: The First Thirty Years* (1978).

Regulatory bodies such as the Department of Labor's Occupational Safety & Health Agency (OSHA), after conducting a comprehensive literature review, have found and reported case reports in scientific literature showing very short exposures being capable of causing mesothelioma: "Asbestos exposures as short in duration as a few days have caused mesothelioma in humans."[27]

As noted above, almost half of the 32 case reports in the seminal 1960 Wagner paper involved environmental or household contact alone. Similarly, one can find individuals within larger cohorts whose exposures were low or intermittent. For example, there is a chrysotile mine in Italy that is free from amphibole asbestos contamination.[28] An early study found two (2) miners with mesothelioma, which the authors conclude is evidence of pure chrysotile causing

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[27] United States Department of Labor, Occupational Safety & Health Administration (OSHA), Safety and Health Topics, *Asbestos,* https://www.osha.gov/SLTC/asbestos/ (accessed March 12, 2015)., citing: E. Skammeritz, et al., *Asbestos Exposure and Survival in Malignant Mesothelioma: A Description of 122 Consecutive Cases at an Occupational Clinic,* 2(4) Int. J. Occup. Environ. Med. 224 (2011).; Morris Greenberg & T.A. Lloyd Davies, *Mesothelioma Register 1967-68,* 31 Brit. J. Ind. Med. 91 (1974).; World Health Organization (WHO), International Agency for Research on Cancer (IARC), *IARC Monographs on the Evaluation of the Carcinogenic Risks to Humans, Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42, Supplement 7* 106-116 (1987); and John T. Hodgson & Andrew Darnton, *The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure,* 44(8) Ann. Occup. Hyg. 565 (2000).

[28] This chrysotile mine has a mineral called balangeroite present as well. This mineral is not a form of asbestos, is not chemically similar to amphibole asbestos, has poor bio-persistence, and upon testing by the researchers was deemed to not be responsible for the excess of mesotheliomas in the area. *See* Franceso Turci, et al., *Role of Associated Mineral Fibres in Chrysotile Asbestos Health Effects: The Case of Balangeroite,* 53(5) Ann. Occup. Hyg. 491 (2009).

mesothelioma but with low potency.[29] Years later and after additional latency had passed, in an updated study looking at not only the miners, but white collar workers and people who lived in the area surrounding the mine, they found a tremendous increase in mesothelioma. Not only were there now fourteen (14) miners with mesothelioma, there were thirteen (13) non-miners including white collar only workers and five (5) environmental cases.[30]

5. Epidemiological Studies.

There is overwhelming consensus in the medical and scientific communities that exposure to all forms of commercial asbestos can induce mesotheliomas based on peer-reviewed, published epidemiological studies.[31] Mesothelioma is caused by asbestos-containing dust, not by a job classification or product type — the issue is simply one of inhalation of respirable asbestos fibers. Just as there is no scientific "rule" that worker exposures be quantified in order for attribution, there is no "rule" that a product specific positive epidemiology study be available for attribution.

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[29] Giorgio Piolatto, et al., *An Update of Cancer Mortality among Chrysotile Asbestos Miners in Balangero, Northern Italy*, 47 Brit. J. Ind. Med. 810 (1990).

[30] D. Mirabelli, et al., *Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy*, 65 Occup. Environ. Med. 815 (2008).

[31] IARC 2009, *supra.*; IARC 2012, *supra.*; *Joint Policy Committee of the Societies of Epidemiology, Position Statement on Asbestos from the Joint Policy Committee of the Societies of Epidemiology (JPC-SE)* (2012).

Nearly all human asbestos epidemiology that has looked at the question has determined that asbestos-induced mesothelioma follows a dose-response pattern, generally with the greater the total dose, the greater the risk of mesothelioma. "One factor that emerges from the Peto model and its modifications is that when there are multiple asbestos exposures, each contributed to cumulative exposure and hence to the risk and causation of MM, within an appropriate latency interval."[32] Researchers have found some additional factors important in analyzing the asbestos mesothelioma dose-response curve, such as time since first exposure, but total cumulative dose consistently is the best indicator of risk: "In this study...the dose-response seemed to be described best by Cumulative Exposure Index."[33]

Moreover, human dose-response studies show significant increased risk at very low levels of exposure. These studies have found increased risk of 2.69 to 7.9-fold based on low-level exposure. *See e.g.* 2014 LaCourt, et al., $\leq$ 0.1 f/cc/years, Odds Ratio (OR) 4.0 (99% Confidence interval (CI) 1.9-3.3)[34]; 2001 Rödelsberger, et al., $\leq$ 0.15 f/cc/years, OR 7.9 (95% CI 2.1-30.0)[35]; 2014

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[32] Hammar, *supra.*

[33] Jean Bignon, et al., *History and Experience of Mesothelioma in Europe, in* Mesothelioma 29-53 (Bruce W.S. Robinson & A. Philippe Chahinan, eds., 2002).

[34] A. LaCourt, et al., *Occupational and Non-Occupational Attributable Risk of Asbestos Exposure for Malignant Pleural Mesothelioma,* Thorax 1 (2014).

[35] Klaus Rödelsperger, et al., *Asbestos and Man-Made Vitreous Fibers as Risk Factors for Diffuse Malignant Mesothelioma: Results from a German Hospital-Based Case-Control Study,* 39 Am. J. Ind. Med. 262 (2001).

Offermans, et al., ≤0.2 f/cc/years, Hazard Ratio (HR) 2.69 (95% CI 1.60 o4.53);[36] and the 1998 Iwatsubo, et al., 0.5 - 0.99 f/cc/years, OR 4.0 (95% CI 2.0-8.8).[37]

And epidemiological studies provide additional support for a weight of the evidence conclusion that asbestos from brakes can and does cause mesothelioma. There are numerous epidemiological studies and case reports of mesothelioma in persons whose primary exposure to asbestos is from asbestos-containing brake materials.[38] [39] [40] [41] [42] [43] From an industrial hygiene perspective, it does not take long, at levels known to exist in garage environments without asbestos dust control, for a person to receive appreciable exposures.

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[36] Nadine S.M. Offermans, et al., *Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherlands Cohort Study,* 56(1) J. Occup. Environ. Med. 6 (2014).

[37] Y. Iwatsubo, et al., *Pleural Mesothelioma: Dose-Response Relation at Low Levels of Asbestos Exposure in a French Population-based Case-Control Study,* 148(2) Am. J. Epidemiol. 133 (1998).

[38] D. McDonald, et al., *Epidemiology of Primary Malignant Mesothelial Tumors in Canada,* 26(4) Cancer 914 (1970).

[39] A. E. Anderson, et al., *Asbestos Emissions from Brake Dynamometer Tests,* SAE Technical Paper 730549 (1973).

[40] Klaus Rödelsperger, et al., *Asbestos Dust Exposure During Brake Repair, 10* Am. J. Ind. Med. 63 (1986).

[41] M. Huncharek, et al., *Pleural Mesothelioma in a Brake Mechanic,* 46 Brit. J. Ind. Med. 69 (1989).

[42] Iwatsubo, *supra.*

[43] Kay Teschke, et al., *Mesothelioma Surveillance to Locate Sources of Exposure to Asbestos,* 88(3) Canadian J. Pub. Health 163 (1997).

These and other asbestos exposure and disease studies in brake workers were reviewed by Dr. Richard Lemen, Assistant Surgeon General (Ret.) in 2004; Dr. Lemen concluded that "Even the so called ''controlled'' use of asbestos containing brakes poses a health risk to workers, users, and their families."[44] Recently, a cancer registry-based study in Massachusetts reported a greater than two-fold excess of mesothelioma in auto mechanics.[45] For another epidemiological example, an analysis of mortality in bus drivers and bus maintenance workers in Genoa, Italy demonstrated significant excess deaths (SMR 3.67) from pleural mesothelioma when compared to Italian males.[46]

Brake manufacturers and their consultants invent their own "scientific rule" demanding that a collection of approximately 18-22 "negative" friction studies and industry-financed meta-analyses thereof trumps all other evidence. But for a study or studies to be truly "negative" in the sense hoped for by industry advocates, the study must be large enough to have sufficient "power" to detect an increase in risk,

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[44] Lemen, *Asbestos in Brakes*, *supra*.

[45] Cora R. Roelofs, et al., *Mesothelioma and Employment in Massachusetts: Analysis of Cancer Registry Data 1988-2003,* 56 Am. J. Ind. Med. 985 (2013).

[46] Domenico Franco Merlo, et al., *A Historical Mortality Study Among Bus Drivers and Bus Maintenance Workers Exposed to Urban Air Pollutants in the City of Genoa, Italy,* 67 Occup. Environ. Med. 611 (2010). There was not an increased risk when compared to the surrounding population, because the surrounding region contained several shipyards that historically used asbestos. So the choice of comparison population can influence the findings regarding mesothelioma in an urban or working population. This needs to be taken into account when interpreting study findings or individual exposure histories.

have reliable work histories to establish exposure or lack thereof, and be followed for a sufficiently long time to account for latency.[47] It is also highly preferable that the study be specifically designed to detect an increase in risk for the disease in question. The studies in questions fail on all of the above counts.[48] As concluded by the highly respected scientific body the National Cancer Institute: "[s]tudies evaluating the cancer risk experienced by automobile mechanics exposed to asbestos through brake repair are limited, but the overall evidence suggests there is no safe level of asbestos exposure."[49]

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[47] Median latency since first exposure for mesothelioma is 38.4 years. *See* A. Reid, et al., *Mesothelioma Risk after 40 Years since First Exposure to Asbestos: a Pooled Analysis,* 0 Thorax, 1 (2014).

[48] A comprehensive survey of the brake literature is not the intent of this brief. However, Amici incorporate by reference: Lemen, *Asbestos in Brakes, supra.*; David S. Egilman & Marion A. Billings, *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability,* 11 Int. J. Occup. Environ. Health 360 (2005); and Laura S. Welch, *Asbestos Exposure Causes Mesothelioma, But Not This Asbestos Exposure: An Amicus Brief to the Michigan Supreme Court, 13* Int. J. Occup. Environ. Health 318 (2007).

[49] National Cancer Institute, *Asbestos Exposure and Cancer Risk* (2009) http://www.cancer.gov/cancertopics/causes-prevention/risk-factors/cancer-causing-substances/asbestos/asbestos-fact-sheet (accessed March 11, 2015).

CONCLUSION

It is axiomatic that every exposure to asbestos, while not necessarily a substantial contributing factor, contributes to the total dose. An expert's concurrence with this scientific fact should not "infect" his testimony such that his causation opinion is negated, especially where, as here, the expert employed the generally accepted method of a multi-faceted review of the diagnosis, medical and occupational history, individual susceptibility, biological plausibility, and relevant case and epidemiological studies.

Dated: April 7, 2015

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Exhibit A

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I hereby certify that on this 7th day of April 2015 that I served the foregoing Brief of Amici Curiae Muge Akpinar-Elci, MD, et al., as follows via first class U.S Mail which service satisfies the requirements of Pa RAP 121 and 3309(a).

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EXHIBIT 30

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ORIGINAL CONTRIBUTIONS

Pleural Mesothelioma: Dose-Response Relation at Low Levels of Asbestos Exposure in a French Population-based Case-Control Study

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A hospital-based case-control study of the association between past occupational exposure to asbestos and pleural mesothelioma was carried out in five regions of France. Between 1987 and 1993, 405 cases and 387 controls were interviewed. The job histories of these subjects were evaluated by a group of experts for exposure to asbestos fibers according to probability, intensity, and frequency. A cumulative exposure index was calculated as the product of these three parameters and the duration of the exposed job, summed over the entire working life. Among men, the odds ratio increased with the probability of exposure and was 1.2 (95% confidence interval (CI) 0.8–1.9) for possible exposure and 3.6 (95% CI 2.4–5.3) for definite exposure. A dose-response relation was observed with the cumulative exposure index: The odds ratio increased from 1.2 (95% CI 0.8–1.8) for the lowest exposure category to 8.7 (95% CI 4.1–18.5) for the highest. Among women, the odds ratio for possible or definite exposure was 18.8 (95% CI 4.1–86.2). We found a clear dose-response relation between cumulative asbestos exposure and pleural mesothelioma in a population-based case-control study with retrospective assessment of exposure. A significant excess of mesothelioma was observed for levels of cumulative exposure that were probably far below the limits adopted in most industrial countries during the 1980s. *Am J Epidemiol* 1998;148:133–42.

asbestos; case-control studies; mesothelioma; occupational exposure

Mesothelioma is a rare cancer that is mainly due to occupational or nonoccupational asbestos exposure. The background level is assumed to be as low as 1–2 per million inhabitants (1). During recent decades, however, its prevalence has been increasing in the general populations of most industrialized countries (2, 3).

In the cohorts of workers occupationally exposed to asbestos that have been followed since the 1960s, the risk of mesothelioma has increased with the level or duration of exposure or both (4–14). The absence of accurate measurements for low exposure levels limits the reliability of any current quantitative assessments of the risk they carry. Furthermore, since only a few subjects in these cohorts were exposed to low levels of asbestos, there is not enough statistical power to show any significant association with mesothelioma.

Case-control studies among the general population and its variety of occupational categories exposed to different asbestos levels are more likely to include subjects whose exposure was low. Despite recent developments in retrospective assessment of exposure (15, 16), the quantitative assessment of low levels remains difficult, since measurements of dust concentration during the relevant periods are not often available.

Previous studies of mesothelioma (4–14) have examined exposure parameters, including cumulative exposure and such time-related variables as time since or age at first exposure. Other exposure parameters, in particular, the time-related pattern of exposure, might be useful. Although the current asbestos exposure pro-

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Abbreviations: CEI, cumulative exposure index; CI, confidence interval; ISCO, International Standard Classification of Occupations (1968 edition); ISIC, International Standard Industrial Classification of All Economic Activities; OR, odds ratio.

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file involves mostly intermittent exposure, the data now available do not allow any conclusion about whether asbestos inhalation at intermittent peaks contributes to the risk of mesothelioma.

The aims of this study were to examine the dose-response relation by using several types of exposure parameters and to study the role of time-related exposure patterns (intermittent compared with continuous) in a large case-control study conducted in France since 1987.

MATERIALS AND METHODS

This report is based on data collected in a hospital-based case-control study of pleural malignant mesothelioma (hereafter referred to as mesothelioma). This study is ongoing, and the present analysis is limited to data collected between January 1, 1987, and December 31, 1993. Five administrative regions of France are currently participating: The study began in the Paris metropolitan area in 1987 and was extended in 1989 to the region of Provence-Alpes-Côte-d'Azur and to Corsica and, in 1992, to Lorraine and Auvergne. The respiratory disease, chest surgery, and oncology departments of all public hospitals and the main private clinics were informed of the study and invited to participate.

Mesothelioma patients in this study met the following criteria: 1) consultation, at any stage of the disease, in a participating hospital; 2) histologically confirmed diagnosis; 3) resident in a participating region at diagnosis; and 4) alive at the time of interview.

The diagnosis of mesothelioma was confirmed by the French Mesothelioma Panel (17, 18). The panel excluded 46 (10 percent) of the subjects initially considered eligible (for whom, after pathology review, the principal diagnosis was adenocarcinoma). In 125 subjects (31 percent of the remaining 405 cases), however, the panel could not reach a conclusion because the histologic sample was insufficient or because the slides had not been sent to the panel. The likelihood of diagnosis was then determined by reviewing clinical data (clinical history, radiologic data), laboratory test reports, and the histologic conclusions of the local pathologists. Hospital controls were individually matched for sex, age (±5 years), place of residence (administrative department), and racial or ethnic origin (black, white, North African, Asian, or other) and were selected in the departments of internal medicine, ophthalmology, and surgery. Patients with a medical history of malignant tumors or asbestos-related diseases (i.e., asbestosis and lung cancer) were excluded as controls. To the extent possible, controls were chosen in the same hospital as their matching cases.

Data collection

An experienced interviewer questioned patients during their hospitalization. In a few cases, the subject was interviewed at home. A standardized questionnaire was used to collect information on work history: work periods, including the starting and ending dates of each job that lasted at least 6 months; the company's economic branch of activity; and a description of the tasks performed by the subject. This information allowed us to classify the subject's job according to the International Standard Classification of Occupations (ISCO) code for occupations (19) and the International Standard Industrial Classification of All Economic Activities (ISIC) code (20) for industrial activities. For each job period, the subjects were asked five specific questions about direct (handled) and indirect (working in the immediate vicinity of colleagues who handled) asbestos exposure.

Exposure assessment

A panel of five experts in industrial hygiene evaluated occupational exposure to asbestos, as follows: 1) all job periods of all subjects (cases and controls) were sorted by economic branch of activity (ISIC codes) and occupation (ISCO codes); 2) the job periods were selected for review according to the likelihood of exposure of the job titles, classified by ISIC and ISCO codes; 3) the job periods for which subjects reported exposure were selected; and 4) occupational exposure to asbestos was evaluated for all job periods selected in either step 2 or step 3, in sequential order of both the ISIC and ISCO codes. Each job period for each subject was thus evaluated independently. This procedure was chosen to minimize errors in the exposure assessment due to knowledge of the subjects' lifetime exposure.

The experts were blinded to the case-control status of each job period, and decisions were made by consensus. The experts had access to all information from the questionnaire, such as job history, tasks performed, and self-report of direct or indirect exposure to asbestos.

This evaluation of each job allowed each job period to be classified according to the probability, intensity, and frequency of exposure. Categories of intensity and frequency were established by the experts before the evaluation began by using the following semiquantitative scale: probability of exposure: not exposed, possible, definite; frequency: sporadic (less than 5 percent of work time); irregular (5–50 percent of work time); continuous (more than 50 percent of work time); intensity: low (less than 1 fiber/ml); medium (1–2 fibers/ml); high (2–10 fibers/ml); very high (>10 fibers/ml).

We attributed weighting factors to each exposure category to calculate an exposure index: probability:

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null = 0, possible = 0.5, definite = 1; frequency: sporadic = 0.025, irregular = 0.25, continuous = 0.75; intensity: low = 0.1 fiber/ml, medium = 1 fiber/ml, high = 10 fibers/ml, very high = 100 fibers/ml.

Because the latency period of the disease is so long, we did not analyze asbestos exposure during the 20 years before the mesothelioma diagnosis (1, 21, 22).

We used the following exposure parameters for each subject.

Highest probability, intensity, and frequency. Each subject's highest probability of exposure was determined by the highest probability of any job period during lifetime work history. Highest intensity and frequency were determined in the same way.

Duration of exposed jobs. Duration of exposed jobs (years) is defined as the total duration of job periods involving possible or definite exposure.

Cumulative exposure index (CEI). CEI is the lifetime sum of the products of probability, frequency, intensity, and duration for each job period. Because no measurements of airborne asbestos levels were available, all estimations of exposure parameters were based on the experts' subjectivity, that is, semiquantification, to which we subsequently assigned weighting factors. This index of cumulative exposure was expressed in terms of fibers/ml-years inside quotation marks ("f/ml-years").

Pattern of exposure in time. We examined the relative risks associated with the pattern of exposure by distinguishing subjects who had undergone only intermittent exposure from those whose exposure was considered continuous. Subjects' exposure was classified as intermittent if it was sporadic or irregular and if they had never worked at a job with continuous exposure. The continuous category was reserved for subjects who had been employed in at least one job with continuous exposure.

In addition to these composite variables, age at first exposure and time since first exposure were also examined.

Statistical analysis

We calculated the odds ratio by using logistic regression and the unconditional maximum likelihood method, with the aid of BMDP software (23). This technique allowed us to include the cases who had no controls. The analysis took the matching variables into account. The relation between asbestos exposure and mesothelioma was examined separately for men and women.

Quantitative parameters were categorized by percentile points. To allow us to consider the effect of some previously used cutoff points, we used additional categories for studying cumulative exposure (5 and 10 "f/ml-years").

The effect of the time-related exposure pattern (that is, intermittent vs. continuous) was analyzed after taking into account cumulative exposure.

RESULTS

The study included 405 cases and 387 controls (table 1). The largest group of cases (69.9 percent) came from the Paris metropolitan area. Cases and controls did not differ significantly by sex (82 percent and 81 percent men, respectively) or age at interview (63.5 and 63.9 years, respectively). Since almost the entire sample was white (96.8 percent of cases and 97.7 percent of controls), we did not adjust for race or ethnic origin. The socioeconomic category of the subject was determined by the last occupation held before the interview and coded using the major groups of the ISCO (table 2). Cases and controls differed significantly, with more blue-collar workers among the cases. Thus, for all comparisons, the odds ratios were adjusted for socioeconomic category.

Table 3 presents the main occupations and industries that entailed asbestos exposure among the 3,498 job periods for men. We consider in this table only activities and professions that contained at least 50 job periods and for which at least 25 percent of the job

TABLE 1. Main characteristics of cases and controls by study area, French Mesothelioma Case-Control Study, 1987–1993

Study area and years of study	Cases					Controls				
	No.	% of males	Age (years)			No.	% of males	Age (years)		
			Mean	(SD)*	Range			Mean	(SD)	Range
Paris metropolitan area (1987–1993)	283	78	62.9	(10.8)	25–88	279	78	63.4	(11.2)	29–93
Provence-Alpes-Côte-d'Azur (1989–1993)	82	92	64.5	(8.9)	44–85	73	89	65.2	(9.4)	43–84
Corsica (1989–1993)	8	75	67.5	(7.0)	60–81	7	86	64.7	(4.9)	56–71
Lorraine (1992–1993)	28	89	64.8	(12.2)	32–85	25	89	65.8	(10.7)	47–87
Auvergne (1992–1993)	4	88	68.0	(4.1)	63–73	3	75	66.0	(4.0)	62–70
Total	405	82	63.5	(10.5)	25–88	387	81	63.9	(10.7)	29–93

* SD, standard deviation.

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TABLE 2. Distribution of mesothelioma cases and controls according to socioeconomic category,* French Mesothelioma Case-Control Study, 1987–1993

	ISCO code (major groups)	Men				Women			
		Cases		Controls		Cases		Controls	
		No.	%	No.	%	No.	%	No.	%
Professional, technical, and related workers	0/1	47	14.3	45	14.5	7	9.6	15	20.3
Administrative and managerial workers	2	22	6.7	17	5.5	1	1.4	0	0
Clerical and related workers	3	35	10.6	40	12.9	28	38.4	25	33.7
Sales workers	4	30	9.1	24	7.7	5	6.9	3	4.1
Service workers	5	14	4.3	32	10.3	13	17.8	20	27.0
Agricultural, animal husbandry, and forestry workers; fishermen; and hunters	6	2	0.6	8	2.6	0		3	4.1
Production and related workers, transport equipment operators, and laborers	7/8/9	179	54.4	145	46.6	19	26.0	8	10.8
No occupational activity		1		1		2		1	

* Socioeconomic category corresponding to the International Standard Classification of Occupations (ISCO) code of the last job held by the subject before interview.

periods were evaluated as possibly or definitely exposed. In the industries and occupations in which we had anticipated asbestos exposure, the proportions of exposure were high. For example, exposure was likely to have occurred in 264 of the 487 (54 percent) men's job periods in the construction industry and in 55 of the 70 (79 percent) men's job periods in the shipbuilding industry. In some occupations, exposure was frequent, e.g., 82 percent among motor vehicle mechanics and 85 percent among plumbers and pipe fitters. The proportion of exposed job periods in the categories of other industrial activities and occupations was low (16 and 19 percent, respectively).

Table 4 indicates the distribution of job periods of male cases and controls according to starting date and exposure intensity for possibly and definitely exposed job periods. Very few job periods were considered as very highly exposed, and those were found mainly among cases (18 job periods in cases vs. four among controls). These were observed after 1950 when the industrial use of asbestos had developed.

Table 5 reports the distribution of male cases and controls according to various exposure parameters. The exposure measures in this table have not been adjusted for the other exposure parameters. Mesothelioma risk increased with exposure probability, intensity, and frequency. The odds ratio for possible exposure was 1.2 (not significant), and for definite exposure, it was 3.6. Risk increased with frequency of exposure, but subjects with sporadic exposure were not at greater risk of mesothelioma than were controls. Risk also increased with the total duration of exposed jobs: The odds ratio for subjects exposed for at least 20 years was 5.4.

The odds ratio for the relation between pleural mesothelioma and asbestos exposure parameters did not increase with time since first exposure, nor was any consistent trend observed with age at first exposure.

As determined by the experts' evaluations and the weighting factors, the cumulative exposure of our population was rather low. Twenty-three percent of the cases and 35 percent of the controls had been exposed to less than 0.5 "f/ml-years." A gradient was observed with the CEI; the odds ratio rose from 1.2 for the subjects with less than 0.5 "f/ml-years" to 8.7 for the category with more than 10 "f/ml-years."

Among women, a significant risk of mesothelioma was observed among those possibly and definitely exposed to asbestos, considered together (odds ratio (OR) = 18.8, 95 percent confidence interval (CI) 4.1–86.2). Because of the small number of women exposed to asbestos, especially among controls (25 cases and two controls, for 33 and 3 percent of their respective categories), we did not analyze the dose-response relation among women any further.

The results about the time-related pattern of exposure reveal a significantly elevated odds ratio among workers whose exposure to asbestos was intermittent (OR = 1.8, 95 percent CI 1.3–2.6). The odds ratio was much greater, however, for continuous exposure (OR = 5.7, 95 percent CI 3.4–9.7). The median CEI within each category considered, i.e., <0.5, 0.5–0.99, 1–9.99, and ≥10 "f/ml-years," was similar among intermittent and continuous exposure cases, except in the highest class of CEI (≥10 "f/ml-years") (0.1, 0.65, 3.5, and 38.7 "f/ml-years" for the intermittent exposure groups and 0.075, 0.65, 3.1, and 71.3 "f/ml-years" for the continuous groups, respectively). We attempted

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TABLE 3. Selected principal industrial activities and occupations entailing asbestos exposure among men, French Mesothelioma Case-Control Study, 1987–1993*

Title	No. of job periods	Proportion of exposed job periods† (%)
Industrial activities (4-digit ISIC‡ code)		
5000 Construction	487	54
3843 Manufacture of motor vehicles	113	27
7111 Railway transport	76	30
3841 Shipbuilding and repairing	70	79
3813 Manufacture of structural metal products	65	49
3511 Manufacture of basic industrial chemicals, except fertilizers	52	54
9513 Repair of motor vehicles and motorcycles	62	71
3823 Manufacture of metal and wood working machinery	58	26
3829 Manufacture of machinery and equipment, except electrical not elsewhere classified	54	30
3845 Manufacture of aircraft	51	31
3710 Iron and steel basic industries	51	61
Other industries and industries not specified ($n = 7$)	2,359	16
Occupations (3-digit ISCO‡ code)		
9–99 Laborers not elsewhere classified	152	26
8–41 Machinery fitters and machine assemblers	110	38
8–55 Electrical wiremen	107	54
8–49 Machinery fitters, machine assemblers, and precision instrument makers (except electrical) not elsewhere classified	99	57
8–73 Sheet-metal workers	85	49
9–54 Carpenters, joiners, and parquetry workers	75	37
8–71 Plumbers and pipe fitters	73	85
9–51 Bricklayers, stonemasons, and tile setters	69	58
8–43 Motor vehicle mechanics	67	82
3–91 Stock clerks	58	28
Other professions	2,603	19
Total job periods	3,498	27

* In these tables, only activities and professions that contained at least 50 job periods and for which at least 25 percent of the job periods were evaluated as possibly or definitely exposed were considered.
† Possible or definite exposure to asbestos without taking into account the 20-year latency period.
‡ ISIC, International Standard Industrial Classification of all Economic Activities; ISCO, International Standard Classification of Occupations.

to separate the possible effect of the exposure delivery pattern from that of cumulative exposure by a stratified analysis. The odds ratios increased with the CEI among subjects with intermittent and with continuous exposures (table 5). The amplitude of the odds ratio differed, however, between these categories. When we examined the odds ratios for subjects within each of our CEI categories, they were almost twice as high for subjects with continuous exposure as for those intermittently exposed, except for the CEI category of 0.5–1 "f/ml-years."

DISCUSSION

This study, one of the larger population-based case-control studies published (24–37) sheds light on several important aspects of mesothelioma and asbestos.

As far as we know, our study is the first conducted in a general population that uses a semiquantitative assessment of exposure to examine the dose-response relation between asbestos exposure and mesothelioma.

The mesothelioma cases of this study were identified in hospitals that had agreed to participate in the case-control survey. Cases seen in other hospitals and those who were not followed within a hospital structure were not included. There is no reason to suppose, however, that the type of health care facility depended on the level of asbestos exposure. We ought to point out another source of selection bias. Mesothelioma diagnosis remains difficult. The patient who has a known history of asbestos exposure is more likely to be diagnosed with mesothelioma than a patient with similar symptoms but no known history of asbestos exposure. This bias could have heightened the dose-response relation between asbestos exposure and mesothelioma. There are probably few cases erroneously diagnosed as mesothelioma, since the French Me-

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TABLE 4. Distribution of job periods among men, according to the intensity of exposure* and decade of beginning, French Mesothelioma Case-Control Study, 1987–1993

Probability of exposure	Distribution job periods													
	Before 1930		1930–1939		1940–1949		1950–1959		1960–1969		1970 and after		Total	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Cases														
Not exposed	83	77.6	172	75.1	332	66.9	280	62.8	202	61.2	215	69.8	1,284	67.0
Low	10	9.4	25	10.9	65	13.1	55	12.3	54	16.4	38	12.3	247	12.9
Medium	12	11.2	26	11.4	74	14.9	64	14.4	50	15.2	29	9.4	255	13.3
High	2	1.9	6	2.6	23	4.6	40	9.0	20	6.1	21	6.8	112	5.9
Very high	0		0		2	0.4	7	1.6	4	1.2	5	1.6	18	0.9
Total	107	100	229	100	496	100	446	100	330	100	308	100	1,916†	100
Controls														
Not exposed	65	82.3	192	84.2	341	80.6	282	80.8	218	79.3	183	83.9	1,281	81.5
Low	2	2.5	21	9.2	46	10.9	33	9.5	29	10.6	17	7.8	148	9.4
Medium	7	8.9	13	5.7	31	7.3	27	7.7	26	9.5	15	6.9	119	7.6
High	4	5.1	2	0.9	4	1.0	6	1.7	1	0.4	3	1.4	20	1.3
Very high	1	1.2	0		1	0.2	1	0.3	1	0.4	0		4	0.3
Total	79	100	228	100	423	100	349	100	275	100	218	100	1,572‡	100

* Intensity for possible or definite exposure to asbestos without taking into account the 20-year latency period.
† Job periods for which the year of beginning is missing = 6.
‡ Job periods for which the year of beginning is missing = 4.

sothelioma Panel excluded, after pathology review, 10 percent of subjects initially considered eligible and confirmed the diagnosis for 62 percent of the cases on the basis of pathology reports. We accepted the remaining 28 percent after reviewing available histologic data and hospital records.

The use of hospital controls could have entailed some bias. In particular, cases and controls differed in socioeconomic status, with the latter group containing fewer blue-collar workers. This difference could have arisen from a selection bias and might reflect the controls' failure to represent adequately the population from which the cases were drawn, or it might be due to a particularly high rate of pleural mesothelioma among blue-collar workers because of their high prevalence of asbestos exposure. In the latter case, taking socioeconomic status into account could have led to overadjustment of the relation between asbestos exposure and pleural mesothelioma. The crude odds ratios, however, were of same order of magnitude as the adjusted values.

The validity of the information about asbestos exposure depends on how well we have avoided three types of errors: difference in the quality of interview data according to disease status (recall bias or interviewer bias), errors by the experts in classifying the subjects into defined categories, and errors related to the accuracy of the weighting factors subsequently assigned to each category. As recently stated in an International Agency for Research on Cancer meeting on retrospective assessment of occupational exposure in epidemiology (38), the validity of expert judgment, which relies on both the knowledge and the experience of industrial hygienists, has rarely been evaluated. Indeed, when no objective method of measuring exposure is available, their judgment is most often considered the gold standard.

Our study assessed frequency and intensity of exposure by using ordinal categories with specific boundaries. This procedure should have minimized the misclassification of subjects between extreme exposure categories. The experts themselves, however, reported sometimes encountering difficulties in distinguishing between sporadic and irregular exposure and between low and moderate exposure.

Moreover, they suggested that the quality of their assessment for the periods under consideration (20 or more years ago) might not be as good as for more recent years because of the lack of published data for these periods. These errors could have led to the nondifferential misclassification of subjects into exposure categories and the possible underestimation of the odds ratios (39).

To avoid the exposure suspicion bias, the experts were blinded to case-control status when they evaluated exposure. Recall bias could have influenced the quality of the answer to the questionnaire and, subsequently, the expert judgment. To test this potential bias, we compared the experts' assessment with results from an asbestos job exposure matrix (40). We found no difference between cases and controls (data not shown), suggesting that it was unlikely that a substantial recall bias had affected the experts' judgment. The interviewers, however, were aware of case-control status and thus might have conducted the interviews of the case subjects more thoroughly than those of controls. Since the experts considered all of the information available, they might have been able to evaluate

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TABLE 5. Odds ratios for relations between pleural mesothelioma and asbestos exposure parameters among men, French Mesothelioma Case-Control Study, 1987–1993, with a latency period of 20 years

Asbestos exposure parameters	No. of cases	No. of controls	OR*	95% CI†
Highest probability of exposure				
Not exposed	95	154	1.0	
Possible	51	71	1.2	0.8–1.9
Definite	184	87	3.6	2.4–5.3
Highest intensity of exposure				
Low	55	74	1.2	0.8–1.9
Medium	106	66	2.8	1.8–4.3
High	74	18	7.1	3.9–12.9
Highest frequency of exposure				
Sporadic	56	86	1.0	0.7–1.6
Irregular	94	46	3.3	2.1–5.1
Continuous	85	26	5.7	3.4–9.7
Duration of exposed job (years)				
1–7	63	64	1.7	1.1–2.6
8–19	74	60	2.0	1.3–3.1
≥20	88	34	5.4	3.2–8.9
Time since first exposure (years)				
20–37	77	53	2.3	1.4–3.6
38–48	83	47	2.8	1.8–4.5
≥49	75	58	2.2	1.4–3.6
Age at first exposure (years)				
<16	66	55	1.9	1.2–3.1
16–22	96	52	3.0	1.9–4.6
≥23	73	51	2.3	1.5–3.7
Cumulative exposure ("f/ml-year"‡)				
0.001–0.49	77	109	1.2	0.8–1.8
0.5–0.99	29	12	4.2	2.0–8.8
1–9.9	80	27	5.2	3.1–8.8
≥10	49	10	8.7	4.1–18.5
	Cumulative exposure ("f/ml-year")			
Temporal exposure pattern§				
Intermittent				
<0.5	66	98	1.1	0.8–1.7
0.5–0.99	19	8	4.0	1.7–9.7
1–9.99	48	21	4.0	2.2–7.2
≥10	17	5	5.9	2.1–16.7
Continuous				
<0.5	11	11	1.9	0.8–4.8
0.5–0.99	10	4	4.6	1.4–15.4
1–9.99	32	6	9.2	3.7–23.1
≥10	32	5	11.3	4.1–30.7

* Odds ratios (ORs) adjusted for age and socioeconomic category.

† CI, confidence interval.

‡ Cumulative exposure index was based on subjective assessment, that is, semiquantification of exposure by the experts and selected weighting factors assigned to each category of exposure, with no objective measurement of airborne asbestos levels. Thus, the exposure unit, f/ml-years, is expressed in quotation marks.

§ Subjects' exposure was classified as intermittent if it was sporadic or irregular and if they had never worked at a job with continuous exposure. The continuous category was reserved for subjects who had been employed in at least one job with continuous exposure.

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exposure more precisely for the cases than for the controls. The frequency of the exposure category "possible," used when the experts could not reach a definite conclusion, was higher among controls than among cases, so that this type of error cannot be excluded. We thus undertook a supplementary analysis to examine, at least in part, the effect of this bias. First, we considered all of the jobs in the possible category as nonexposed. The pattern of dose-response relation was very similar to that observed: no significant risk for subjects in the category of less than 0.5 "f/ml-years" and an odds ratio of 7.8 (95 percent CI 3.8–16.2) for those in the category of more than 10 "f/ml-years." Classifying all of the possibly exposed subjects as definitely exposed did not change the dose-response relation pattern very much either (OR = 1.0, 95 percent CI 0.7–1.6 for the lowest category and OR = 7.7, 95 percent CI 3.8–15.7 for the highest).

The validity of the dose-specific risks in our study also depends greatly on the values of the weighting factors selected for each exposure category. For this purpose, we attempted to retain the intervals used by the experts. Although this procedure is assumed to provide more precise exposure evaluation than would a relative ranking of subjects by an ordinal scale without specified boundaries, some misclassification of subjects according to dose-specific exposure probably occurred. Indeed, all jobs classified in the same exposure category were assigned the same weighting value without consideration of the variability of exposure within the category. Such nondifferential misclassification of the subjects usually attenuates the relation between exposure and disease and flattens the dose-response curve (39). We should note that the intervals used by the experts for the categories of intensity were rather dissymetric—narrow for medium exposure and large for very high exposure. There were few job periods with very high exposure, however, so that errors due to the variability in this category should have had little effect on the dose-response relation observed.

We observed a dose-response relation with cumulative exposure. Because, as stated, the exposure assessment for the earliest periods might have been underestimated and because of the imprecision of intensity weighting factors, we tested two models using two other series of coefficients for weighting intensity of exposure: 1) second model: 0.5, 1.5, 6, and 550 fibers/ml, for low, medium, high, and very high exposures, respectively (midpoints of boundaries), and 2) third model: 0.5, 5, 50, and 500 fibers/ml for low, medium, high, and very high exposures, respectively. These models showed a dose-response relation with the CEI similar to that in the first model, but they did not show as clear a dose-response trend as the first model. In the second model, the odds ratio was 1.0 (95 percent CI 0.7–1.6) for the lowest dose and 6.4 (95 percent CI 3.4–12.2) for the highest. The corresponding odds ratios for the third model are 0.9 (95 percent CI 0.5–1.4) for the lowest and 7.1 (95 percent CI 4.2–11.9) for the highest.

The pattern of the dose-response curve could have depended on the length of latency period selected. We have used a 20-year latency period, as suggested by McDonald and McDonald (1, 21), who concluded that latency is seldom less than 20 years and usually 30–40 years. We also examined the effects of 10- and 30-year latency periods. The results obtained with the former were very similar to those we found with the 20-year latency period. A 30-year latency period resulted in a lower odds ratio and a less clear dose-response relation, suggesting that exposure misclassification occurred using such a long latency period.

Because no objective measurement was available to test the validity of the experts' evaluation, we express the cumulative exposure using units of f/ml-years in quotation marks. Even in cohort studies, however, precise measurement of exposure is difficult (2, 41).

In this study, we used several surrogate parameters for dose to examine dose-response relation, as suggested by Blair and Stewart (42) and Suarez-Almazor et al. (43). We considered separately the intensity, frequency, and duration of exposure, and each was significantly related to mesothelioma. The relative risk increased along with each parameter. In addition, when each of these parameters was adjusted for the others, the relative risk of each, although lower, remained significant. These results suggest that each exposure parameter contributed to some extent to the occurrence of mesothelioma, although the dose-response relation seemed to be described best by the CEI.

The existence of a causal association between asbestos exposure and mesothelioma was first demonstrated in 1960 (44). Both cohort (6–9, 11–14, 45) and case-control (32, 34–37, 46, 47) studies focusing on mesothelioma and examining surrogate parameters for dose have reported a dose-response relation.

However, because of the rarity of mesothelioma, even among asbestos workers, little quantitative information is available from which the dose-response relation can be precisely estimated (1, 41, 48).

Peto et al. (49), using mathematical models, observed that the risk of mesothelioma in one occupationally exposed cohort (North American insulators) was best described by a model in which the risk increases with the third or fourth power of time since first exposure. They also concluded that their data were compatible with a linear dose-response relation

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between the level of asbestos exposure and the risk of mesothelioma. Our data for the higher categories of CEI also support this conclusion. The pattern of a dose-response relation is more doubtful at low doses because the uncertainties of exposure evaluation are highest for low doses.

Some indication of the effect of exposure that is low level by the brevity of its duration comes from industrial cohort studies. Very few cases of mesothelioma have been observed among those whose exposure was very brief: There were no cases of mesothelioma among members of the cohort of Australian Blue Asbestos workers who were exposed for less than 3 months (47), none among the North American insulators whose exposure lasted less than 15 months (4), and only one, rather than the 25 expected, among Rochdale textile workers exposed for less than 10 years (8). These cohorts do not, however, provide data that allow us to examine the effect of low-intensity exposure.

Illgren and Browne (50) considered whether a threshold exposure might exist and concluded that mesothelioma was unlikely in persons exposed for less than 5 f/ml-years. Our results indicate, however, that mesothelioma cases occurred below a cumulative exposure of 5 f/ml-years and perhaps below 0.5 f/ml-years.

Very few studies have focused on the time-related pattern of exposure as a factor in mesothelioma. Schenker et al. (51) examined the risk of mesothelioma among railroad workers, distinguishing between "intermittent" and "regular" asbestos exposure on the basis of job categories. No significant risk was observed for those whose exposure was intermittent, but those in the regular exposure category were at high risk.

Our study examined the temporal exposure pattern according to the frequency of exposure and the CEI. We observed a dose-response relation with cumulative exposure for both intermittent and continuous patterns of exposure. Much more attention to the role of these temporal patterns is needed, adjusting for cumulative exposure. Our results suggested that intermittent exposure does not entail as high a risk of mesothelioma as does continuous exposure. Assessment of this apparent excess risk of continuous compared with intermittent exposure, however, should bear in mind the likelihood that more subjects with intermittent exposure are misclassified.

We could not examine mesothelioma risk according to fiber types because our study design (i.e., case-control study in a general population) did not allow us to identify those subjects whose exposure was only to chrysotile fibers.

The odds ratio between exposure to asbestos and mesothelioma was much higher for the women in our study than for the men. No evidence of individual, sex-related susceptibility to mesothelioma has been found (52). One explanation for this might be the different distributions of asbestos-related occupations between men and women. Since asbestos-related occupations were rarely held by women, any exposure that did occur may have been very well characterized, leading, in turn, to fewer misclassification errors than for males, particularly among controls.

We found a clear dose-response relation between cumulative exposure to asbestos and pleural mesothelioma in a population-based case-control study with retrospective assessment of exposure. A significant excess of mesothelioma was observed for levels of cumulative exposure that were probably far below the limits adopted in many industrial countries during the 1980s.

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EXHIBIT 31

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Asbestos and Man-Made Vitreous Fibers as Risk Factors for Diffuse Malignant Mesothelioma: Results From a German Hospital-Based Case–Control Study

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Background *This study examines the role of occupational factors in the development of diffuse malignant mesothelioma with special emphasis on the dose–response relationship for asbestos and on the exposure to man-made vitreous fibers (MMVFs).*
Methods *One hundred and twenty-five male cases, diagnosed by a panel of pathologists, were personally interviewed concerning their occupational and smoking history. The same number of population controls (matched for sex, age and region of residence) underwent similar interviews by trained interviewers. Odds ratios (OR) were calculated for an expert-based exposure index using conditional logistic regression.*
Results *Exposure to asbestos shows the expected sharp gradient with an OR of about 45 for a cumulative exposure > 1.5 fiber years (arithmetic mean 16 fiber years). A significant OR was calculated even for the lowest exposure category "> 0–≤ 0.15 fiber years". Although the mean cumulative exposure to MMVF is roughly 10% of the exposure to asbestos, an increased OR is observed in an ever/never evaluation. This observation is heavily hampered by methodical problems. A corresponding case–control study was performed using a lung tissue fiber analysis in addition to interviews. Both interviews and the lung tissue analysis yielded similar OR levels between the reference and the maximum exposure intervals.*
Conclusions *Despite a possible influence as a result of selection and information bias, our results confirm the previously reported observation of a distinct dose–response relationship even at levels of cumulative exposure below 1 fiber year. Moreover, the study confirms that asbestos is a relevant confounder for MMVF. A causal relationship between exposure to MMVF and mesothelioma could neither be detected nor excluded, as in other studies.* Am. J. Ind. Med. 39:262–275, 2001. © *2001 Wiley-Liss, Inc.*

***KEY WORDS:** mesothelioma; case–control; asbestos; MMVFs; occupational history; lung tissue*

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INTRODUCTION

Diffuse malignant mesothelioma (DMM) is predominantly caused by asbestos fibers [HEI-AR, 1991; Mark and Yokoi, 1991; Rösler et al., 1994; Spirtas et al.,1994; Gun, 1995]. In the past, its incidence in the general population was low, but it has been increasing for decades in industrialized countries and it may take another 20 years before its peak is passed [Peto et al., 1999]. In addition to asbestos exposure at the workplace, contact in the household and environmental exposure to asbestos are established causes of DMM [Hain and Dalquen, 1974; Vianna and Polan, 1978; Großgarten and Woitowitz, 1993; Rödelsperger et al., 1996; Schneider et al.,1996; Magnani et al., 1997; Rees et al., 1999]. It has been demonstrated that the time since initial exposure and the type of asbestos are important for the quantification of the risk [Doll and Peto, 1985]. A risk estimate based on accurate workplace measurements is not yet available. Nevertheless, it has recently been demonstrated that an increase of risk may occur even below a cumulative exposure of a few fiber years (fibers/mL × years) [Iwatsubo et al., 1998]. However, a reliable dose–response relationship between the concentration of long amphibole fibers and the risk of mesothelioma has been consistently established by several research groups on the basis of lung tissue fiber analysis, while no relationship was observed for chrysotile fibers [Rödelsperger et al., 1999]. This is true even though the amphibole fibers in the lung tissue do not seem to be a good indicator of the fiber content of the pleura, where chrysotile fibers are predominantly observed [Smith and Wright, 1996].

Animal experiments show that other kinds of fibers, with a minimal length above 5 μm, may also induce mesothelioma after direct application [Pott, 1991]. They confirm the hypothesis that fibers, which are sufficiently long, thin, and durable, are carcinogenic [DFG, 1997]. Besides asbestos sufficient evidence for this hypothesis in humans has only been obtained for erionite [IARC, 1988; DFG, 1997]. In inhalation experiments in rats only erionite fibers caused mesothelioma whereas asbestos and ceramic fibers, but not other man-made vitreous fibers (MMVF), provoked lung cancer. However, even for asbestos this effect was observed only at very high fiber concentrations, which are not feasible for MMVF, since they are much longer and thicker than asbestos fibers and would, therefore, require a much higher mass concentration. This model is, therefore, considered too insensitive for predicting carcinogenic effects in humans [Pott and Roller, 1993; Infante et al., 1994, 1996; Rödelsperger and Woitowitz, 1995]. The question of the carcinogenicity of MMVF is of utmost public health relevance. Epidemiological observations have revealed an increased lung cancer mortality in producers of glass, stone, and slag wool, but the causal relationship to MMVF remained unclear [Infante et al., 1994, 1996; DeVuyst et al., 1995; DFG, 1997]. The concentration of MMVF is higher for processing than for production [IARC, 1988; WHO, 1988; Corn et al., 1992]. However, epidemiologic investigations in craftsmen processing these fibers are difficult since they also process asbestos.

The original aim of this study was to investigate not only asbestos, but also MMVF and other inorganic fibers as causal factors of the DMM [Woitowitz et al., 1993; Rödelsperger, 1996; Rödelsperger et al.,1998, 1999]. Since it was necessary to carry out a lung tissue fiber analysis, in addition to recording occupational histories, patients undergoing surgical resection for a diagnosis other than mesothelioma were selected as controls in the participating hospitals. Most of them suffered from lung cancer. In addition, a suitable group of population controls was recruited for the cases from the area of Hamburg. The results obtained for pairs of cases and population controls from the area of Hamburg are reported in this paper. It was from the first results of this study that there was a strong association between exposure to asbestos and to MMVF. Hence, it was first necessary to make a thorough examination of the influence of asbestos. Meanwhile, in connection with the study of Iwatsubo et al., [1998], a critical commentary on the use and the methodological problems of population-based mesothelioma case–control studies has emphasized the importance of direct risk estimation in a low-dose population [Siemiatycki and Boffetta, 1998]. Accordingly, we have examined the relationship between exposure to asbestos and the risk of mesothelioma and compared this with the relationship which was obtained from lung tissue fiber analysis [Rödelsperger et al., 1999].

MATERIAL AND METHODS

Subjects

The 415 incident patients with suspected diagnosis of diffuse malignant mesothelioma (DMM), recruited between January 1, 1988 and December 31, 1991 from clinics in Hamburg, Heidelberg, Essen, Munich, and Berlin, included 324 cases (275 male, 49 female) with a definite diagnosis confirmed by a panel of pathologists.

The present study was restricted to cases from Hamburg, which were individually matched to population controls. They had to be of German nationality, willing and able to give a personal interview and to provide written informed consent. Of 137 male and 37 female cases (almost all patients of DMM being treated in two specialized hospitals in Hamburg during the recruitement period) it was possible to include 125 male cases in the final analyses after matching with controls (according to region of residence, sex, year of birth ± 5 years). Females were not included on account of the small sample available. Population controls

TABLE I. Characteristics of Male Cases and Controls from Hamburg

	Cases	Controls
No.	125	125
Age (years)		
Median	62.0	64.0
Mean	62.6	64.2
SD	9.4	10.0
No. of jobs		
Median	6.0	6.0
Mean	6.0	5.6
Ever smoked	99 (79.2%)	107 (85.6%)

were randomly drawn from the mandatory registries of suitable administrational units [Woitowitz et al., 1993]. The response rate was 63%. One case/control pair had a year-of-birth difference >5 years. However, this did not influence the results presented in this paper. Some basic characteristics of cases and controls are shown in Table I.

Data Collection

In the first step, a structured questionnaire was used by trained interviewers to obtain information on job history and, in a second step, to obtain further specific information on occupational exposure to asbestos, MMVF, and other mineral fibers. Additionally, smoking, residential exposure, medical history, leisure-time activities and basic demographic characteristics were registered. Interviewers participated in several intensive training seminars during the recruitment phase. In a subset of variables from the questionnaire—(e.g., the duration of the interview and the number of job periods) the data were entered immediately in order to monitor and reduce possible interviewer effects.

Quantification of Occupational Exposure to Asbestos and MMVF

The description which in the first step was obtained for every job held (duration of at least 12 months) was supplemented with information from the second step (without limit of duration) and an expert industrial hygienist used these data to assess the cumulative dose of exposure to Asbestos and to man-made vitreous fibers.

Expert judgment was obtained blind with respect to the case–control status. Fiber concentration was quantified by assignment to one, two or even three of five exposure categories (Table II).

The geometric mean of the experts' estimate of the maximal and minimal value of fibers/ml per working shift

TABLE II. Definition of the Categories of the Asbestos Fiber Concentration and the Reproducibility of the Estimates of Two Experts

Categories of fiber concentration: definition by concentration intervals

Category	Fiber concentration fibers/ml				
	None	Low	Middle	High	Very high
Minimal value	0	0.005	0.025	0.25	2.0
Maximal value	0	0.025	0.25	2.0	10.0

Expert 1	Expert 2				
	none	low	middle	high	total
	Estimation of the minimal concentration value[a]				
None	191	10	4	—	205
Low	10	19	6	3	37
Middle	1	2	7	2	12
High	1	2	8	—	11
Total	203	32	25	5	265
	Estimation of the maximal concentration value[a]				
None	191	—	9	5	205
Low	3	1	—	3	7
Middle	5	2	7	4	18
High	3	1	6	24	34
Total	203	4	22	36	265

[a]Number of job periods per exposure category. If the experts were not able to assign the exposure to a certain category, they were allowed to combine two or more of them. In this case the minimal value resulted from the lowest and the maximal value from the highest category. Agreement is 82% for minimal and 84% for maximal concentration value.

was multiplied by the number of the working shifts with exposure. Integrating over the life span of each individual yielded the cumulative exposure to fibers (fiber years). For job periods with exposure to both asbestos fibers and MMVF the duration of exposure was only recorded once for both fiber species together. Because of the large differences between the minimal and maximal value of the concentration estimate and of the category of the frequency of working shifts with exposure, the arithmetic mean concentration, averaged over all job periods and correspondingly the cumulative exposure in fiber years, is taken as five times the geometric mean value (compare Table III and the section "Error of the Expert Estimate of the exposure to asbestos").

The quantitation is based on our own experience in measurement [Woitowitz and Rödelsperger, 1983; Arbeiger et al., 1984; Rödelsperger et al., 1980, 1986, 1991], the international literature for asbestos [Woitowitz et al., 1983] and for man-made fibers [Walton and Coppock, 1987; IARC, 1988; WHO, 1988]. A second industrial hygienist reevaluated 265 job periods of 50 patients in order to check the reproducibility of the concentration estimates. The agreement for both rates is described in Table II.

The experts agreed in assigning the category "none" for 191 job periods (72%). For 48 periods (18%) asbestos exposure was scored by both while 26 times (10%) this was scored by only one of them. In all, the experts scored asbestos exposure for 74 job periods. When these are evaluated separately only 34% of the minimal values and 43% of the maximal values are classified in the same exposure category, while 15% of the minimal and 36% of the maximal values differ for more than one category. The weighted κ values are 0.61 (95% CI 0.53–0.70) and 0.68 (95% CI 0.59–0.78) for the minimal and maximal concentration values, respectively, (SAS-procedure proc freq). If distinction is only made between exposed and unexposed people, both values increase to 0.72 (95% CI 0.62–0.82). For each expert the number of overestimates was nearly equal to the number of underestimates. Finally, the following parameters were selected for each person:

- Time since first exposure (years) defined as years since the beginning of the first job period with exposure to asbestos.
- Duration of an asbestos exposure (years) defined as duration of all job periods with exposure to asbestos.
- Highest intensity of asbestos exposure (f/ml) estimated during any period of the working life.
- Cumulative exposure to asbestos and MMVFs (f/ml × years) defined as fiber dose. For asbestos the cumulative exposure was calculated until the date of the interview and to time points 10 and 20 years previously.

Statistical Analysis

Odds ratio (OR) and 95% confidence intervals (95% CI) were calculated by conditional logistic regression using the SAS procedure PHREG [SAS institute, 1992]. Occupational risks were analyzed separately for job categories and industries, and for the parameters of exposure.

Job titles and industries were coded according to standard classifications [Statistisches Bundesamt, 1975, 1979]. The analysis of job histories was based on these

TABLE III. Frequency of the Geometric Mean Fiber Concentrations per Working Shift with an Exposure to Asbestos, Estimated for 454 Single Job Periods with an Exposure to Asbestos. Possible Concentrations are Obtained from Table II by Taking the Geometric Mean Values for Various Combinations of Lower and Upper Limits of the Concentration Ranges

Job periods				Cases		Controls	
				n	%	n	%
All periods				746		697	
Periods with exposure to asbestos							
No.	GM f/ml	Range f/ml		324	100	130	100
1	0.0112	0.005	0.025	108	33.3	51	39.2
2	0.0354	0.005	0.25	99	30.6	47	36.2
3	0.0791	0.025	0.25	25	7.7	8	6.2
4	0.1	0.005	2	12	3.7	4	3.1
5	0.2236	0.025	2	41	12.7	13	10.0
6	0.5	0.025	10	16	4.9	0	0.0
7	0.7071	0.25	2	1	0.3	0	0.0
8	1.581	0.25	10	22	6.8	7	5.4
9	4.472	2	10	0	0.0	0	0.0

codes which were grouped into 32 job categories and 21 branches of industry [Jöckel et al., 1994, 1998]. An ever vs. never exposure concept was used, in addition to information on the longest-held job, which had been commenced at least 20 years before the onset of disease; on evaluation this yielded comparable results.

In order to adjust for the established impact of asbestos on mesothelioma risk, 4–5 categories of the different parameters of exposure were set up and included into the logistic regression as additional dummy variables. Five categories of cumulative exposure were used for MMVFs in a similar manner. An ever/never evaluation was also performed.

RESULTS

Job History

The mean duration of lifetime employment was 42 years for cases and 43 years for controls. Asbestos exposure was registered for 454 of a total of 1,443 single job periods of cases and controls. Table III gives the geometric mean value (GM) and the corresponding range of fiber concentration per shift for the five categories of exposure defined in Table II and their combinations together with the number of job periods assigned to these categories. The percentage of the job periods with asbestos exposure amounts to 43.4% for cases compared to 18.3% for controls. The frequency distribution of job periods with asbestos exposure is similar for cases and for controls.

Table IV gives the percentage of job periods with asbestos exposure, the duration of exposure and the arithmetic mean fiber concentration calculated as five times the GM value. After 1950 the frequency of exposure and fiber concentration, but not the mean duration of exposure, are higher for the cases than for the controls.

Tables V and VI show the number of cases and controls together with the odds ratios (ORs) for 22 of the 32 predefined occupations and for 20 of the 21 predefined industries, where at least five cases or five controls were exposed. Again the percentages of job periods with asbestos exposure and the estimate of the arithmetic mean fiber concentrations during these periods are also presented.

Significantly increased ORs and the highest numbers of mesothelioma cases were observed for the occupation of mechanics, fitters, and plumbers (n = 62), and in the industry of engine and vehicle building (n = 82).

TABLE IV. Job Periods Starting During Different Periods of Calendar Time

Timeperiod	Cases		Controls	
Number of job periods and % with asbestos exposure				
Before 1950	343	39.7%	340	14.1%
1950–1970	352	50.3%	261	24.6%
1970–	51	21.6%	76	17.1%
Mean duration of exposure (years)				
Before 1950	6.02		6.14	
1950–1970	10.94		10.04	
1970–	5.85		7.29	
Mean fiber concentration (fibers/ml)				
Before 1950	0.75		0.97	
1950–1970	1.08		0.51	
1970–	0.90		0.13	

Characterization of job periods according to the percentage with an asbestos exposure, the mean duration of exposure and the estimate of the (arithmetic) mean of the fiber concentration, which is averaged amongst all shifts with an exposure.

Asbestos Exposure

We considered years since first exposure, years of duration of exposure, highest intensity of exposure estimated during any period of the working life, and cumulative asbestos exposure as parameters of an occupational asbestos exposure (Table VII). None of these parameters has been adjusted for the effects of the others. Comparing exposed persons to not-exposed ones yields a significantly increased OR for any of the categories of any of these parameters. For each of the parameters, with the exception of time since first exposure, the OR even increases among exposed persons, when the lower intervals of exposure are compared to the higher ones. A steep risk gradient up to OR = 47 is observed for the highest intensity of exposure, for years of exposure, and for the cumulative dose estimate. This result does not alter very much by introducing cut-off points 10 or 20 years before the end of observation for the calculation of the cumulative exposure.

Exposure to MMVFs

Table VIII reveals that elevated risks have been found for three intervals of cumulative exposure to MMVFs, although their concentration range is lower, by a factor of 10, than the corresponding range for asbestos exposure (see Table VII). Adjustment for asbestos exposure, however, causes a distinct reduction of the OR and the results are no longer significant. Instead of analyzing on the basis of a dose estimation, an ever/never evaluation may be performed by comparing the first exposure group (0 fiber years = never) to the three upper groups altogether (= ever). In this case the OR remains significant even after adjustment for asbestos.

Additionally, in Table IX exposure estimates and ORs are compared for four different groups of persons with and without exposure to asbestos or MMVFs. A significantly increased OR is registered for cases and

TABLE V. Number of Cases and Controls and Odds Ratio from an Ever/Never Evaluation of 22 of 32 Occupations Where At Least Five Cases or Five Controls were Exposed. Within Each of the Occupations the Job Periods are Characterized by the Percentage of Jobs with an Asbestos Exposure and by the Arithmetic Mean of the Fiber Concentration

					Jobs periods of cases and controls		
					All periods		Only periods with an asbestos exposure
Key[a]	Occupation[a]	Cases No.	Controls No.	OR[b]	% of all periods		Fiber concentration GM × SF/ml
11,41–43	Farmer	17	25	0.60	92	0	0.00
21–32, 44,52	Forestry worker, fisherman, Animal husbandry worker	4	6	0.67	15	13.3	0.10
12,51	Gardener, vineyard worker	2	5	0.40	16	0	0.00
71–91	Miner	6	8	0.75	21	0	0.00
141–150	Chemical processor and related worker	13	11	1.18	48	60.4	1.39
181–184, 501–504	Joiner, wood processing worker	9	8	1.12	79	21.5	0.34
191–252	Metal production and processing worker	26	14	2.09*	81	45.7	0.80
261–306	Mechanician, fitter, plumber	62	21	2.82*	359	72.1	0.79
311–315	Electrician	15	5	3.00*	88	37.5	0.41
391–433	Food production and processing worker	3	5	0.60	47	0	0.00
441–453	Carpenter, bricklayer, roofer	8	10	0.78	96	34.4	0.69
461–472	Road construction worker, pipe layer, well digger, Unskilled construction worker	17	17	1.00	96	15.6	0.73
481–492	Tile setter, plasterer, paviour, upholsterer	11	3	3.67*	30	63.3	2.94
531	Unskilled worker not elsewhere classified	5	8	0.57	19	31.6	0.14
541–549	Stationary engine and heavy equipment operator	19	7	3.40*	41	65.9	0.70
601–635	Technician engineer	19	9	2.25	89	46.1	0.28
681–706	Sales assurance agent	11	26	0.38*	80	3.8	0.06
711–744	Transportation & store worker	48	39	1.32	227	22.9	0.20
751–784	Administrative & organization clerk	34	49	0.57*	201	4.0	0.07
791–805	Protective service worker	59	71	0.56	240	7.5	0.09
861–893	Teacher, scientist, social worker	4	7	0.57	41	14.6	0.10
901–937	Housekeeper, cleaner, hairdresser, bartender	5	7	0.71	28	14.3	0.62

[a] A priori defined occupational groups, see [Jöckel et al., 1994, 1998], code according to standard classification of industries [Statistisches Bundesamt, 1975, 1978].
[b] Odds ratio matched for age and region of residence.
[c] Cases, population controls and control patients.
*$p < 5\%$, two-sided.

controls, which were exclusively exposed to MMVFs, if they are compared to those exposed to neither MMVFs nor asbestos.

DISCUSSION

Retrospective exposure assessment is one of the main problems arising from case–control studies [Finkelstein, 1995; Siemiatycki, 1996, 1997; Benke et al., 1997]. Exposure estimates are often based on job exposure matrices (JEM) where estimates describing probability, frequency, and intensity of exposure are not related to specific persons but to specific combinations of occupations and industries. Different types of exposures are often estimated side by side [Siemiatycki, 1996; Benke et al., 1997; Cocco, 1999]. The mesothelioma case–control study of Iwatsubo et al. [1998] for example, is based on this concept. In our study, however, the frequency and the upper and lower limits of intensity of exposure to asbestos and MMVFs were estimated semi-quantitatively for the single job periods of individual persons. Other types of fiber-containing materials, such as talc or attapulgite, were considered at least qualitatively. Besides asbestos, MMVF was by far the most frequent cause source of exposure to fibers and it was only for MMVF that

TABLE VI. Number of Cases and Controls and Odds Ratio from an Ever/Never-Evaluation of 20 of 21 Industries Where at least Five Cases or Five Controls were Exposed. Within Each of the Industries the Job Periods are Characterized by the Percentage of Jobs with an Asbestos Exposure and by the Arithmetic Mean of the Fiber Concentration

					Jobs: periods of cases and controls		
					All periods		only periods with an asbestos exposure
Key[a]	Industries[a]	Cases No.	Controls No.	OR[b]	% of all periods		fiber concentration GM × 9 f/ml
011–077	Fishing, forestry, farming and horticulture	20	31	0.58	124	1.61	0.10
100–118	Energy and mining	20	11	1.82	61	34.4	1.89
200–205	Chemical and oil industry	13	16	0.79	65	30.6	0.18
210–216	Rubber and plastics	9	7	1.33	36	83.3	1.62
221–227	Stone and glass	15	9	2.00	35	55.6	2.73
230–239	Metal production	20	9	3.20*	72	52.8	0.30
240–249	Engine and vehicle building	82	45	3.18*	351	65.5	1.01
250–259	Electrical and sheet metal	15	10	1.71	61	14.8	0.23
260–269	Paper, wood, and print	11	17	0.57	97	5.2	0.13
270–279	Leather and textile	6	7	0.86	19	10.5	0.65
281–289	Food and tobacco	16	14	1.17	77	2.6	0.06
300–308	Construction	36	36	1.00	232	25.9	0.43
310–316	Installation	27	9	4.00*	92	57.6	0.29
401–439	Trade	11	17	0.63	115	7.0	0.11
511–517	Transportation	36	29	1.33	152	32.9	0.18
551–555	Stock-keeping and shipment	14	12	1.15	74	32.4	0.25
600–657	Financial service and insurance	4	9	0.38	33	3.0	0.18
731–745, 93	Cleaning service, barbershop, house-keeping, waste disposal	4	8	0.50	25	16.0	0.59
751–799, 94, 96	Education, sport, health	11	20	0.53	75	12.0	0.20
811–860	Public service and non-profit organizations	73	80	0.74	352	9.9	0.11

[a] A priori defined industries, see [Jockel et al., 1994, 1998], codes according to standard classification of industries [Statistisches Bundesamt, 1975, 1979].
[b] Odds ratio matched for age and region of residence.
[c] cases, population controls and control patients.
* $p < 5\%$, two-sided.

an increased risk was estimated (Tables VIII, IX). However, exposure to MMVFs is heavily confounded with exposure to asbestos; therefore this result has to be discussed very carefully. First, a critical examination of the study design has to be performed [Siemiatycki and Boffetta, 1998] and in addition, comparisons may be made with results which have previously been obtained for a second series of hospital controls using the same method, and with results of lung tissue fiber analysis [Woitowitz et al., 1993; Rödelsperger, 1996; Rödelsperger et al., 1999].

Selection Bias

It has been argued that the diagnosis of mesothelioma may be made more probable if asbestos exposure is evident [Siemiatycki and Boffetta, 1998]. This diagnostic bias would increase the risk estimate for asbestos. In our study a panel of pathologists was installed to exclude diagnostic errors. Each diagnosis obtained by the pathologist of a participating hospital had to be confirmed by a member of this panel. The whole panel was then included in the decision for 24% of the diagnoses, where the decisions were discrepant. A total of 15% of the cases considered was discarded (5% in agreement between the two pathologists and 10% by a panel decision).

Selection bias for the selection of population controls is minimized by the matching procedure. In contrast, many of the hospital controls of our previous study suffered from lung cancer [Woitowitz et al., 1993], which is well known to be caused by asbestos.

TABLE VII. Odds Ratios for the Relationship between Mesothelioma and Asbestos Exposure Together with the Number of Cases and Controls According to Different Parameters of the Asbestos Exposure [a]

	125 Cases	125 Controls	Odds ratio [a]	95%-CI
Time since first exposure (years)				
not exposed	11	67	1	
≤30	12	4	22.5	4.3–119
>30–40	40	22	18.9	5.3–67.3
>40	62	32	19.6	5.7–67.2
Duration of exposure (years)				
not exposed	11	67	1	
>0–10	24	21	13.4	2.9–37.1
>10–20	22	14	16.5	4.1–65.6
>20–30	19	8	27.7	5.8–132
>30	49	15	43.7	10.8–177
Highest intensity of exposure [b]				
not exposed	11	67	1	
low	14	12	9.2	2.3–35.9
medium	37	25	17.9	5.0–64.4
high	63	21	46.3	12.1–178
Cumulative exposure up to end of observation (fiber years)				
not exposed	11	67	1	
>0–0.15	14	12	7.9	2.1–30.0
>0.15–1.5	38	25	21.9	5.7–83.8
>1.5–15	46	16	47.1	11.5–193
>15	16	5	45.4	8.1–257
Cumulative exposure up to 10 years before end of observation				
not exposed	11	67	1	
>0–0.15	15	13	7.9	2.1–29.5
>0.15–1.5	39	24	24.0	6.2–93.0
>1.5–15	45	16	51.8	12.4–216
>15	15	5	42.6	7.3–249
Cumulative exposure up to 20 years before end of observation				
not exposed	14	68	1	
>0–0.15	15	13	9.2	2.4–35.0
>0.15–1.5	44	24	20.5	5.6–72.6
>1.5–15	40	16	32.2	8.5–122
>15	12	4	43.8	7.1–269

[a] Odds ratio matched for age and region of residence.
[b] "Low" (<0.01 fibers/ml), "high" (>1 fiber/ml) or "medium" (otherwise) according to five times the geometric mean fiber concentration given in Table II.

Information Bias

Information bias may be caused by the different situation of the interview for mesothelioma patients compared to that for the healthy reference population [Siemiatycki and Boffetta, 1998]. In the total for our population controls it is likely that there will be a great deal of information bias increasing the risk estimate while selection bias ought to be low. This may lead to an overestimate. In contrast, the interview situation should be comparable both for the cases and for the hospital controls of the previous study, who have been treated by pulmonary resection. Hence, information bias, which may increase the risk estimate, should be low, while selection bias, which is expected to decrease the risk, should be high. The overall result might be to underestimate the risk. In order to reduce this type of bias, biographical

TABLE VIII. Odds Ratios and Number of Cases and Controls in Males and Exposure to MMVF

Geometric mean × 5 (fiber years)	Cases	Controls	Odds ratio[a]	95%-CI	Odds ratio[b]	95%-CI
never = 0	70	111	1.00	—	1.00	—
>0–0.015	10	6	2.96	0.92–9.57	0.78	0.16–3.77
>0.015–0.15	11	4	4.19*	1.17–15.00	3.11	0.55–17.2
>0.15–1.5	20	1	26.28*	3.39–203.75	7.95	0.88–72.3
>1.5	14	3	6.50*	1.47–28.80	9.43	0.72–41.0
ever >0	55	14	6.12*	2.90–12.93	3.08*	1.17–8.07

[a] Odds ratio matched for age and region of residence.
[b] Odds ratio adjusted for asbestos fiber years by means of four indicator variables, as defined in Table III.
* $p < 5\%$, two-sided.

history of all job periods was obtained in a first step, then a check list of asbestos and other fibrous products, including brand names, working processes and photos, was presented in a second step, but only minor additions were obtained.

Exposure Assessment Bias

An underestimate of the higher past exposure levels may be caused by information bias or even by change in the methods of fiber counting [Doll and Peto, 1985; Siemiatycki and Boffetta, 1998]. A magnification of the dose–response relationship may result. On the other hand, random errors as a result of misclassification of exposure usually bias the risk toward null value (no association) [Armstrong, 1998].

In Table IV, where exposure estimates of exposure to asbestos for different time periods are presented, the percentage of jobs with asbestos exposure, the mean duration of exposure, and fiber concentration for cases is higher between 1950 and 1970 than in the time periods before and afterwards. This pattern, apart from the fiber concentration, is similar for population controls. A similar pattern in time was observed in the French mesothelioma case–control study [Iwatsubo et al., 1998]. In this study the highest rate of job periods with asbestos exposure also was observed between 1950 and 1970, but the percentage of about 38% in cases and 20% in controls lies below the German results. In contrast, the prevalence of exposure is higher in France from 1970. This observation might be explained by the restrictions in the use of asbestos, which were introduced in Germany at the end of the 1970s.

In our study quantitative estimates may be subject to error by using only one common duration of exposure in job periods where asbestos and MMVFs were used side by side. For job periods with exposure to asbestos the percentage for an additional exposure to MMVFs was only 22% in cases and 9% in controls. In periods with an exposure to MMVFs the percentage of an additional asbestos exposure was 85% in cases and 67% in controls. Hence, an overestimate must be expected, particularly for the cumulative exposure to MMVFs.

TABLE IX. Exposure to Asbestos and MMVF in Males, Estimate of MMVF and Asbestos Fiber Dose, Numbers of Cases and Controls and Odds Ratio

Exposure	Mean fiber years	125 Cases	125 Controls	Odds ratio*	95%-CI
MMVF –	0	9	65	1.00	—
Asbestos –	0				
MMVF +	0.6	2	2	15.4*	1.05–218
Asbestos –	0				
MMVF –	0	61	46	19.8*	4.7–83
Asbestos +	7.1				
MMVF +	2.4	53	12	61.3*	12.9–292
Asbestos –	16.2				

* Odds ratio matched for age and region of residence.
* $p < 5\%$, two-sided.

Error of the Expert Estimate of the Exposure to Asbestos

According to Table III the factor between the GM value of fiber concentration per shift and the upper and lower limits, respectively, of the exposure category which corresponds to this GM value may vary between 2.2 (No.9), and 20 (No.4) with weighted mean values of 6.4 for cases and 5.5 for controls. This expert estimate of the range of uncertainty largely describes the random error for single job periods. The mean bias for the exposure estimates of all job periods should lie below these factors. A similar but lower uncertainty results from classifying the number of working shifts with exposure to asbestos into only three categories where the highest category, e.g., even everyday exposure which is registered as "more than once per week," only contributes half of the working days of a year.

For a single job period the cumulative exposure to asbestos is obtained by the years of duration of exposure normally multiplied with the arithmetic mean values (AM) of fiber concentration per shift and the rate of working shifts with the exposure. Here AM amounts to half the sum of the lower and the upper limit of the expert estimate and therefore, an upper limit much higher than the lower limit would yield an AM which is roughly half the upper limit. A better description of the range of uncertainty is obtained using the GM, but GM values are systematically lower than AM values. The AM of fiber concentration is estimated five times the GM in order to compensate this bias, which results from the uncertainties of the estimate of the fiber concentration (factor 3) and of the frequency of asbestos exposure (factor 1.5). The weighed average of the AM values for all jobs with exposure to asbestos is 0.93 f/ml for the cases while it is 0.66 f/ml for the controls (Table III).

Consistency Between Exposure and Risk Estimates

OR significantly increases (Table VII) for each of the three parameters of cumulative exposure even within the first exposure interval > 0–≤ 0.15 fiber years. Exceptional behavior is observed for the time after first exposure. Here the OR remains constant, although a steep relationship should exist for this parameter. However, this discrepancy may be explained from study design: Controls were matched to cases with respect to the year of birth. There is a good correlation between cases and controls for the age and the time of first exposure.

In Tables V and VI, the percentage of job periods with asbestos exposure and the concentration estimate as parameters of exposure are compared to the OR. The correlation is better with the percentage of job periods with an asbestos exposure ($R = 0.810$ for occupations and $R = 0.769$ for industries, both $P < 0.001$) than with mean fiber concentration ($R = 0.765$, $P < 0.001$ and $R = 0.524$, $P = 0.018$). The most distinct discrepancy appears between the installation and the stone and glass industries. For the former, the highest OR of 4 (statistically significant) is associated with a small average concentration of 0.29 f/ml. For the latter, an OR of 2 (not significant) is associated with the highest concentration estimate of 2.7 f/ml.

In the "*installation industry*" a total of 56 job periods of *27 cases* and 21 job periods of *nine controls* was observed. They worked as tin smith or plumber (12 cases and 2 controls), carried out heating installation (5 cases and 3 controls), air conditioning installation (2 cases and 1 control) or worked as electrician (4 cases and 2 controls) or, interior designer or painter (4 cases and 1 control). In this industry working procedures were distributed homogeneously among cases and controls and, therefore, estimates of asbestos exposure were similar for both of them. The low exposure estimates are reliable since exposure mainly resulted from pipe insulation with asbestos, asbestos cement, welding protection, and sealing.

In the "*glass and stone*" *industries* a total of only 27 job periods of *15 cases* and *nine controls* was observed. Nine cases but only one control ($P = 0.02$) worked as insulators and definitely used asbestos in most periods; three times this was spray asbestos. One further case had mined asbestos in the Urals as a prisoner of war and two others had worked in the asbestos industry. The latter is also true for one control but he mainly worked inside the office as a designer. One of the remaining three cases was a boilerman in a glass factory. The other two worked as locksmiths in the cement industry. In contrast, the seven remaining controls worked in quarries ($n = 4$) and in concrete production ($n = 3$). The work periods of cases and controls are very obviously different in the "glass and stone" industry. While the high exposure estimate is convincingly justified by the work place descriptions of the cases, the OR is reduced by a large number of controls with places of work where a much lower degree of asbestos exposure should be expected.

Comparison with Lung Tissue Fiber Analysis

The fiber burden of the pulmonary tissue has been analyzed for a total of 66 cases (60 male and 6 female) and 66 hospital controls (primarily lung cancer cases) of the original study, among them 27 cases and 39 controls are from Hamburg as reported elsewhere [Rödelsperger, 1996; Rödelsperger et al., 1999]. The dose estimates for these cases on the average (AM) are 1.7 times higher, than for the cases from the present study. A subsample of 20 male cases was included in both studies, among them was the one with the highest exposure estimate of 167 fiber years.

For the patients of the lung tissue study, cumulative asbestos exposure, as derived from the interview, correlates with the concentration of asbestos fibers longer than 5 μm in

the lung tissue for amphibole ($R = 0.44$, $P < 0.001$), but not for chrysotile fibers [Rödelsperger, 1996]. Regression analysis reveals that a fiber dose of one fiber year roughly corresponds to a concentration of 80,000 amphibole fibers longer than 5 μm per gram dry lung tissue (g dry). This relationship is in good agreement with other estimates [Consensus Report, 1997].

A clear dose–response relationship could be observed between the concentration of these long amphibole fibers and the risk of mesothelioma [Rödelsperger and Woitowitz, 1995; Rödelsperger et al., 1999]. An OR of almost 100 was obtained from the almost linear relationship, when 10 of 66 cases (15%) in the reference interval < 50,000 fibers/g dry were compared to 29 of 66 cases (44%) in the uppermost interval ≥ 500,000 fibers/g dry.

Correspondingly, from the present study (Table VII) an OR of about 45 is observed, if the 11 cases (9%) of the reference interval (0 fiber years) are compared to the 62 cases (50%) of the two uppermost intervals ≥ 1.5 fiber years.

Within these uppermost exposure intervals the average dose estimate combined for cases and controls amounts to 25 fiber years for the lung burden study and to 16 fiber years for the interview study. Obviously, the results of both studies are very similar. This is true, though the OR of the lung burden study should be reduced in comparison to the present study because of the choice of hospital controls instead of population controls. On the other hand, the diminution of the OR by random error may be much stronger for the interview study.

Comparison With Other Case–Control Studies

In this study some 91% of the cases compared to 54% of the controls were occupationally exposed to asbestos (Table VII). Since only male individuals from Hamburg were included, the results are not representative for the German population as a whole. For example, higher exposure to asbestos can be expected in Hamburg due to shipyards and asbestos-processing industries [Hain and Dalquen, 1974]. The rates correspond to the upper limits of the ranges of 12–95% for mesothelioma cases and 2–48% for controls, which have been reported from international mesothelioma case–control studies [Brochard et al., 1993].

Iwatsubo et al. [1998] observed a pattern very similar to the results of Table VII for the different parameters of exposure, but in the uppermost intervals OR generally only reached 5 to 9 compared to about 45 in the German study. Again the OR remained almost constant for the time since first exposure. Its value is 2.2–2.8 in the French and 18.9–22.5 in the German study. The percentage of cases exposed is much higher for the German study (91% compared to 71%). In contrast, the rate of exposure among the German population controls is somewhat lower than for the French hospital controls (46% compared to 51%).

The ORs observed in our study fully support the well-established epidemiologic evidence for the carcinogenicity of asbestos with respect to mesothelioma. However, due to the low number of pairs with exposed controls and non-exposed cases, the absolute magnitude of the OR should be regarded with caution: the maximum OR decreased from about 100 in the original study [Woitowitz et al., 1993] to 45 in the present paper when the definition for "not exposed to asbestos" slightly was altered from "< 0.015 fiber years" to "0 fiber years" according to Iwatsubo et al., [1998]. According to Table VII the lower reference category contained 11 cases and 67 controls, among them eight pairs. The three remaining cases formed pairs with exposed controls.

Furthermore, if our results obtained for 125 population controls are compared to the results obtained for 125 additionally available hospital controls consisting primarily of lung cancer patients [Woitowitz et al., 1993], the maximum OR decreases from 100 to about 10. If, however, the matching is broken and a *stratified unconditional logistic regression* is applied the maximum OR decreases to 17 for the population controls while it remains almost constant for the hospital controls (OR = 9.4 in the maximum). A clear dose–response relationship is obtained even for these hospital controls and even for an exposure "> 0.15–1.5 fiber years" there is a significantly increased OR of 3.2 (95% CI: 1.7–6.1).

Asbestos Fibers and MMVFs

The estimate of the exposure to MMVFs is only about 10% of the exposure estimated for asbestos (Table IX) and, in addition, the effect of MMVF is greatly affected by this exposure. By adjusting for asbestos, a significantly increased OR only remains in the ever/never evaluation, which does not depend on the dose estimate (Table VIII). Considering the two cases and two controls, who were only exposed to MMVF but not to asbestos yield a significantly increased OR of 15.1 (Table IX). Despite the difference in fiber years the risk estimate is very similar for those exposed to asbestos alone. Therefore, MMVF even might be more hazardous than asbestos. However, this conclusion is severely hampered by the problems of estimation of exposure, which influence both the adjustment for asbestos exposure and the definition of non-exposed. In addition, the type of asbestos—chrysotile or amphibole—is unknown in spite of its well-known importance [Rödelsperger et al., 1999]. Further difficulties arise from the small sample size in cells with differing exposure with respect to either agent. Accordingly in the original report [Woitowitz et al. 1993], restriction to cases and controls without exposure to asbestos did not yield an increased OR since—as was discussed in the last section—the definition of the reference category was "< 0.015 fiber years" instead of "0 fiber

years." Therefore, in agreement with other studies, there is insufficient evidence to establish a causal relationship between the exposure to MMVF and mesothelioma. Nevertheless, even those studies, which do not show significantly increased incidence of mesothelioma, cannot exclude the possibility MMVF being carcinogenic with sufficient precision [Doll, 1987; Simonato et al., 1987; Marsh et al., 1990; Marsh et al., 1996; Boffetta et al., 1997].

In agreement with other studies [McDonald et al., 1990], our lung burden study did not reveal increased concentrations of MMVF, even after heavy exposure to glass or rock wool [Rödelsperger, 1996; Rödelsperger et al., 1998]. Yet, as for chrysotile, it cannot be excluded that these fibers may have caused a tumor, even if they are not present in lung tissue, when it is diagnosed [Baker, 1991; Woitowitz et al., 1991].

CONCLUSIONS

For all measures of asbestos exposure the OR increases significantly up to about 45 in the uppermost intervals. Even within the first exposure interval "> 0–≤ 0.15 fiber years" the OR significantly increases. This relationship may be influenced by information bias, exposure assessment bias, and the random error. Nevertheless, a stratified analysis, where matching is broken, and a further series of hospital controls yields a lower but still substantial OR.

The OR estimate shows a plausible relationship to the estimate of fiber concentration and to the percentages of jobs with asbestos exposure for different occupations and industries. The highest numbers of mesothelioma together with a significantly increased OR are found in "mechanics, fitters and plumbers" and for the "engine- and vehicle-building" industry. Discrepancies appear between the "glass and stone", and the "installation" industry since OR is reduced for the latter despite a much higher concentration estimate. This may be explained by a substantial difference in the type of exposure of cases and controls in this industry.

Although exposure to MMVF is much lower than the exposure to asbestos, an increased OR is observed in an ever/never evaluation. It even remains significant, if confounding by asbestos is considered by adjustment or if evaluation is restricted to cases and controls without any exposure to asbestos. However, when considering the problems of dose estimation and the sample size, a causal relationship can neither be proven nor excluded.

A further case–control analysis, based on lung tissue fiber concentrations in addition to the interview, yields similar ORs, if reference intervals and uppermost exposure intervals contain similar percentages of all cases. These results confirm the distinct dose–response relationship of the interview study even at a cumulative exposure below 1 fiber year. They clearly support the outcome of the French mesothelioma case–control study.

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EXHIBIT 32

Friday, October 20, 2006

Friday, October 20, 2006 **8:00–10:05**

ORAL SESSION

Epidemiology I

32 South Africa: The first link between asbestos and mesothelioma, but not the last word?

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The link between asbestos and mesothelioma was established by the South Africans Wagner, Marchand and importantly Sleggs, whose clinical acumen recognised both the association and that the entity was separate from fulminant tuberculosis. All three main commercial types of asbestos were mined in South Africa – white chrysotile, blue crocidolite and brown amosite, the latter two being particularly mesotheliomagenic. Peak production occurred in 1975, with more than 97% of the global crocidolite and all of the amosite (an acronym for Asbestos Mines of South Africa) produced by South Africa. A rapid decline and closure of these mines occurred within 20 years. More than 90% of all 10.2 million tons of asbestos produced in South Africa was exported, and to 70 countries at peak.

The mesotheliomagenicity of the different fibre types is crocidolite > amosite > chrysotile. The rarity of mesothelioma in chrysotile exposed workers may occur because the chrysotile contains little tremolite, a pattern which repeats in neighbouring Swaziland and Zimbabwe. While data on incidence is sparse, our best information indicates that about 75% of mesothelioma cases are occupational, with 25% associated with environmental exposure. The mining/industrial split is 45/55. The measured incidence in Prieska, a crocidolite mining town, is similar to that of Wittenoom in Australia. There is an ongoing under-diagnosis of mesothelioma in black workers, because being largely rural and poor, they have lesser access to diagnostic and therapeutic services, and also because of the masking effect of tuberculosis (worsened by the HIV pandemic), which harks back to the experience of Sleggs.

Recent litigation settlements in South African courts – first with individual claimants and later with two larger class action suits - have established significant compensation funds of - USD 100 million for asbestos-related diseases. With South Africa being the first link and having exported most of its asbestos, we are still hearing the last words as export destinations are experiencing their own epidemics of mesothelioma.

33 The mesothelioma epidemic: Occupational, environmental and spontaneous cases

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Background: Asbestos use fell abruptly in Britain around 1980, but there are now 1,900 mesothelioma deaths per year, slightly more than melanoma and almost twice the number for cervical cancer. The total number of British mesothelioma deaths is predicted to be about 90,000, most of which are still to occur, with 250,000 throughout Western Europe.

Methods: We have interviewed 624 mesothelioma patients (513 men, 111 women), 425 resected lung cancers (294 men, 131 women), and 1,420 population controls (1,112 men, 308 women) to obtain lifetime occupational and residential histories.

Results: Defining the reference group as <5 years of low-risk industrial work, odds ratios (95% CI) for 5+ years of exposure before age 35 years were 33.8 (17.0-67.2) for carpenters, 12.2 (7.2-20.8) for plumbers, electricians, painters and decorators, 3.9 (2.0-7.8) for other construction workers, and 12.8 (7.2-22.6) for other recognized high-risk occupations. The male mesothelioma rate was much lower in other occupations, corresponding to a lifetime risk of the order of 1 in 600 men in the reference group.

Conclusions: The lifetime mesothelioma risk for men born before about 1950 will thus be about 5% for carpenters, 2% for other high-risk occupations and almost 1% in general construction. Lung samples obtained from the resected lung cancers and at post-mortem from mesothelioma patients we have interviewed will define the relationship between lung fibre burden and mesothelioma risk. These data, together with lung samples obtained from young construction workers undergoing surgery for pneumothorax, will determine the risks caused by current exposures. Most female mesotheliomas have no known exposure, so the fivefold increase in female rates since the 1960s may be due largely to environmental exposure. The effects of brief and prolonged chrysotile exposure will be discussed in relation to these findings, and to developing countries where uncontrolled asbestos use is still widespread.

34 The global asbestos epidemic and its reflections in the recent Japanese experience

R-T. Lin, K. Takahashi. *Department of Environmental Epidemiology, UOEH, Kitakyushu, Japan*

Background: Japan experienced an "asbestos-panic" this past year, triggered by news that a major company was responsible for scores of asbestos-related disease (ARD) cases among former employees. Social criticism gained momentum as it became apparent that many residential victims (without job exposure) were also involved. Many other companies having used asbestos came forward to disclose their responsible share of ARDs. Administration faced allegations that control measures lagged behind Western countries: e.g., crocidolite and amosite was banned in 1995; chrysotile was banned in 2004 "in principle." The consumption curve of asbestos in Japan trailed that of many Western countries, concurrent with the "economic catch-up period," and lingered into the late 1990s. Consequently, mesothelioma mortality started to show an upsurge fairly recently (953 deaths or 7.6 per million in 2004). The recent Japanese experience is likely to be a reflection of the global asbestos epidemic.

Methods: We explored the global situation by 1) conducting an ecological study of national-level data between historical asbestos consumption and recent mortality rates of ARDs [mesothelioma (m), pleural m, peritoneal m, and asbestosis]. There is concern that, as regards ARDs, industrializing countries may be following the paths of industrialized countries. To address this issue, we followed-up on the above global analyses by 2) assessing the time-trend patterns of historical asbestos consumption and their relation to the time-trend patterns of ARDs.

Results: 1) Clear and plausible correlations were found between the two sets (consumption and mortality) of cross-sectional national data, which were marked for mesothelioma in both sexes and male asbestosis (Lancet, in press). 2) Preliminary results suggest that the global asbestos epidemic is indeed unfolding, but in a manner that can be characterized by distinctive historical time-patterns of consumption (to be submitted).

Conclusions: The burden of ARDs, which has been/is becoming apparent among industrialized countries, will likely shift towards the industrializing countries in a sequential manner.

35 Risk of pleural mesothelioma: A French population-based case-control study (1998-2002)

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Background: In industrialised countries, asbestos has been widely used for decades and occupational exposure to asbestos has been identified as the most important risk factor for pleural mesothelioma. The aims of this study are to analyse the risk of mesothelioma according to occupations and industries, to estimate the attributable risk to asbestos exposure, and to research other factors (synthetic fibres, ionizing radiations, SV40 virus, previous diseases). Only results on occupational exposure are presented in this report.

Methods: A population-based case-control study was conducted between 1998 and 2002 in 19 French districts, within the National mesothelioma surveillance program (covering 25% of the French population). This report is based on 467 cases histologically confirmed (80% males, 41-93 years old) and 868 controls matched for sex, age and district. Data were collected by trained interviewers with a standardised questionnaire, and lifetime occupational exposure was as-

sessed by experts. Logistic regression was used to estimate odds ratios (OR) and 95% confidence intervals (CIs).
Results: Among men, the highest risks were observed for the occupations of plumbers, pipe-fitters, sheet-metal workers, and for the industries of ship repair, asbestos products, metal products, and construction. A significant dose-response relationship was found between cumulative occupational asbestos exposure and pleural mesothelioma, even for the lowest exposure category >0-0.07 fibres/ml-years (OR 2.8, 95% CI 1.7-4.7). The attributable risk for occupational asbestos exposure was 63.5% (95% CI 77.2-69.8%) for men and 41.9% (95% CI 29.8-54.0%) for women.
Conclusions: This report enabled us to classify occupations and industries according to the risk of pleural mesothelioma. It provides guidance for preventive actions among specific groups of workers, and allows a continuous evaluation of their efficiency. The analyses are ongoing with a focus on non-occupational exposed subjects to asbestos (women in particular).

36 Health effects among World Trade Center responders

S.M. Levin, R. Herbert, J. Moline. *Mount Sinai School of Medicine, New York, USA*

Background: Responders to the World Trade Center disaster on 9/11 and in subsequent months were exposed to a wide range of physical and chemical hazards and to psychological stressors. Anecdotal clinical evidence suggested that many developed aerodigestive disorders and psychological sequelae.
Methods: Over 16,000 rescue and recovery workers have undergone clinical evaluation with the use of a standardized assessment protocol that includes pre- and post-bronchodilator spirometry.
Results: High rates of persistent upper and lower airway abnormalities, gastroesophageal reflux, and post-traumatic stress and depression have been identified among the examinees.
Conclusions: WTC-related exposures encountered by WTC responders caused upper and lower respiratory abnormalities, GERDS, and psychological sequela that are highly prevalent and persistent. Exposure to asbestos in the rubble and settled dust, especially among specific categories of responders, poses a risk of mesothelioma in the future, the magnitude of which is uncertain.

37 Pleural abnormalities and exposure to asbestos-contaminated vermiculite from Libby, Montana: A twenty year follow-up study

J.E. Lockey, A.M. Rohs, K. Dunning, T. Hilbert, R. Shipley, C. Meyer, J. Wiot, R. Shukla. *University of Cincinnati, Cincinnati, USA*

Background: Vermiculite mined in Libby, Montana was shown to be contaminated with asbestiform fibers, best described as tremolite-actinolite-richterite-winchite transition fiber. This study is a follow up investigation of a 1980 cross-sectional study of 513 workers exposed predominantly to Libby vermiculite ore. The rationale was to determine the long-term pulmonary effects of exposure to Libby vermiculite used in a secondary manufacturing setting.
Methods: Of the original 513 workers, 82 were deceased and eight could not be located. Of the remaining 423, 70.4% (298) participated in the study, 11.6% (49) refused, and 18.0% (76) did not respond. Of the 298 participants, 94% (280) completed both the radiograph and the interview for inclusion in this analysis. Primary outcomes were pleural and interstitial changes as determined from chest radiography. Pleural thickening was defined as unilateral or bilateral thickening, excluding solitary blunting of the costophrenic angle(s). Parenchymal changes were defined as irregular opacities with a profusion category >1/0. Chest radiographs were interpreted by three board certified radiologists (B readers). For a radiograph to be classified as abnormal, two of the three B readers must agree. Exposure to vermiculite ore was defined cumulatively (fiber/cc/years).
Results: Initial analysis of the first 194 participants indicated an increase in the number of workers with pleural changes. Pleural plaques were identified in 25% compared to 4% in the original 1980 cohort. Final results will be presented examining the association of pleura and interstitial findings with exposure indices.
Conclusions: Vermiculite contaminated with asbestiform fibers has the propensity to cause an increase in pleural changes at low exposure levels. The public health implications of these preliminary findings are important in view of the national distribution of the Libby vermiculite ore for home and commercial uses.
This abstract has not undergone external peer review as required by the funding agency.
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38 Mesothelioma update for Libby, Montana: Occupational and non-occupational

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Background: In 1986, a McGill University study of vermiculite miners/millers in Libby, Montana reported 4 mesothelioma cases in workers hired before 1965. In 2004 mortality was updated to 1999, adding 8 deaths. No study including workers after 1965 or any para-occupational or non-occupational mesothelioma in Libby has been published previously.
Methods: Case data for mesothelioma in Libby was obtained from 3 sources: a) the published McDonald (McGill) (1986, 2004) follow-up studies of W.R. Grace workers at Libby, Montana; b) information from attorneys representing mesothelioma victims and c) Dr. Brad Black on case numbers, occupational/non-occupational origin and basis of diagnosis at the CARD clinic at Libby, which began in 2000. There is no overlap between the latter and the published studies. Information obtained included years of work at the mine or mill (if any); pathology reports and death certificates.
Results: Four occupational cases were found additional to the 12 published who started work before 1965. One worked for only 6 weeks and 3 died later than 1999. Mesothelioma proportional mortality among workers is over 5%; a proportion seen previously only in crocidolite asbestos miners/millers and amosite-exposed insulation workers. Three more cases were diagnosed among those who began work after 1965, for a total of 19 to date. Ten non-occupational cases were identified; for 7 diagnosed after 2000, (6 confirmed pathologically, 1 clinically) there was also no history of living with a worker. Latency averaged 35 years from first exposure in workers. Latency in non-occupational cases, if equated with time of residence in Libby, exceeds this. Non-occupational cases incident earlier than 2000, while likely, have never been fully ascertained.
Conclusions: Mesothelioma in Libby workers and those without occupational exposure has been under-estimated. Caused by "Libby amphibole", it has ominous implications for known continuing amphibole exposure sources throughout the USA, including widely distributed waste products and 'naturally-occurring' amphiboles not commercially exploited.

Friday, October 20, 2006 10:30–12:20

ORAL SESSION

Epidemiology II

39 Australia: Wittenoom and beyond

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Background: The epidemic of mesothelioma in Australia was initially driven by the crocidolite mine and mill at Wittenoom in Western Australia where long-established asbestos hygiene principles and regulations were routinely ignored. Australia was also the largest per capita user of asbestos in the 1950s, resulting in the highest rate of mesothelioma in the world in the 1990s and beyond.
Methods: Rates of mesothelioma in different Australian states over time and overall consumption of asbestos were examined, and different sources of exposure to asbestos were described.
Results: Rates of mesothelioma in Australia appear to be leveling off while use of asbestos has declined. Importation of new asbestos into Australia was finally banned at the end of 2003, but the vast quantities of materials from previous use still remain. Australia's chief manufacturer of asbestos products during the last century tried to avoid its future liabilities by transferring its operations to the Netherlands, but was shamed into establishing a more realistic compensation fund. While the mesothelioma surveillance of the National Occupational Health and Safety Commission appears to have lost impetus, the National Health and Medical Research Council of Australia has recently set aside substantial funds for asbestos-related disease research.
Conclusions: Given that the highest future exposures to asbestos are likely to occur among workers in small demolition and removal operations, the dismantling of workers' protection regulations for small business, through Australia's "new" industrial relations legislation, is likely to result in future high rates of mesothelioma lasting well into this new century.

40 Age and sex differences in malignant mesothelioma after residential exposure to blue asbestos (crocidolite)

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Background: Blue asbestos (crocidolite) was mined and milled at Wittenoom,

EXHIBIT 33

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ORIGINAL ARTICLE

Occupational and non-occupational attributable risk of asbestos exposure for malignant pleural mesothelioma

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ABSTRACT

Objectives To estimate the proportion of pleural mesothelioma cases that can be attributed to asbestos exposure in France including non-occupational exposure.

Methods A population-based case-control study including 437 incident cases and 874 controls was conducted from 1998 to 2002. Occupational and non-occupational asbestos exposure was assessed retrospectively by two expert hygienists. ORs of pleural mesothelioma for asbestos-exposed subjects compared to non-exposed subjects, and population-attributable risk (ARp) of asbestos exposure were estimated using a conditional logistic regression.

Results A clear dose-response relationship was observed between occupational asbestos exposure and pleural mesothelioma (OR=4.0 (99% CI 1.9 to 8.3) for men exposed at less than 0.1 f/mL-year vs 67.0 (99% CI 25.6 to 175.1) for men exposed at more than 10 f/mL-year). The occupational asbestos ARp was 83.1% (99% CI 74.5% to 91.7%) for men and 41.7% (99% CI 25.3% to 58.0%) for women. A higher risk of pleural mesothelioma was observed in subjects non-occupationally exposed to asbestos compared to those never exposed. The non-occupational asbestos ARp for these subjects was 20.0% (99% CI −33.5% to 73.5%) in men and 38.7% (99% CI 8.4% to 69.0%) in women. When considering all kinds of asbestos exposure, ARp was 87.3% (99% CI 78.9% to 95.7%) for men and 64.8% (99% CI 45.4% to 84.3%) for women.

Conclusions Our study suggests that the overall ARp in women is largely driven by non-occupational asbestos exposure arguing for the strong impact of such exposure in pleural mesothelioma occurrence. Considering the difficulty in assessing domestic or environmental asbestos exposure, this could explain the observed difference in ARp between men and women.

Key messages

What is the key question?
- What is the overall impact of asbestos exposure including non-occupational exposure on pleural mesothelioma?

What is the bottom line?
- Occupational asbestos exposure explains less than half the pleural mesothelioma cases among women, thus, there is a need to quantify the impact of non-occupational asbestos exposure for pleural mesothelioma.

Why read on?
- This study provides insights regarding non-occupational exposure impact in pleural mesothelioma occurrence, especially among women.

INTRODUCTION

Malignant mesothelioma is a rare tumour mostly located in the pleura and associated with poor survival. To date, asbestos and erionite fibres are the only recognised risk factors for pleural mesothelioma.[1 2]

Asbestos had been extensively used in many industries all around the world, and was progressively banned in most industrialised countries, with such action taking place first in Europe in the 1980s and specifically in 1997 in France. However, asbestos is still produced and used in many countries, most of them being developing countries. This massive use of asbestos fibres has been correlated with an increase in the incidence of asbestos-related diseases and, in particular, pleural mesothelioma.[3]

The causal relationship between occupational asbestos exposure and pleural mesothelioma is already well established,[4–13] and it is estimated that approximatively 80% of male cases may be attributed to occupational asbestos exposure. Although the role of non-occupational asbestos exposure (para-occupational, domestic or environmental) in the occurrence of pleural mesothelioma has already been demonstrated,[14–16] there is a need to quantify the risk attributable to such exposure, especially in women in whom only 40% of cases can be explained by occupational exposure.[10] Knowledge about overall asbestos exposure (occupational and/or non-occupational exposure) attributable risk could provide further information regarding the aetiology of pleural mesothelioma and could be useful to help authorities for prevention in situations where asbestos-containing materials still persist in developed countries, and where asbestos is still in use in developing countries.

In France, the National Mesothelioma Surveillance Program (PNSM) was set up in 1998

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to provide relevant information on the health effects of asbestos exposure on the French population. It relies on the exhaustive recording of all incident primary pleural tumours in specified districts.[17]

The present study reports the occupational, non-occupational and overall asbestos exposure-attributable risk for pleural mesothelioma from a population-based case-control study carried out between 1998 and 2002.

MATERIALS AND METHODS

Study design

The study design was previously reported in detail.[18] Cases were identified through the PNSM from 1998 to 2002 and certified by a standardised diagnostic confirmation procedure.[17] Two controls, selected from the general population, were matched with cases for sex, age (±5 years) and district of residence. Participation rates of cases and controls were 61.1% and 20.8%, respectively.

Trained interviewers administered a standardised questionnaire to each subject. Only subjects alive at the time of the interview were included. Information about lifetime residential, educational and occupational history (including details on job tasks and do-it-yourself activities), demographic characteristics including the socioeconomic category defined by the last occupation held, previous respiratory diseases, occupations of partners and parents, and family cancer history were collected. A more specific part of the questionnaire focused on specific lifetime situations that might have involved asbestos exposure, for example, spraying fibres, asbestos cement disposal and removal, other insulation with asbestos-containing products (sheets, ropes, gaskets, etc), brake and clutch repairs, and washing asbestos-contaminated clothes. Additional questions concerned other suspected risk factors such as ionising radiations, and man-made mineral fibres (glass fibres and mineral wool, refractory ceramic fibres). Each job held for more than 6 months was coded according to international and national classifications for occupations and industries.

The PNSM obtained the appropriate institutional review board authorisations. Written informed consent for participation in the study was obtained from all participants.

Asbestos exposure assessment

Asbestos exposure was assessed by retrospective expertise.[8 18] Two experts (an industrial hygienist and an occupational physician) analysed each subject's questionnaire blinded to case-control status.[19]

For occupational exposure, each job held for at least 6 months by a subject was translated into four semiquantitative occupational asbestos exposure parameters, including the probability of exposure (possible, definite), frequency of exposure (sporadic, intermittent, frequent or continuous), intensity of exposure (low, medium, high, very high), and the route of exposure (direct, indirect). Table 1 describes these exposure parameters. A subject occupationally exposed to asbestos was defined as a subject who had held at least one job with a non-null probability of exposure. For each subject, the maximal probability of exposure, duration of exposure (years), age at first exposure (years), time since first exposure (years), year at first exposure and a cumulative exposure index (CEI, expressed in fiber/mL-years) were defined. The CEI was calculated by summing the products of probability, frequency, intensity and duration of exposure of each job held by a given subject. Since job occupational exposure parameters are semiquantitative, numerical values were assigned to each of them (table 1). These assigned values were the same as those defined in a previous mesothelioma French case-control study,[8] where authors concluded that these values had the best ability to show a clear dose-response relationship.

Table 1 Description of the exposure parameters used to assess asbestos exposure at job level

Asbestos exposure parameters	Definition	Numerical values*
Probability of exposure	Degree of confidence that exposure really occurred, expressed in percent (%)	
Not exposed	0	0
Possible	>0–50	0.5
Definite	>50–100	1
Frequency of exposure	Expressed in percent of work time (%)	
Sporadic	>0–5	0.025
Occasional	>5–30	0.25
Frequent or continuous	>30–100	0.75
Intensity of exposure	Expressed in f/mL	
Low	>0–0.1	0.1
Medium	>0.1–1	1
High	>1–10	10
Very high	>10	100

*Numerical values assigned to each category in order to calculate cumulative exposure index.

Lifetime non-occupational asbestos exposure was also assessed by expertise using information reported by subjects on the use of asbestos-containing materials or performed tasks. The probability, frequency and intensity of exposure, and the route of exposure (domestic, para-occupational and environmental) were assigned to each subject. The definitions of probability, frequency and intensity of exposure were the same as for occupational exposure, except for intensity where the 'low' category was split into 'very low' and 'low'. Domestic exposure was defined by do-it-yourself activities that might have involved asbestos-containing products, for example, home improvements, and brake and clutch repairs. Para-occupational exposure was considered with regard to asbestos-contaminated clothes of relatives, partners and parents who had jobs well known to be associated with asbestos exposure, or with a high risk for pleural mesothelioma. For environmental exposure, only self-reported living near an industrial source of asbestos was considered.[18]

Statistical analysis

Analyses were performed separately for men and women. ORs and 99% CIs were estimated for the occupational and non-occupational asbestos exposure parameters using conditional logistic regression models for matched sets. All continuous exposure variables were arbitrarily categorised: duration of exposure; 1–10/>10–20/>20 years; age at first exposure: ≤15/>15–20/>20 years; time since first exposure: >19–40/>40–50/>50 years; year at first exposure: >1960/>1960–1977/>1977; CEI: >0–0.1/>0.1–1/>1–10/>10 'f/mL-years'(table 4). All population-attributable risks (ARp) and 99% CIs were calculated using the Mantel–Haenszel estimation.[20] The non-occupational asbestos exposure ARp was estimated among matched sets where cases and controls were not occupationally exposed to asbestos. Considering all asbestos exposure, the overall ARp was estimated using OR comparing

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subjects occupationally and/or non-occupationally exposed to those never exposed at all. Given the long latency period of pleural mesothelioma, exposures occurring less than 20 years before diagnosis were not taken into account.[8] Although asbestos exposure was retrospectively assessed by two experts in the domain, this expertise was mainly based on subjects' self-reported tasks or asbestos-containing materials used. Thus, we conducted several sensitivity analyses by setting as not exposed: (1) subjects initially assessed as possibly exposed; (2) subjects initially assessed as exposed at a very low intensity; (3) subjects initially assessed as possibly exposed at a very low intensity; (4) subjects initially assessed as possibly exposed or exposed at a very low level intensity. We also performed additional analyses by setting the lag period at 0, 10 and 30 years.

RESULTS

The study sample comprised 437 cases (362 men, 75 women) and 874 controls (724 men, 150 women). Table 2 presents the main subjects' characteristics. Cases were ascertained in average near 12 months after they had been identified, but they were interviewed before the certification of the diagnosis and, on average, 3 months after they had been recorded. The socio-economic category differed between cases and controls: 59.4% of male cases were blue-collar workers versus 32.2% for controls. Among females, 41.1% of cases were clerical and related, workers versus 28.5% of controls.

Table 3 presents the 10 most frequent occupations and industries among males. There was a slight variation in the proportion of occupations held by male cases and controls. For some occupations, the occurrence of asbestos exposure within those occupations differed between cases and controls; that is, 90.7% of cases who ever held an occupation as sheet-metal workers were exposed to asbestos within this occupation versus 77.8% of controls.

The relationship between pleural mesothelioma and occupational asbestos exposure is reported in table 4. Among men, ORs for pleural mesothelioma were significantly increased for all exposure parameters, and a dose-response relationship was observed for all of them. OR for men exposed at less than 0.1 f/mL-years compared to those never exposed was 4.0 (99% CI 1.9 to 8.3) vs 67.0 (99% CI 25.6 to 175.1) in men exposed at more than 10 f/mL-years. Among women, a significant association between occupational asbestos exposure and pleural mesothelioma was also observed, OR for women ever exposed compared to those never exposed was 12.0 (99% CI 3.5 to 41.7). The estimated occupational asbestos exposure ARp was 83.1% (99% CI 74.5% to 91.7%) in men and 41.7% (95% CI 25.3% to 58.0%) in women.

Table 5 presents the association between non-occupational asbestos exposure and pleural mesothelioma among subjects never occupationally exposed, that is, 9 matched sets among males (9 cases and 18 controls) and 36 matched sets among females (36 cases and 72 controls). OR for men ever non-occupationally exposed to asbestos compared to those never exposed was 2.4 (99% CI 0.2 to 26.7) and increased to 4.3 (99% CI 1.2 to 15.1) for women. The non-occupational asbestos ARp for subjects never occupationally exposed was 20.0% (99% CI −33.5% to 73.5%) in men and 38.7% (99% CI 8.4% to 69.0%) in women.

The ARp related to overall asbestos exposure (occupational and non-occupational) is presented in table 6. The risk of pleural mesothelioma was strongly associated with asbestos exposure with a slight difference between men (OR=13.0 (99% CI 6.2 to 27.5)) and women (OR=8.0 (99% CI 2.9 to 21.8)).

Table 2 Main characteristics of subjects: 437 cases (362 men; 75 women) and 874 controls (724 men; 150 women), French case-control study, 1998–2002

	Men (1 086)				Women (225)			
	Cases (362)		Controls (724)		Cases (75)		Controls (150)	
	n	%	n	%	n	%	n	%
Age (years)								
Mean (SD)	67.6 (9.3)		66.8 (9.1)		66.6 (9.1)		65.8 (8.9)	
Range	41–93		41–89		41–92		41–89	
Education level (years)								
≤14	185	51.1	275	38.0	33	44.0	60	40.0
>14	177	48.9	449	62.0	42	56.0	90	60.0
Diagnostic-ascertainment period (months)								
Mean (SD)	12.7 (18.8)				12.1 (15.2)			
Range	0–97.1				0.2–65.9			
Diagnostic-interview period (months)								
Mean (SD)	3.4 (3.9)				3.4 (4.4)			
Range	0–43.7				0–36.3			
Last held occupation (ISCO 1968 major group)*								
0/1 Professional, technical and related workers	49	13.5	146	20.2	5	6.9	31	21.5
2 Administrative and managerial workers	15	4.1	68	9.4	3	4.1	1	0.7
3 Clerical and related workers	34	9.4	91	12.6	30	41.1	41	28.5
4 Sales workers	23	6.4	69	9.5	7	9.6	13	9.0
5 Service workers	16	4.4	48	6.6	16	21.9	34	23.6
6 Agricultural, animal husbandry and forestry workers; fishermen; hunters	10	2.8	69	9.5	3	4.1	4	2.8
7/8/9 Production and related workers, transport equipment operators and labourers	215	59.4	233	32.2	9	12.3	20	13.9

*Among women, two cases and six controls had never worked.

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Table 3 Occupational asbestos exposure: proportion of exposed men in the most represented occupations and industries, French case-control study, 1998–2002

	Cases (362)				Controls (724)			
	n	%	Exposed within job		n	%	Exposed within job	
			n	%			n	%
Occupations (ISCO Edition 1968)								
8-73 Sheet-metal workers	54	14.9	49	90.7	27	3.7	21	77.8
8-49 Machinery fitters, machine assemblers n.e.c.*	46	12.7	31	67.4	37	5.1	25	67.6
7-00 Production supervisors and general foremen	44	12.2	31	70.5	56	7.7	40	71.4
8-71 Plumbers and pipe fitters	39	10.8	36	92.3	14	1.9	12	85.7
8-41 Machinery fitters and machine assemblers	37	10.2	21	56.8	30	4.1	13	43.3
9-99 Labourers n.e.c.*	36	9.9	24	66.7	27	3.7	15	55.6
6-21 General farm workers	31	8.6	0	0.0	86	11.9	4	4.7
9-51 Bricklayers, stonemasons and tile setters	29	8.0	25	86.2	31	4.3	26	83.9
8-34 Machine-tool operators	26	7.2	14	53.8	29	4.0	7	24.1
9-54 Carpenters, joiners and parquetry workers	26	7.2	21	80.8	27	3.7	20	74.1
Industries (ISIC Revision 2)								
5000 Construction	129	35.6	109	84.5	140	19.3	105	75.0
3841 Ship building and repairing	65	18.0	58	89.2	20	2.8	19	95.0
3813 Manufacture of structural metal products	52	14.4	43	82.7	30	4.1	20	66.7
1110 Agriculture and livestock production	48	13.3	5	10.4	145	20.0	32	22.1
6200 Retail trade	33	9.1	8	24.2	94	13.0	19	20.2
6100 Wholesale trade	29	8.0	9	31.0	74	10.2	13	17.6
3819 Manufacture of fabricated metal products except machinery and equipment n.e.c.*	24	6.6	13	54.2	24	3.3	6	25.0
9100 Public administration	24	6.6	6	25.0	71	9.8	11	15.5
3845 Manufacture of aircraft	23	6.4	10	43.5	27	3.7	10	37.0
9310 Education services	23	6.4	9	39.1	82	11.3	19	23.2

*n.e.c.: not elsewhere classified.

The overall asbestos exposure ARp was 87.3% (99% CI 78.9% to 95.7%) in men and 64.8% (99% CI 45.4% to 84.3%) in women.

From the sensitivity analysis, in men, the overall ARp was 68.6% (99% CI 58.0 to 79.1), 85.9% (99% CI 77.2 to 94.7), 86.0% (99% CI 77.2 to 94.7) and 68.6% (99% CI 58.0 to 79.1) for analyses 1, 2, 3 and 4, respectively. In women, the overall ARp was 26.7% (99% CI 12.0 to 41.3), 47.4% (99% CI 30.0 to 65.0), 52.0% (99% CI 34.1 to 69.8) and 22.0% (99% CI 8.0 to 36.0) for analyses 1, 2, 3 and 4, respectively (see online supplementary material).

DISCUSSION

This study permits to quantify the role of some aspects of non-occupational asbestos exposure in the occurrence of pleural mesothelioma. When taking into account all kinds of asbestos exposure, 87.3% of male cases and 64.8% of female cases were attributable to asbestos.

In table 3, we observed an intravariability of asbestos exposure within each occupation and industry category. This is due to the exposure assessment method that incorporated experts' knowledge about that occupation or industry, as well as specific tasks performed, and circumstances of asbestos exposure reported by the subject himself.

The relationship between occupational asbestos exposure and occurrence of pleural mesothelioma is now well known. To date, several case-control studies have analysed this relationship, and all confirmed the causal link between occupational asbestos exposure and pleural mesothelioma,[4 5 7–13 21] but few have estimated the population-attributable risk of asbestos exposure for pleural mesothelioma.[4 5 10 13] Results from the present study are consistent with the literature. However, they may not be generalisable to countries, such as developing countries, countries with different pattern of industrialisation, or which used different type of asbestos fibres.

In France, almost all the asbestos used was imported, and chrysotile seems to have been the most common used type. Sources of asbestos exposure came from mainly construction industry, shipbuilding and repairing industry, manufacture of asbestos cement products, metal-working industry and motor vehicles manufacture or repairing.[8]

Our results support the role of non-occupational asbestos exposure in the aetiology of pleural mesothelioma and make it possible to quantify the weight of such exposure in the occurrence of pleural mesothelioma, especially among women. There is now stronger evidence that pleural mesothelioma may be induced by non-occupational exposure. Several population-based case-control studies have established an association between para-occupational exposure and the occurrence of pleural mesothelioma,[22–24] as well as for household exposure.[7 25] A meta-analysis of eight studies provided a summary relative risk of 8.1 (95% CI 5.3 to 12.0).[26] While there is evidence of an association between asbestos exposure from a geological source and the increased incidence of pleural mesothelioma,[27] recent studies have shown an increased risk of pleural mesothelioma associated with environmental exposure related to proximity with an asbestos plant,[28 29] as well as environmental exposure due to residence near asbestos mines.[11 30] Bourdes *et al* reported a summary relative risk for environmental exposure due either to asbestos mines or an asbestos

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Table 4 Occupational asbestos exposure and risk of pleural mesothelioma, 437 cases (362 men; 75 women) and 874 controls (724 men; 150 women), French case-control study, 1998–2002

		Men (1 086)							Women (225)					
		Cases (362)		Controls (724)					Cases (75)		Controls (150)			
Occupational asbestos exposure		n	%	n	%	OR	99% CI		n	%	n	%	OR	99% CI
Not exposed		28	7.7	327	45.2	1.0	–		41	54.7	139	92.7	1.0	–
Exposed		334	92.3	397	54.8	11.4	6.1 to 21.4		34	45.3	11	7.3	12.0	3.5 to 41.7
Highest probability of exposure	Possible	43	11.9	102	14.1	5.6	2.6 to 12.1	Possible	19	25.3	8	5.3	9.6	2.4 to 38.1
	Definite	291	80.4	295	40.7	13.2	7.0 to 25.0	Definite	15	20.0	3	2.0	18.2	2.9 to 112.9
Duration of exposure (years)	1–10	71	19.6	119	16.4	7.9	3.8 to 16.1	1–10	19	25.3	4	2.7	36.2	2.6 to 513.3
	>10–20	78	21.6	95	13.1	11.2	5.4 to 23.3	>10	15	20.0	7	4.6	6.6	1.6 to 27.8
	>20	185	51.1	183	25.3	13.9	7.2 to 27.0							
Age at first exposure (years)	≤15	77	21.3	63	8.7	15.8	7.4 to 33.8	≤20	14	18.7	2	1.3	38.2	2.4 to 601.3
	>15–20	122	33.7	115	15.9	14.6	7.3 to 29.4	>20	20	26.6	9	6.0	8.3	2.2 to 31.5
	>20	135	37.3	219	30.2	8.5	4.4 to 16.4							
Time since first exposure (years)	>19–40	81	22.4	119	16.4	8.3	4.0 to 17.2	>19–40	15	20.0	5	3.3	10.2	2.0 to 50.5
	>40–50	114	31.5	131	18.1	12.6	6.2 to 25.6	>40	19	25.3	6	4.0	14.2	2.7 to 75.0
	>50	139	38.4	147	20.3	13.4	6.7 to 26.8							
Year at first exposure	≤1960	256	70.7	275	38.0	13.8	7.2 to 26.7	≤1960	19	25.3	6	4.0	14.3	2.7 to 75.8
	>1960–1977	75	20.7	108	14.9	8.4	4.0 to 17.6	>1960–1977	13	17.3	4	2.7	10.6	2.1 to 55.9
	>1977	3	0.9	14	1.9	1.9	0.3 to 11.8	>1977	2	2.7	1	0.6	7.9	0.2 to 258.8
Cumulative exposure (« f/mL-years »)	>0–0.1	54	14.9	181	25.0	4.0	1.9 to 8.3	>0 to 0.1	13	17.3	6	4.0	6.9	1.5 to 30.9
	>0.1–1	68	18.8	121	16.7	8.3	3.8 to 17.7	>0.1	21	28.0	5	3.3	23.7	3.3 to 168.53
	>1–10	115	31.8	68	9.4	22.5	10.4 to 48.7							
	>10	97	26.8	27	3.7	67.0	25.6 to 175.1							
Attributable risk (99% CI) (%)						83.1 (74.5 to 91.7)							41.7 (25.3 to 58.0)	

Table 5 Non occupational asbestos exposure and risk of pleural mesothelioma among subjects non-exposed to occupational asbestos exposure, 45 cases (9 men; 36 women) and 90 controls (18 men; 72 women), French case-control study, 1998–2002

	Men						Women					
	Cases (9)		Controls (18)				Cases (36)		Controls (72)			
Non-occupational asbestos exposure	n	%	n	%	OR	99% CI	n	%	n	%	OR	99% CI
Not exposed	6	66.7	15	83.3	1.0	–	17	47.2	56	77.8	1.0	–
Exposed	3	33.3	3	16.7	2.4	0.2 to 26.7	19	52.8	16	22.2	4.3	1.2 to 15.1
Highest probability of exposure												
Possible	0	0.0	1	5.6	–	–	11	30.6	13	18.1	3.2	0.8 to 13.2
Definite	3	33.3	2	11.1	4.6	0.2 to 96.9	8	22.2	3	4.1	7.5	1.2 to 48.0
Attributable risk (99% CI) (%)					20.0 (−33.5 to 73.5)						38.7 (8.4 to 69.0)	

processing plant of 7.0 (95%CI 4.7 to 11.0). To date, there is only weak evidence of a possible effect of passive exposure in buildings containing asbestos on the occurrence of pleural mesothelioma.[15]

As in most case-control studies, the major limitation of this study is the retrospective assessment of exposure. While we are confident about the reliability of the occupational and para-occupational asbestos exposure assessment, this is not the case for domestic and environmental asbestos exposure. In the questionnaire, there were specific questions about domestic use of asbestos, or the proximity to an asbestos-processing plant. The reliability of such assessment is poor and is highly subject to recall bias.[31] Besides the proximity to an asbestos-processing plant, we did not consider other potential sources of environmental exposure, such as residence in buildings containing asbestos, or proximity to construction sites involving asbestos. Moreover, since we did not assess environmental exposure through lifetime residential distance from an asbestos-processing plant or other industrial sources but through self-report, we may have underestimated this kind of exposure, thereby leading to an underestimation of non-occupational and overall ARp. This hypothesis could explain the observed difference in ARp between men and women. Indeed, since 92% of male cases and only 45% of female cases were occupationally exposed, the ARp among women should be more affected by the bias induced by the underestimation of environmental exposure than that among men. A recent French study on the spatial distributions of male and female incidence of mesothelioma cases with and without any identified occupational or non-occupational asbestos exposure suggested the major influence of asbestos in female mesothelioma, likely through unknown environmental exposure.[32] However, we cannot exclude a possible overestimation of the non-occupational ARp and, thus, the overall ARp due to recall bias if we hypothesise that controls tended to under-report environmental or domestic exposure compared to cases. This effect might have been more extreme among women, since most cases were not occupationally exposed leading to a possible over-reporting of environmental or domestic exposure. This plausible hypothesis may also explain the observed difference between men and women for the non-occupational ARp. Indeed, from the sensitivity analyses conducted which aimed to assess the extent of this recall bias, the overall ARp varied from 68.6% to 87.3% among men, and from 22.0% to 64.8% among women. Only 11.6% of male cases were assessed as possibly exposed and 0.6% at a very low intensity versus 42.7% and 28.6% of female cases, respectively. Female cases were mainly considered as exposed at a very low intensity through domestic exposure, that is, use of baking trays, cooking gloves, toasters and ironing boards. Results from these sensitivity analyses demonstrate the difficulty of estimating precisely the overall asbestos ARp among women. Indeed, since the ARp among men is mostly driven by occupational exposure, and because the assessment of such exposure relies on the knowledge of experts in industrial hygiene, there is little impact

Table 6 All asbestos exposure and risk of pleural mesothelioma, 437 cases (362 men; 75 women) and 874 controls (724 men; 150 women), French case-control study, 1998–2002

	Men (1 086)						Women (225)					
	Cases (362)		Controls (724)				Cases (75)		Controls (150)			
All asbestos exposure	n	%	n	%	OR	99% CI	n	%	n	%	OR	99% CI
Not exposed	17	4.7	263	36.3	1.0	–	19	25.3	104	69.3	1.0	–
Exposed	345	95.3	461	63.7	13.0	6.2 to 27.5	56	74.7	46	30.7	8.0	2.9 to 21.8
Occupational only	264	76.5	317	68.7			26	46.4	9	19.6		
Non occupational only	11	3.2	64	13.9			22	39.3	35	76.1		
Occupational and non-occupational	70	20.3	80	17.4			8	14.3	2	4.3		
Highest probability of exposure												
Possible	42	11.6	104	14.4	6.8	2.8 to 16.2	32	42.7	38	25.3	5.8	2.0 to 17.0
Probable	303	83.7	357	49.3	14.8	7.0 to 31.6	24	32.0	8	5.4	14.7	4.0 to 54.2
Attributable risk (99% CI) (%)					87.3 (78.9 to 95.7)						64.8 (45.4 to 84.3)	

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of the sensitivity of the 'ever exposed' definition on the ARp estimation. However, since only 40% of female cases are attributable to occupational asbestos exposure, the overall ARp in women is driven by occupational and non-occupational asbestos exposure. Thus, among women, the 'ever exposed' definition, and especially, the 'non-occupationally ever exposed' definition, has an important impact on the estimation of ARp.

The PNSM relies on the exhaustive recording of all incident primary pleural tumours in representative districts of France.[17] Only incident cases who were alive at the time of the interview were included in these analyses. From a substudy on non-response bias, it appeared that the sociodemographic characteristics of cases who died before interview or refused to answer questions, did not differ from those of the cases included in this study.[18] Cases who had died before the interview seemed to be less occupationally exposed than those included (among males, 80.3% vs 92.3%, respectively; among women, 18.7% vs 45.3%, respectively). However, occupational asbestos exposure was derived from simplified questionnaires and mainly assessed through job titles. According to controls selection, it appeared that there were more blue-collar workers among refusing controls and more white-collar workers among participating controls, leading to a possible underestimation of occupational asbestos exposure among our control population.[33]

As in all previous published population-based mesothelioma case-control studies, the prevalence of blue collar workers was higher among cases than among controls. We did not adjust for this difference in socioeconomic status between cases and controls, since this is a variable that is linked to asbestos exposure but cannot be considered as potential causal factor of pleural mesothelioma.[34]

The definition of a lag period of 20 years had no impact on results since they were similar when we implemented a lag period strategy of 0, 10 and 30 years (see online supplementary material).

Since we have performed several comparisons, we have decided to present results with 99% CIs instead of the traditional 95% CIs. In order to estimate the 99% CIs for ARp, we used a method proposed by Greenland,[20] which is based on the Mantel–Haenszel estimation and is consistent for sparse data as found in individually matched studies. Since it exists several variance formulas in the literature, we also derived 99% CIs using the bootstrap method,[35] and results were very similar (see online supplementary material).

CONCLUSION

After quantification of the role of non-occupational asbestos exposure in the occurrence of pleural mesothelioma, approximately 35% of female cases were still not attributable to asbestos exposure. Since domestic and environmental exposures are hardly identifiable, the overall ARp could be underestimated owing to the underestimation of non-occupational asbestos exposure. Explanations, such as other factors involved in the aetiology of pleural mesothelioma, are also possible.

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Occupational and non-occupational attributable risk of asbestos exposure for malignant pleural mesothelioma

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COUNTY OF SPARTANBURG	)	
	)	
Beverly Dale Jolly and Brenda Rice Jolly,	)	Civil Action No. 2016-CP-42-01592
	)	
Plaintiffs,	)	**FISHER CONTROLS INTERNATIONAL LLC'S MOTION TO QUASH PLAINTIFFS' TRIAL SUBPOENA**
	)	
v.	)	
	)	
General Electric Company, et al.,	)	
	)	
Defendants.	)	
	)	
	)	

COMES NOW DEFENDANT, Fisher Controls International LLC ("Fisher"), and respectfully moves this court for an order quashing the trial subpoena received by its counsel. This Motion is made pursuant to Rule 45 of the South Carolina Civil Procedure and relevant case law. The Subpoena does not comply with Rule 45 in that Fisher is not a resident of South Carolina and was not properly served pursuant to the Rule. This Motion is to be further supported by a memorandum of law to be filed hereinafter. The undersigned counsel states that, pursuant to Rule 11, SCRCP, Fisher and its counsel has attempted to resolve the issue prior to the filing of this Motion.

Respectfully submitted,

LEATH, BOUCH & SEEKINGS, LLP

By: /s/ Yancey A. McLeod

Timothy W. Bouch
Yancey A. McLeod, III
Amy Melvin McLeod
92 Broad Street
Charleston, South Carolina 29401
(843) 937-8811

Counsel for Defendant Fisher Controls International LLC

ELECTRONICALLY FILED - 2017 Jul 20 3:43 PM - SPARTANBURG - COMMON PLEAS - CASE#2016CP4201592

ELECTRONICALLY FILED - 2017 Jul 20 9:08 PM - SPARTANBURG - COMMON PLEAS - CASE#2016CP4201592

STATE OF SOUTH CAROLINA	)	IN THE COURT OF COMMON PLEAS
	)	FOR THE SEVENTH JUDICIAL CIRCUIT
COUNTY OF SPARTANBURG	)	
	)	
Beverly Dale Jolly and Brenda Rice Jolly,	)	Civil Action No. 2016-CP-42-01592
	)	
Plaintiffs,	)	**FISHER CONTROLS INTERNATIONAL LLC'S MEMORANDUM IN SUPPORT OF ITS MOTION TO QUASH PLAINTIFFS' TRIAL SUBPOENA**
	)	
v.	)	
	)	
General Electric Company, et al.,	)	
	)	
Defendants.	)	
	)	
	)	

Fisher Controls International LLC ("Fisher"), by and through its undersigned counsel, files the instant Memorandum in Support of its Motion for Sanctions. The Subpoena does not comply with Rule 45 of the South Carolina Rules of Civil Procedure because Fisher is an out of state party and was not properly served. Fisher is not aware of any authority compelling it to appear at trial in the Plaintiffs' case.

ARGUMENT

I. FISHER WAS NOT SERVED IN COMPLIANCE WITH RULE 45 OF THE SOUTH CAROLINA RULES OF CIVIL PROCEDURE AND FISHER CANNOT BE COMPELLED TO ATTEND TRIAL IN SOUTH CAROLINA.

The Plaintiffs' subpoena is not directed to a specific individual, but is rather directed to Fisher. (See Fisher Subpoena, attached hereto as **Exhibit A**). Fisher is not aware of any authority that compels it to attend trial. Fisher is not a South Carolina resident and was not properly served pursuant to Rule 45, SCRCP. According to Rule 45, "Service of a subpoena upon a person named therein shall be made in the same manner prescribed for service of a summons and complaint in Rule 4(d) or (j)." Rule 45(b)(1), SCRCP. The subpoena issued to Fisher's counsel does not comply with the Rules' service requirements because Fisher's counsel is

not authorized to accept service on behalf of Fisher for Rule (d) or (j) pleadings. *See* Rule 4(d), (j). Plaintiffs' counsels' attempted service on Fisher's counsel is not surprising, however, given the requirement that "a subpoena may be served at any place within the State." See Rule 45(b)(2). Plaintiffs are aware that they cannot serve a South Carolina subpoena anywhere outside of this state.

Indeed, the requirement that Plaintiffs' must serve the subpoena within this State is clearly why no attempt was made to serve Fisher's 30(b)(6) deponent, Mr. Ron Duimstra. Mr. Duimstra does not reside in South Carolina and is therefore not within the subpoena power of the court. (*See* Affidavit of Phillip Reid, attached hereto as **Exhibit B**). Plaintiffs' cover letter attempts to cure the deficiency by claiming the subpoena is not an attempt to establish the unavailability of any witness in order to read deposition testimony into the record. (See Exhibit A). However, reading Mr. Duimstra's testimony into the record is the precisely what the South Carolina Rules of Civil Procedure provides. Rule 32 is clear:

> The deposition of a party or of anyone who at the time of taking the deposition was an officer, director, or managing agent, or a person designated under Rule 30(b)(6) or 31(a) to testify on behalf of a public or private corporation, partnership or association or governmental agency which is a party may be used by an adverse party for any purpose.

SCRCP 32(a)(2). Rule 32 is the vehicle that is available to the Plaintiffs in under the instant circumstances. Rule 32 provides for situations where, as here, the party to the case is unavailable to testify at trial:

> The deposition of a witness, **whether or not a party**, may be used by any party for any purpose if the court finds:
>
> (A) that the witness is dead; or
>
> (B) that the witness is at a greater distance than 100 miles from the place of trial or hearing, **or is out of the State**, unless it appears that the absence of the witness was procured by the party offering the deposition; or

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Rule 32(a)(3), SCRCP. In the instant case, the Plaintiffs' subpoena does not comply with the Rules. The Plaintiff chose to sue Fisher in South Carolina. Fisher is not a South Carolina corporation. Mr Duimstra, Fisher's 30(b)(6) witness, does not live in South Carolina. Under these circumstances, the Plaintiff is required to properly Notice and take Mr. Duimstra's deposition for either reading or playing at trial. The Rules simply do not provide Plaintiffs or this Court with the authority to command Fisher or Mr. Duimstra to attend trial. Fisher is unaware of any authority to the contrary. *See, e.g., In re C.R. Bard, Inc., Pelvic Repair Sys. Prod. Liab. Litig.*, No. 2:11-CV-00195, 2013 WL 3367715, at *2 (S.D.W. Va. July 5, 2013); *Amoco Chem. Co. v. Certain Underwriters at Lloyd's of London*, 34 Cal. App. 4th 554, 561, 40 Cal. Rptr. 2d 80, 85 (1995); *Fitzwater v. Speco Corp.*, No. 2916, 1992 WL 302444, at *3 (Ohio Ct. App. Oct. 26, 1992); *Hill v. Homeward Residential, Inc.*, 799 F.3d 544, 553 (6th Cir. 2015). To secure Fisher's attendance at trial, the Parties needed to come to an agreement. Fisher attempted in good faith to do so, and is left no choice but to file its Motion to Quash and the instant memorandum. The Plaintiffs' subpoena does not comply with the Rules and should be quashed.

Respectfully submitted,

LEATH, BOUCH & SEEKINGS, LLP

By: /s/ Yancey A. McLeod

Timothy W. Bouch
Yancey A. McLeod, III
Amy Melvin McLeod
92 Broad Street
Charleston, South Carolina 29401
(843) 937-8811

Counsel for Defendant Fisher Controls International LLC

ELECTRONICALLY FILED - 2017 Jul 20 9:08 PM - SPARTANBURG - COMMON PLEAS - CASE#2016CP4201592

EXHIBIT A

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DEAN | OMAR | BRANHAM

3900 Elm Street
Dallas, Texas 75226
p: 214.722.5990
f: 214.722.5991

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July 11, 2017

Timothy W. Bouch, Esq.
Yancey A. McLeod, III, Esq.
Amy M. McLeod, Esq.
Leath, Bouch & Seekings, LLP
92 Broad Street
Charleston, South Carolina 29401

RE: ***Beverly Dale Jolly and Brenda Rice Jolly v. GE Company et al.***
C.A. No.: 2016-CP-42-01592

Dear Counsel of Record for Defendant Fisher Controls International, LLC

Please find enclosed for the purposes of service on your client, an executed subpoena commanding Fisher Controls International, LLC to appear at trial in this matter for the purpose of giving testimony. Service of this subpoena is not a formality or prerequisite for establishing unavailability of any witness in order to read deposition testimony into the record. Rather, Plaintiffs serve this subpoena on Defendant in order to secure live testimony from Defendant at trial.

At the same time, Plaintiffs have no interest in making any corporate representative sit around for days in the courtroom waiting to testify at trial. Therefore, Plaintiffs are willing to work with Defendant to coordinate a specific day and time for Defendant's live testimony in Plaintiffs' case in chief.

Sincerely,

Jonathan M. Holder

JMH/bj
enclosures

STATE OF SOUTH CAROLINA

ISSUED BY THE COMMON PLEAS COURT IN THE COUNTY OF SPARTANBURG

BEVERLY DALE JOLLY, et al., Plaintiffs — SUBPOENA IN A CIVIL CASE

v. — Case Number: 2016-CP-42-01592

GE Company, et al., Defendants — Pending in Spartanburg County

TO: **DEFENDANT FISHER CONTROLS INTERNATIONAL, LLC; through Counsel of Record**

☒ YOU ARE COMMANDED to appear in the above-named court at the place, and time specified below to testify in the above case.

PLACE OF TESTIMONY	COURTROOM To be determined
Spartanburg County Courthouse; 180 Magnolia Street; Spartanburg, SC 29302	DATE AND TIME July 24, 2017, 9:00 AM and continuing from day to day until the completion of trial.

☐ YOU ARE COMMANDED to appear at the place, date, and time specified below to testify at the taking of a deposition in the above case.

PLACE OF DEPOSITION	DATE AND TIME , AM

☐ YOU ARE COMMANDED to produce and permit inspection and copying of the following documents or objects in your possession, custody or control at the place, date and time specified below (list documents of objects:

PLACE	DATE AND TIME , AM

☐ YOU ARE COMMANDED to permit inspection of the following premises at the date and time specified below.

PREMISES	DATE AND TIME , AM

ANY SUBPOENAED ORGANIZATION NOT A PARTY TO THIS IS HEREBY DIRECTED TO RULE 30(b)(6), SOUTH CAROLINA RULES OF CIVIL PROCEDURE, TO FILE A DESIGNATION WITH THE COURT SPECIFYING ONE OR MORE OFFICERS, DIRECTORS, OR MANAGING AGENTS, OR OTHER PERSONS WHO CONSENT TO TESTIFY ON ITS BEHALF, SHALL SET FORTH, FOR EACH PERSON DESIGNATED, THE MATTERS ON WHICH HE WILL TESTIFY OR PRODUCE DOCUMENTS OR THINGS. THE PERSON SO DESIGNATED TESTIFY AS TO MATTERS KNOWN OR REASONABLY AVAILABLE TO THE ORGANIZATION

I CERTIFY THAT THE SUBPOENA IS ISSUED IN COMPLIANCE WITH RULE 45(c)(1), AND THAT NOTICE AS REQUIRED BY RULE 45(b)(1) HAS BEEN GIVEN TO ALL PARTIES.

Attorney/Issuing Officer's Signature / Indicate if Attorney for Plaintiff or Defendant	July 11, 2017 Date	Jonathan M. Holder, SC Bar No. 77935 Print Name

Attorney's Address and Telephone Number:
Dean Omar Branham, LLP
302 N. Market Street, Ste. 300
Dallas, Texas 75202
214.722.5990

Clerk of Court/Issuing Officer's Signature	Date	Print Name

Pro Se Litigant's Name, Address and Telephone Number:

SCCA 254 (05/2015)

(See Rule 45, South Carolina Rules of Civil Procedure, Parts (c) & (d) on pages 2 and 3)

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PROOF OF SERVICE

SERVED	DATE	FEES AND MILEAGE TO BE TENDERED TO WITNESS UPON DAILY ARRIVAL
	PLACE	☐YES ☐NO AMOUNT $
SERVED ON Counsel of Record for Defendant		MANNER OF SERVICE
SERVED BY		TITLE

DECLARATION OF SERVER

I certify that the foregoing information contained in the Proof of Service is true and correct.

Executed on_____

SIGNATURE OF SERVER

ADDRESS OF SERVER

Rule 45, South Carolina Rules of Civil Procedures, Parts (c) and (d):

(c) Protection of Persons Subject to Subpoenas.

(1) A party or an attorney responsible for the issuance and service of a subpoena shall take reasonable steps to avoid imposing undue burden or expense on a person subject to that subpoena. The court on behalf of which the subpoena was issued shall enforce this duty and impose upon the party or attorney in breach of this duty an appropriate sanction, which may include, but is not limited to, lost earnings and a reasonable attorney's fee.

(2)(A) A person commanded to produce and permit inspection and copying of designated electronically stored information, books, papers, documents or tangible things, or inspection of premises need not appear in person at the place of production or inspection unless commanded to appear for deposition, hearing or trial. A party or an attorney responsible for the issuance and service of a subpoena for production of books, papers and documents without a deposition shall provide to another party copies of documents so produced upon written request. The party requesting copies shall pay the reasonable costs of reproduction.

(B) Subject to paragraph (d)(2) of this rule, a person commanded to produce and permit inspection and copying may, within 14 days after service of the subpoena or before the time specified for compliance if such time is less than 14 days after service, serve upon the party or attorney designated in the subpoena written objection to inspection or copying of any or all of the designated materials or of the premises—or to producing electronically stored information in the form or forms requested. If objection is made, the party serving the subpoena shall not be entitled to inspect and copy the materials or inspect the premises except pursuant to an order of the court by which the subpoena was issued. If objection has been made, the party serving the subpoena may, upon notice to the person commanded to produce, move at any time in the court that issued the subpoena for an order to compel the production. Such an order to compel production shall protect any person who is not a party or an officer of a party from significant expense resulting from the inspection and copying commanded.

(3)(A) On timely motion, the court by which a subpoena was issued, or regarding a subpoena commanding appearance at a deposition, or production or inspection directed to a non-party, the court in the county where the non-party resides, is employed or regularly transacts business in person, shall quash or modify the subpoena if it:

(i) fails to allow reasonable time for compliance; or

(ii) requires a person who is not a party nor an officer, director or managing agent of a party, nor a general partner of a partnership that is a party, to travel more than 50 miles from the county where that person resides, is employed or regularly transacts business in person, except that, subject to the provisions of clause (c)(3)(B)(iii) of this rule, such a person may in order to attend trial be commanded to travel from any such place within the state in which the trial is held; or

(iii) requires disclosure of privileged or otherwise protected matter and no exception or waiver applies; or

(iv) subjects a person to undue burden.

(B) If a subpoena:

(i) requires disclosure of a trade secret or other confidential research, development, or commercial information, or

(ii) requires disclosure of an unretained expert's opinion or information not describing specific events or occurrences in dispute and resulting from the expert's study made not at the request of any party, or

(See Rule 45, South Carolina Rules of Civil Procedure, Parts (c) & (d) on pages 2 and 3)

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(iii) requires a person who is not a party nor an officer, director or managing agent of a party, nor a general partner of a partnership that is a party, to incur substantial expense to travel from the county where that person resides, is employed or regularly transacts business in person, the court may, to protect a person subject to or affected by the subpoena, quash or modify the subpoena or, if the party in whose behalf the subpoena is issued shows a substantial need for the testimony or material that cannot be otherwise met without undue hardship and assures that the person to whom the subpoena is addressed will be reasonably compensated, the court may order appearance or production only upon specified conditions.

(d) Duties in Responding to Subpoena.

(1)(A)A person responding to a subpoena to produce documents shall produce them as they are kept in the usual course of business or shall organize and label them to correspond with the categories in the demand.

(B) If a subpoena does not specify the form or forms for producing electronically stored information, a person responding to a subpoena must produce the information in a form or forms in which it is ordinarily maintained or in a reasonably usable form or forms.

(C) A person responding to a subpoena need not produce the same electronically stored information in more than one form.

(D) A person responding to a subpoena need not provide discovery of electronically stored information from sources that the person identifies as not reasonably accessible because of undue burden or cost. On motion to compel discovery or to quash, the person from whom discovery is sought must show that the information sought is not reasonably accessible because of undue burden or cost. If that showing is made, the court may nonetheless order discovery from such sources if the requesting party shows good cause, considering the limitations of Rule 26(b)(6)(B). The court may specify conditions for the discovery.

(2)(A) When information subject to a subpoena is withheld on a claim that it is privileged or subject to protection as trial preparation materials, the claim shall be made expressly and shall be supported by a description of the nature of the documents, communications, or things not produced that is sufficient to enable the demanding party to contest the claim.

(B) If information produced in response to a subpoena is subject to a claim of privilege or of protection as trial preparation material, the person making the claim may notify any party that received the information of the claim and the basis for it. After being notified, a party must promptly return, sequester, or destroy the specified information and any copies it has and may not use or disclose the information until the claim is resolved. A receiving party may promptly present the information to the court under seal for a determination of the claim. If the receiving party disclosed the information before being notified, the receiving party must take reasonable steps to retrieve the information. The person who produced the information must preserve the information until the claim is resolved.

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EXHIBIT B

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STATE OF SOUTH CAROLINA COUNTY OF SPARTANBURG	)))	IN THE COURT OF COMMON PLEAS FOR THE SEVENTH JUDICIAL CIRCUIT
Beverly Dale Jolly and Brenda Rice Jolly, Plaintiffs, v. General Electric Company, et al., Defendants.	)))))))))))	Civil Action No. 2016-CP-42-01592 **AFFIDAVIT OF PHILIP C. REID** (Jury Trial Demanded)

Philip C. Reid swears the following is true:

1. I am an attorney licensed to practice law in Wisconsin and have been admitted in this case *pro hac vice*.

2. I have known Ron Duimstra, who will appear in this matter as the representative of Fisher Controls International LLC, for 10 years. Mr. Duimstra resides in Marshalltown, Iowa.

Philip C. Reid

Subscribed and sworn to before me
this 20th day of July, 2017

Kathleen E. Lamontagne
Notary Public, State of Wisconsin
My Commission Expires: 12-25-20

28269623_1 DOCX

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ELECTRONICALLY FILED - 2017 Jul 20 3:39 PM - SPARTANBURG - COMMON PLEAS - CASE#2016CP4201592

STATE OF SOUTH CAROLINA	)	IN THE COURT OF COMMON PLEAS
	)	FOR THE SEVENTH JUDICIAL CIRCUIT
COUNTY OF SPARTANBURG	)	
	)	
Beverly Dale Jolly and Brenda Rice Jolly,	)	Civil Action No. 2016-CP-42-01592
	)	
Plaintiffs,	)	**CROSBY VALVE, LLC'S**
	)	**MOTION TO QUASH**
v.	)	**PLAINTIFFS' TRIAL SUBPOENA**
	)	
General Electric Company, et al.,	)	
	)	
Defendants.	)	
	)	
	)	

COMES NOW DEFENDANT, Crosby Valve, LLC ("Crosby"), and respectfully moves this court for an order quashing the trial subpoena received by its counsel. This Motion is made pursuant to Rule 45 of the South Carolina Civil Procedure and relevant case law. The Subpoena does not comply with Rule 45 in that Crosby is not a resident of South Carolina and was not properly served pursuant to the Rule. This Motion is to be further supported by a memorandum of law to be filed hereinafter. The undersigned counsel states that, pursuant to Rule 11, SCRCP, Crosby and its counsel has unsuccessfully attempted to resolve the issue prior to the filing of this Motion.

Respectfully submitted,

LEATH, BOUCH & SEEKINGS, LLP

By: /s/ Yancey A. McLeod

Timothy W. Bouch
Yancey A. McLeod, III
Amy Melvin McLeod
92 Broad Street
Charleston, South Carolina 29401
(843) 937-8811

Counsel for Defendant Crosby Valve, LLC

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STATE OF SOUTH CAROLINA	)	IN THE COURT OF COMMON PLEAS
	)	FOR THE SEVENTH JUDICIAL CIRCUIT
COUNTY OF SPARTANBURG	)	
	)	
Beverly Dale Jolly and Brenda Rice Jolly,	)	Civil Action No. 2016-CP-42-01592
	)	
Plaintiffs,	)	**CROSBY VALVE, LLC'S MEMORANDUM IN SUPPORT OF ITS MOTION TO QUASH PLAINTIFFS' TRIAL SUBPOENA**
	)	
v.	)	
	)	
General Electric Company, et al.,	)	
	)	
Defendants.	)	
	)	
	)	

Crosby Valve, LLC'S ("Crosby"), by and through its undersigned counsel, files the instant Memorandum in Support of its Motion for Sanctions. The Subpoena does not comply with Rule 45 of the South Carolina Rules of Civil Procedure because Crosby is an out of state party and was not properly served. Crosby is not aware of any authority compelling it to appear at trial in the Plaintiffs' case.

ARGUMENT

I. CROSBY WAS NOT SERVED IN COMPLIANCE WITH RULE 45 OF THE SOUTH CAROLINA RULES OF CIVIL PROCEDURE AND CROSBY CANNOT BE COMPELLED TO ATTEND TRIAL IN SOUTH CAROLINA.

The Plaintiffs' subpoena is not directed to a specific individual, but is rather directed to Crosby. (See Crosby Subpoena, attached hereto as **Exhibit A**). Crosby is not aware of any authority that compels it to attend trial. Crosby is not a South Carolina resident and was not properly served pursuant to Rule 45, SCRCP. According to Rule 45, "Service of a subpoena upon a person named therein shall be made in the same manner prescribed for service of a summons and complaint in Rule 4(d) or (j)." Rule 45(b)(1), SCRCP. The subpoena issued to Crosby's counsel does not comply with the Rules' service requirements because Crosby's counsel

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is not authorized to accept service on behalf of Crosby for Rule (d) or (j) pleadings. *See* Rule 4(d), (j). Plaintiffs' counsels' attempted service on Crosby's counsel is not surprising, however, given the requirement that "a subpoena may be served at any place within the State." See Rule 45(b)(2). Plaintiffs are aware that they cannot serve a South Carolina subpoena anywhere outside of this state.

Indeed, the requirement that Plaintiffs' must serve the subpoena within this State is clearly why no attempt was made to serve Crosby's 30(b)(6) deponent, Mr. Robert Martin. Mr. Martin does not reside in South Carolina and is therefore not within the subpoena power of the court. (*See* Affidavit of Robert Martin, attached hereto as **Exhibit B**). Plaintiffs' cover letter attempts to cure the deficiency by claiming the subpoena is not an attempt to establish the unavailability of any witness in order to read deposition testimony into the record. (See Exhibit A). However, reading Mr. Martin's testimony into the record is the precisely what the South Carolina Rules of Civil Procedure provides. Rule 32 is clear:

> The deposition of a party or of anyone who at the time of taking the deposition was an officer, director, or managing agent, or a person designated under Rule 30(b)(6) or 31(a) to testify on behalf of a public or private corporation, partnership or association or governmental agency which is a party may be used by an adverse party for any purpose.

SCRCP 32(a)(2). Rule 32 is the vehicle that is available to the Plaintiffs in under the instant circumstances. Rule 32 provides for situations where, as here, the party to the case is unavailable to testify at trial:

> The deposition of a witness, **whether or not a party**, may be used by any party for any purpose if the court finds:
>
> (A) that the witness is dead; or
>
> (B) that the witness is at a greater distance than 100 miles from the place of trial or hearing, **or is out of the State**, unless it appears that the absence of the witness was procured by the party offering the deposition; or

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. . .

Rule 32(a)(3), SCRCP. In the instant case, the Plaintiffs' subpoena does not comply with the Rules. The Plaintiff chose to sue Crosby in South Carolina. Crosby is not a South Carolina corporation. Mr Martin, Crosby's 30(b)(6) witness, does not live in South Carolina. The Plaintiffs chose not to pursue Mr. Martin's deposition in this case. Under these circumstances, the Plaintiff is required to properly Notice and take Mr. Martin's deposition for either reading or playing at trial. The Rules simply do not provide Plaintiffs or this Court with the authority to command Crosby or Mr. Martin to attend trial. Crosby is unaware of any authority to the contrary. *See, e.g., In re C.R. Bard, Inc., Pelvic Repair Sys. Prod. Liab. Litig.*, No. 2:11-CV-00195, 2013 WL 3367715, at *2 (S.D.W. Va. July 5, 2013); *Amoco Chem. Co. v. Certain Underwriters at Lloyd's of London*, 34 Cal. App. 4th 554, 561, 40 Cal. Rptr. 2d 80, 85 (1995); *Fitzwater v. Speco Corp.*, No. 2916, 1992 WL 302444, at *3 (Ohio Ct. App. Oct. 26, 1992); *Hill v. Homeward Residential, Inc.*, 799 F.3d 544, 553 (6th Cir. 2015).

In the instant case, the Plaintiffs did not Notice or take any Crosby 30(b)(6) deposition. There is simply no authority that Crosby is aware to compel it or its out of state employees to attend trial in South Carolina. The Plaintiffs' subpoena does not comply with the Rules and should be quashed.

Respectfully submitted,

LEATH, BOUCH & SEEKINGS, LLP

By: /s/ Yancey A. McLeod

Timothy W. Bouch
Yancey A. McLeod, III
Amy Melvin McLeod
92 Broad Street
Charleston, South Carolina 29401
(843) 937-8811

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Counsel for Defendant Crosby Controls International LLC

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EXHIBIT A

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DEAN | OMAR | BRANHAM

3900 Elm Street
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July 11, 2017

Timothy W. Bouch, Esq.
Yancey A. McLeod, III, Esq.
Amy M. McLeod, Esq.
Leath, Bouch & Seekings, LLP
92 Broad Street
Charleston, South Carolina 29401

RE: ***Beverly Dale Jolly and Brenda Rice Jolly v. GE Company et al.***
C.A. No.: 2016-CP-42-01592

Dear Counsel of Record for Defendant Crosby Valves, LLC

Please find enclosed for the purposes of service on your client, an executed subpoena commanding Crosby Valves, LLC to appear at trial in this matter for the purpose of giving testimony. Service of this subpoena is not a formality or prerequisite for establishing unavailability of any witness in order to read deposition testimony into the record. Rather, Plaintiffs serve this subpoena on Defendant in order to secure live testimony from Defendant at trial.

At the same time, Plaintiffs have no interest in making any corporate representative sit around for days in the courtroom waiting to testify at trial. Therefore, Plaintiffs are willing to work with Defendant to coordinate a specific day and time for Defendant's live testimony in Plaintiffs' case in chief.

Sincerely,

Jonathan M. Holder

JMH/bj
enclosures

STATE OF SOUTH CAROLINA

ISSUED BY THE COMMON PLEAS COURT IN THE COUNTY OF SPARTANBURG

BEVERLY DALE JOLLY, et al., Plaintiffs

v.

GE Company, et al., Defendants

SUBPOENA IN A CIVIL CASE

Case Number: 2016-CP-42-01592

Pending in Spartanburg County

TO: **DEFENDANT CROSBY VALVES, LLC; through Counsel of Record**

☒ YOU ARE COMMANDED to appear in the above-named court at the place, and time specified below to testify in the above case.

PLACE OF TESTIMONY	COURTROOM To be determined
Spartanburg County Courthouse; 180 Magnolia Street; Spartanburg, SC 29302	DATE AND TIME July 24, 2017, 9:00 AM and continuing from day to day until the completion of trial.

☐ YOU ARE COMMANDED to appear at the place, date, and time specified below to testify at the taking of a deposition in the above case.

PLACE OF DEPOSITION	DATE AND TIME , AM

☐ YOU ARE COMMANDED to produce and permit inspection and copying of the following documents or objects in your possession, custody or control at the place, date and time specified below (list documents of objects:

PLACE	DATE AND TIME , AM

☐ YOU ARE COMMANDED to permit inspection of the following premises at the date and time specified below.

PREMISES	DATE AND TIME , AM

ANY SUBPOENAED ORGANIZATION NOT A PARTY TO THIS IS HEREBY DIRECTED TO RULE 30(b)(6), SOUTH CAROLINA RULES OF CIVIL PROCEDURE, TO FILE A DESIGNATION WITH THE COURT SPECIFYING ONE OR MORE OFFICERS, DIRECTORS, OR MANAGING AGENTS, OR OTHER PERSONS WHO CONSENT TO TESTIFY ON ITS BEHALF, SHALL SET FORTH, FOR EACH PERSON DESIGNATED, THE MATTERS ON WHICH HE WILL TESTIFY OR PRODUCE DOCUMENTS OR THINGS. THE PERSON SO DESIGNATED TESTIFY AS TO MATTERS KNOWN OR REASONABLY AVAILABLE TO THE ORGANIZATION

I CERTIFY THAT THE SUBPOENA IS ISSUED IN COMPLIANCE WITH RULE 45(c)(1), AND THAT NOTICE AS REQUIRED BY RULE 45(b)(1) HAS BEEN GIVEN TO ALL PARTIES.

	July 11, 2017	Jonathan M. Holder, SC Bar No. 77935
Attorney/Issuing Officer's Signature	Date	Print Name

Indicate if Attorney for Plaintiff or Defendant
Attorney's Address and Telephone Number :
Dean Omar Branham, LLP
302 N. Market Street, Ste. 300
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Clerk of Court/Issuing Officer's Signature	Date	Print Name

Pro Se Litigant's Name, Address and Telephone Number :

SCCA 254 (05/2015)

(See Rule 45, South Carolina Rules of Civil Procedure, Parts (c) & (d) on pages 2 and 3)

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PROOF OF SERVICE

SERVED	DATE	FEES AND MILEAGE TO BE TENDERED TO WITNESS UPON DAILY ARRIVAL
	PLACE	☐YES ☐NO AMOUNT $

SERVED ON Counsel of Record for Defendant	MANNER OF SERVICE
SERVED BY	TITLE

DECLARATION OF SERVER

I certify that the foregoing information contained in the Proof of Service is true and correct.

Executed on_____

SIGNATURE OF SERVER

ADDRESS OF SERVER

Rule 45, South Carolina Rules of Civil Procedures, Parts (c) and (d):

(c) Protection of Persons Subject to Subpoenas.

(1) A party or an attorney responsible for the issuance and service of a subpoena shall take reasonable steps to avoid imposing undue burden or expense on a person subject to that subpoena. The court on behalf of which the subpoena was issued shall enforce this duty and impose upon the party or attorney in breach of this duty an appropriate sanction, which may include, but is not limited to, lost earnings and a reasonable attorney's fee.

(2)(A) A person commanded to produce and permit inspection and copying of designated electronically stored information, books, papers, documents or tangible things, or inspection of premises need not appear in person at the place of production or inspection unless commanded to appear for deposition, hearing or trial. A party or an attorney responsible for the issuance and service of a subpoena for production of books, papers and documents without a deposition shall provide to another party copies of documents so produced upon written request. The party requesting copies shall pay the reasonable costs of reproduction.

(B) Subject to paragraph (d)(2) of this rule, a person commanded to produce and permit inspection and copying may, within 14 days after service of the subpoena or before the time specified for compliance if such time is less than 14 days after service, serve upon the party or attorney designated in the subpoena written objection to inspection or copying of any or all of the designated materials or of the premises—or to producing electronically stored information in the form or forms requested. If objection is made, the party serving the subpoena shall not be entitled to inspect and copy the materials or inspect the premises except pursuant to an order of the court by which the subpoena was issued. If objection has been made, the party serving the subpoena may, upon notice to the person commanded to produce, move at any time in the court that issued the subpoena for an order to compel the production. Such an order to compel production shall protect any person who is not a party or an officer of a party from significant expense resulting from the inspection and copying commanded.

(3)(A) On timely motion, the court by which a subpoena was issued, or regarding a subpoena commanding appearance at a deposition, or production or inspection directed to a non-party, the court in the county where the non-party resides, is employed or regularly transacts business in person, shall quash or modify the subpoena if it:

(i) fails to allow reasonable time for compliance; or

(ii) requires a person who is not a party nor an officer, director or managing agent of a party, nor a general partner of a partnership that is a party, to travel more than 50 miles from the county where that person resides, is employed or regularly transacts business in person, except that, subject to the provisions of clause (c)(3)(B)(iii) of this rule, such a person may in order to attend trial be commanded to travel from any such place within the state in which the trial is held; or

(iii) requires disclosure of privileged or otherwise protected matter and no exception or waiver applies; or

(iv) subjects a person to undue burden.

(B) If a subpoena:

(i) requires disclosure of a trade secret or other confidential research, development, or commercial information, or

(ii) requires disclosure of an unretained expert's opinion or information not describing specific events or occurrences in dispute and resulting from the expert's study made not at the request of any party, or

(See Rule 45, South Carolina Rules of Civil Procedure, Parts (c) & (d) on pages 2 and 3)

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(iii) requires a person who is not a party nor an officer, director or managing agent of a party, nor a general partner of a partnership that is a party, to incur substantial expense to travel from the county where that person resides, is employed or regularly transacts business in person, the court may, to protect a person subject to or affected by the subpoena, quash or modify the subpoena or, if the party in whose behalf the subpoena is issued shows a substantial need for the testimony or material that cannot be otherwise met without undue hardship and assures that the person to whom the subpoena is addressed will be reasonably compensated, the court may order appearance or production only upon specified conditions.

(d) Duties in Responding to Subpoena.

(1)(A)A person responding to a subpoena to produce documents shall produce them as they are kept in the usual course of business or shall organize and label them to correspond with the categories in the demand.

(B) If a subpoena does not specify the form or forms for producing electronically stored information, a person responding to a subpoena must produce the information in a form or forms in which it is ordinarily maintained or in a reasonably usable form or forms.

(C) A person responding to a subpoena need not produce the same electronically stored information in more than one form.

(D) A person responding to a subpoena need not provide discovery of electronically stored information from sources that the person identifies as not reasonably accessible because of undue burden or cost. On motion to compel discovery or to quash, the person from whom discovery is sought must show that the information sought is not reasonably accessible because of undue burden or cost. If that showing is made, the court may nonetheless order discovery from such sources if the requesting party shows good cause, considering the limitations of Rule 26(b)(6)(B). The court may specify conditions for the discovery.

(2)(A) When information subject to a subpoena is withheld on a claim that it is privileged or subject to protection as trial preparation materials, the claim shall be made expressly and shall be supported by a description of the nature of the documents, communications, or things not produced that is sufficient to enable the demanding party to contest the claim.

(B) If information produced in response to a subpoena is subject to a claim of privilege or of protection as trial preparation material, the person making the claim may notify any party that received the information of the claim and the basis for it. After being notified, a party must promptly return, sequester, or destroy the specified information and any copies it has and may not use or disclose the information until the claim is resolved. A receiving party may promptly present the information to the court under seal for a determination of the claim. If the receiving party disclosed the information before being notified, the receiving party must take reasonable steps to retrieve the information. The person who produced the information must preserve the information until the claim is resolved.

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EXHIBIT B

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STATE OF SOUTH CAROLINA	)	IN THE COURT OF COMMON PLEAS
	)	FOR THE SEVENTH JUDICIAL CIRCUIT
COUNTY OF SPARTANBURG	)	
	)	
Beverly Dale Jolly and Brenda Rice Jolly,	)	Civil Action No. 2016-CP-42-01592
	)	
Plaintiffs,	)	
	)	(Jury Trial Demanded)
v.	)	
	)	
General Electric Company, et al.,	)	
	)	
Defendants.	)	
	)	
	)	

AFFIDAVIT OF ROBERT J. MARTIN

STATE OF NORTH CAROLINA)
) ss
COUNTY OF BRUNSWICK)

Robert J. Martin swears the following is true to the best of my knowledge and belief:

1. I am a current resident of Southport, North Carolina.

Robert J. Martin

Robert J. Martin

Subscribed and sworn to before me
this 26 day of July, 2017

Suzanne M Contente
Notary Public, State of North Carolina
My commission expires June 5 2021

SUZANNE M. CONTENTE
My Commission Expires
NOTARY PUBLIC
June 5, 2021
BRUNSWICK COUNTY, NC

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STATE OF SOUTH CAROLINA COUNTY OF SPARTANBURG **BEVERLY DALE JOLLY and BRENDA RICE JOLLY,** Plaintiffs, v. **CROSBY VALVE, LLC,** et al. Defendants.	)))))))))))))	IN THE COURT OF COMMON PLEAS FOR THE SEVENTH JUDICIAL CIRCUIT **C/A No. 2016-CP-42-1592**

PLAINTIFFS' RESPONSE IN OPPOSITION TO DEFENDANT FISHER CONTROLS INTERNATIONAL, LLC AND CROSBY VALVE, LLC'S MOTIONS TO QUASH PLAINTIFFS' TRIAL SUBPOENAS

Plaintiffs Beverly Dale Jolly and Brenda Rice Jolly oppose Defendants Fisher Controls International LLC ("Fisher") and Crosby Valve, LLC's ("Crosby" or collectively "Defendants") motions to quash Plaintiffs' trial subpoenas.

BACKGROUND

Plaintiffs served Defendant Fisher and Defendant Crosby with trial subpoenas on July 12, 2017 for trial beginning July 24, 2017. *See* **Exhibit-A**, Plaintiffs Trial Subpoenas. The trial subpoenas were served via hand delivery by Fed-Ex to Leath Bouch and Seekings, 92 Broad St., Charleston, SC 29401, and were signed for by *McLeod* (misspelled McCloud by carrier). *See* **Exhibit-B**, proof of service with McLeod signature.

Over a week has passed and pre-trial motions hearing took place July 18, 2017, at which the issue of trial subpoenas was never raised by Defendants. On July 20, 2017, Defendants filed motions to quash the trial subpoenas of both Defendant Fisher and Defendant Crosby. Plaintiffs respond in opposition as detailed herein.

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ARGUMENTS AND AUTHORITIES

Defendants claim Plaintiffs' trial subpoenas are not valid "in that Fisher (and Crosby) is not a resident of South Carolina and was not served pursuant to the rule." Defs' Mot. to Quash at 1. Defendants' argument fails to recognize that Defendants Fisher and Crosby are parties to this action who have submitted to the jurisdiction of this Court. They are not merely non-party witnesses.

Rule 45, SCRCP, only allows *non-parties* to quash a trial subpoena. The rule states in pertinent part, as follows:

> (3)(A) On timely motion, the court by which a subpoena was issued, or regarding a subpoena commanding appearance at a deposition, or production or inspection directed to a non-party, the court in the county where the non-party resides, is employed or regularly transacts business in person, shall quash or modify the subpoena if it:
>
> (i) fails to allow reasonable time for compliance; or
>
> (ii) requires *a person who is not a party nor an officer, director or managing agent of a party, nor a general partner of a partnership that is a party*, to travel more than 50 miles from the county where that person resides, is employed or regularly transacts business in person, except that, subject to the provisions of clause (c)(3)(B)(iii) of this rule, such a person may in order to attend trial be commanded to travel from any such place within the state in which the trial is held; or
>
> (iii) requires disclosure of privileged or otherwise protected matter and no exception or waiver applies; or
>
> (iv) subjects a person to undue burden.

Rule 45(c)(3)(A), SCRCP (emphasis added). Rule 45 thus provides that only a non-party may quash a subpoena for geographic or travel reasons, and even then a non-party may be subpoenaed to attend trial from anywhere in the state.

The rule does provide a mechanism for quashing a trial subpoena, but again only for non-parties:

> (B) If a subpoena . . .

> (iii) requires *a person who is not a party nor an officer, director or managing agent of a party, nor a general partner of a partnership that is a party, to incur substantial expense to travel from the county where that person resides, is employed or regularly transacts business in person, the court may, to protect a person subject to or affected by the subpoena, quash or modify the subpoena* or, if the party in whose behalf the subpoena is issued shows a substantial need for the testimony or material that cannot be otherwise met without undue hardship and assures that the person to whom the subpoena is addressed will be reasonably compensated, the court may order appearance or production only upon specified conditions.

Rule 45(c)(3)(B), SCRCP (emphasis added). There is simply no provision for a party to quash a trial subpoena because the party resides outside the county or even the state.

In contending that they cannot be compelled to attend trial because they are corporations, Fisher and Crosby ignore the key fact that they are parties to this case. Their status as nonresident corporations does not change the fact that they have submitted to the jurisdiction of this Court by making a general appearance and litigating this case to trial. A general appearance constitutes a voluntary submission to the jurisdiction of the court. *See, e.g., Stickland v. Consol. Energy Prod. Co.*, 274 S.C. 554, 555, 265 S.E.2d 682, 683 (1980); *Brays Island Plantation, Inc. v. Harper*, 245 S.C. 399, 405, 140 S.E.2d 781, 784 (1965).

This issue was addressed earlier this year in Louisiana state court, with the same arguments being made by nonresident corporations who claimed that they could not be subpoenaed to trial. The Louisiana Court of Appeal held that because the nonresident corporations were parties, the court had subpoena power and could compel their attendance at trial. *See Hayden v. 3M Co.*, 211 So. 3d 528, 532 (La. App. 2017), *writ denied, stay denied,* 216 So. 3d 799 (La. 3/3/17), a courtesy copy of which is attached here as **Exhibit-C**. The Louisiana court explained that "[i]n the same way that Louisiana exercises personal jurisdiction over parties participating in litigation in the state, those parties may, upon the discretion of the court, be compelled to appear in Louisiana for discovery depositions, hearings, and/or trial." *Id.*

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The Court should also reject Defendants' arguments that the only mechanism to secure trial testimony from Fisher and Crosby's corporate representatives is through a Rule 30(b)(6) deposition. There is no authority for the proposition that Plaintiffs cannot obtain live testimony from a *party* to this case. Rule 32 references the use of depositions of "witnesses" that reside more than 100 miles from the courthouse, not parties. Plaintiffs have subpoenaed Defendants' corporate representatives to testify at trial in large part because presenting their testimony through depositions taken in other cases is less than ideal. These depositions are piecemeal, from other cases in other jurisdictions, and generally must be read in from paper transcripts. It is only fair that Plaintiffs be allowed to question Defendants, through their representatives, through live testimony at trial. In-person testimony will certainly aid the jury's understanding of the relatively complex issues presented in this case regarding Defendants' wrongful conduct in manufacturing and marketing asbestos products without a warning and their knowledge of the danger.

Finally, there is also no merit to Fisher and Crosby's contention that Plaintiffs' method of service was deficient. The Editor's Notes to the 2002 Amendments to Rule 45 confirm that a subpoena may be served on a corporation and that such service may be made on the corporation's attorney:

> The first 2002 amendment amends Rule 45(b)(1) to permit service of subpoenas by the same method as used to serve a summons and complaint.
>
> First, in addition to in hand service of the subpoena, service on an individual could be made by leaving the subpoena at the person's home or usual place of abode with a person of suitable age and discretion then residing there as provided in Rule 4(d)(1). Second, *a subpoena could be served on* an individual, *a corporation*, or a partnership by registered or certified mail, return receipt requested and delivery restricted to the addressee under Rule 4(d)(8). *In addition, the person or the person's attorney may accept service under Rule 4(j).*

Notes to 2002 Amendments, Rule 45, SCRCP (emphasis added).

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Further, subsection (j) of Rule 4 defines acceptance of service and provides that "[n]o other proof of service shall be required *when acceptance of service is acknowledged in writing and signed by the person served or his attorney*, and delivered to the person making service. The acknowledgement shall state the place and date service is accepted." Rule 4(j), SCRCP (emphasis added).

In addition, Rule 5(b), SCRCP applies to all service requirements listed in any South Carolina Rule of Civil Procedure. Subsection (b)(1) of Rule 5 provides that unless the Court orders that service on the party must be made personally, then whenever "service is required to be made upon a party represented by an attorney the service shall be made upon the attorney." *Id.* Rule 5 even defines delivery as meaning "handing it to the attorney or the party or leaving it at his office with his clerk or other person in charge thereof." *Id.*

Here, the Court never ordered that either Defendant Fisher or Defendant Crosby were to be served personally in circumvention of Rule 5(b)(1), therefore Defendant Fisher and Defendant Crosby "shall" be served by their attorney of record in the pending case pursuant to Rule 5(b)(1). Secondly, Plaintiffs have provided the Court with Defendants' acknowledgment of acceptance of service in writing and signed by the person served or his attorney. *See* **Exhibit-B**, signed by "McLeod" at the Leath Bouch & Seekings law firm July 12, 2017. Finally, Plaintiffs served timely valid subpoenas for attendance at trial, issued pursuant to Rule 45, served on Defendants pursuant to rule Rule 5(b)(1) and acknowledged by Defendant through signature of acceptance of service pursuant to Rule 4(j). Rules 4, 5 and 45, SCRCP.

CONCLUSION

Plaintiffs are informed and believe they are entitled to an Order denying Defendants' motions to quash Plaintiffs' trial subpoenas and for an order compelling Defendant Fisher and

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Defendant Crosby to comply with Plaintiffs' trial subpoenas and appear at trial to give live testimony in Plaintiffs' case-in-chief. Additionally, Plaintiffs request an award of costs and attorney's fees attendant to this motion as provided by Rule 37, SCRCP.

Respectfully submitted,

s/Jonathan M. Holder
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July 20, 2017
Dallas, Texas.

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FedEx ® Tracking

779603947446

Ship date:
Tue 7/11/2017

Dean Omar Branham
Megan Williams
302 N. Market St. Suite 300
DALLAS, TX US 75202
214 722-5990

Delivered
Signed for by: M.MCLOUD

Actual delivery:
Wed 7/12/2017 8:18 am

Leath Bouch & Seekings, LLP
Timothy Bouch
92 Broad Street
CHARLESTON, SC US 29401
214 722-5990

Travel History

Date/Time	Activity	Location
7/12/2017 - Wednesday		
8:18 am	Delivered	CHARLESTON, SC
7:22 am	On FedEx vehicle for delivery	CHARLESTON, SC
7:14 am	At local FedEx facility	CHARLESTON, SC
6:43 am	At destination sort facility	CHARLESTON, SC
4:11 am	Departed FedEx location	MEMPHIS, TN
12:20 am	Arrived at FedEx location	MEMPHIS, TN
7/11/2017 - Tuesday		
9:46 pm	Left FedEx origin facility	IRVING, TX
5:24 pm	Picked up	IRVING, TX
2:04 pm	Shipment information sent to FedEx	

Shipment Facts

Tracking number	779603947446	**Service**	FedEx First Overnight
Weight	0.5 lbs / 0.23 kgs	**Delivery attempts**	1
Delivered To	Receptionist/Front Desk	**Total pieces**	1
Total shipment weight	0.5 lbs / 0.23 kgs	**Terms**	Not Available
Shipper reference	Serve	**Packaging**	FedEx Envelope
Special handling section	Deliver Weekday	**Standard transit**	7/12/2017 by 8:00 am

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STATE OF SOUTH CAROLINA COUNTY OF SPARTANBURG	)))	IN THE COURT OF COMMON PLEAS SEVENTH JUDICIAL CIRCUIT
Beverly Dale Jolly and Brenda Rice Jolly, Plaintiff, vs. General Electric Company, et al., Defendants.	))))))))))	Civil Action No. 2016-CP-42-01592 **FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE LLC'S JOINT MOTION TO RECONSIDER THE ORAL DENIAL OF MOTION TO QUASH PLAINTIFFS' TRIAL SUBPOENA**

Fisher Controls International LLC ("Fisher") and Crosby Valve LLC ("Crosby") respectfully submit this joint motion to reconsider the Court's oral denial of Fisher's and Crosby's motion to quash the plaintiff's trial subpoena on the grounds that the Court failed to rule upon Crosby and Fisher's argument that they were not properly served with the trial subpoenas and this Court lacks the power to command an out-of-state representative of a non-resident corporation to appear and testify at trial. The movants' other arguments are included here as if incorporated herein for the Court's reconsideration as well.

ARGUMENT

I. Plaintiffs failed to effectuate valid service of the subpoenas on Crosby or Fisher.

A. Plaintiffs failed to personally serve on Crosby or Fisher.

Crosby and Fisher argued in their motions to quash and at the July 21, 2017 hearing that Plaintiffs failed to properly serve the subpoenas. (Fisher Motion to Quash pp. 1–2; Crosby Motion to Quash pp. 1–2; Tr. dated July 21, 2017 p. 6:18-23). The Court did not expressly rule on the service-related arguments and instead based its oral ruling on the authority delineated in Rule 45

of the South Carolina Rules of Civil Procedure. (Tr. dated July 21, 2017 pp. 13–16). Crosby and Fisher respectfully request that the Court reconsider its ruling and grant the motions to quash on the ground that Plaintiffs failed to effectuate proper service.

South Carolina's subpoena rule—Rule 45, SCRCP—incorporates the service requirements in Rules 4(d) and (j) of the South Carolina Rules of Civil Procedure. Rule 4(d) requires personal service. For a corporation, personal service is defined as delivering a copy of a document to "an officer, a managing or general agent, or any other agent authorized by appointment or by law to receive service of process." Rule 4(d)(3), SCRCP. Crosby and Fisher are out-of-state companies that are not registered to do business in South Carolina and, therefore, have no registered agent in the state. The companies, therefore, have no "agent authorized by appointment or by law to receive service" within South Carolina.

In an attempt to avoid the limitations of Rule 4(d), Plaintiffs served their subpoenas on Crosby and Fisher's trial counsel. Service on Plaintiff's trial counsel is not valid service. *See Hamilton v. Davis*, 300 S.C. 411, 414, 389 S.E.2d 297, 298 (Ct. App. 1990) (explaining the mere fact that a person is an agent of a company for some purpose does not render that person an authorized agent for the company for service of process); *see also Harrison v. Prather*, 404 F.2d 267, 273 (5th Cir. 1968) (finding a subpoena served on a plaintiff's attorney did not meet the personal service requirements in Federal Rule of Civil Procedure 45); *Ellis v. Louisiana ex rel. Governor's Office of Homeland Sec. & Emergency Preparedness*, No. CIV.A. 13-27-BAJ-RLB, 2013 WL 6272294, at *2 (M.D. La. Dec. 4, 2013) ("Under the plain language of the rule, as well as Fifth Circuit precedent, service is improper if the person himself is not served with a copy of the subpoena. The Fifth Circuit has found that service of a Rule 45 subpoena on a party's attorney, instead of the party, 'renders such service a nullity.'" (citation omitted)); *Waddle v. Waddle*, 868

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P.2d 751, 753 (Okla. Ct. App. 1994) (finding service of a subpoena is personal and the trial court did not err in refusing to enforce a subpoena that was served only on a party's counsel); 9A Charles Allen Wright & Arthur R. Miller, *Federal Practice & Procedure* § 2454 (3d ed.) ("The longstanding interpretation of Rule 45 has been that personal service of subpoenas is required. . . . Moreover, unlike service of most litigation papers after the summons and complaint, service on a person's lawyer will not suffice.").

Thus, to serve Crosby and Fisher with subpoenas, Plaintiffs must serve a person who is authorized by the companies to accept service of process—the companies' registered agents. Because Crosby and Fisher have no registered agents in South Carolina, Plaintiffs must get a court in a state in which the companies have a registered agent to serve the subpoenas on a registered agent. Service of the subpoenas is therefore invalid, and the witnesses thus cannot be compelled to testify.

B. Rule 4(j), SCRCP, does not allow service of a subpoena upon an out-of-state litigant's attorney.

At the hearing, the parties argued whether Rule 4(j) of the South Carolina Rules of Civil Procedure allow service on Crosby and Fisher's attorney. The Court did not rule upon this argument. Rule 4(j) does not allow service of a subpoena on an out-of-state party's attorney.

Rule 4(j) provides, "No other proof of service shall be required when acceptance of service is acknowledged in writing and signed by the person served or his attorney, and delivered to the person making service." Rule 4(j), SCRCP. Rule 4(j) relates back to Rule 4(g), which sets forth the requirements for proof of service. *Langley v. Graham*, 322 S.C. 428, 431, 472 S.E.2d 259, 261 (Ct. App. 1996). Proof of service is simply the method by which a serving party proves that it delivered a document and to whom it delivered the document; it "does not affect the validity of the service." Rule 4(g), SCRCP. "Rule 4(j) *establishes no new procedure for service of process*;

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rather, it is but a recognition of the long standing practice that acknowledgement or acceptance of service is equivalent to personal service." *Langley*, 322 S.C. at 431–32, 472 S.E.2d at 261 (emphasis added).

Rule 4(j) does not change the requirement in Rule 4(d)(3) that service upon a corporation must be made to "an officer, a managing or general agent, or to any other agent authorized by appointment or by law" by adding a corporation's attorney to the list of people who may be effectively served. Instead, it simply supports the proposition that Plaintiffs need not offer additional proof that they delivered the subpoenas to Crosby and Fisher's counsel if they have counsel's signature showing that he received the subpoenas through the mail. Service must be made to a registered agent to be effective; the attorney's acknowledgement of receipt does not make service effective. Further, as counsel argued at the hearing, he did not concede the validity of Plaintiffs service attempt by simply signing for a FedEx package without knowing the contents of the package.[1]

II. The Court lacks power to compel out-of-state witnesses to testify, regardless of whether the witnesses are parties.

A. The Court's exercise of personal jurisdiction over the parties does not automatically provide it with subpoena power over the parties.

This Court denied Crosby and Fisher's motions to quash on the ground that a party that has "submitted to the jurisdiction of the Court . . . can be compelled to submit testimony in the case." (Tr. dated July 21, 2017 pp. 13–14). This ruling was erroneous. The Court's exercise of personal jurisdiction over parties to litigation is separate from its subpoena power; personal jurisdiction and

[1] Rule 5(b) of the South Carolina Rules of Civil Procedure does not apply to the subpoena issue. Rule 45 incorporates Rule 5(b) only as it relates to "prior notice in writing of any commanded production of documents and things or inspection of premises before trial." Rule 45(b)(1), SCRCP.

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subpoena power are distinct, unrelated concepts. *See, e.g.*, *Syngenta Crop Prot., Inc. v. Monsanto Co.*, 908 So. 2d 121, 128 (Miss. 2005) ("[T]he concepts of personal jurisdiction and subpoena power are altogether different."). Moreover, no South Carolina authority links the exercise of personal jurisdiction to subpoena power, and Rule 45 does not provide an exception allowing South Carolina courts to exercise subpoena power over out-of-state parties. *See* Rule 45, SCRCP.

In a similar fashion, the Federal Rules of Civil Procedure do not provide federal courts with unlimited power to subpoena parties from outside the jurisdiction. Instead, those rules provide that a court may command a party or party's officer to attend trial if the trial is within 100 miles of where the person resides, is employed, or personally transacts business *or* if the trial is within the state where the person resides, is employed, or personally transacts business. Fed. R. Civ. P. 45(c)(1). Thus, even if a person is a party or a party's officer, the court cannot compel the person to travel over 100 miles and across state lines to attend trial. Other jurisdictions agree with this approach. *See, e.g.*, 6 Ind. Prac., Trial Handbook For Indiana Lawyers § 16:4 (stating there are no provisions for compulsory attendance of out-of-state residents in civil actions in Indiana); California Subpoena Handbook § 3:3 ("[T]he California civil litigant who needs the testimony of an out-of-state witness who will not come to California to testify must go to that state and take the deposition or proceed without that testimony.").

Accordingly, as Fisher and Crosby argued in their motions to quash, if Plaintiffs wished to present new testimony from Fisher and Crosby's corporate representatives, they should have noticed and taken a deposition of each and presented the deposition testimony at trial.[2] Instead,

[2] *See* Rhonda Wasserman, *The Subpoena Power: Pennoyer's Last Vestige*, 74 MINN. L. REV. 37, 41 (1989) ("The states' failure to exercise extraterritorial subpoena power has not made the testimony of out-of-state witnesses totally unavailable at trial. To compensate for the lack of extraterritorial subpoena power, the states have adopted complicated statutory schemes that permit a litigant to take a deposition in the nonresident witness's home state and introduce the transcript

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because Plaintiffs are aware that this Court cannot compel the out-of-state witnesses to sit for a deposition or appear at trial, they have concocted an alternate scheme to hail those two witnesses into court by improperly issuing subpoenas to Fisher and Crosby.

B. Rule 45 does not include any exceptions that provide this Court with power to compel testimony from non-officer employees of out-of-state parties.

In its ruling on the motions to quash, this Court interpreted Rule 45 to include an exception to the out-of-state limitations for "agents, managing directors, and other representatives of a corporation." (Tr. dated July 21, 2017 p. 16:14-23) ("I think that interpretation is further borne out -- I'm not looking at the language of Rule 45 right now, but in the part where it has the exception the fact that it [excepts] from the nonparty rules about out-of-state witnesses, agents, managing directors and other representatives of the corporation is further indication that corporate representatives of parties are not affected by the out-of-state limitations in the rule."). This Court's ruling was erroneous.

The "exception" applies to "a party or an officer, director or managing agent of a party." Rule 45(a)(3), SCRCP. There are no exceptions for non-officer employees or "other representatives" of a corporation. As counsel for Crosby and Fisher explained at the hearing, "neither Mr. Duimstra nor Mr. Martin are officers, directors, or managing agents as called for in Rule 45." (Tr. dated July 21, 2017 p. 19:4-8). Therefore, regardless of whether Rule 45 can be interpreted as carving out a party exception, that exception does not apply to either Crosby's or Fisher's non-officer, non-director, and non-managing agent corporate representatives. There is no exception in Rule 45 that allows a South Carolina court to command an out-of-state person to

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into evidence in the trial state."); Ryan W. Scott, *Minimum Contacts, No Dog: Evaluating Personal Jurisdiction for Nonparty Discovery*, 88 MINN. L. REV. 968, 1009 (2004) ("[P]arties could always secure the testimony of an out-of-state innocent bystander through a subpoena issued by a home forum that can properly exercise general jurisdiction.").

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attend a trial in South Carolina, regardless of whether that person is a party to litigation occurring in South Carolina.

Respectfully submitted,

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July 26, 2017

CERTIFICATE OF SERVICE

It is hereby certified that on this date the foregoing ***Fisher Controls International LLC and Crosby Valve LLC's Joint Motion To Reconsider The Oral Denial Of Motion To Quash Plaintiffs' Trial Subpoena*** in the above-captioned civil action was served upon each party to this cause or that party's counsel of record, by serving a copy upon counsel of record via email and hand delivery, postage prepaid, to the following address:

Theile B. McVey, Esq.
Kassel McVey Attorneys at Law
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Charleston, SC
July 26, 2017

/s/ Yancey A. McLeod III
Timothy W. Bouch
Yancey A. McLeod III

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STATE OF SOUTH CAROLINA	)	
	)	IN THE COURT OF COMMON PLEAS
COUNTY OF SPARTANBURG	)	SEVENTH JUDICIAL CIRCUIT
Beverly Dale Jolly and Brenda Rice Jolly,	)	Civil Action No. 2016-CP-42-01592
	)	
Plaintiffs,	)	
	)	
vs.	)	**FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE LLC'S JOINT MOTION FOR DIRECTED VERDICT**
	)	
General Electric Company, et al.,	)	
	)	
Defendants.	)	
	)	

Pursuant to Rule 50(a) of the South Carolina Rules of Civil Procedure, Defendants Fisher Controls International LLC ("Fisher") and Crosby Valve LLC ("Crosby") submit this Joint Motion for Directed Verdict. As set forth below, Plaintiffs Beverly Dale Jolly ("Mr. Jolly") and Brenda Rice Jolly (together, "Plaintiffs") have failed to meet their burden of proof as to each of their claims, and Fisher and Crosby are entitled to judgment as a matter of law.

SUMMARY OF ARGUMENT

Fisher and Crosby are entitled to a directed verdict because, even viewing all of the evidence in the light most favorable to Plaintiffs, no reasonable jury could find in their favor. Specifically, Fisher and Crosby are entitled to a directed verdict on the following grounds:

- Plaintiffs have failed to meet their burden of proof as to their failure to warn claims because the hazards of asbestos were open and obvious and known to Mr. Jolly, and Fisher and Crosby are protected by the sophisticated intermediary doctrine.
- Plaintiffs have failed to meet their burden of proof as to their design defect claims because they did not present evidence of a reasonable, feasible alternative design.
- Plaintiffs have failed to meet their burden of proof as to their negligence claims because they did not present evidence of the applicable standard of care or evidence that Fisher and Crosby deviated from the applicable standard of care.

- Regardless of their theory of recovery, Plaintiffs have presented insufficient evidence that Mr. Jolly developed mesothelioma from exposure to products placed into the stream of commerce by Fisher and Crosby. The evidence instead points to Mr. Jolly's exposure to asbestos-containing products manufactured and sold by third parties.

- Plaintiffs have failed to show Mr. Jolly's exposure to asbestos in connection with a Fisher or Crosby product was a proximate cause of his mesothelioma. The only specific causation evidence Plaintiffs offered was expert testimony from Dr. Arthur Frank. Dr. Frank's testimony is unreliable and inconsistent with the law, and it therefore should be excluded, consistent with a recent ruling from a South Carolina federal court.

- Finally, Plaintiffs' claims for punitive damages must be dismissed. Punitive damages are not attainable on strict liability or breach of warranty theories, and Plaintiffs' negligence claims fail as a matter of law. Furthermore, Plaintiffs failed to demonstrate, by clear and convincing evidence, that punitive damages are warranted as to either Defendant.

For all of the above reasons and those that follow, Plaintiffs cannot prevail on any of their claims, and Fisher and Crosby are entitled to a directed verdict.

LEGAL STANDARD

"When considering a directed verdict motion, the trial court should view the evidence and all reasonable inferences in the light most favorable to the non-moving party." *S.C. Fed. Credit Union v. Higgins*, 394 S.C. 189, 193–94, 714 S.E.2d 550, 552 (2011) (citations omitted). If more than one reasonable inference can be drawn from the evidence, the court should submit the case to the jury. *Id.* The court "should be 'concerned only with the existence or nonexistence of evidence,' not its credibility or weight. *Id.* (citation omitted). "Essentially, this Court must resolve whether it would be reasonably conceivable to have a verdict for [Plainitffs] under the facts as liberally construed in [Plainitffs'] favor." *Pye v. Estate of Fox*, 369 S.C. 555, 564, 633 S.E.2d 505, 509 (2006) (citations omitted).

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ARGUMENT

I. Plaintiffs Have Failed To Meet Their Burden of Proof On Their Failure To Warn Claims.

Plaintiffs have the burden of proving the elements of their failure to warn claims. This burden applies to each of their theories of recovery—negligence, strict liability, and breach of warranty. To prove liability under *any* products liability theory, Plaintiffs must show: (1) they were injured by the product; (2) "the injury occurred because the product was in a defective condition, unreasonably dangerous to the user;" and (3) "that the product at the time of the accident was in essentially the same condition as when it left the hands of the defendant." *Bragg v. Hi-Ranger, Inc.*, 319 S.C. 531, 539, 462 S.E.2d 321, 326 (Ct. App. 1995); *Dema v. Shore Enters., Ltd.*, 312 S.C. 528, 530, 435 S.E.2d 875, 876 (Ct. App. 1993). In addition, a manufacturer of a product is liable for failure to warn if (1) it knows or has reason to know the product is or is likely to be dangerous for its intended use, (2) it has "no reason to believe the user will realize the potential danger," and, (3) it fails to "exercise reasonable care to inform of its dangerous condition or of the facts which make it likely to be dangerous." *Livingston v. Noland Corp.*, 293 S.C. 521, 525, 362 S.E.2d 16, 18 (1987). If Plaintiffs fail to prove these elements, they cannot succeed on their failure to warn claim under any theory of recovery.

Plaintiffs have failed to establish any liability on the part of Fisher and Crosby as a matter of law, for several reasons. First, Mr. Jolly admitted that the dangers posed by asbestos-containing products were open and obvious to him at the time he worked around Fisher and Crosby products. Second, the undisputed evidence shows that Fisher and Crosby supplied their products to a sophisticated intermediary (Mr. Jolly's employer, Duke Power Company) and reasonably relied upon the sophisticated intermediary to comply with *its* duty to warn its employees of any risks posed by the products. Third, Plaintiffs have provided no evidence that the products at the time of

the alleged exposure were in essentially the same condition as when they left the hands of the Defendants. Finally, Plaintiffs have failed to show Fisher and Crosby's failure to warn about the hazards of asbestos associated with their valves proximately caused their injuries.

A. The potential hazards posed by asbestos allegedly used in connection with Defendants' valves were open and obvious, as Mr. Jolly admitted during his testimony.

A manufacturer has no duty to warn of an "open and obvious" danger created by its products nor one that the product's users generally recognize. *Moore v. Barony House Rest., LLC*, 382 S.C. 35, 41–42, 674 S.E.2d 500, 504 (Ct. App. 2009). Similarly, the failure to provide a warning is inconsequential where a plaintiff admits he knew of the dangers. *Rife v. Hitachi Const. Mach. Co.*, 363 S.C. 209, 220, 609 S.E.2d 565, 571 (Ct. App. 2005) (citing *Marchant v. Mitchell Distrib. Co.*, 270 S.C. 29, 37, 240 S.E.2d 511, 514 (1977)); *see also Owings v. Moneynick Oil Mill*, 55 S.C. 483, 33 S.E. 511, 512–13 (1899) ("There was not only no testimony in the case tending to show that the plaintiff was ignorant of the danger to which he was exposed in being put to work near the electric wire, but it shows, on the contrary, that the plaintiff was fully aware of the danger, and hence no warning was necessary. The plaintiff's own testimony shows that he was fully aware of the danger of coming in contact with the electric wires . . . . Any warning which might have been given would have been entirely useless, and therefore the defendant neglected no duty in failing to give such warning.").

Mr. Jolly knew of the potential hazards of working with asbestos-containing products. Numerous exhibits show Duke Power Company was warning its employees about the hazards of asbestos years before Jolly began working at Duke, and it continued to warn its employees during

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Jolly's tenure.[1] Moreover, Mr. Jolly admitted, based on information Duke conveyed to him, that he always knew asbestos could be hazardous.[2] Nonetheless, Mr. Jolly continued to work with asbestos-containing products. Even viewing the evidence and all reasonable inferences in the light most favorable to Plaintiffs, a reasonable fact finder could reach only one conclusion: the hazards of asbestos-containing products were open and obvious because Mr. Jolly admitted that he and his co-workers plainly knew of those hazards. Thus, to paraphrase *Owings, supra*, Mr. Jolly was

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[1] *See, e.g.*, Defendants' Exhibits 134 (1977 Duke Power document detailing safe working practices when working with asbestos); 135 (revising safe working practices for working with asbestos); 136 (1984 Duke Power guidelines on how to protect employees from asbestos); 137 (January 1, 1985 revised safe working practices for asbestos exposure at Duke Power).

[2] *See* 7/25/17 Trial Tr., at 119:13–120:8 ("Q Warning signs shall be provided and displayed at each location where asbestos dust hazards are present did Duke have such signs? A Well -- ***well they warned us.*** I'll put it like this. I couldn't tell you if a piece of dust on the floor came from insulation. Could I not tell you. Could I not tell you -- I couldn't identify it. And I wasn't looking for it. So I wasn't looking for it. ***I mean we know I think asbestos was bad for you*** but I didn't know how bad it was for me at the time. And a lot of other people didn't. But I didn't specifically go around looking down at the floor, could you get killed. You had to watch where you was going and I couldn't tell you -- and we were specifically – in fact, a guy on another crew almost got fired because he took off a piece of installation himself, he removed it. We could not remove it. That wasn't our job. We didn't remove insulation. ***We didn't touch it if we didn't have to. We didn't have to and we didn't touch it so you knew those restrictions were there you know. We didn't – we tried to avoid the insulation you know.***") (emphasis added); *id.* at 126:10-17 ("Q So is it fair to say in terms of warning that was Duke taking on responsibility to provide warnings for anything on its premise correct? A ***Yes there were warning signs. They had them everywhere. You see them everywhere. I mean everywhere you go*** you wouldn't believe the size of these building. Some of them are football fields long and wide you know.") (emphasis added); id. at , 126:20–127:15 ("Q Duke was a user of all these asbestos products from insulation to asbestos containing gaskets or packing correct? . . . Q Duke used all these products correct? A Yes. Q Insulation gaskets packing? A Yes at one time or the other yes. . . . Q Duke was knowledgeable about the hazards of asbestos correct? A I would say so. ***Q They had a warning after all? A Yes, they did.*** Q They were a sophisticated company? A I would say so yes I think so. ***Q Now when you saw these warning labels and you saw work being done on gaskets did you get a respirator? A Well if they had a sign up they did if they didn't I didn't you know.*** We didn't have to because it wasn't you know we were told when to use the respirators.") (emphasis added); *id.* at 130:14-20 ("Q In any event you were never handed a procedure on how to deal with asbestos in that 80 to 84 period correct? A ***We were told not to take off any insulation you know or be around when it they were taking it out or installing it. We knew that now you know. I knew not to do that. We all knew that.***") (emphasis added).

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fully aware of the danger, and hence no warning by Fisher or Crosby was necessary, nor would one have been heeded by him had it existed. For those reasons, Fisher and Crosby are entitled to a directed verdict with respect to Plaintiffs' failure to warn claims.

B. Fisher and Crosby are protected from liability by the sophisticated intermediary doctrine.

i. The Court should apply the *Webb* sophisticated intermediary doctrine.

This Court should apply the sophisticated intermediary doctrine to this case, as recently described by the California Supreme Court in *Webb v. Special Electric Co.*, 370 P.3d 1022, 202 Cal. Rptr. 460 (2016). The sophisticated intermediary doctrine is based on the principle that, when there is no effective way for a manufacturer to convey a product warning to the ultimate consumer or user, the manufacturer "should be permitted to rely on downstream suppliers to provide the warning." *Id.* at 1034. Judge Hill correctly permitted this defense to go to the jury in the *Garvin* matter.

The sophisticated intermediary doctrine allows a seller to "discharge its duty to warn end users about known or knowable risks in the use of its product" if it (1) "provides adequate warnings to the product's immediate purchaser, ***or*** sells to a sophisticated purchaser that it knows is aware or should be aware of the specific danger," and (2) "reasonably relies on the purchaser to convey appropriate warnings to downstream users who will encounter the product."[3] *Id.* (emphasis added). A manufacturer can satisfy the first prong by warning the intermediary of the hazard. *Id.* at 1035. However, a manufacturer is not required to warn the intermediary if the intermediary is "so knowledgeable about the material supplied that it knew or should have known about the

[3] This doctrine applies to both negligence and strict liability failure to warn claims because "there is little functional difference between the two theories in the failure to warn context." *Id.* at 1035 (citation omitted).

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particular danger." *Id.* As to the second prong, courts generally consider three factors: (1) the gravity of the risk posed by the product, (2) the likelihood that the intermediary will warn those downstream, and (3) the feasibility or effectiveness of the manufacturer giving a warning directly to the user. *Id.* at 1036.

The first factor weighs the reasonableness of the manufacturer's conduct against the potential severity of the harm. *Id.* at 1037. The second factor focuses on the reliability of the intermediary. *Id.* The manufacturer's knowledge about the reliability of the intermediary is determined using an objective standard based on what a reasonable manufacturer would have known under the circumstances. *Id.* In an analysis of the second factor, it is significant if the intermediary itself had a legal duty to warn end users about the hazard. *Id.* "In general, 'every person has a right to presume that every other person will perform his duty and obey the law.'" *Id.* (citation omitted). The third factor focuses on what the manufacturer can reasonably accomplish to warn end users of the risks. *Id.*

ii. An application of the sophisticated intermediary doctrine compels a ruling that Fisher and Crosby cannot be found liable on any of Plaintiffs' claims.

Applying the sophisticated intermediary doctrine to this case, Fisher and Crosby cannot be held liable for failing to warn Duke Power employees like Mr. Jolly. Fisher and Crosby sold valves to Duke Power. It cannot reasonably be disputed that Duke is a sophisticated entity. Further, the evidence shows Duke Power was knowledgeable about the hazards of asbestos leading up to and during Mr. Jolly's career at its power plants.[4]

[4] *See* Note 1, *supra*.

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Mr. Jolly agreed that Duke Power was a sophisticated entity who warned him and his co-workers about the hazards of asbestos from the very beginning of his tenure at Duke.[5] Similarly, Plaintiffs' own expert, Dr. Arthur Frank, agreed that Duke Power documents indicate that Duke was warning its employees about the hazards of asbestos, consistent with its policies and mandates under OSHA, prior to and during the time that Mr. Jolly worked for Duke.[6] In fact, when specifically asked by Plaintiffs' own counsel what a company in Fisher and Crosby's position could have done to warn Duke or its employees, Dr. Frank suggested four things the companies could have done:

> Q: So the question I have is *what is your opinion Dr. Frank as to what Crosby valve company or Fisher Controls could have told Duke Power in 1979 or 1980 about whether there was a hazard from the regular foreseeable maintenance of valves that they had sold to Duke Power*?
>
> * * *
>
> MR. BOUCH: Same objection I've been making for the record assumes facts not in evidence incomplete hypothetical.
>
> THE COURT: Overruled.
>
> THE WITNESS: *What could have been shared by the companies supplying asbestos containing materials was that their product contained asbestos and that asbestos was known to be hazardous and that the people working with their product should be cautioned about it about using the product about liberating dust and if they were going to do that to do it in a manner that would reduce their exposures.*[7]

Dr. Frank then admitted that Duke Power *already knew each of these things* by the time Mr. Jolly arrived to work there.[8] Accordingly, Duke Power qualifies as a sophisticated buyer "so

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[5] *See* Note 2, *supra*.
[6] *See* 7/26/17 Trial Tr., at pp. 160–75.
[7] *Id.* at 108:9 – 109:4 (emphasis added).
[8] *See id.* at 181:3-20 (admitting Duke Power ordered and knew it was receiving asbestos gaskets, knew the hazards of asbestos since at least 1964, was under a responsibility to reduce asbestos hazards pursuant to OSHA regulations beginning at least in 1972, and knew how to reduce exposures through various methods).

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knowledgeable about the material supplied that it knew or should have known about the particular danger," and Fisher and Crosby have satisfied the first prong of the sophisticated intermediary doctrine. *Webb*, 370 P.3d at 1035.

Fisher and Crosby also satisfied the second prong of the sophisticated intermediary doctrine. Duke Power was a reliable intermediary. *See id.* at 1037. The documentary evidence and admissions by Plaintiffs' own experts show that Duke Power had a duty to warn (and protect) its employees of the potential hazards of asbestos-containing products pursuant to OSHA regulations, and it complied with that duty during Mr. Jolly's entire career at Duke. Fisher and Crosby were entitled to presume that Duke would comply with its own obligations. *See id.* Finally, Fisher and Crosby could not realistically warn Mr. Jolly and other Duke employees about the alleged hazards of asbestos connected to their valves. Fisher and Crosby therefore satisfy the requirements of the sophisticated intermediary doctrine as a matter of law, and they are entitled to directed verdict on all of Plaintiffs' claims.

iii. An application of the sophisticated intermediary doctrine is in accordance with South Carolina law.

In 1995, the South Carolina Court of Appeals considered a trial court's decision to charge a jury on the "sophisticated user defense."[9] *Bragg*, 319 S.C. at 550, 462 S.E.2d at 332. The Court of Appeals noted the defense, which was outlined in section 388 of the Restatement (Second) of

[9] The sophisticated user defense and the sophisticated intermediary doctrine, although bearing slightly different names, are the same doctrine. *See Webb*, 370 P.3d at 1034; *Bragg*, 319 S.C. at 550, 462 S.E.2d at 332 ("[T]he sophisticated user defense may be permitted in cases involving an employer who is aware of the inherent dangers of a product which the employer purchases for use in its business; if the employer/purchaser has 'equal knowledge' of the product's dangers, then the manufacturer may be able to rely on the employer/purchaser to protect its own employees from harm.") (citing *Willis v. Raymark Indus., Inc.*, 905 F.2d 793 (4th Cir. 1990). Fisher and Crosby believe the "sophisticated intermediary doctrine" is the more appropriate name to avoid confusion between the intermediary (the employer) and the user (the plaintiff-employee).

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Torts, "has been adopted by numerous jurisdictions." *Id.* The Court of Appeals affirmed the trial court's charging of the doctrine. *Id.*

Despite the Court of Appeals' explicit approval of the doctrine in *Bragg* and later in *Lawing v. Trinity Mfg., Inc.*, 406 S.C. 13, 23, 462 S.E.2d 126, 131 (Ct. App. 2013) (*Lawing I*), the South Carolina Supreme Court held in 2015 that no South Carolina appellate court had adopted the doctrine. *Lawing v. Univar, USA, Inc.*, 415 S.C. 209, 226, 781 S.E.2d 548, 557 (2015) (*Lawing II*). *But see Lawing II*, 415 S.C. at 229, 781 S.E.2d at 559 (Kittredge, J., concurring) ("The doctrine was clearly recognized in *Bragg* . . . ."). Nonetheless, the Supreme Court did not expressly adopt or reject the sophisticated intermediary doctrine in *Lawing II*. 415 S.C. at 226, 781 S.E.2d at 557. Instead, the court explained it "need not formally adopt the doctrine at this time because . . . the facts of this case do not implicate the sophisticated user defense." *Id.*

The facts in *Lawing* are distinguishable from this case. Therefore, *Lawing* does not control whether this Court should or should not apply the sophisticated intermediary doctrine. In *Lawing*, the issue was whether bags of a flammable chemical were properly labeled in a manner that would allow employees of a plant to identify what was in the bags. *Id.* at 227, 781 S.E.2d at 548. Because the bags were not labeled in a manner that allowed the employees to identify them as flammable chemical, the employees did not move the bags to a safe distance before performing work with an oxyacetylene torch nearby. *Id.* at 214–16, 781 S.E.2d at 551–52. The bags ignited, injuring the employees. *Id.* The court held the proper focus under the facts of *Lawing* was the *labeling* on the bags of chemical, not the use of the chemical in the plant. *Id.* at 227, 781 S.E.2d at 558. The employer's knowledge of the dangers of the chemical did not affect the supplier's duty to label the chemical in a manner that would allow an employee to identify it as a hazardous and flammable

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product, "because the knowledge of [the chemical's] inherent qualities [is] useless to a person who comes into contact with the chemical but cannot identify it." *Id.*

The Court explained, "there is a critical distinction between an intermediary's knowledge of the dangerous qualities and nature of a product, and the ability of the third party user to identify and recognize that product on its face. When considering only [the employer's] use of [the chemical] in its manufacturing process, it follows that [the employer] is a 'sophisticated user.'" *Id.* at 227–28, 781 S.E.2d at 558. When labeling is the underlying issue, however, the adequacy of the labeling does not require a sophisticated user analysis. *Id.* Thus, in *Lawing*, the employer's knowledge of the dangers of the chemical was irrelevant. Instead, the critical issue was whether the product's labeling allowed employees to recognize the chemical as a product that needed to be moved out of range of the oxyacetylene torch before using the torch.

In this case, however, the issue is Duke Power's *use* of asbestos-containing products given their potentially hazardous nature. The issue is not whether the products were labeled in a manner that allowed Duke's employees to identify it as asbestos. Therefore, *Lawing* is not controlling, and this Court should apply the sophisticated intermediary doctrine because Duke had a duty to warn its employees about the hazardous nature of asbestos.

C. Plaintiffs have failed to show that the Fisher and Crosby valves allegedly at issue were in essentially the same condition as when they left the hands of the Defendants.

It is well settled that plaintiffs in a products liability case, regardless of their theory of recovery, must show that the allegedly defective product "was in essentially the same condition as when it left the hands of the defendant." *Madden v. Cox*, 284 S.C. 574, 579, 328 S.E.2d 108, 112 (Ct. App. 1985) (citing W. Prosser, Law of Torts 671-72 (4th ed. 1970); S.C. Code Ann. §§ 15-73-10, 36-2-318, 36-2-715; *Claytor v. General Motors Corp.*, 277 S.C. 259, 286 S.E. 2d 129

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(1982); *Logan v. Montgomery Ward & Co., Inc.*, 216 Va. 425, 219 S.E. (2d) 685 (1975)); *see also Graves v. CAS Med. Sys.*, 401 S.C. 63, 79, 735 S.E.2d 650, 658 (2012) ("In any products liability action, a plaintiff must establish three things: (1) he was injured by the product; (2) the product was in essentially the same condition at the time of the accident as it was when it left the hands of the defendant, and (3) the injury occurred because the product 'was in a defective condition unreasonably dangerous to the user.'") (citing *Madden*).

Here, Plaintiffs have made no showing that the valves that Plaintiffs claim exposed Mr. Jolly to asbestos were in essentially the same condition as when they left the hands of the Defendants. In fact, it appears that the opposite is true: Mr. Jolly testified that he believes he was exposed to asbestos from flange gaskets that were placed on the Defendants' valves, and the undisputed evidence shows that these flange gaskets were placed on those valves by Duke Power employees long after they left the hands of Fisher and Crosby. As set forth more fully herein, these Defendants cannot be liable for these products, and the Court should properly direct a verdict in favor of these Defendants on this additional ground.

D. Plaintiffs have failed to show Fisher and Crosby's alleged failure to warn was the proximate cause of their injuries.

For reasons similar to those set forth above, Plaintiffs' failure to warn claims must also be dismissed because Plaintiffs have not shown that the Defendants' alleged failure to warn about the hazards of asbestos associated with their valves proximately caused their injuries.

To prove their failure to warn claims, Plaintiffs must show that a warning given by Fisher and Crosby would have made a difference and prevented the harm. Plaintiffs have not made this required showing. Plaintiffs elicited no evidence regarding 1) what the warning should have contained, 2) where it should have been displayed, 3) who would have seen the warning, 4) what the person seeing the warning would have done in response to seeing the warning, 5) how that

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response would have impacted plaintiff's alleged exposure, or 6) whether the response to the warning would have impacted causation (i.e. that if warned, Mr. Jolly would not have contracted mesothelioma).

There is no presumption in South Carolina that a plaintiff would have heeded a warning if it had been given. Instead, "[t]he plaintiff has the burden of showing that a warning would have made a difference in the conduct of the person warned." *Allen v. Long Mfg. NC, Inc.*, 332 S.C. 422, 432, 505 S.E.2d 354, 359 (Ct. App. 1998). If the plaintiff would not have heeded a warning, the failure to warn cannot be the proximate cause of the plaintiff's injuries. *See id.* Mr. Jolly made clear during his testimony that he knew about the hazards of asbestos. *See* Part I.B.ii, *supra*. Plaintiffs' evidence also proves that Duke Power knew asbestos could be hazardous and repeatedly warned its employees. *Id.* Despite Duke's warnings and Mr. Jolly's knowledge, he continued to work with and around asbestos-containing products and Fisher and Crosby valves.

<u>As a matter of fact, Plaintiffs submitted no evidence that Mr. Jolly would have heeded a warning had one been given; they did not even attempt to elicit such testimony and for this reason alone their failure to warn claim should be dismissed</u>. By contrast, because Mr. Jolly admitted that Duke Power warned him about asbestos, and he failed to take precautions anyway, the only evidence in the record is that Mr. Jolly did not heed warnings generally.

Because Mr. Jolly in fact *did not* heed warnings from Duke Power and continued to work around Fisher and Crosby valves despite his knowledge of the hazards, it would defy logic for Plaintiffs to argue that Mr. Jolly would have stopped working around Fisher and Crosby valves if he had received a warning directly from Fisher and Crosby. No reasonable jury could find that Mr. Jolly would have heeded such warnings from Fisher and Crosby on their valves. Consequently, as a matter of law, Fisher and Crosby's failure to warn was not the proximate cause

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of Plaintiffs' injuries. *See Owings*, 33 S.E. at 512–13 (1899) ("Any warning which might have been given would have been entirely useless, and therefore the defendant neglected no duty in failing to give such warning."); *see also, e.g.*, *Billiar v. Minnesota Min. & Mfg. Co.*, 623 F.2d 240, 243 (2d Cir. 1980) ("When the user is fully aware of the nature of the product and its dangers, however, the supplier cannot be held liable for failure to warn him."). Fisher and Crosby are entitled to a directed verdict on the failure to warn claims.

II. Plaintiffs Have Failed To Meet Their Burden of Proof On Their Design Defect Claims.

Plaintiffs' design defect claim[10] also fails as a matter of law. For a plaintiff to succeed on a design defect claim, "he must show that the design of the product caused it to be 'unreasonably dangerous.'" *Branham v. Ford Motor Co.*, 390 S.C. 203, 218, 701 S.E.2d 5, 13 (2010). South Carolina courts apply the risk-utility test in analyzing design defect claims. *Id.* at 220, 701 S.E.2d at 14. The risk-utility test requires a plaintiff asserting design defect claims must prove the availability of a reasonable alternative design. *Id.*; *Holland ex rel. Knox v. Morbark, Inc.*, 407 S.C. 227, 237, 754 S.E.2d 714, 720 (Ct. App. 2014) ("[T]he requirement of proving a reasonable alternative design in a design defect case is mandatory."). A plaintiff must "point[] to a design flaw in the product and show[] how his alternative design would have prevented the product from being unreasonably dangerous." *Graves v. CAS Med. Sys., Inc.*, 401 S.C. 63, 79, 735 S.E.2d 650, 658 (2012) (quoting *Branham*, 390 S.C. at 225, 701 S.E.2d at 16). To prove a reasonable alternative design, a plaintiff must "set forth some evidence of an 'alternative design,' which necessarily included the 'consideration of costs, safety, and functionality associated with the alternative design.'" *Holland*, 407 S.C. at 237–38, 754 S.E.2d at 720 (citation omitted). Plaintiffs

[10] Plaintiffs have not asserted (or attempted to prove) a manufacturing defect claim in this case. Consequently, if their failure to warn and design defect claims are dismissed, the entire case must be dismissed.

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must satisfy this requirement to prove a product was in a defective condition unreasonably dangerous to the user; therefore, the following analysis applies to all three of Plaintiffs' theories of recovery. *See Bragg*, 319 S.C. at 539, 462 S.E.2d at 326; *Dema*, 312 S.C. at 530, 435 S.E.2d at 876.

Plaintiffs have not put forth any evidence whatsoever showing that a reasonable alternative design existed during the time that Mr. Jolly allegedly worked around the Defendants' valves. In fact, with respect to asbestos-containing thermal insulation that may have gone near or around some valves (which are not at issue in this case for purposes of these Defendants' potential liability, as set forth in Section IV, *infra*), Plaintiffs' own expert, Dr. Frank, admitted that asbestos was the *only* insulating option available in hot settings at issue (i.e., applications greater than 1,000 degrees).[11] This requirement for alternative feasible design proof applies to a failure to warn claim. *Holland ex rel. Knox v. Morbark, Inc.*, 407 S.C. 227, 237, 754 S.E.2d 714, 720 (Ct. App. 2014).

III. Plaintiffs' Negligence Claims Fail Because They Have Not Shown Fisher or Crosby Deviated From the Standard of Care.

A plaintiff asserting products liability claims under a negligence standard—in addition to the elements described above—bears the burden of demonstrating the defendant failed to exercise due care. *See Bragg*, 319 S.C. at 539, 462 S.E.2d at 326. This requires the plaintiff to present evidence showing the applicable standard of care and that the defendant deviated from the standard of care. *See Sunvillas Homeowners Ass'n, Inc. v. Square D Co.*, 301 S.C. 330, 333, 391 S.E.2d 868, 870 (Ct. App. 1990) ("To establish negligence the plaintiff must prove the defendant failed to exercise due care in some respect."); *see also AT & T Corp. v. Med. Review of N.C., Inc.*, 876

[11] *See* 7/27/17 Trial Tr. at 147:24 – 148:5 ("Q Do you know the temperature ranges between the [fiberglass insulation] and the asbestos ranges? A Asbestos will go up to higher heat ranges I don't know the exact numbers. Q So if you had something going up to a thousand degrees you couldn't use fiberglass could you? A There's probably a limit a little bit below that.").

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F. Supp. 91, 93 (E.D.N.C. 1995) (providing that the elements of a products liability claim for negligence include "evidence of a standard of care owed by the reasonably prudent person in similar circumstances" and "breach of that standard of care"). Plaintiffs have not presented evidence of the applicable standard of care at the time of Mr. Jolly's alleged exposure to asbestos in connection with the Defendants' valves. *Bragg*, 319 S.C. at 548–49, 462 S.E.2d at 331 (stating that, under South Carolina products liability law, "a product must be measured against a standard existing at the time of sale"); *id.* at 548, 462 S.E.2d at 331 ("A manufacturer . . . has no duty to notify previous purchasers of its products about later developed safety devices or to retrofit those products if the products were nondefective under the standards existing at the time of the manufacture or sale."). Therefore, Plaintiffs have failed to prove a necessary element of their negligence claims.

Furthermore, regardless of whether Plaintiffs have properly presented evidence of the standard of care, they have not proven that Fisher or Crosby deviated from the standard of care. As explained in the previous section, to prove a negligent design defect, Plaintiffs must present evidence of a reasonable alternative design. *Branham*, 390 S.C. at 225, 701 S.E.2d at 16; *Holland*, 407 S.C. at 237, 754 S.E.2d at 720. Accordingly, Plaintiffs must show Fisher and Crosby designed products in an unreasonably dangerous manner despite the existence of a feasible, less dangerous alternative design. *Id.*; Allen, 332 S.C. at 426–27, 505 S.E.2d at 356 (providing "[l]iability for negligence requires, . . . proof that the manufacturer breached its duty to exercise reasonable care to adopt a safe design"). To prove a negligent failure to warn, Plaintiffs must show Fisher and Crosby had actual or constructive knowledge of the risk posed by their products. *Webb*, 370 P.3d at 1030. The standard of care is whether a prudent seller of a product who knew of a risk would

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have acted reasonably in not giving a warning. *Id.* A seller of a product who reasonably does not give a warning is not liable for negligent failure to warn.

Here, as explained in Part II, Plaintiffs did not present evidence of a reasonable alternative design; therefore, they have offered no proof Fisher and Crosby deviated from the standard of care as to Plaintiffs' design defect claim. As explained in Part I, Fisher and Crosby had no duty to warn Mr. Jolly of the hazards of asbestos. Consequently, Plaintiffs have presented no evidence that Fisher and Crosby deviated from the standard of care as to Plaintiffs' negligent failure to warn claims.

Moreover, it is unrefuted that any potential exposure to asbestos in connection with Fisher and Crosby valves would not have exceeded applicable permissible exposure limits in place at the time Mr. Jolly worked around those valves. Compliance with government safety standards—such as OSHA standards—constitutes strong and substantial evidence that a product is not defective. *Gideon v. Johns-Manville Sales Corp.*, 761 F.2d 1129, 1144 (5th Cir. 1985). Plaintiffs cannot prove their negligence claims, and Fisher and Crosby are entitled to a directed verdict on those claims.

IV. Plaintiffs' Claims Related To Products Fisher and Crosby Did Not Sell, Design, or Place Into The Stream of Commerce Must Be Dismissed.

Fisher and Crosby maintain that they are entitled to a directed verdict on all of Plaintiffs' claims, as explained above. Additionally, Fisher and Crosby assert that they are entitled to a directed verdict on Plaintiffs' claims related to products Fisher and Crosby did not sell, design, or place into the stream of commerce.

Many of the valves Fisher and Crosby manufactured and sold were asbestos-free when they left Fisher and Crosby's hands. Moreover, Plaintiffs have presented no evidence that Mr. Jolly worked around *original* asbestos-containing components of a Fisher or Crosby valve nor that he

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worked around these *internal* components at all. Instead, Mr. Jolly's only testimony alleging exposure to asbestos in connection with Fisher or Crosby valves related to *flange gaskets*, which these Defendants did not sell or recommend.[12] Thus, there is *no evidence* Mr. Jolly was ever exposed to asbestos in connection with a product actually sold by Fisher or Crosby. Instead, Fisher and Crosby sold "bare metal" valves, and Duke Power Company later added asbestos-containing (or non-asbestos-containing) flange gaskets (and, in some cases, insulation) to the outside of those valves. However, because Fisher and Crosby did not make, sell, specify, or place into the stream of commerce any such flange gaskets and/or insulation that Duke Power employees may have used in conjunction with Fisher or Crosby valves, courts across the country have held that Fisher and Crosby owed no legal duty to Plaintiffs with respect to those products.

Indeed, an overwhelming majority of courts in recent years have concluded that they will not hold manufacturers responsible for allegedly defective component products supplied by third parties. This trend appears to have begun with the Fourth Circuit, which reached such a holding under South Carolina law, and it has more recently been extended into the asbestos context for companies just like Fisher and Crosby. *See Baughman v. General Motors Corp.*, 780 F.2d 1131, 1132–33 (4th Cir. 1986) (applying South Carolina law in holding that a defendant cannot be liable for a defective component part where the defendant manufacturer did not incorporate the defective component part into its finished product and did not place the defective component into the stream of commerce"); *see also, e.g.*, *O'Neil v. Crane Co.*, 266 P.3d 987, 991, 1005 (Cal. 2012) (holding pump and valve defendants were not liable for harm caused by asbestos products they did not manufacture or distribute, regardless of the theory of recovery articulated, and stating "California

[12] *See, e.g.*, 7/25/17 Trial Tr. at pp. 75-77 (Mr. Jolly testifying that he was present when others were scraping flange gaskets off Crosby and Fisher valves); *see also* 7/28/17 Trial Tr. at pp. 107-108 (testifying that Fisher never provided or recommended flange gaskets to customers).

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law does not impose a duty to warn about dangers arising entirely from another manufacturer's products, even if it is foreseeable that the products will be used together"); *Simonetta v. Viad Corp.*, 197 P.3d 127 (Wash. 2008). South Carolina has also expressed this requirement in the asbestos context in *Henderson*, in which the Supreme Court held that in order to establish a cause of action for an asbestos injury, a plaintiff must show, *inter alia*, that he or she "was exposed to the [*defendants*'] asbestos containing products." *Henderson v. Allied-Signal, Inc.*, 373 S.C. 179, 185, 644 S.E.2d 724, 727 (2007) (emphasis added) (altered from "Respondents" to "defendants").

Judge Norton, of the District of South Carolina, initially agreed with this notion, as he ruled in late 2014 that a pump manufacturer could not be liable for other companies' asbestos components that were incorporated into its pumps post-sale. *See Sparkman v. A.W. Chesterton Co.*, No. 2:12-cv-02957-DCN, 2014 U.S. Dist. LEXIS 177560 (D.S.C. Dec. 29, 2014) ("*Sparkman I*"). As Judge Norton explained:

> ***The rationale for strict liability under South Carolina product liability law comports with the principles of the bare metal defense.*** Specifically, under *comment c of 402A*, Goulds should not be held liable for products which it did not manufacture, supply or market. See *Restatement (Second) of Torts § 402A cmt. c* (1965). In light of this rationale, and the persuasive weight of authority from other jurisdictions adopting the bare metal defense, ***the South Carolina Supreme Court would likely find a manufacturer is not subject to a duty to warn about potential dangers from exposure to parts of its product which it did not manufacture or otherwise supply. Accordingly, Goulds cannot be liable for exposure to asbestos from products that it did not manufacture or otherwise supply.***

Id. at *19–20 (emphasis added).

Judge Norton subsequently reversed course on this point based on a South Carolina circuit court's holding in its post-trial ruling in *Garvin v. Agco Corp.*, Case No. 2012-CP-40-6675 (2014) (the "*Garvin* Order"), because there was evidence that the defendant in *Sparkman* "specified the use of asbestos for packing and gaskets in its pumps." *Sparkman v. Goulds Pumps, Inc.*, No. 2:12-cv-02957, 2015 U.S. Dist. LEXIS 19579 (D.S.C. Feb. 19, 2015) ("*Sparkman II*"). However,

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Fisher and Crosby respectfully submit that Judge Norton's initial holding was correct and the appropriate ruling. Moreover, the *Garvin* Order is not binding upon this Court and, as set forth below, the rationale for imposing liability under the *Garvin* Order is not present here.

First, the *Garvin* Order indicated that the plaintiff there may have been exposed to "original" asbestos-containing materials supplied with the defendant's valves. No such evidence exists here with respect to Fisher and Crosby products. Second, the *Garvin* Order indicated that the replacement parts used with the defendant's valves at issue were similar to the original parts. The only testimony in this case relates to asbestos-containing flange gaskets supplied by Duke, which purchased them from third parties and incorporated them into Fisher and Crosby gaskets post-sale—after the valves left Fisher and Crosby. There is no evidence in this case that the gaskets Duke Power incorporated into Fisher and Crosby valves were substantially similar to those Fisher and Crosby had originally incorporated as *internal* components into their valves. Finally, unlike the defendant in *Garvin*, there is no evidence in this case that Fisher and Crosby had any interaction with Duke Power or somehow required or specified the use of asbestos-containing components with their valves. Rather, the evidence demonstrates the opposite: *Duke Power engineers* specified which parts they would use in connection with the Defendants' products.

The specification by Duke Power engineers places this case squarely within the analysis in the *O'Neil* case authored by the California Supreme Court. There, the court found the United States Navy was responsible for using asbestos-containing replacement parts in the defendants' valves. 266 P.3d at 996. The court found "there was no evidence that defendants' products *required* asbestos-containing gaskets or packing in order to function." *Id.* In fact, "*[a]part from the Navy's specifications*, no evidence showed that the design of defendants' products required the use of asbestos components, and their *mere compatibility* for use with such components is not

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enough to render them defective." *Id.* (emphasis added). The court further held, "[w]here the intended use of a product *inevitably* creates a hazardous situation, it is reasonable to expect the manufacturer to give warnings. Conversely, where the hazard arises entirely from another product, and the defendant's product does not create or contribute to that hazard, liability is not appropriate." *Id.* at 1004 (emphasis added).

Similarly, in cases decided more recently than *Garvin*, South Carolina federal courts have declined to adopt a rule that defendants are liable whenever the use of asbestos in their products is foreseeable. *See, e.g.*, *Andrews v. Crane Co.*, Case No. 2:13-cv-02055-RMG, D.E. 454 at 16 (D.S.C. June 2, 2015). Instead, the courts have rejected application of the bare metal defense only "where the defendant has specified or recommended the use of another product with its own." *Id.* (finding a defendant could not be liable for insulation used with its pumps because the plaintiff did not put forward evidence that the defendant "recommended or specified asbestos-containing insulation for use with its valves"); *see also Andrews v. Crosby Valve LLC*, Case No. 2:13-cv-02055-RMG, D.E. 418 at 13 (D.S.C. May 26, 2015) ("There is no evidence that Crosby specified, recommended or required the use of these external asbestos gaskets or the external asbestos jacket insulation. Therefore, summary judgment is appropriate.").

Accordingly, because Plaintiffs presented no evidence that Fisher and Crosby specified or required the use of asbestos-containing replacement parts with their valves or that it was inevitable that original parts would be replaced with asbestos-containing parts, Fisher and Crosby are entitled to a directed verdict.

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V. Plaintiffs Have Failed To Show Mr. Jolly's Exposure To Asbestos In Connection with the Defendants' Products Was a Proximate Cause of His Mesothelioma.

A. Plaintiffs Have Failed to Establish Specific Causation Through Relevant, Reliable Expert Testimony That Would Assist Jury In Reaching a Verdict.

Regardless of their theory of recovery, Plaintiffs have not shown that Mr. Jolly's exposure to asbestos proximately caused his mesothelioma. In a case where a medical causal relationship is at issue and not within common knowledge (such as the relationship between asbestos exposure and mesothelioma), Plaintiffs must prove both general and specific causation through expert testimony. *Fisher v. Pelstring*, 817 F. Supp. 2d 791, 814 (D.S.C. 2011) ("Where a medical causal relation issue is not one within the common knowledge of the layman, proximate cause cannot be determined without expert medical testimony.") (citing *In re Bausch & Lomb Inc. Contacts Lens Solution Prod. Liab. Litig.*, 693 F.Supp.2d 515, 518 (D.S.C.2010)). "To establish medical causation in a product liability case, a plaintiff must show both general causation and specific causation." *Id.* "General causation is whether a substance is capable of causing a particular injury or condition in the general population, while specific causation is whether a substance caused a particular individual's injury." *Id.*

The testimony of Plaintiffs' experts fails to prove specific causation because they gave opinions that were unreliable and at inadmissible under Rules 702 and 403 of the South Carolina Rules of Evidence. Specifically, Plaintiffs offered the testimony of Dr. Frank, who testified that each and every exposure causes mesothelioma and the cumulative effect of Mr. Jolly's "exposures to asbestos contribute[d] to his cumulative exposure that gave Mr. Jolly his mesothelioma."[13] Dr. Frank repeatedly emphasized his opinions that every single exposure to asbestos was a cause of Mr. Jolly's mesothelioma, and, because he cannot "rule out" a particular exposure as the cause of

[13] *See* 7/26/17 Trial Tr. at 65:24–68:24.

the mesothelioma, he must "rule in" every exposure as being the cause.[14] This opinion is clearly unreliable, as several courts have held. In fact, at one point Dr. Frank even admitted that he held

[14] *See* 7/25/17 Trial Tr. pp. 41:19–44:5 ("There is one safe level of exposure to asbestos: Zero. It's the only level at which you're absolutely safe. . . . One day of exposure to asbestos, both in animal studies and a few human cases that have been reported, have occurred with a single day of exposure. Now it's unusual and it's rare but it tells you how little exposure it takes to give somebody a mesothelioma. Or a lung cancer. And for mesothelioma, one day has done it. We know a month or less of exposure to asbestos has doubled the risk of lung cancer for example. That's epidemiological data in humans but the animal data, one day and mesothelioma individual cases of one day of exposure."); *id.* at 57:2–58:19 ("Q: And if those products are removed from hand wire brushing electric grinders the gaskets and someone gets mesothelioma some decades later are those exposures an important and substantial part of the cause of that mesothelioma? A: Yes. Q: Okay can you Dr. Frank say there's one exposure to removal of an steel asbestos jacket on one day for half an hour a person gets mesothelioma 30 years later and say you know what that sounds so puny let's just put that to the side in terms causation? . . . THE WITNESS: ***What we have to say at the end of the day if someone gets a mesothelioma is that the cumulative exposure they have had to asbestos from whatever products over whatever time is what gave it to them. . . . Some products contribute more some less but you can't leave any of them out*** because it is a cumulative exposure, the totality of what people were exposed to that gave them their disease so all of them have to be included.") (emphasis added); *id.* at 65:24–68:24 ("Q: Do you have an expert opinion with a reasonable degree of medical certainty as to whether or not that exposure even if it were just one occasion would be a substantial contributing factor to what caused the mesothelioma? A: I do have an opinion. Q: And what is that? A: And ***my opinion is since there is no exposure that can be left out for reasons we discussed a bit earlier that would be a substantial contributing factor. It was part of one's cumulative exposure.*** Q: Dr. Frank, if all of the things I've asked you to assume, if that were the only asbestos exposure, in and of itself, would it be sufficient to cause mesothelioma? A: By that are you saying his one day of exposure or just the exposure over four years to. Q: The one day of exposure standing there doing inspection work on a gasket on a flange face of a valve -- an asbestos containing gasket on a flange face of a valve is being removed with a power tool hand tools and scraper? A: ***Again I have not ever seen such a case but if that was the facts of case given to me I would have to say that that exposure because we know in other settings one day of exposure was sufficient that that was the cause of someone's mesothelioma if they developed one.*** Q: So if that person developed mesothelioma, that exposure in and of itself would be sufficient is that what you're saying? A: Yes. . . . Q: Let me ask you to make all of those same assumptions about exposures and ask you to assume that that is the exposure of Mr. Dale Jolly. Do you have an opinion with a reasonable degree of medical certainty as to whether or not Dale Jolly's mesothelioma was substantially contributed to by exposure to asbestos liberated from asbestos gaskets on the flange face of valves during removal using the processes that I described? A: I do have an opinion. Q: And what is that? A: My opinion would be that ***Mr. Jolly had those exposures contribute to his cumulative exposure*** that gave Mr. Jolly his mesothelioma. Q: And Dr. Frank but for those exposures, meaning if you try to take them out, would you be able to say that Mr. Jolly would have still gotten mesothelioma? A: When you say is that ***all of them contributed.*** What occurs when you take them out any one of them is unclear

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this opinion with regard to a single asbestos exposure one might receive as a bystander to work being done to the flange of a valve on one day being the cause of a later-developed mesothelioma, ***even though he was not aware of such a case ever occurring.***[15] Moreover, as the Honorable Judge David C. Norton held earlier this month in South Carolina federal court, this opinion is directly at odds with Plaintiffs' burden of proof as set forth in *Henderson v. Allied-Signal, Inc.*, 373 S.C. 179, 644 S.E.2d 724 (2007), and it should therefore be excluded and given no weight on that basis pursuant to Rule 403.

The admissibility of expert testimony is generally governed by Rule 702 of the South Carolina Rules of Evidence, which provides that "[i]f scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training or education, may testify thereto in the form of an opinion or otherwise." In determining whether to admit expert testimony under Rule 702, the Court must make three inquiries: (1) whether the subject matter is beyond the ordinary knowledge of the jury, thus requiring an expert to explain the matter to the jury; (2) whether the expert has acquired the requisite knowledge and skill to qualify as an expert in the particular subject matter; and (3) whether the substance of the testimony is reliable. *See Graves v. CAS Medical Systems, Inc.*, 401 S.C. 63, 74, 735 S.E.2d 650, 655 (2012); *Watson v. Ford Motor Co.*, 389 S.C. 434, 446–47, 699 S.E.2d 169, 175–76 (2010). Plaintiffs' experts' "no safe level/every exposure" causation opinions fail to meet the reliability requirement of Rule 702.

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what other exposures may have done without those may I have changed the time or the setting in which you get a mesothelioma. Some of my own research point out that additional exposures increased cell division so if you leave out one exposure or another, maybe this cells don't divide and the diseases show up a bit later but one has to say that whatever exposures one has when one gets disease was contributory.") (emphasis added).

[15] *See id.* at 65:24–68:24.

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If an expert's testimony is scientific in nature, then the trial court must determine its reliability pursuant to the factors set forth in *State v. Council*, 335 S.C. 1, 19, 515 S.E.2d 508, 517 (1999).[16] *Graves*, 401 S.C. at 74, 735 S.E.2d at 655. There can be little doubt that testimony that there is no safe level of exposure to asbestos or that "every exposure" to asbestos causes mesothelioma is scientific in nature; therefore, the Court must look to the following factors:

(1) the publications and peer view of the technique;

(2) prior application of the method to the type of evidence involved in the case;

(3) the quality control procedures used to ensure reliability; and

(4) the consistency of the method with recognized scientific laws and procedures.

Id.; *Council*, 335 S.C. at 19, 515 S.E.2d at 517.

The "no safe level/every exposure/cumulative exposure" opinion offered by Dr. Frank does not satisfy *any* of the *Council* factors because (1) it is neither published nor peer reviewed; (2) it has been rejected by other courts when plaintiffs have attempted to apply it to the type of evidence involved here; (3) there are no quality control procedures or other methods used to ensure reliability; and (4) the method is wholly inconsistent with recognized scientific laws and procedures. *See Yates v. Ford Motor Co.*, 113 F. Supp. 3d 841, 846 (E.D.N.C.) (rejecting the "each and every exposure" theory as unreliable because it "cannot be tested, has not been published in peer-reviewed works, and has no known error rate"). In fact, this theory is not a "method" at all, but rather an end run around the Plaintiffs' burden of proof.

[16] Although these are sometimes referred to as the *Jones* factors (or the *Jones* standard), from *State v. Jones*, 273 S.C. 723, 259 S.E.2d 120 (1979), the Supreme Court of South Carolina articulated these factors in the *Council* decision.

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This opinion is seductive in its simplicity. Now that the jury has heard it, the jury will be unlikely to engage in any critical assessment of the exposure and medical evidence adduced to assess causation, as it is required to do under South Carolina law. Rather, the jury likely will accept Dr. Frank's invitations and short circuit any meaningful deliberations as to specific causation. *See Yates*, 113 F. Supp. 3d at 850 (E.D.N.C.) (excluding opinions similar to Dr. Frank's and explaining "given the potential persuasiveness of expert testimony, proffered evidence that has a greater potential to mislead than to enlighten should be excluded"). Therefore, this opinion testimony should be stricken[17] and given no weight in the Court's causation analysis, in line with the countless number of courts across the country who have considered and rejected this opinion in differing forms.[18]

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[17] In addition to the arguments set forth herein, these Defendants hereby also refer the Court back to their Motion *in Limine* and Motion to Strike this testimony, which were made with the Court prior to and contemporaneously with Dr. Frank offering the above testimony.

[18] *See, e.g.*, *Vedros v. Northrop Grumman Shipbuilding, Inc.*, 119 F.Supp.3d 556, 560 n.2 (E.D. La. 2015) (rejecting this theory and noting that "[t]he theory that Defendants refer to [as the "'Every Exposure theory'"] has other names, including 'No Safe Level of Exposure,' 'Each and Every Exposure,' 'Any Exposure,' and 'Single Fiber.' All theories have as their basis the principle that all exposures to asbestos should be included as a cause of mesothelioma because there is no way to know which exposures caused it and which ones did not."); *Bell v. Foster Wheeler Energy, Corp.*, No. 15-6394, 2016 U.S. Dist. LEXIS 138817, at *11 (E.D. La. Oct. 5, 2016) ("The Court sees no material difference between the 'every exposure' theory and the 'every significant exposure' theory.") (citing *Vedros*); *Krik v. Crane Co.*, 76 F. Supp. 3d 747, 750–51 (N.D. Ill. 2014) ("Any Exposure," "Each and Every Exposure," "Single Fiber," and "no safe level of exposure" are all different "monikers" for the same theory); *Anderson v. Ford Motor Co.*, 950 F. Supp. 2d 1217, 1225 (D. Utah 2013) ("[A] growing number of courts have determined that the ["no safe level/every exposure"] theory is not proper under Daubert and Rule 702."); *Smith v. Ford Motor Co.*, No. 2:08–cv–630, 2013 U.S. Dist. LEXIS 7861, at *6 (D. Utah Jan. 18, 2013) (concluding that the "no safe level/every exposure" theory does not hold up under careful examination, noting that "[i]t is questionable whether it can even properly be called a theory, inasmuch as a theory is commonly described as a coherent collection of general propositions used to describe a conclusion, and while there are some general propositions used by [plaintiff's expert], they fall far short of supporting the legal liability he attempts to reach with them."); *Suoja v. Owens-Illinois, Inc.*, 211 F. Supp. 3d 1196 (W.D. Wis. 2016) (rejecting the "cumulative exposure" theory, also described as the "no safe level" theory or "every exposure contributes" theory, and concluding the opinion was "no different from the 'any exposure' theory" or from the impermissible conclusion that "if there

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In one recent federal case, *Smith v. Ford Motor Co.*, the court held that even if a plaintiff's expert's "no safe level/every exposure" theory were deemed admissible under Rule 702, it would exclude the testimony pursuant to Rule 403 because "the probative value of such unsupported speculation by [the expert] is substantially outweighed by the danger of unfair prejudice, as well as being confusing, and presenting a danger of misleading the jury." No. 2:08–cv–630, 2013 U.S. Dist. LEXIS 7861, at *6 (D. Utah Jan. 18, 2013). In reaching this conclusion, the court noted its agreement with "the growing number of published opinions from other courts that have reached a similar result: that the every exposure theory as offered as a basis for legal liability is inadmissible speculation that is devoid of responsible scientific support." *Id.* Similarly, in the recent case of *Rockman v. Union Carbide Corp.*, C.A. No. RDB-16-1169, at 8–22 (D.Md. July 17, 2017), a Maryland federal district court excluded the "each and every exposure" and "cumulative exposure" opinions of two experts—including Dr. Frank—and granted summary judgment for the defendants on the ground that the plaintiffs failed to establish causation.

In line with this notion, the Honorable David C. Norton of the District of South Carolina also recently excluded expert opinions almost identical to Dr. Frank's opinions in this case. *See Haskins v. 3M Co., et al.*, No. 2:15-cv-02086-DCN, D.E. 312 (D.S.C. July 21, 2017).[19] In *Haskins*,

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is exposure, then there is causation"); *Comardelle v. Pa. Gen. Ins. Co.*, 76 F. Supp. 3d 628, 634 (E.D. La. 2015) ("Although there may be no known safe level of asbestos exposure, this does not support [plaintiff's expert's] leap to the conclusion that therefore every exposure Comardelle had to asbestos must have been a substantial contributing cause of his mesothelioma. The Court agrees that this 'is not an acceptable approach for a causation expert to take.'"); *Sclafani v. Air & Liquid Sys Corp.*, 14 F. Supp. 3d 1351, 1356 (C.D. Cal. 2014) (noting that the "'every exposure' theory could not be tested and had not been published in any peer-reviewed literature [and] therefore failed to satisfy at least two of the four Daubert criteria").

[19] The case is a combination of two different cases, each with its own caption. The companion case is *Chesher v. 3M Co., et al.*, No. 2:15-cv-02086-DCN, D.E. 312 (D.S.C. July 21, 2017). This brief will refer to the case as *Haskins*.

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an expert espoused the "cumulative exposure" theory: "each of the defendants' products which contained asbestos added to the total cumulative dose of Chesher's asbestos exposure." *Id.* at 4. The *Haskins* expert's opinions, as described by the court, closely mirrored Dr. Frank's opinions in this case. *Id.* at 11–13 ("Bedrossian asserts that an individual's 'total cumulative dose' is the 'best indicator' of an individual's risk of developing mesothelioma and views the 'total cumulative dose' as the 'cause' of [the plaintiffs'] injuries. . . . Bedrossian further explains that mesothelioma may result from very small exposures, citing a number of authorities that have concluded that 'there is no safe level of asbestos exposure.' . . . [B]ecause [the plaintiffs'] exposures to defendants' asbestos-containing products were—by definition—part of their respective 'cumulative doses,' such exposures significantly contributed to their development of mesothelioma. . . . [I]t appears that Bedrossian assumes that, because every 'occupational' exposure contains exponentially more fibers than any background exposure, every 'occupational' exposure significantly contributes to the total cumulative dose. . . . *Thus, in Bedrossian's view, whenever the total cumulative dose results in mesothelioma, every 'occupational' exposure should be considered causative, no matter how small.*") (emphasis added).

Judge Norton found the issue of Bedrossian's opinions "requires very little analysis" because "Bedrossian's opinions evaluate causation in a manner that is inconsistent with the appropriate legal standard."[20] *Id.* at 14–15. Therefore, the court found the opinions were "essentially irrelevant, and any probative value they may have is easily outweighed by their tendency to confuse or mislead the jury" under rule 403. *Id.* at 15. The court further explained,

> [F]or Bedrossian's opinion to have any legal significance, the court must accept the proposition that each exposure can be considered a "substantial cause" of

[20] Although the court applied maritime law, it found there "does not appear to be any significant difference between the specific causation requirements of maritime law and the *Lohrmann* test." *Id.* at 13 n.7.

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> mesothelioma if it could have independently caused the disease. But this approach would be problematic from both a factual and legal perspective. Every exposure to asbestos *could* cause mesothelioma, but lower levels of exposure carry lower risk. Therefore, Bedrossian's logic quickly devolves into the sort of "every exposure" liability that was specifically reject in *Lindstrom* [*v. A-C Prod. Liab. Trust*, 424 F.3d 488 (6th Cir. 2005)], and leads to the bizarre conclusion that *any* exposure that carries *any* chance of causing mesothelioma—however miniscule—constitutes a "substantial cause," regardless of the other exposures that may have contributed to the total cumulative exposure.

Id. at 16–17. The court further emphasized that Bedrossian "showed very little knowledge of, or interest in, [the plaintiffs'] actual exposures to defendants' products." *Id.* at 18 n.11. Instead, he focused solely on the "no safe level of exposure" theory. *Id.* The court therefore was "convinced that Bedrossian's opinions are not based on a meaningful, defendant-specific analysis of intensity, frequency, and regularity of [the plaintiffs'] exposures to defendants products." *Id.* The court found Bedrossian's opinions could not be used to support a finding of causation and excluded the opinions. *Id.* at 20.

Like Bedrossian's opinions in *Haskins*, Dr. Frank's opinions are not based on any analysis of causation specific to Fisher or Crosby. Instead, they rely solely on the idea that any miniscule exposure to asbestos in association with Fisher or Crosby's products contributed to the cumulative result of Mr. Jolly contracting mesothelioma and, therefore, the exposure was a cause of Mr. Jolly's injury. These opinions are inconsistent with the appropriate legal standard and are useless to a jury. Instead, they will serve merely to confuse or mislead the jury as to the standard it should apply to Mr. Jolly's claims. Accordingly, the opinions do not comport with Rule 403 and should be excluded and/or stricken from the record. Without Dr. Frank's opinions, Plaintiffs have presented no evidence of specific causation, and Fisher and Crosby are entitled to a directed verdict.

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B. Plaintiffs Also Cannot Prove Causation Under the *Henderson/Lohrmann* Standard.

Plaintiffs have also failed to prove causation pursuant to the "frequency, regularity, and proximity" standard. In *Henderson v. Allied-Signal, Inc.*, 373 S.C. 179, 644 S.E.2d 724 (2007), the South Carolina Supreme Court adopted the following standard as a requirement for a plaintiff to maintain an actionable claim of asbestos exposure:

> In determining whether exposure is actionable, we adopt the "frequency, regularity, and proximity test" set forth in *Lohrmann v. Pittsburgh Corning Corp.*, 782 F.2d 1156, 1162 (4th Cir. 1986): "To support a reasonable inference of substantial causation from circumstantial evidence, there must be evidence of exposure to a ***specific product on a regular basis over some extended period of time in proximity to where the plaintiff actually worked***."

Id. at 185, 644 S.E.2d 727 (emphasis added). Moreover, after finding the plaintiff had failed to present evidence of regular and frequent exposure to asbestos-containing products in proximity to where the plaintiff worked, the *Henderson* court specifically held "presence in the vicinity of static asbestos" is not actionable exposure. *Id.*

Here, Plaintiffs have failed to show Mr. Jolly was exposed to asbestos dust from a "specific [Fisher or Crosby asbestos-containing] product on a regular basis over some extended period of time." *Henderson*, 373 S.C. at 185, 644 S.E.2d at 727. Put another way, the evidence and testimony in this case is not sufficient to satisfy the "regular," "frequent," and "proximate" exposure standard required by *Henderson* and its parent case, *Lohrmann*. *See Lohrmann*, 782 F.2d at 1163 (noting that the plaintiff "testified that he was exposed to an asbestos-containing pipe covering called Unibestos on *ten to fifteen occasions of between one and eight hours duration* during the term of his employment," and holding that "[t]he trial court correctly ruled that, viewing the evidence in the light most favorable to the plaintiff, his exposure to Unibestos *was not sufficient* to raise a permissible inference that such exposure was a substantial factor in the development of

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his asbestosis") (emphasis added); *see also Pace v. Air & Liquid Sys. Corp.*, 642 Fed. Appx. 244 (4th Cir. 2016) (affirming summary judgment under South Carolina law and *Henderson/Lohrmann*, despite the fact that one witness testified that he believed the Plaintiffs worked with the defendant's pumps "a lot," because an examination of the witness's total testimony demonstrated that this statement amounted to pure speculation). Plaintiffs do not meet their burden of proof by showing general exposure to asbestos-containing material, or general exposure to the Defendants' valves, or both. Plaintiffs must meet the *Henderson/Lohrmann* test with respect to exposure to asbestos-containing products (with the asbestos in a form that can be breathed) placed into the stream of commerce by Defendants. Plaintiffs did not meet this burden, nor did they show such exposure with respect to products required, foreseen, or "specified" (whatever the meaning of that word).

C. Plaintiffs Also Cannot Prove Causation Because the Undisputed Evidence Shows that Mr. Jolly's Mesothelioma was Caused By Other Means that Bar a Finding of Liability Against These Defendants.

Plaintiffs also cannot meet their causation burden, because the undisputed evidence demonstrates that (1) Mr. Jolly was comparatively negligent which at least exceeds fifty percent of any supposed comparative fault of the Defendants, and (2) Duke Power's decision to procure asbestos-containing gaskets and insulation for use at its power plant facilities is an intervening and superseding cause of Plaintiffs' harm.

As noted above, Mr. Jolly testified that Duke Power alerted him and his co-workers that asbestos could be dangerous from the first day that he began working for Duke Power in 1979. Despite that warning and his understanding of the potential hazards, Mr. Jolly continued to work with or around asbestos-containing products at the facility, sometimes wearing a dust mask and sometimes not wearing a dust mask. Mr. Jolly's decision to continue working at Duke Power in

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spite of his knowledge of the hazards of asbestos places him at fault for Plaintiffs' injuries. Furthermore, because the evidence of his fault is overwhelming, Mr. Jolly's comparative fault exceeds fifty percent of the comparative fault of the defendants, and therefore, Plaintiffs are entitled to no recovery. *See, e.g.*, *Bloom v. Ravoira*, 339 S.C. 417, 424, 529 S.E.2d 710, 714 (2000) ("Where evidence of the plaintiff's greater negligence is overwhelming, evidence of slight negligence on the part of the defendant is simply not enough for a case to go to the jury.") (citing *Hopson v. Clary*, 321 S.C. 312, 314, 468 S.E.2d 305, 307 (Ct. App. 1996)).

Additionally, as noted above, documentary evidence entered in this case demonstrates that Duke Power knew of the hazards of asbestos, yet it continued to purchase and utilize asbestos-containing products (including insulation, gaskets, packing, etc.) for decades. Duke Power's decision to procure asbestos-containing gaskets and insulation for use in its facilities is a superseding cause of Plaintiffs' alleged harm, and because these Defendants could reasonably expect Duke Power to protect its employees, these Defendants could not reasonably have anticipated or foreseen that superseding cause. Duke Power's actions therefore break the causal chain with respect to any purported negligence or product defect of these Defendants, relieving them from liability. *See, e.g.*, *Roddey v. Wal-Mart Stores East, LP*, 415 S.C. 580, 590, 784 S.E.2d 670, 676 (2016) ("An intervening force may be a superseding cause that relieves an actor from liability, but for there to be relief from liability, the intervening cause must be one that could not have been reasonably foreseen or anticipated.") (citing *Rife v. Hitachi Const. Mach. Co.*, 363 S.C. 209, 217, 609 S.E.2d 565, 569 (Ct. App. 2005). Therefore, Plaintiffs' cannot show that Fisher and Crosby's valve design or failure to warn is the proximate cause of Plaintiffs' injuries.

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VI. Plaintiffs Have Failed To Prove Their Causes of Action For Premises Liability, Fraudulent Misrepresentation, and Loss of Consortium.

Plaintiffs' Complaint also lays out causes of action for premises liability, fraudulent misrepresentation, and loss of consortium. Plaintiffs have put forth no evidence whatsoever tending to prove a claim for premises liability or fraudulent misrepresentation. Indeed, there are no allegations (and there is certainly no evidence) that Mr. Jolly was injured as the result of being on the Defendants' premises, nor are there allegations that the Defendants made any sort of representation (much less a fraudulent misrepresentation) to Mr. Jolly. Those claims therefore must be dismissed.[21]

Moreover, it is well settled in South Carolina that, where an injured person's claim is barred as a matter of law, his or her spouse's asserted claim for loss of consortium is also barred. *See, e.g.*, *Lee v. Bunch*, 373 S.C. 654, 663, 647 S.E.2d 197, 202 (2007) ("Generally, a plaintiff spouse's claim for loss of consortium fails if the impaired spouse's claim fails, whether the claim is considered separate and independent from the impaired spouse's claim or derivative in nature.") (quoting 41 Am.Jur.2d *Husband and Wife* § 227 (2007) and citing *Daves v. Cleary*, 355 S.C. 216, 234, 584 S.E.2d 423, 432 (Ct. App. 2003)). Because Mr. Jolly's underlying claims against these Defendants fail as a matter of law, as detailed herein, so too do Mrs. Jolly's claims, for the same reasons. All of above grounds for directed verdict are hereby asserted as to Mrs. Jolly's claims, as if set forth herein verbatim.

VII. Plaintiffs' Claims for Punitive Damages Must Be Dismissed.

"On the issue of punitive damages, the highest burden of proof known to the civil law is applicable." *Mellen v. Lane*, 377 S.C. 261, 290, 659 S.E.2d 236, 251 (Ct. App. 2008). "To receive

[21] Based on the jury charges submitted by Plaintiffs to the Court, it appears they have abandoned these two theories of recovery.

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an award of punitive damages, the plaintiff has the burden of proving by ***clear and convincing evidence*** the defendant's misconduct was ***willful, wanton, or in reckless disregard*** of the plaintiff's rights." *Solanki v. Wal-Mart Store No. 2806*, 410 S.C. 229, 236-37, 763 S.E.2d 615, 618-19 (Ct. App. 2014) (emphasis added).

The issue of punitive damages "may not be submitted to the jury for review" where the court determines that the defendant's conduct does not "rise[] to the level of culpability warranting a punitive damage award." *S.C. Farm Bureau Mut. Ins. Co. v. Love Chevrolet, Inc.*, 324 S.C. 149, 154, 478 S.E.2d 57, 59 (1996). Moreover, punitive damages should only be submitted to the jury where "more than one reasonable inference can be drawn from the evidence as to whether the defendant's behavior was reckless, willful, or wanton." *Solanki*, 410 S.C. at 236, 763 S.E.2d at 618.

There is "no formula or standard to be used as a measure for assessing punitive damages." *Mellen*, 377 S.C. at 290, 659 S.E.2d at 251. However, relevant factors for consideration include:

(1) the defendant's degree of culpability;

(2) the severity of the harm caused by the defendant;

(3) the extent to which the plaintiff's own conduct contributed to the harm;

(4) the duration of the conduct, the defendant's awareness, and any concealment by the defendant;

(5) the existence of similar past conduct;

(6) the profitability of the conduct to the defendant;

(7) the defendant's ability to pay;

(8) the likelihood the award will deter the defendant or others from like conduct;

(9) the awards of punitive damages against the defendant in any state or federal court action alleging harm from the same act or course of conduct complained of by the plaintiff;

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(10) any criminal penalties imposed on the defendant as a result of the same act or course of conduct complained of by the plaintiff; and

(11) the amount of any civil fines assessed against the defendant as a result of the same act or course of conduct complained of by the plaintiff.

S.C. Code Ann. § 15-32-520(E); *see also Mitchell v. Fortis Ins. Co.*, 385 S.C. 570, 586-87, 686 S.E.2d 176, 184-85 (2009) ("[W]e [have] identified eight considerations that trial courts should apply in conducting a post-judgment due process review of any punitive damages award. These considerations are: (1) the defendant's degree of culpability; (2) the duration of the conduct; (3) the defendant's awareness or concealment; (4) the existence of similar past conduct; (5) the likelihood the award will deter the defendant or others from like conduct; (6) whether the award is reasonably related to the harm likely to result from such conduct; (7) the defendant's ability to pay; and (8) any other factors deemed appropriate.") (citing *Gamble v. Stevenson*, 305 S.C. 104, 111-12, 406 S.E.2d 350, 354 (1991)).

South Carolina appellate courts have repeatedly approved of the trial court's grant of a directed verdict where a claim for punitive damages fails to meet this high burden. *See, e.g.*, *Cartee v. Lesley*, 290 S.C. 333, 337–38, 350 S.E.2d 388, 390 (1986) ("Here, the trial judge correctly directed a verdict for the trustees on the issue of punitive damages."); *Leonard v. Broad River Power Co.*, 174 S.C. 199, 177 S.E. 152, 154 (1934) ("There is no merit in the contention that the court erred in directing a verdict for the defendant as to punitive damages."); *Mendelsohn v. Whitfield*, 312 S.C. 17, 25-26, 430 S.E.2d 524, 530 (Ct. App. 1993), *aff'd in part as modified*, 312 S.C. 226, 439 S.E.2d 845 (1994) ("Since Whitfield failed to prove claims for fraud and conspiracy as a matter of law, he was not entitled to recover punitive damages on those claims. . . . Accordingly, the granting of a directed verdict on the issue of punitive damages was not prejudicial error.").

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Here, Plaintiffs cannot recover punitive damages as to any of their theories of liability. First, punitive damages are not recoverable under a theory of strict liability. *Barnwell v. Barber-Colman Co.*, 301 S.C. 534, 536-37, 393 S.E.2d 162, 163 (1989). Rather, damages for a strict liability claim are limited to actual damages, "which compensate 'for physical harm caused.'" *Id.* (quoting *Laird v. Nationwide Ins. Co.*, 243 S.C. 388, 134 S.E.2d 206 (1964)). Additionally, punitive damages are also not recoverable in connection with a breach of warranty claim. *Rhodes v. McDonald*, 345 S.C. 500, 504, 548 S.E.2d 220, 222 (Ct. App. 2001). Therefore, both of these avenues are foreclosed for Plaintiffs' attempt to recover punitive damages.

Furthermore, Plaintiffs cannot recover punitive damages on their negligence theory because, as detailed above, that claim fails as a matter of law. Plaintiffs have not shown how Crosby or Fisher deviated from the standard of care. Therefore, because this claim fails, it cannot form the basis of a claim for punitive damages either. *Cf. Mendelsohn v. Whitfield*, 312 S.C. 17, 25–26, 430 S.E.2d 524, 530 (Ct. App. 1993) (noting that where a defendant was exonerated from malpractice and conversion claims, the plaintiff "could not recover punitive damages on those claims").

Finally, it is unconstitutional to award punitive damages against Fisher and Crosby based on a comparative fault scheme that does not permit the jury to fully apportion fault between and among all responsible persons, regardless of whether they are (or were) defendants in this case. *See, e.g.*, *Willow Inn, Inc. v. Pub. Serv. Mut. Ins. Co.*, 399 F.3d 224, 233–34 (3d Cir. 2005) ("[T]he ratio of punitive damages to the harm caused by the defendant is a tool to ensure that the two bear a reasonable relationship to each other."); *Searcy v. R.J. Reynolds Tobacco Co.*, No. 3:09-CV-13723, 2013 WL 5421957, at *7 (M.D. Fla. Sept. 12, 2013) ("[A]lthough Defendants are jointly

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and severally liable for the amount of the compensatory damages, this Court must assess the punitive damages award against each Defendant separately.").

CONCLUSION

For the foregoing reasons, Fisher and Crosby respectfully request that this Court grant their motion for a directed verdict as to all of Plaintiffs' claims.

Respectfully submitted,

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July 31, 2017

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STATE OF SOUTH CAROLINA **COUNTY OF SPARTANBURG** **BEVERLY DALE JOLLY and BRENDA RICE JOLLY,** **Plaintiffs,** **v.** **CROSBY VALVE, LLC and FISHER CONTROL INTERNATIONAL, LLC, et al.** **Defendants.**	))))))))))))))))	**IN THE COURT OF COMMON PLEAS FOR THE SEVENTH JUDICIAL CIRCUIT** **C/A No. 2016-CP-42-1592** **PLAINTIFFS' MOTION FOR NEW TRIAL *NISI ADDITUR***

Plaintiffs Beverly Dale Jolly and Brenda Rice Jolly hereby move the Court for a New Trial *Nisi Additur*, and would respectfully show the Court that the damages awarded by the jury are inadequate and that compelling reasons support an increase of the damages award in accordance with the evidence presented at trial.

INTRODUCTION

At trial, the primary issues of dispute were the extent of Plaintiff Dale Jolly's exposure to asbestos from valves manufactured by Crosby Valve ("Crosby") and Fisher Controls International ("Fisher"), and the causation of his malignant mesothelioma from that exposure. The jury resolved those issues in Plaintiffs' favor, finding that both Crosby and Fisher were negligent and breached implied warranties and that their wrongful conduct was a substantial factor in causing Mr. Jolly's mesothelioma. **Ex. 1**, Verdict Form. While liability was hotly contested, the evidence of the Jollys' damages was not disputed in any way. The expert testimony regarding Mr. Jolly's medical expenses was never contradicted, nor was the evidence of his pain and suffering, loss of enjoyment of life, and mental anguish, and Mrs. Jolly's loss of consortium. In fact, when pressed, even Defendants' medical causation expert, Dr. James Crapo,

confirmed the horrific nature of Mr. Jolly's anticipated pain, suffering, and other significant non-economic damages.

The Court should find that the jury's damages award of $300,000 is inadequate, and that compelling reasons warrant increasing the award to at least $2.5 million. The Jollys' economic and noneconomic damages, and the fact that those damages arose from Mr. Jolly's mesothelioma diagnosis, were *completely undisputed* issues in this case. The award to Mr. Jolly should be increased from $200,000 to at least $2,000,000. The undisputed evidence was that his past and future medical expenses would be $1 million. The jury's award of $200,000 to Mr. Jolly did not even cover his medical expenses, much less his pain and suffering and other noneconomic damages. Mr. Jolly should be awarded $1 million in economic damages, as well as at least $1 million in noneconomic damages given the record establishing that he has lived in pain for a year and a half, endured major invasive surgery, and that, according to experts on both sides, is expected to die a very painful death from mesothelioma.

The award to Mrs. Jolly should be increased from $100,000 to at least $500,000. Plaintiffs asked the jury to award Mrs. Jolly half of the amount awarded to Mr. Jolly, which they did. She has been his caregiver for the past year and a half, at the expense of her own health. The expert testimony is that Mr. Jolly will pass away within a year, which is ten years short of his life expectancy. Mrs. Jolly's losses are therefore very significant, and not adequately compensated by the jury's relatively small award.

Therefore, for the reasons set forth herein, Plaintiffs ask the Court to grant a new trial *nisi additur* and increase the verdict to at least $2.5 million.

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STATEMENT OF FACTS

Testimony bearing on damages was offered by Plaintiffs Dale and Beverly Jolly, their daughter, Tracy Pavlish, Plaintiffs' expert Dr. Arthur Frank, and defense expert Dr. James Crapo.

Mesothelioma Diagnosis, Treatment, and Prognosis

It is undisputed that Mr. Jolly suffers from malignant mesothelioma. (**Ex. 3**, TT 7/26, at 95, 96; **Ex. 4**, TT 8/1 at 82, 149; **Ex. 5**, TT 8/2 at 187, 190).[1] He was diagnosed with mesothelioma in January 2016, at the age of 71. (**Ex. 2**, TT 7/25 at 58-59; TT 7/26 at 100). He initially presented with shortness of breath and fluid in his lungs. (TT 7/26 at 96). The fluid had to be drained from his lungs, and then he had a biopsy of his tissue in order for the doctors to make a diagnosis. (TT 7/26 at 96). The tumor was so large at that time that Mr. Jolly underwent three rounds of chemotherapy to shrink the tumor. (TT 7/25 at 159; TT 7/26 at 96-97). The chemotherapy made his feet swell so much he could not walk, he lost a lot of weight, and he became very fragile and depressed. (TT 7/25 at 159, 175). He was nauseous and could not really eat. (TT 7/25 at 175).

In June 2016, Mr. Jolly had major surgery to remove the mesothelioma tumor and the lining of his lung. (TT 7/25 at 145; TT 7/26 at 97, 102). The surgery involved taking out one of his ribs and spreading his rib cage apart to scrape out the tumor. (TT 7/26 at 97, 102). His daughter testified that he was in "unbelievable" pain after the surgery. (TT 7/25 at 160). He was released from the hospital with a drainage tube still located in his back. (TT 7/25 at 160; TT 7/26 at 97, 102-03).

[1] All cited trial transcripts are the daily (rough) transcripts. The transcripts are cited as "TT," followed by a designation of the date and page number.

Within two days of his release from the hospital, Mr. Jolly fell and had to be re-admitted because he had blood poisoning. (TT 7/25 at 160; TT 7/26 at 97). He was so weak from the surgery and the complications that he had to go to a rehabilitation facility. (TT 7/25 at 161). He was in a lot of pain, had to use a walker, and could not make it to the bathroom by himself. (TT 7/25 at 161). He also had to use an oxygen tank. (TT 7/25 at 161). Mr. Jolly went to respiratory rehabilitation and with a lot of hard work he recovered enough to walk and breathe without assistance. (TT 7/25 at 161-62).

When he testified, Mr. Jolly was visibly weak and was having trouble breathing. He testified that he has lost 45 pounds. (TT 7/25 at 60). Four months ago, he found out that the tumor was back. (TT 7/25 at 145). His daughter said he was "devastated" by this news after all he has been through. (TT 7/25 at 162). He is now on an experimental drug therapy because he has no other treatment options. (TT 7/25 at 145, 162; TT 7/26 at 100). Mr. Jolly said that the pain and difficulty of this treatment "brings you to your knees." (TT 7/25 at 145). Mrs. Jolly testified that the treatment really wears him out and he has no strength. (TT 7/25 at 174). He does not like to take pain medication but has started to take it in the last month or so to give himself some strength. (TT 7/25 at 167-68).

Experts on both sides of this case described mesothelioma as an unusually painful disease. (TT 7/26 at 98; TT 8/1 at 150-53). Defense expert Dr. Crapo testified:

> Q. As a typical clinical course mesothelioma is [characterized] by significant pain?
>
> A. It's common yes [to] have bad pain.
>
> Q. [T]he closer one gets as the disease progresses, the pain is such that narcotic, heavy doses of narcotic medication is necessary to try to control that pain?

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A. Yes that's a very common experience.

Q. A very common experience with mesothelioma is significant breathlessness correct?

A. That's also correct.

(TT 8/1 at 150).

It is undisputed that Mr. Jolly will more likely than not die from mesothelioma. (TT 7/26 at 99; TT 8/2 at 190). Dr. Frank testified that Mr. Jolly would likely die in a year or less. (TT 7/26, at 99-100).

Defense expert Dr. Crapo repeatedly described dying from mesothelioma as very difficult:

Q. Mesothelioma, as the disease progresses, the tumor characteristically grows into what should be air space?

A. That's common that it does extend into the lung.

Q. And death by suffocation is character[is]tic for some people if something else doesn't kill them first correct?

A. It's actually not the usual cause -- well there are many causes of death but usually it's one side and they have adequate ventilation on the other side but in no way do I want to you think it's a comfortable death it's a very bad tumor.

* * *

Q. And a typical course in terms of the ways and the complications that people expire is what?

A. Usually it's a very severe disease involving lungs the ability to breathe and a[s] typical of most cancer it's a process that ca[u]ses a wasting in a classic cancer death it's not a very good death.

* * *

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Q. And you don't dispute the medical evidence that before Mr. Jolly passes away, it would be highly reasonable that he [will] have more hospitalizations correct?

A. That's very likely.

Q. Become completely incapacitated and unable to care for himself even as to a basic of daily living?

A. That's a likely outcome.

Q. He will [n]eed supplement amount oxygen?

A. That's likely.

Q. He will be in hospice which will require 24-hour a day round the clock nursing care?

A. I would say that's likely too.

Q. Among other things right?

A. It's a bad death yes.

(TT 8/1 at 151, 152, 153). All this according to *Defendants'* expert.

Both doctors indicated that Mr. Jolly will continue to require medical care until his death. (TT 7/26 at 101-02; TT 8/1 at 153). Dr. Frank testified that Mr. Jolly's shortness of breath will be constant. (TT 7/26 at 98). He will need increasing amounts of pain medication over time as the disease continues to progress. (TT 7/26 at 98). The more his condition deteriorates, the more medical care he will need toward the end of his life. (TT 7/26 at 101). He may need to be fed with a feeding tube or intravenously. (TT 7/26 at 102). He may also need hospice care. (TT 7/26 at 102).

Dr. Frank testified that all of the medical treatment received by Mr. Jolly has been medically necessary. (TT 7/26 at 100). Even before the surgery, he had medical bills of

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$142,000. (TT 7/26 at 193). The cost of the surgery alone was hundreds of thousands of dollars. (TT 7/26 at 101). Based on the extensive treatment Mr. Jolly has already had, and his expected future treatment, Dr. Frank testified that the total cost of Mr. Jolly's medical care would reasonably be $1,000,000 or more. (TT 7/26 at 101, 179).

Adverse Impact on the Jollys' Life

Mr. Jolly's life has been irreparably ruined by his mesothelioma diagnosis. At the time of his diagnosis, he was still working as an ultrasonic technician at Duke. (TT 7/25 at 59-60, 63-64). He had retired in 2002 but two months later they asked him to come back and work with his old crew. (TT 7/25 at 64, 158). He testified that he "loved" working, primarily because of the people he got to work with. (TT 7/25 at 64). He had to stop working when he became ill with mesothelioma. (TT 7/25 at 59). He could no longer climb the stairs at Duke's Catawba nuclear plant because of his shortness of breath. (TT 7/25 at 59).

When asked about how his life had changed after he got mesothelioma, Mr. Jolly became so emotional crying that he was unable to talk about it. (TT 7/25 at 107-08). He could only say that it was hard and that it was hurting him. (TT 7/25 at 108). Mr. Jolly's non-verbal display of the mental anguish of despair and hopelessness with his condition appears in a single line of the Trial Transcript at page 108. Between direct and cross examination, counsel for both parties conferred with the Court at a bench conference. While all counsel and the Court were focused on the legal issues at the bench conference, Mr. Jolly sat alone on the stand with his head in his hands weeping uncontrollably; "*Reporter's Note: He became very upset and cried." (TT 7/25 at 108:15–18).

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His daughter and his wife described the kind of active man that her father was before his diagnosis. (TT 7/25 at 169). Typically, after working all day he would come home and after dinner he would work outside in his garden or in the yard. (TT 7/25 at 158, 169). He loved to garden and kept a three-acre vegetable garden. (TT 7/25 at 153). He kept a garden for 48 years. (TT 7/25 at 169-70). His garden fed the family, their neighbors, and supplied a local meals-on-wheels program. (TT 7/25 at 170-71).

Mr. Jolly loves spending time with his grandchildren. (TT 7/25 at 155). His daughter said his grandchildren are "his life." (TT 7/25 at 155). He taught two of them to say his nickname, "Papa," as their first word. (TT 7/25 at 155).

When asked about how life had changed for Mr. Jolly and his wife after his diagnosis, their daughter said "their life came to a halt." (TT 7/25 at 156-57). Mr. Jolly was "heartbroken" by the diagnosis. (TT 7/25 at 157). He can no longer do the things that bring him joy. (TT 7/25 at 157). He cannot garden or cut grass. (TT 7/25 at 165). He cannot go to church or have friends over for dinner. (TT 7/25 at 173). They used to go to the beach for a vacation every year and they can no longer do that. (TT 7/25 at 173). When he tries to do things that he used to do, Mr. Jolly finds out that he is not physically up for it. (TT 7/25 at 165). Now when they try to go out, he just wants to come back home. (TT 7/25 at 176).

Mr. Jolly's physical appearance has changed drastically in the year and a half he has been fighting mesothelioma, and he now looks like a much older version of himself. (TT 7/25 at 157). He spends most of his time at doctor's appointments for his treatment. (TT 7/25 at 162-63, 172).

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When the cancer came back recently, Mrs. Jolly was really scared. (TT 7/25 at 162). She described herself as his caregiver as much as his wife. (TT 7/25 at 173). She has witnessed him in a lot of pain. (TT 7/25 at 167).

About two weeks before trial, Mr. and Mrs. Jolly were staying in Durham over the weekend because Mr. Jolly had medical appointments on Friday and the following Monday. (TT 7/25 at 163). While they were there, Mrs. Jolly collapsed from a heart attack (TT 7/25 at 163-64). Their daughter testified that Mrs. Jolly had not been taking care of herself because she was so focused on caring for Mr. Jolly. (TT 7/25 at 164).

Mr. and Mrs. Jolly have been married for 51 years. (TT 7/25 at 170). For years they have had a ritual where Mr. Jolly asks her if she is ready for her coffee in the morning and he brings it to her in bed. (TT 7/25 at 171). He continues to do this for her even now.

STANDARD OF REVIEW

A new trial *nisi additur* may be granted when the trial court finds the amount of the verdict to be "merely inadequate" in light of the evidence presented at trial. *Green v. Fritz*, 356 S.C. 566, 570, 590 S.E.2d 39, 41 (2003); *Vinson v. Hartley*, 324 S.C. 389, 406, 477 S.E.2d 715, 723 (Ct. App. 1996). When granting a motion for *additur*, the court must offer compelling reasons. *Green*, 356 S.C. at 570, 590 S.E.2d at 41; *Bailey v. Peacock*, 318 S.C. 13, 14, 455 S.E.2d 690, 691 (1995). "Great deference is given to the trial judge . . . [because] the trial judge who heard the evidence and is more familiar with the evidentiary atmosphere at trial possesses a better-informed view of the damages than [the appellate courts]." *Waring v. Johnson*, 31 S.C. 248, 257, 533 S.E.2d 906, 911 (2000).

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"A new trial *nisi* is one in which a new trial will be granted unless the party opposing it complies with a condition set by the court." *Waring*, 31 S.C. at 257, 533 S.E.2d at 911. The trial court has the authority to grant a new trial to the plaintiff unless the defendant agrees to the payment of an additional amount. *Id.* at 258, 533 S.E.2d at 911.

ARGUMENTS AND AUTHORITIES

I. The jury's verdict was inadequate under the evidence and the applicable law.

South Carolina courts have upheld *additur* in circumstances, like this one, where the jury did not award compensatory damages in an amount shown by the undisputed evidence. A new trial *nisi additur* is warranted when a jury has awarded medical damages for injuries caused by the defendant but has failed to consider the plaintiff's pain and suffering associated with those injuries. *See Waring*, 31 S.C. at 260, 533 S.E.2d at 912. In *Waring*, the jury only awarded the exact amount of the plaintiff's medical expenses, $23,237.28, associated with her injuries from a car wreck. The Supreme Court found that it was proper to grant a *nisi additur* of $40,000 given the evidence that the plaintiff's pain was caused by her injury and that she was unable to continue her previously active lifestyle. *Id.* at 260, 533 S.E.2d at 912; *see also id.* at 258-59, 533 S.E.2d at 911-12 (collecting South Carolina cases upholding trial court orders granting new trial *nisi additur* under the abuse of discretion standard).

Even when some award for noneconomic damages has been given, a trial court may still reasonably find the verdict to be inadequate. In *Riley v. Ford Motor Co.*, 414 S.C. 185, 188, 777 S.E.2d 824, 826 (2015), the Supreme Court held that the trial court had not abused its discretion in granting a *nisi additur* in the amount of $600,000. This was a product liability case against Ford brought by the estate of a man who was killed when his door latch failed in a car accident.

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Id. at 189, 777 S.E.2d at 826-27. The uncontradicted evidence was that the family had $228,000 in economic damages, plus a number of witnesses testified about the significant noneconomic damages suffered by his family. *Id.* at 193, 777 S.E.2d at 829. The trial court found the jury's verdict of $300,000 to be inadequate, granted a *nisi additur* of $600,000, and entered judgement for $900,000. *Id.* at 189, 777 S.E.2d at 827.

In upholding the grant of a new trial *nisi additur*, the Supreme Court clarified that a *nisi additur* may be appropriate even when the jury has awarded some amount for noneconomic damages. *Id.* at 194, 777 S.E.2d at 829-30. The trial court had articulated compelling reasons for granting the *nisi additur*, including "a thorough recitation of the 'uncontested, and emotionally compelling' evidence, including testimony and supporting exhibits that demonstrated both the pecuniary losses suffered by the Riley family and also the noneconomic compensable elements of loss that are recoverable in a wrongful death action." *Id.*; *see also Graham v. Whitaker*, 282 S.C. 393, 405, 321 S.E.2d 40, 45 (1984) (upholding new trial *nisi additur* of several times the jury verdict, increasing actual damages from $10,000 to $67,500).

Under this case law, it is clear that the Jollys' verdict is inadequate and should be increased to more accurately reflect the great extent of their losses. A fair reading of the verdict is that the jury only awarded a fraction of Mr. Jolly's medical expenses and nothing for pain and suffering. *Waring* instructs that there should be a new trial *nisi additur* in such circumstances. *See Waring*, 31 S.C. at 260, 533 S.E.2d at 912. *Riley* is also instructive, as the court upheld a *nisi additur* that tripled the jury's award from $300,000 to $900,000. 414 S.C. 185, 189, 777 S.E.2d 827. There, the economic damages were $228,000, and the trial court awarded about three times that amount in noneconomic damages. Here, Plaintiffs are only asking for a 1:1 ratio of Mr.

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Jolly's economic and noneconomic damages. Plaintiffs request the minimal amount of damages necessary to adequately compensate the Jollys for their losses.

A. Mr. Jolly's damages should be increased.

The jury was instructed that, if they found Crosby and Fisher liable, they should award "compensatory" or "actual" damages. (**Ex. 6**, TT 8/3 at 139). The instruction stated that, "Actual damages are designed to compensate a party for their loss and to make that party as whole as near as money can do and to put him or her in the same position they were in before the accident or incident occurred. Actual damages would be the actual losses and expenses the Plaintiffs have incurred." (TT 8/3 at 139).

The jury was instructed on four categories of actual damages – medical expenses, pain and suffering, loss of enjoyment of life, and mental anguish. (TT 8/3 at 139-40). The jury was also instructed that future damages should be awarded for the duration of Mr. Jolly's life expectancy, which is a little more than eleven (11) years given his age of 73. (TT 8/3 at 141-42).

The evidence supported damages in all of these categories, and for future damages. The jury's award of only $200,000 was not sufficient to make Mr. Jolly whole for the magnitude of his losses resulting from his mesothelioma diagnosis, treatment, and expected death.

1. Medical expenses – past and future.

The jury was instructed that the Plaintiffs could recover for past and future medical expenses:

> Actual damages include several areas recognized by the law. In person[al] injury actions, the Plaintiffs may recover for the necessary and reasonable expenses caused by the injury[, s]uch [as] amounts necessarily incurred for medical treatment. A person who suffers personal injuries because of the misconduct of another is entitled to recover the reasonable value of medical care and expenses incurred for the treatment of the injuries to the time of trial as well as those reasonably certain to be incurred in the future. Accordingly, a Plaintiff may

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> recover amounts incurred for past and future medical care, including hospital[,] physician [and] nursing care costs[,] medicine [and] other out-of-pocket expenses such as vocational[,] physical or other rehabilitation efforts.

(TT 8/3 at 139-40).

The Court's instructions reiterated that future damages should be awarded if supported by the evidence:

> Now in determining the amount of compensation for someone personally injured by another misconduct it's proper to consider past present and future aspects of the injury the injured party may recover for those future damages that are reasonably certain to result. The principal underlying compensation is that only one action can be brought and therefore only one recovery had so it may be depending on your view of the evidence proper to include the estimate of future damages, future medical expenses and pain and suffering that will with reasonable certainty result. The plaintiff is never entitled to recover speculative damages, that is damages through arrive at by guesswork. The plaintiff has the burden of proving the existence and the causation and the amount of damages which he does not have to prove to a mathematical certainty. Instead the plaintiff must produce evidence sufficient to allow to you make a reasonable approximation of damages and the plaintiff must prove their damages to a reasonable degree of certainty. Any future damages you may award must be reasonably calculated to a result of the – reasonably calculated to be the result of an alleged injury or damage sustained and they must be reduced to their present cash value. I will charge you that in this case Mr. Jolly is a 73-year old male with a life expectancy according to the South Carolina code of 11.39 years. This is a factor you may consider along with any other facts and circumstances that may bear on his health[,] long[evity,] and occupations and [habits] of life.

(TT 8/3 at 141-42).

As noted, Dr. Frank testified that all of the medical treatment received by Mr. Jolly was medically necessary. (TT 7/26 at 100). Dr. Frank testified, *without dispute*, that the total cost of Mr. Jolly's past and future medical care, from the time of his diagnosis to the time of his death, would reasonably be $1,000,000 or more. (TT 7/26 at 101, 179). This undisputed testimony took into account some of Mr. Jolly's past medical bills of $142,000, plus the cost of his surgery that was hundreds of thousands of dollars. (TT 7/26 at 101, 193).

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The jury heard evidence that Mr. Jolly is currently undergoing an experimental therapy that requires him to go for treatments and doctor visits several times a week. (TT 7/25 at 145, 162-63, 172; TT 7/26 at 100). Experts on both sides agreed that Mr. Jolly's would likely die from mesothelioma and that his medical needs would increase as he got sicker and closer to death. (TT 7/26 at 101-02; TT 8/1 at 153).

Given this undisputed evidence of Mr. Jolly's past and future medical needs and expenses, the Court should increase his medical expenses award to $1 million.

2. Pain and suffering – past and future.

The jury was also instructed on pain and suffering damages:

> Pain and suffering is also recognized as an element of damages properly considered under the law. Pain and suffering compensates the plaintiff for physical discomfort and emotional response to the sensation of pain caused by the injury. There's no definite standard I can give you to determine the amount of damages[.] [Y]ou the jury will of to use your calm and reasonable judgment to determine them in the light[] of your even experiences and in the light of the testimony and evidence presented in this case.

(TT 8/3 at 140).

It would appear that the jury awarded nothing for Mr. Jolly's pain and suffering, even though the evidence established that his pain has been extreme in the past year and a half and will only get worse as his health continues to decline. There was evidence that after his diagnosis Mr. Jolly suffered greatly through three rounds of chemotherapy, and that the surgery that involved removing his rib and scraping out the tumor was really bad. (TT 7/25 at 145, 159-60, 175; TT 7/26 at 96-97, 102). He lost 45 pounds. (TT 7/25 at 60). His recovery from the surgery required months of rehabilitation to help him regain his ability to walk and to breath without oxygen. (TT 7/25 at 161-62). Now, he is on an experimental treatment that he said "brings you to your knees." (TT 7/25 at 145). He has to take pain medication to get through the day. (TT 7/25 at

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167-68). Dr. Frank and defense expert Dr. Crapo both confirmed that the pain associated with mesothelioma is quite bad. (TT 7/26 at 98; TT 8/1 at 150-53).

This is not to mention Mr. Jolly's expected death, which Dr. Crapo described at length as "in no way . . . a comfortable death [because it is] a very bad tumor," "not a very good death," and "a bad death." (TT 8/1 at 151, 152, 153). Mr. Jolly will continue to waste away and by the end of his life will likely require feeding by a feeding tube or intravenously, and will be on hospice care. (TT 7/26 at 102).

The inordinate and prolonged pain Mr. Jolly has suffered, and will continue to suffer until his death, warrants a significant damages award. Together with his loss of enjoyment of life and his mental anguish, discussed below, noneconomic damages should be at least $1 million.

3. Loss of enjoyment of life – past and future.

The jury received the following instruction on loss of enjoyment of life: "Another form of damages is loss of enjoyment of life. [T]his compensates the plaintiff for limitation on the ability to participate in and derive pleasure from normal activities of daily life." (TT 8/5 at 140).

The evidence of Mr. Jolly's loss of enjoyment of life is quite compelling. The evidence is that he was a very healthy, active person prior to his diagnosis and that because of his mesothelioma he can no longer do any of the things that bring him joy. (TT 7/25 at 157). The testimony was that his life "came to a halt" after his diagnosis. (TT 7/25 at 156-57). He was still working at Duke at the time of his diagnosis, at the age of 71, because he "loved" his work. (TT 7/25 at 64). He had to stop because of his illness. (TT 7/25 at 59). He used to keep a vegetable garden and spend a lot of time gardening and mowing the grass, which he is not up for anymore. (TT 7/25 at 165). He cannot go to church or have friends over for dinner. (TT 7/25 at 173). They

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used to go to the beach for a vacation every year and they can no longer do that. (TT 7/25 at 173).

Given this evidence of the substantial decline in the quality of Mr. Jolly's life, and his inability to do the things in life he really loves to do, the jury's award was inadequate for failing to award any damages for this category of loss.

4. Mental anguish – past and future.

The jury was also instructed on damages that can be awarded for mental anguish:

> Another category is mental suffering that is compensation for shock, fright[,] anxiety[,] emotional upset. Actual damage for the plaintiff who has been physically injured include pain and suffering, both past and future as well as mental anguish. Mental anguish is more than mere disappointment or anger or worry or embarrassment or resentment. Although it may include all of those it also includes mental sensation of pain resulting from painful emotions such as grief[,] wounded pride[,] shame[,] and humiliation. It may also be composed of fright[,] nervousness[,] grief[,] and anxiety and worry for [an ordeal].

(TT 8/3 at 140-41).

The jury did not award damages for mental anguish even though these losses were undisputed. As mentioned supra, Mr. Jolly's non-verbal display of the mental anguish of despair and hopelessness with his mesothelioma appears in a single line of the Trial Transcript at page 108. Mr. Jolly was so over whelmed with despair he could not speak, therefore no testimony appears in the record describing this terrible suffering, except for a short Reporter's note. Between direct and cross examination of Mr. Jolly on July 25th, attorneys for both parties conferred with the Court at a bench conference. While all counsel and the Court were focused on the legal issues at the bench conference, Mr. Jolly sat alone on the stand with his head in his hands weeping uncontrollably, which was noted in the transcript at 108, stating simply "*Reporter's Note: He became very upset and cried." (TT 7/25 at 108:15–18).

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Furthermore, Mr. Jolly's daughter testified that he was "heartbroken" by the diagnosis, (TT 7/25 at 157), and became "depressed" after going through chemotherapy. (TT 7/25 at 159, 175). She explained that he was "devastated" by the recent news that his cancer had come back. (TT 7/25 at 162). Mr. Jolly's mental anguish was evident for the jury to witness, even if not verbally, as described above, he became so choked up with tears that he could not even talk about his medical condition. (TT 7/25 at 107-08). This evidence, along with all the other evidence of his physical and mental pain and suffering and loss of enjoyment of life, supports a noneconomic damages award of at least $1 million.

B. Mrs. Jolly's damages should be increased.

Mrs. Jolly's loss of consortium damages should be increased along with Mr. Jolly's damages. The loss of consortium instructions were as follows:

> Now the plaintiff Brenda Jolly also claims loss of consortium because of the injuries received by her husband Dale Jolly. The companionship or society of a spouse are not articles of [commerce] that can be [weighed] or measured or bought and sold. No expert is competent to testify as to their value and there's no way we can go out in the marketplace to determine their market price of course. And the consideration on which they're based is not monetary but the plaintiff is entitled to compensation and money for their loss. Loss of consortium includes afforded services provided by the other spouse[,] love[,] [companionship,] affection[,] society[,] sexual relations[,] Comfort[,] [solace,] guidance. Plaintiff is entitled to reco[ver] the value of [those] services of [her] []spouse which were lost including the loss of her spouse'[s] society and [companion]ship in her home and for any expenses for the care and treatment of her spouse.

(TT 8/3 at 142).

Mrs. Jolly has substantial loss of consortium damages—both past and future—as a result of Mr. Jolly's mesothelioma. Mr. and Mrs. Jolly have been married for 51 years. (TT 7/25 at 170). She has turned into Mr. Jolly's caregiver and has witnessed him in a lot of pain. (TT 7/25 at 167, 173). As noted, he cannot really do any of the things they used to enjoy together. Her

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own health has been neglected to the point that she had a heart attack a few weeks before trial. (TT 7/25 at 163-64). Her daughter described her as "really scared" by the return of the cancer. (TT 7/25 at 162). Given everything that the Jollys have endured, and that her time with Mr. Jolly will be cut short by at least ten years, the jury's award of $100,000 in loss of consortium damages is inadequate to compensate Mrs. Jolly for her losses. The evidence supports an award of at least $500,000. A new trial *nisi additur* should therefore be granted to raise the loss of consortium damages to at least this amount.

II. The jury's verdict was inadequate in comparison to awards in comparable cases.

When considering a new trial *nisi*, it is proper to compare the jury's verdict to damages awards in comparable cases. *See Lucht v. Youngblood*, 266 S.C. 127, 136, 221 S.E.2d 854, 858 (1976); *see also Kapuschinsky v. U.S.*, 259 F. Supp. 1, 8 (D.S.C. 1966) ("Admittedly not controlling, but worthy of note are treatments of verdicts from all over this country").

It is particularly helpful to compare the jury verdict in this case with that in *Garvin v. Agco Corp.*, No. 2012-CP-40-6675 (S.C. Ct Comm. Pleas), a mesothelioma case tried before the Honorable D. Garrison Hill in Richland County in 2013. **Ex. 7**. The plaintiff in that case, Lloyd Garvin, was 74 years old at the time of trial, with a life expectancy of almost 11 years. *Id.* at 38. There were a number of facts that made his damages less compelling than Mr. Jolly's, however. He was not working at the time of his diagnosis, and unlike Mr. Jolly, he did not enjoy good health prior to his diagnosis. *Id.* Mr. Garvin remained cancer-free after surgery to remove the tumor, and because he had a very rare type of mesothelioma the evidence was that there was a 50% chance that the mesothelioma would never return and he would not have any further medical expenses. *Id.* at 4. His past medical expenses were only $149,000, and any possible

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future medical expenses were not expected to exceed $150,000 to $300,000. *Id.* at 38. Judge Hill noted that Mr. Garvin's total economic damages were no more than $300,000 to $450,000. *Id.*

Despite these facts, the jury awarded Mr. Garvin $10 million in compensatory damages and his wife $1 million in loss of consortium damages. *Id.* at 4-5. Judge Hill granted a new trial *nisi remitter* and reduced Mr. Garvin's award to $1.5 million (the loss of consortium award was not challenged). *Id.* at 38-39. In so doing, Judge Hill reasoned that, "based upon Mr. Garvin's pecuniary damages, the nature of his injury, his treatments, his age, and his life expectancy, compelling reasons justify reducing his actual damages award from $10 million to $1.5 million." *Id.* at 39.

Judge Hill's analysis in the *Garvin* matter indicates that a *nisi additur* in this case is warranted. Even though Mr. Garvin's damages were not nearly as extreme as Mr. Jolly's, the court settled on a compensatory award of $1.5 million, more than $1 million of which was for noneconomic damages. Even assuming the maximum future medical expenses, the noneconomic portion of the award was more than twice the economic. Here Plaintiffs are only asking for a 1:1 ratio between Mr. Jolly's medical expenses of $1 million and his noneconomic damages of $1 million. Further, Plaintiffs are asking for only half of what Mrs. Garvin received in loss of consortium damages although, again, at the time of trial the Garvins did not have quite the same level of damages as the Jollys.

Plaintiffs' request is on the low end of pain and suffering awards in mesothelioma cases nationally. Damages awards for pain and suffering in comparable mesothelioma cases range from $1.5 million to more than $20 million. *See, e.g., Bobo v.* Tennessee Valley Authority, 855 F.3d 1294 (11th Cir. 2017) (affirming in relevant part the district court's award of $3 million in

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pain and suffering damages for woman with asbestos-related mesothelioma); *Romano v. Metropolitan Life Ins. Co.*, --So.3d --, 2017 WL2265424 (La. Ct. App. 2017) (increasing pain and suffering award from $500,000 to $1.5 million in mesothelioma case where plaintiff had endured invasive surgery and was expected to die from his disease); *John Crane, Inc. v. Linkus*, 190 Md. App. 217, 988 A.2d 511 (Md. Ct. App. 2010) (upholding $15,335,274 verdict for living shipyard worker diagnosed with mesothelioma at the age of 70, of which $335,000 was medical expenses and $15 million was noneconomic loss); *In re New York City Asbestos Litigation (Re D'Ulisse)*, 16 Misc.3d 945, 842 N.Y.S.2d 333 (N.Y. Sup. Ct. 2007) (denying motion to reduce award of $20 million in past and future pain and suffering to mesothelioma victim and $5 million to his wife of 51 years); *Williams v. CSX Transp., Inc.*, 176 N.C.App. 330, 626 S.E.2d 716 (N.C. Ct. App. 2006) (affirming verdict in which railroad worker with mesothelioma was awarded $7.5 million in pain and suffering damages); *Goede v. Aerojet Gen. Corp.*, 143 S.W.3d 14, 17, 27-28 (Mo. Ct. App. 2004) (upholding award of $2 million for pre-death pain, suffering and emotional distress where 43-year-old had survived one year with mesothelioma).

The South Carolina Supreme Court has held that the goal of compensatory damages is "to restore the injured party, as nearly as possible through the payment of money, to the same position he or she was in before the wrongful injury occurred." *Clark v. Cantrell*, 339 S.C. 369, 378-379, 529 S.E.2d 528, 533 (2000). The jury's $300,000 verdict in this case is not sufficient to restore the Jollys to the same position they were in before Mr. Jolly was diagnosed with mesothelioma. While no amount of money can accomplish such a restoration, the inadequacy of the jury's award should be remedied by this Court through a new trial *nisi additur*. The *additur* should increase the award to at least $2.5 million given the undisputed evidence of Mr. Jolly's

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extensive medical procedures and expenses, his extraordinary pain and emotional anguish, and his greatly shortened life expectancy.

CONCLUSION

For the foregoing reasons, Plaintiffs ask the Court to grant a new trial *nisi additur*.

Respectfully submitted,

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August 11, 2017
Dallas, Texas

CERTIFICATE OF SERVICE

I hereby certify that a true and correct copy of the above and foregoing instrument was e-filed with the Spartanburg County court's e-filing system, and thereby served electronically on all counsel of record, this 11th day of August, 2017.

/s/ *Jonathan M. Holder*
Jonathan M. Holder
Attorney for Plaintiff

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EXHIBIT 7

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FORM 4

STATE OF SOUTH CAROLINA
COUNTY OF RICHLAND
IN THE COURT OF COMMON PLEAS

JUDGMENT IN A CIVIL CASE

CASE NUMBER: 2017CP4008675

Lloyd Strom Garvin
Velda Garvin
PLAINTIFF(S)

BW/IP International Inc
Byron Jackson Pump Company
DEFENDANT(S)

FILED
2014 DEC 10 AM 11:08
JEANETTE W. McBRIDE
C.C.P. & G.S.
RICHLAND COUNTY

Submitted by: _____ Attorney for: ☐ Plaintiff ☐ Defendant or ☐ Self-Represented Litigant

DISPOSITION TYPE (CHECK ONE)

☐ **JURY VERDICT.** This action came before the court for a trial by jury. The issues have been tried and a verdict rendered.

☐ **DECISION BY THE COURT.** This action came to trial or hearing before the court. The issues have been tried or heard and a decision rendered.

☐ **ACTION DISMISSED** (*CHECK REASON*): ☐ Rule 12(b), SCRCP; ☐ Rule 41(a), SCRCP (Vol. Nonsuit); ☐ Rule 43(k), SCRCP (Settled); ☐ Other _____

☐ **ACTION STRICKEN** (*CHECK REASON*): ☐ Rule 40(j), SCRCP; ☐ Bankruptcy; ☐ Binding arbitration, subject to right to restore to confirm, vacate or modify arbitration award; ☐ Other _____

☐ **DISPOSITION OF APPEAL TO THE CIRCUIT COURT** (*CHECK APPLICABLE BOX*): ☐ Affirmed; ☐ Reversed; ☐ Remanded; ☐ Other _____

NOTE: ATTORNEYS ARE RESPONSIBLE FOR NOTIFYING LOWER COURT, TRIBUNAL, OR ADMINISTRATIVE AGENCY OF THE CIRCUIT COURT RULING IN THIS APPEAL.

IT IS ORDERED AND ADJUDGED: ☒ See attached order (formal order to follow) ☐ Statement of Judgment by the Court:

ORDER INFORMATION

This order ☐ ends ☐ does not end the case.
Additional Information for the Clerk : _____

INFORMATION FOR THE JUDGMENT INDEX

Complete this section below when the judgment affects title to real or personal property or if any amount should be enrolled. If there is no judgment information, indicate "N/A" in one of the boxes below.

Judgment in Favor of (List name(s) below)	Judgment Against (List name(s) below)	Judgment Amount To be Enrolled
		$
		$
		$

If applicable, describe the property, including tax map information and address, referenced in the order:

The judgment information above has been provided by the submitting party. Disputes concerning the amounts contained in this form may be addressed by way of motion pursuant to the SC Rules of Civil Procedure. Amounts to be computed such as interest or additional taxable costs not available at the time the form and final order are submitted to the judge may be provided to the clerk. **Note: Title abstractors and researchers should refer to the official court order for judgment details.**

Circuit Court Judge _____ **Judge Code** _____ **Date** _____

For Clerk of Court Office Use Only

This judgment was entered on the ____ day of _____, 20 ____ and a copy mailed first class or placed in the appropriate attorney's box on this 10th day of Dec., 20 14 to attorneys of record or to parties (when appearing pro se) as follows:

Theile Branham McVey
ATTORNEY(S) FOR THE PLAINTIFF(S)

Mark H. Wall
Timothy W. Bouch
Robert O. Meriwether
ATTORNEY(S) FOR THE DEFENDANT(S)

Court Reporter _____

Clerk of Court Jeanette W. McBride

SCRCP Form 4C (10/2011)

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STATE OF SOUTH CAROLINA	§	IN THE COURT OF COMMON PLEAS
	§	
COUNTY OF RICHLAND	§	
	§	C/A No. 2012-CP-40-6675
LLOYD STROM GARVIN, and	§	
VELDA GARVIN,	§	
Plaintiff,	§	
	§	
v.	§	
	§	
AGCO CORPORATION f/k/a ALLIS	§	
CHALMERS COMPANY, et al.,	§	
Defendants.	§	

2014 DEC 10 AM 11:08
JEANETTE W. McBRIDE
C.C.P. & G.S.
RICHLAND COUNTY
FILED

ORDER DENYING IN PART AND GRANTING IN PART
DEFENDANT CRANE CO.'S MOTION FOR POST-TRIAL RELIEF

In this asbestos case, Defendant Crane Co. ("Crane") moves for relief from the jury's September 11, 2013 verdict. For the reasons set forth below, Crane's motions for judgment notwithstanding the verdict and new trial absolute are DENIED, but Crane's motion for new trial nisi remittitur is GRANTED.

I. The Evidence at Trial

Viewing the record in the light most favorable to Plaintiffs, sufficient evidence supports the jury's finding that Plaintiff Lloyd Garvin suffers from malignant mesothelioma proximately caused by exposure to asbestos. Mr. Garvin and his wife, Velda, brought this action against companies that made asbestos products Mr. Garvin allegedly worked with during his career as a maintenance mechanic and millwright. Evidence at trial indicated that during his employment at the Graniteville textile plant from 1964 to 1972 and the Carolina Eastman plastics plant from 1974 to 2003, he was exposed to asbestos gaskets and packing while installing and repairing pumps and valves, including Crane valves.

From 1964 to 1972 at Graniteville, Mr. Garvin installed and maintained new pumps and valves. The Graniteville site included many different buildings, and Mr. Garvin worked at all of the site's facilities.

After leaving Graniteville, Mr. Garvin worked at Carolina Eastman for about 30 years. He started as a millwright in the construction division and eventually became a millwright foreman and then a millwright general foreman. The site manager at Carolina Eastman, Doyle Ridgell, testified that there were seven or eight plants that each made a different product, and Mr. Garvin worked in all of them at various times. He worked on the construction of new buildings, installing new valves and pumps. He also did maintenance and repair work on valves and pumps. There was testimony that even after Mr. Garvin became a foreman in the 1970s, he was a "working foreman," who did hands on work on valves and pumps with his crew.

There was evidence that Mr. Garvin installed new Crane valves and repaired them at both Graniteville and Carolina Eastman. He first worked on Crane valves in 1964, and last worked around them in the 1990s. He recalled that Crane valves were "all over" Carolina Eastman. (Garvin Discovery Depo., at 115:2-8.) Documentary evidence, including Crane Co. specifications for various types of valves used at Carolina Eastman, confirmed that Crane's asbestos-containing valves were present at Carolina Eastman. Mr. Ridgell testified that Mr. Garvin worked on all the Crane valves at the plant, including rebuilding Crane valves.

Evidence showed Crane standardized the use of asbestos gaskets and packing on all of its bronze, iron and steel valves. Asbestos was the standard offering from Crane up into the 1980s. Crane's manuals and drawings from Carolina Eastman list asbestos gaskets and/or packing in the Crane valves present at that facility. The evidence showed that every Crane valve at Carolina

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Eastman had asbestos-containing gaskets or packing. Mr. Garvin testified that he knew how to tell the difference between gaskets made of asbestos and gaskets made of other materials, and he identified the valve gaskets as asbestos. Plaintiffs' occupational medicine expert, Dr. Edwin Holstein, and defense expert Dr. Victor Roggli testified that Crane valves utilized asbestos gaskets and packing.

Mr. Garvin removed gaskets and packing from Crane valves. About 80% of the valves had flange gaskets. Crane valves also had an internal gasket on the top of the valve. It was part of Mr. Garvin's job as a mechanic to remove and replace asbestos gaskets on valves. He estimated that the gaskets stuck to the metal valve about 75% of the time. When the gaskets stuck, he had to scrape them off with a putty knife, scraper, hammer, wire brush, power wire brush, or end grinder. It took up to half an hour to scrape off a gasket, and it was a dusty process.

Mr. Garvin also removed and replaced asbestos packing on Crane valves. All new valves came with packing that had to be removed during maintenance work, using a special tool called a packing puller. The old packing came out in pieces. Sometimes Mr. Garvin had to use compressed air to blow out the old packing. Removing packing created visible dust, as did cleaning up the packing debris.

Crane supplied its valves with asbestos gaskets and packing, and there was evidence that Mr. Garvin performed maintenance on new Crane valves that he or others had installed. When Mr. Garvin started at Carolina Eastman in 1974, he was involved in constructing a seven-story building, called 17L01 or the polymer building, and all of his work as millwright on this construction involved installing new equipment. Mr. Ridgell testified that Mr. Garvin installed new pumps and valves in that building, and once construction was complete Mr. Garvin and

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another millwright maintained and repaired the pumps and valves. Mr. Garvin did first maintenance on new valves that came with packing. He performed this work himself, and was also in close proximity to work on pumps and valves done by others.

Dr. Holstein testified regarding causation of Mr. Garvin's mesothelioma from his exposure to asbestos while working on Crane valves. Dr. Holstein testified that the gaskets and packing in Crane valves contained asbestos. This testimony was based on Crane's specifications for the plants where Mr. Garvin worked as well as historical evidence that asbestos gaskets were virtually always used in valves in industrial operations during the relevant time period.

Dr. Holstein testified that Mr. Garvin's exposure to asbestos gaskets and packing supplied directly by Crane was a substantial factor in causing his mesothelioma. Dr. John Maddox, a pathologist who specializes in asbestos-related disease, agreed that Mr. Garvin's exposure to asbestos from Crane valves was a substantial factor in causing his mesothelioma. Crane presented testimony from pathologist Dr. Victor Roggli, who testified Mr. Garvin had insufficient exposure to asbestos from Crane valves to cause his mesothelioma.

Mr. Garvin has an especially rare form of mesothelioma that originated in his testicles called mesothelioma of the tunica vaginalis testis, a subset of the more common peritoneal mesothelioma. There was evidence that Mr. Garvin's mesothelioma may be fatal, although there was also testimony that with mesotheliomas of this type there is a 50% chance of recovery. He was in remission at the time of trial.

A jury trial was held against Crane and two other defendants, Durco and Byron Jackson, in August and September 2013. The jury was charged the law of negligence, strict product liability, and breach of implied warranty. They returned a verdict of $10 million in compensatory

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damages to Lloyd Garvin and $1 million in compensatory damages for loss of consortium to Velda Garvin. After a bifurcated trial on punitive damages, the jury awarded $11 million in punitive damages against Crane, $11 million against Durco, and $5 million against Byron Jackson.[1]

Crane now seeks judgment notwithstanding the verdict, a new trial absolute, and a new trial nisi remittitur. It contends that there was no competent evidence that Mr. Garvin was exposed to asbestos gaskets and packing supplied by Crane. Crane further challenges the jury's willful and wanton finding, and the amounts of both the compensatory and punitive damages awards.

II. Ruling on Motion for Judgment Notwithstanding the Verdict and New Trial Absolute

A. Standard of Review

In ruling on a motion for JNOV, the trial judge must view the evidence and all reasonable inferences therefrom in the light most favorable to the non-moving party. JNOV should be granted when only one reasonable inference can be drawn from the evidence and no reasonable jury could have reached the challenged verdict. The trial judge does not have authority to decide credibility or resolve conflicts in the evidence. Curcio v. Caterpillar, Inc., 355 S.C. 316, 585 S.E.2d 272 (2003); Welch v. Epstein, 342 S.C. 279, 536 S.E.2d 408 (Ct. App. 2000).

If the amount of the verdict is so grossly inadequate or excessive that it shocks the conscience of the court and clearly indicates the amount was the result of passion, caprice, prejudice, partiality, corruption, or some other improper motive, the trial judge is required to

[1] Plaintiffs resolved their claims with Durco and Byron Jackson post-verdict.

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grant a new trial absolute. Cock-N-Bull Steak House, Inc., v. Generali Ins. Co., 321 S.C. 1, 466 S.E.2d 727 (1996); Vinson v. Hartley, 324 S.C. 389, 477 S.E.2d 715 (Ct. App. 1996).

B. Exposure

Crane contends there was insufficient evidence for the jury to find Mr. Garvin was substantially exposed to asbestos gaskets and packing provided by Crane at the time the valves were sold to his work sites. The court disagrees. There is substantial evidence that Crane supplied asbestos gaskets and packing in the valves it sold to the Graniteville and Carolina Eastman plants, including Crane documents specific to those plants. There was testimony from Mr. Garvin and Mr. Ridgell indicating that Mr. Garvin did the first maintenance job on Crane valves that were newly installed by either Mr. Garvin or other workers. This was not an isolated or unique scenario, as it was part of Mr. Garvin's job to repair and rebuild Crane valves. When he repaired a Crane valve, Mr. Garvin had to scrape off the asbestos gaskets and remove the asbestos packing in a manner that created visible dust in the air.

Crane argues that Mr. Garvin only had exposure to asbestos flange gaskets and points to Mr. Garvin's testimony that he did not work on bonnet gaskets. The jury also heard evidence, however, that he worked on gaskets internal to Crane valves, including Mr. Garvin's testimony that he worked on the gasket on the top of the Crane valve, Mr. Ridgell's testimony that Mr. Garvin had to clean the face of the valve, and Dr. Holstein's review of the evidence showing that Crane valves came from the factory with internal gaskets in place and that Mr. Garvin would have been exposed to those internal gaskets when working on Crane valves. Crane's corporate witness, Anthony Pantaleoni, acknowledged there was evidence of Mr. Garvin's work on internal gaskets on valves.

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Crane contends Dr. Holstein engaged in improper speculation in testifying that Crane valves contained asbestos and that Mr. Garvin was exposed to asbestos materials originally supplied with Crane equipment. Dr. Holstein's testimony was not speculative. It was based on a proper foundation in the fact testimony and documentary evidence in this case, as well as his knowledge and experience of the historical use of asbestos materials in valves in industrial settings.[2] Dr. Holstein has spent years evaluating asbestos health hazards in plants and factories, interviewing hundreds of workers for epidemiological research. He teaches industrial hygienists and relies on their work to evaluate exposures that cause disease. This broad knowledge about the use of asbestos gaskets and packing as the only available material for hot systems during the relevant time period was sufficient to support his testimony that Crane valves contained asbestos. Dr. Holstein was relying on numerous other sources, however, including Mr. Garvin's testimony that he was working with asbestos gaskets, Crane testimony that its valves were designed for use with asbestos, and site-specific documents that indicated all the Crane valves at Carolina Eastman contained asbestos gaskets or packing. His testimony conformed to the requirement that an expert may base an opinion on facts or data "perceived by or made known to the expert at or before the hearing." Rule 703, SCRE.

Dr. Holstein's testimony about Garvin's exposure was helpful to the jury and did not implicate any Rule 403 concerns. His specialized knowledge assisted the jury in understanding the evidence and determine an important fact in issue. Rule 702, SCRE. Dr. Holstein's expertise in teaching others about asbestos exposure and disease helped the jury understand the testimony

[2] A defense expert (Dr. Roggli) similarly testified that gaskets used in steam applications, like those described by Mr. Garvin, were likely asbestos-containing in the 1960s and 1970s.

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and documents regarding Mr. Garvin's work with Crane valves and exposure to original asbestos materials from that work. Dr. Holstein's testimony complied with all requirements for scientific expert witnesses and was properly presented to the jury. See Watson v. Ford, 389 S.C. 434, 699 S.E.2d 169 (2010); State v. White, 382 S.C. 265, 676 S.E.2d 684 (2009).

Although Dr. Holstein's testimony was directed at one of the central issues in this case, "[t]estimony in the form of an opinion or inference otherwise admissible is not objectionable because it embraces an ultimate issue to be decided by the trier of fact." Rule 704, SCRE. Dr. Holstein's testimony aided the jury but did not tell them what result to reach or otherwise usurp their function. Hughes v. Children's Clinic, P.A., 269 S.C. 389, 403, 237 S.E.2d 753 (1977). The court notes that defense expert Charles Blake offered opinions about Mr. Garvin's work practices and asbestos exposures, reaching the opposite conclusion that there was no evidence of any exposure to asbestos products made or supplied by Crane. The jury properly considered both sides of this issue, and its resolution in Plaintiffs' favor is supported by competent evidence, including the testimony of Dr. Holstein.

C. Stream of Commerce / Foreseeable Use

Crane claims that it was error to instruct the jury that "[a] Defendant cannot, however, be liable for an allegedly defective product it did not design, recommend, specify, require, manufacture, sell or place in the stream of commerce." (TT 9/11/13, at 207). Crane maintains that it can only be liable for products it placed in the stream of commerce, and has no liability for asbestos materials it only recommended, specified, or required.

A close, fair reading of the charge as a whole does not support Crane's strained position. The court charged the jury repeatedly that a Defendant manufacturer could only be held

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responsible for a product that (a) was defective at the time it left the Defendant's hands and (b) Defendant placed in the stream of commerce. The jury was told at least 14 different times about these concepts. South Carolina law provides, and the jury was charged, that under any products liability theory, the plaintiff must show that "the product at the time of the accident was in essentially the same condition as when it left the hands of the defendant," 5 Star, Inc. v. Ford Motor Co., 408 S.C. 362, 759 S.E.2d 139 (2014); Bragg v. Hi-Ranger, Inc., 319 S.C. 531, 539, 462 S.E.2d 321, 326 (Ct. App. 1995); (TT 9/11/13, at p. 199), and the product is "expected to and does reach the user or consumer without substantial change." S.C. Code Ann. § 15-73-10(1)(b); (TT 9/11/13, at p. 199).

At no point was the jury instructed that a manufacturer could be liable for products it did not itself place in the stream of commerce. In fact, the sentence Crane criticizes follows a sentence discussing a manufacturer's liability for incorporating a defective component part into its own product. Despite Crane's concern, nowhere in the charge is the phrase "replacement parts" used.

Crane further claims the jury charge improperly used the word "specify." To the extent Crane's argument can be followed, it appears to claim the word "specify" would befuddle the jury. Crane argues that "specify" is "inherently vague and subject to multiple interpretations." Crane Motion for Post-Trial Relief at 7 n. 5. Crane may find the word "specify" to be hopelessly confusing, but no reasonable juror would. The word has a well-established, lucid meaning that has been passed down for centuries. The American Heritage Dictionary defines it as meaning "[t]o state explicitly," Webster's Third New International as "to mention or name in a specific or explicit manner; tell or state precisely or in detail," and The Oxford English Dictionary as "[t]o

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mention, speak of, or name (something) definitely or explicitly." Blackstone did not find the word troublesome in a legal setting. See Blackstone, Commentaries on the Laws of England II at 319 (1766) ("But then there must many requisites be observed, which the statute specifies, otherwise such leases are not binding."). Crane fails to explain how there could be any confusion about the meaning of the word "specify" in the context in which it was used. The Court is confident the jury understood manufacturers may be specific about the component parts to be used in their products, just as manufacturers may also "recommend" or "require" particular components.

Crane argues the charge instructed the jury that it could find a manufacturer liable if the manufacturer merely specified, recommended or required a product it neither made, sold or placed in the stream of commerce. Accepting Crane's premise would entail a finding that the jury failed to follow the charge as a whole (which repeated ad nauseum the stream of commerce and defendant control requirements), and an order by this trial court defying South Carolina's longstanding product liability law regarding foreseeable use. What Crane is really driving at is adoption of the "bare metal defense" by a South Carolina court. The term "bare metal" is misleading and, as used here, is semantic advocacy rather than a useful doctrinal description. There is no evidence Crane sold "bare metal" valves or pumps; in fact, the evidence is to the contrary. The record demonstrates that Garvin was exposed to substantial and medically causative levels of asbestos dust when working on or replacing original Crane products that contained asbestos placed by Crane directly into the stream of commerce. Despite this stubborn fact, Crane contends that when its original asbestos parts wore out, the pump or valve somehow disappeared from the stream of commerce, even though it remained as an integral, working part

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of a massive industrial plant. Crane argues its legal responsibility ceased when the asbestos it supplied no longer accompanied the product.

Crane seeks to align this case with O'Neil v. Crane Co., 53 Cal.4th 335, 266 P.3d 987 (Cal.2012) where the California Supreme Court held that "a product manufacturer may not be held liable in strict liability or negligence for harm caused by another's product unless the defendant's own product substantially contributed substantially to the harm, or the defendant participated substantially in creating a harmful combined use of the products." Id., 266 P.3d at 991. The immovable obstacle to Crane's efforts is the record, particularly evidence that Mr. Garvin experienced substantial asbestos exposure to original Crane asbestos products. Even if this exposure never occurred, it was undisputed Crane placed into the stream of commerce products that not only contained asbestos, but specified they be replaced by asbestos parts. Such a scenario was explicitly mentioned by O'Neil as a problem its ruling did not address.[3] Viewed objectively, the manufacturer's product—defective when it left Crane's hands because it contained asbestos and specified asbestos for future use—remained defective and unreasonably dangerous when its specifications were heeded not only by its owner/user, but by another

[3] O'Neil noted that there was no evidence the Plaintiff was exposed to original asbestos products manufactured by Defendants. It also emphasized that the lack of evidence that Defendants specified asbestos be used for replacement gaskets or packing. O'Neil, 266 P.3d at 996 (finding Defendants "did not mandate or advise that [asbestos] be used with their products... the products were designed to meet the Navy's specifications. Moreover, there was no evidence that Defendant's products required asbestos-containing gaskets or packing in order to function."). These observations led the court to carefully explain the limited nature of its holding: "...[I]f the product manufactured specified or required the use of a defective replacement part, a stronger case could be made that the manufacturer's failure to warn was a proximate cause of resulting injury... These difficult questions are not presented in the case before us, and we express no opinion on their appropriate resolution." Id. at n. 6.

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manufacturer. These facts would enable a jury to find a "combined use" sufficient to remove Mr. Garvin's exposure from O'Neil's reach.

No jurisdiction has approved the "bare metal" defense in the teeth of such specification evidence. The only federal court of appeals that has approved the defense did so on the basis that the Plaintiff could show no exposure to asbestos products manufactured by the Defendants. Lindstrom v. A-C Product Liability Trust, et al., 424 F.3d 488 (6th Cir. 2005). But Lindstrom did not involve or address the issue of whether the Defendants specified asbestos-containing replacement parts. Judge Robreno, the MDL judge for federal asbestos claims, relied heavily on Lindstrom in holding the bare metal defense applicable in maritime cases, Conner v. Alfa Laval, Inc., 842 F.Supp.2d 791 (E.D.Pa.2012), but there too no evidence of exposure to Defendants' original asbestos products or specifications appeared in the record. Id. at 802. Likewise, the sharply divided "bare metal" decisions of the Washington supreme court did not confront this precise issue, and that court has sheepishly realized the awkward logical limits of the principle. *Compare* Braaten v. Saberhagen Holdings, Inc., 198 P.3d 493 (Wash. 2008) (no evidence Defendants manufactured, recommended or specified replacement asbestos gaskets or packing) *with* Macias v. Saberhagen Holdings, Inc., 282 P.3d 1069 (Wash. 2012) (respirator manufacturer not entitled to summary judgment even though it did not manufacture asbestos products to which plaintiff exposed; distinguishing Braaten and finding that respirator was specifically designed for use that would inherently cause exposure).

O'Neil and other cases adopting the "bare metal" defense lean on Baughman v. General Motors Corp., 780 F.2d 1131 (4th Cir. 1986). Baughman dealt with tire rims, not asbestos. It is a decision best restricted to its facts. Mr. Baughman was injured when a CR-2 rim assembly

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exploded while he was changing a tire on a 1979 GMC truck. It was undisputed that GM did not design, manufacture, require or specify the use of the CR-2. The original rim assembly was a CR-3, which has a different design. The Fourth Circuit held as follows:

> Where, as here, the defendant manufacturer did not incorporate the defective component part into its finished product and did not place the defective component into the stream of commerce, the rationale for imposing liability is no longer present. The manufacturer has not had an opportunity to test, evaluate, and inspect the component; it has derived no benefit from its sale; and it has not represented to the public that the component part is its own.

780 F.2d at 1132-33. The court did not discuss whether the result would have been different had GM specified the defective rim assembly. This is a fundamental point, for there was no claim that the original GM rim assembly was defective.

This latter fact also upends Crane's reliance on the policy rationales often articulated in support of the "bare metal" concept. Unlike GM, Crane had the opportunity to test and evaluate the asbestos gaskets and packing used in its valves and which injured Mr. Garvin (the jury was even given factors to use in deciding Crane's duty to test). There was no evidence the replacement gaskets and packing manufactured by others were different in material or design from Crane's original supplied products or specifications. The evidence showed Crane long knew of the risk asbestos posed, and it occupied the best position in the chain of distribution to warn consumers of those risks. Crane knew asbestos gaskets and packing would not last as long as its bare metal valves or pumps; consequently it knew those parts would have to be replaced, and replaced with similar if not identical parts whose manufacture was guided by Crane's design and specifications. To say Crane was no longer part of the "chain of distribution" when the original gaskets and packing wore out on its still-functioning product would be artificial, if not

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silly. The drafters of the Restatement of Torts (Second) § 402A well understood these practicalities:

> ... the justification for the strict liability has been said to be that the seller, by marketing his product for use and consumption, has undertaken and assumed a special responsibility toward any member of the consuming public who may be injured by it; that the public has the right to and does expect, in the case of products which it needs and for which it is forced to rely upon the seller, that reputable sellers will stand behind their goods; that public policy demands that the burden of accidental injuries caused by products intended for consumption be placed upon those who market them, and be treated as a cost of production against which liability insurance can be obtained; and that the consumer of such products is entitled to the maximum of protection at the hands of someone, and the proper persons to afford it are those who market the products.

Id. at Comment c. These comments have been incorporated by reference and codified as the official legislative intent of our strict liability statute for over 40 years. S.C. Code § 15- 73-30.

In a recent thoughtful opinion, a federal district court in Chicago set forth the most sensible approach to the "bare metal" defense. Quirin v. Lorillard Tobacco Co., 2014 WL 804305 (N.D. Ill. 2014). Mr. Quirin claimed his mesothelioma arose in part from exposure to asbestos while working with Crane valves as a machinist on the *USS Tolovana* from 1954-1957. The *Tolovana* utilized over 1000 Crane valves, some of which contained internal asbestos gaskets and packing. Crane also sold asbestos gaskets and packing, some of it purchased from third parties. The original gaskets and packing Crane supplied had likely been replaced before Quirin began service, and he could not specifically recall whether he had ever changed out packing original to a Crane valve. Indeed, there was "no direct evidence that Crane Co. supplied any replacement gaskets or packing that Mr. Quirin used on board." Id. at 3.

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Crane moved for summary judgment, asserting it had no legal duty to warn Mr. Quirin of the danger of asbestos materials manufactured and supplied by others. Crane argued there was no evidence Mr. Quirin had any exposure to asbestos-containing products it had placed in the stream of commerce. Applying maritime law, the court held:

> In general, consistent with the bare metal defense, a manufacturer is not liable for materials it did not supply. But a duty may attach where the defendant manufactured a product that, by necessity, contained asbestos components, where the asbestos-containing material was essential to the proper functioning of the defendant's product, and where the asbestos-containing material, whether supplied by the original manufacturer or someone else.
> ***
> Furthermore, even if replacement gaskets and packing for the Crane Co. valves were supplied by a third party, it is reasonable to infer that those replacement parts would have been substantially identical to the components originally supplied by Crane Co. Given these facts, a reasonable jury could find that Crane Co. had a duty to warn about the hazards of asbestos exposure resulting from work on its valves...
> ***
> The court acknowledges that its conclusion differs from that reached by those courts that have relied on the "bare metal defense" to strictly limit liability where a manufacturer did not make the actual product allegedly causing asbestos exposure. It is not convinced, however, that a manufacturer should avoid liability on a failure-to-warn theory, where it designed its products to be used with asbestos-containing materials into the products it sold. On those facts, a jury could conclude that it was not just foreseeable, but inevitable, that the product would subject those working with it to the possible hazards of asbestos exposure. The manufacturer of the product was in a position to warn of dangers apparent at the time the product was sold.

Id. at 8.

Although Crane protests there was no evidence that it required the use of asbestos gaskets and packing with its valves, the evidence was sufficient to create a fact issue for the jury. Plaintiffs presented Crane manuals and drawings from Carolina Eastman that list asbestos

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gaskets or packing as component parts in every valve Crane sold to that facility. There was also evidence that Crane generally designed its valves to be used with asbestos gaskets and packing, and also designed its valves with flanged connections that required asbestos gaskets, versus other connection designs not requiring gaskets.

Crane anticipated that maintenance workers would break down valves, open them up, and change the gaskets and packing. There was also evidence that Crane sold asbestos packing, internal gaskets, flange gaskets, and insulation as replacement parts for use with its valves, including the brand of gaskets used by Mr. Garvin. Mr. Ridgell testified that he would go to the valve manufacturer for replacement gaskets. Carolina Eastman records show Crane included asbestos gaskets and packing in spare parts kits. Crane recommended that work sites keep asbestos gaskets and packing as spare parts for Crane valves. Crane recommended the use of asbestos gaskets, particularly on steam systems, which is the exact type of applications fact witnesses described were in place at Carolina Eastman and Graniteville. Such evidence raises a reasonable inference that asbestos gaskets and packing were required component parts of Crane valves.

The Court finds evidence sufficient for the jury to conclude that when Mr. Garvin worked on Crane valves, the valves were in "essentially the same condition as when [they] left the hands of the defendant." Bragg, 319 S.C. at 539. Crane placed the injury-causing product in the stream of commerce, and there was ample evidence that Mr. Garvin worked on original Crane valves and gaskets. Crane's liability arises because its own valves caused injury as manufactured, supplied, and designed by Crane with asbestos gaskets and packing. When the asbestos gaskets and packing were replaced with the same materials that came with the valve, it was not a

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"substantial change" in the condition of the product. The product remained the same as when sold—valves containing asbestos gaskets and packing.

Mr. Garvin's injury was caused by gaskets and packing that were no different than the gaskets and packing that came with the valves originally. Crane designed and sold its product with asbestos parts that were wear items that Crane knew had to be replaced, there was evidence that the replacement process itself created exposure to toxic asbestos dust, and the replacement parts were to Crane's design specifications.

In sum, the court finds the jury charge conformed to established South Carolina law that a manufacturer's liability extends to foreseeable dangers resulting from the anticipated or intended use of its products. See Spruill v. Boyle-Midway, Inc., 308 F.2d 79, 84 (4th Cir. 1962); Fleming v. Borden, Inc., 316 S.C. 452, 458, 450 S.E.2d 589, 593; Claytor v. General Motors Corp., 277 S.C. 259, 262, 286 S.E.2d 129, 131 (1982); Carolina Home Builders, Inc. v. Armstrong Furnace Co., 259 S.C. 346, 359-60, 191 S.E.2d 774, 870 (1972); Mickle v. Blackmon, 252 S.C. 202, 233 (1969). A jury evaluates whether a product is unreasonably dangerous to the ordinary consumer according to the "conditions and circumstances that foreseeably attend the use of the product." Bragg, 319 S.C. at 543; Claytor, 277 S.C. at 262. A manufacturer is "'expected to anticipate the environment which is normal for the use of his product and . . . must anticipate the reasonably foreseeable risks of the use of his product in such environment.'" Mickle, 252 S.C. at 233 (quoting Spruill, 308 F.2d at 83- 84). "[L]iability may be imposed upon a manufacturer or seller notwithstanding subsequent alteration of the product when the alteration could have been anticipated by the manufacturer or seller." Small v. Pioneer Mach., 329 S.C. 448, 466, 494 S.E.2d 835, 844 (Ct. App. 1997).

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The issues of whether Crane's valves were in essentially the same condition when Mr. Garvin worked on them, and whether Crane could reasonably foresee that the asbestos gaskets and packing in its valves would be replaced with asbestos materials, were for the jury. If a manufacturer recommends, specifies, or requires that asbestos gaskets and packing be replaced with like materials, then the jury may conclude that the product is in essentially the same condition as when it left the manufacturer, that the manufacturer anticipated the subsequent alteration of the product, and that the manufacturer anticipated the reasonably foreseeable risks of using the product in an environment where routine repair and maintenance would require replacement of asbestos components.

Crane had wide berth to convince the jury it was not liable for defective replacement parts, and its skilled trial counsel attempted to do so in a thorough and professional manner. In addition to the instructions set forth above, the jury was instructed on legal cause, which focuses on foreseeability. They were also charged on intervening cause, and the requirement that to be liable the Defendant's product must have been defective at the time it left Defendant's hands, was placed in the stream of commerce by Defendant, and its condition was not substantially changed up to the time it injured Mr. Garvin. The jury applied this law to the facts and found for Mr. Garvin on all issues.

D. Causation

Crane argues that there was insufficient evidence that Mr. Garvin's mesothelioma was caused by his exposure to asbestos from Crane products. The Court does not find merit in any of Crane's arguments.

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1. Frequency, Regularity, Proximity

First, Crane contends that there was no admissible evidence of the frequency, regularity, and proximity of Mr. Garvin's work with asbestos materials in Crane valves, as required by Henderson v. Allied Signal, Inc., 373 S.C. 179, 644 S.E.2d 724 (2007). The frequency, regularity, and proximity factors are designed to evaluate whether there was "actionable exposure" to dust from the defendant's asbestos product. Id. at 185. Dr. Holstein and Dr. Maddox testified that Mr. Garvin has an asbestos-related mesothelioma, that mesothelioma can be caused by relatively brief and low level cumulative exposures, and that there is no known threshold of exposure below which mesothelioma does not occur.

Plaintiffs presented sufficient evidence to support the jury's causation finding against Crane in this case. This is not a situation of mere "presence in the vicinity of static asbestos." Henderson, 373 S.C. at 185. Rather, Mr. Garvin scraped asbestos gaskets from the face and flanges of Crane valves, and removed asbestos packing from Crane valves with a packing puller. Mr. Ridgell described seeing visible dust when Mr. Garvin used a grinder to remove asbestos gaskets. All of the Crane valves at Carolina Eastman contained asbestos, and asbestos was also used on the Crane valves at Graniteville. Dr. Holstein's testimony about Mr. Garvin's exposure to Crane-supplied asbestos materials had an adequate foundation and assisted the jury.

In terms of the duration of this work, there was evidence that Mr. Garvin worked on Crane valves throughout his career, at both Graniteville and Carolina Eastman, from 1964 until the 1990s. Mr. Ridgell testified it was Mr. Garvin's job to rebuild and change out pumps and valves for weeks at a time.

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Plaintiffs' experts discussed studies measuring asbestos fiber release from gaskets and packing up to 100,000 times higher than what is present in the background air, which are levels associated with doubling the risk for asbestos disease. Defense expert Dr. Roggli testified that there can be airborne asbestos at the level of 5 million particles per cubic foot and asbestos would still be invisible to the naked eye. He conceded that if a person can see asbestos dust, the level is well above background levels in the outside air.

Dr. Holstein testified about scientific research showing exposure to asbestos gaskets and packing is quite hazardous. Defense experts Dr. Roggli and Mr. Blake relied on the same studies, in which the authors state the need for protective measures when working with asbestos gaskets, describe exposures thousands of times higher than background, and quantify exposure levels from 1 fiber per cubic centimeter of air (f/cc) to 30 f/cc. Industry studies found similar results of 26 to 30 f/cc of asbestos released when working with asbestos gaskets. Other studies found the use of a grinder to remove asbestos gaskets, as Mr. Garvin did, releases asbestos at a level of 6.8 f/cc. Ten to twenty years before Mr. Garvin was last exposed to asbestos, the Bremerton study concluded that "asbestos, a fibrous mineral silicate which is a component of many gasket materials is a documented health hazard causing cancer and chronic lung disease known as asbestosis." (TT 9/9/13 at 145-46.) A study relied on by Crane's own experts found that gasket work releases asbestos at levels that "exceed all current and historical occupational safety and health administration excursion limits and some permissible excursion limits based on an 8 hour time weighted average." (TT 9/3/13 at 147-148; TT 9/9/13 at 136-137). Dr. Roggli also conceded that Material Safety Data Sheets from asbestos gasket suppliers Durabla, John Crane,

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and Garlock, which supplied asbestos components sold by Crane, indicate that asbestos gaskets cause disease.

Based on the details of Mr. Garvin's exposure, Dr. Maddox concluded that asbestos exposures from scraping gaskets are orders of magnitude higher than the pollution in the ambient air (or background level). Dr. Maddox concluded Mr. Garvin's asbestos exposure from Crane valves was a substantial contributing cause of the development of his mesothelioma. Answering a hypothetical question based on facts later established by the evidence—including exposure to Garlock gaskets that contain 85%+ chrysotile asbestos, used over the span of three decades repeatedly with a grinder —defense expert Dr. Roggli testified that such exposure would substantially contribute to cause mesothelioma.

The evidence of Mr. Garvin's exposure to asbestos from Crane valves was more than sufficient to create a fact issue for the jury on the issue of causation.

2. Admissibility of Expert Causation Testimony

Crane challenges Plaintiffs' experts' causation opinion testimony as inadmissible and insufficient to establish causation under South Carolina law. The admissibility of expert testimony is governed by South Carolina Rule of Evidence 702, which provides that "[i]f scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise." Courts evaluating the admissibility of scientific expert evidence "must find the evidence will assist the trier of fact, the expert witness is qualified, and the underlying science is reliable." State v. Council, 335 S.C. 1, 20, 515 S.E.2d 508 (1999); see also Watson v. Ford, 389 S.C. 434,

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699 S.E.2d 169 (2010). While the trial court assesses the reliability of the expert's methodology, it is for the jury to determine the soundness of the facts underlying the expert's opinion and the correctness of his conclusions. See Milward v. Acuity Specialty Prods. Group, Inc., 639 F.3d 11, 22 (1st Cir. 2011).

Plaintiffs presented expert testimony from Dr. Arnold Brody, a cellular biologist specializing in the biological mechanisms of asbestos-related disease, Dr. Maddox, a board-certified pathologist with expertise in asbestos-related disease, and Dr. Holstein, a physician board-certified in occupational medicine and preventive medicine. The court finds each of these experts to be well-qualified to offer causation opinions, and finds their testimony reliable and helpful to the trier of fact.

Dr. Brody offered only general causation opinions and explained that it is the cumulative dose of asbestos that causes mesothelioma, because asbestos is a carcinogen and it is not possible to say that some doses of the carcinogen were not causative in the disease process. He also testified that numerous government agencies, including the EPA, OSHA, the National Cancer Institute, the World Health Organization (WHO), and the American Thoracic Society, have concluded that there is no safe level of exposure to asbestos.

Dr. Maddox is a board-certified anatomical and clinical pathologist who specializes in the diagnosis and attribution of mesothelioma. His specific causation opinions are based on fact witness testimony regarding the frequency, duration, and proximity of Mr. Garvin's exposure, Mr. Garvin's medical and pathology materials, Crane-specific documents, studies of the fiber release from asbestos gaskets and packing, and the peer-reviewed medical literature regarding the causation of mesothelioma. Mr. Garvin's exposures to asbestos gaskets from Crane valves

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were several orders of magnitude above background levels, and constituted a significant component of his cumulative asbestos exposure that caused his mesothelioma. Dr. Maddox also testified about the reasonableness of Mr. Garvin's medical care, including that his medical bills to date were $149,000, and that his medical expenses could be as much as $300,000 more in the future.

Dr. Holstein is board-certified in occupational medicine and preventive medicine and has spent more than 40 years studying and teaching about the causation of asbestos-related disease in working environments. In addition to his consulting practice, he has been a Clinical Assistant Professor in the Division of Environmental Sciences at Mount Sinai School of Medicine since 1983 and has also served as a consultant to the National Cancer Institute and NIOSH. He evaluated Mr. Garvin's exposure according to the description of his work practices, the evidence of asbestos content in the valves present at Mr. Garvin's work sites, fiber release studies, and epidemiological studies of asbestos-related disease. He testified that Mr. Garvin's exposures from working with asbestos gaskets were 10,000 to 100,000 times above background levels. He determined that Mr. Garvin had frequent and regular exposure to original gaskets supplied with Crane valves and that these exposures were a substantial contributing cause of his mesothelioma.

Crane argues that Plaintiffs' expert medical witnesses testified that "every exposure to asbestos that Mr. Garvin sustained substantially contributed to cause his disease." The trial record does not contain any such testimony from any of Plaintiffs' experts. Ironically, the general acceptance of the opinion challenged by Crane is evidenced by testimony from its own expert, Dr. Victor Roggli, who has previously expressed the opinion that "each and every exposure to asbestos than an individual [with] mesothelioma experienced in excess of a background level is a

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substantial contributing factor to the disease." TT 9/3/13 at 70. Dr. Roggli also indicated that there is no known safe level of exposure to asbestos and that very low levels of exposure above background have been repeatedly demonstrated to cause mesothelioma, including peritoneal mesothelioma. Dr. Roggli testified that it is the total dose of asbestos, regardless of fiber type, that the patient experiences that causes the disease. These opinions were relevant, reliable, and admissible evidence for the jury to consider in evaluating causation.

There was evidence that the opinions expressed by Plaintiffs' experts and Dr. Roggli, including that there is no safe level of exposure to asbestos and that mesothelioma can be caused by brief and low level exposures, are shared by numerous public health and regulatory agencies. These agencies include the EPA ("scientists have not been able to determine a 'safe' or threshold level for exposure to airborne asbestos"); OSHA ("There is no 'safe' level of asbestos exposure for any type of asbestos fiber. Asbestos exposures as short in duration as a few days have caused mesothelioma in humans."); NIOSH ("[E]xcessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a 'safe' level of asbestos exposure."); the Consumer Products Safety Commission ("The Commission has noted that in the scientific literature there is general agreement that there is no known threshold level below which exposure to respirable free-form asbestos would be considered safe."); the WHO ("there is no safe threshold level of exposure"); and the World Trade Organization (finding "on the basis of scientific evidence that 'no minimum threshold of level of exposure or duration of exposure has been identified with regard to the risk of pathologies associated with chrysotile, except for asbestosis"). These authorities support the court's finding that Plaintiffs' experts' opinions are not only reliable and helpful to the trier of

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fact, but also supported the jury's finding that Mr. Garvin developed mesothelioma from breathing asbestos fibers from Crane valves.

3. But-For Cause

The jury was instructed that to prove proximate causation in a negligence case, it must find that the injury would not have occurred "but for" the defendant's negligence. Such evidence was not lacking in this case.

The jury heard expert testimony that there is an inverse relationship between the dose or amount of asbestos a person breaths and the latency period of the disease. If the cumulative dose of asbestos is increased by exposure to asbestos products, then that shortens the latency period and causes the disease to develop more quickly. The court finds that this evidence that the latency of Mr. Garvin's disease was affected by his exposure to asbestos from Crane valves is sufficient to raise a genuine issue of material fact for the jury regarding but-for causation.

E. Knowledge Evidence

Crane contends that certain statements in trade association journals and other documents about the hazards of asbestos were inadmissible hearsay. The statements had been received by Crane from outside sources and were offered on the issue of notice. These statements were therefore not hearsay, as they were not offered for the truth of the matter asserted. Rule 801(c), SCRE. The hearsay rule does not apply to evidence offered solely to prove notice, as that relates to state of mind. Player v. Thompson, 259 S.C. 600, 193 S.E.2d 531 (1972) (testimony that motor vehicle inspector told defendants prior to accident that car's tires were unsafe); Smedra v. Stanek, 187 F.2d 892 (10th Cir. 1951) (testimony "that Dr. Stanek had been told by someone in the operating room that the sponge count did not come out right"). Testimony to show notice of a

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fact and not the fact itself is not hearsay. Baber v. Greenville Cty., 327 S.C. 31, 41, 488 S.E.2d 314, 319 (1997).

Crane specifically complains about the admission of testimony from its corporate representative, Mr. Pantaleoni, regarding a letter from a Crane customer, Foster Wheeler, stating that health hazards had been shown from minimal exposures. Evidence that Crane received that letter and had notice of the contents of that letter was not hearsay. The Court excluded the letter under Rule 403, SCRE, however. Although it was inadvertently shown to the jury in violation of this ruling, this was a harmless error because it was cumulative of other notice evidence already in the case. See Ramos v. Hawley, 316 S.C. 534, 536, 451 S.E.2d 27, 28 (Ct. App. 1994); Sligh v. Johnson, 288 S.C. 364, 367, 342 S.E.2d 620, 622 (Ct. App. 1986). In addition, Crane later opened the door to this evidence by presenting testimony from Mr. Pantaleoni claiming that Crane had no knowledge that asbestos gaskets and packing were harmful. The Foster Wheeler letter was therefore admissible as impeachment evidence to contradict Mr. Pantaleoni's claim, in addition to being relevant on the issue of notice.

Crane also objects that a document from the American Petroleum Institute (API) was inadmissible hearsay. The receipt and contents of this document were properly discussed on cross-examination of Mr. Pantaleoni and Dr. Roggli because the evidence was offered to show that Crane had access to information from API about asbestos hazards. Communications from trade organizations such as API to a defendant or management-level employee of the company are not hearsay when offered to show the information that Crane received about health concerns associated with asbestos exposure. The question of what was known and knowable to asbestos manufacturers is always a highly relevant area of inquiry in any asbestos case, and

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communications to the defendant from a variety of sources may be offered to show notice to a defendant.

F. S.C. Code § 15-38-15

Crane contends that that settled parties should have been allocated fault at trial. The short answer to this is that § 15-38-15 is inapplicable because the jury determined Crane's conduct to be willful and wanton. S.C. Code § 15-38-15(E). Even were this not true, the plain language of S.C. Code § 15-38-15 allows apportionment of fault only among the trial defendants. The legislature repeatedly refers to "defendants" in the pertinent sections of the statute. See S.C. Code Ann. § 15-38-15(A) ("[I]f indivisible damages are determined to be proximately caused by more than one *defendant*, joint and several liability does not apply to any *defendant* who conduct is determined to be less than fifty percent of the total fault for the indivisible damages as compared with the total of: (1) the fault of all the *defendants*; and (ii) the fault (comparative negligence), if any, of the plaintiff."); § 15-38-15(A) ("A *defendant* whose conduct is determined to be less than fifty percent of the total fault shall only be liable for that percentage of the indivisible damages determined by the jury or trier of fact."); § 15-38-15(B) ("Apportionment of percentages of fault among *defendants* is to be determined as specified in subsection (c)."); § 15-38-15(C)(3) ("The jury . . . shall . . . upon a motion by at least one *defendant*, where there is a verdict . . . against two or more *defendants* for the same indivisible injury . . . specify in a separate verdict . . . the percentage of liability . . . that is attributable to each *defendant*."); § 15-38-15(C)(3) ("In determining the percentage attributable to each *defendant*, any fault of the plaintiff . . . will be included so that the total of the percentages of fault attributed to the plaintiff and to the *defendants* must be one hundred percent."); § 15-38-15(E) ("Notwithstanding the

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application of this section, setoff from any settlement received from any potential tortfeasor prior to the verdict shall be applied in proportion to each *defendant's* percentage of liability as determined pursuant to subsection (c).").

The Legislature specifically chose the term "defendant" rather than "nonparty," "released party," or some other term. The Legislature meant what it said. Connecticut Nat. Bank v. Germain, 503 U.S. 249, 253-54 ("Courts must presume that a legislature says in a statute what it means and means in a statute what it said there."). Giving the term its plain and ordinary meaning, a defendant is "[a] person sued in a civil proceeding . . . ." Black's Law Dictionary 482 (9th ed. 2009). Placing nonparties on the verdict form for apportionment of fault would contravene the express terms of the statute.

While it is true that § 15-38-15 (D) provides that Defendants "retain the right to assert that another potential tortfeasor, whether or not a party, contributed to the alleged injury...," the provisions of the statute dealing with verdict forms and how to practically apportion fault restrict their application to "defendants," i.e. parties. Subsection (D) retains a Defendant's right to argue non-party fault at trial even though the non-party does not appear on the verdict form, a right Crane exercised.

Any ambiguity in the statute may be resolved by considering its purpose when it was passed in 2005 to modernize joint and several liability. Construing the statute as a whole, and interpreting it in a practical, reasonable manner in accord with its purpose, see State v. Sweat, 688 S.E.2d 569, 575 (2010), the court finds that § 15-38-15 does not permit a jury to assess the fault of non-parties on the verdict form. The legislature chose its words carefully, and while subsection (D) mentions non-parties, there is no mechanism allowing them a place on the verdict

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from. When a legislature includes particular language in one section of a statute but omits it in another, it is presumed to have done so deliberately. Russello v. United States, 464 U.S. 16, 23 (1983).

The legislative history sheds further light. Two unadopted versions of the Bill proposed language that Crane now seeks to have this court make the law. See Shaw, Limited Joint and Several Liability Under Section 15-38-15: Application of the Rule and the Special Problem Posed by Nonparty Fault, 58 S.C. Law. Rev. 627, 633-34 (2007) (referring to prior versions of H.B. 3008 referred to House Judiciary Committee). The legislature's "rejection of the very language that would have achieved the result" Crane urges "weighs heavily" against Crane's interpretation. Hamdan v. Rumsfeld, 548 U.S. 557, 579-80 (2006).[4]

A statute's words must be construed in context and in light of the intended purpose of the statute in a manner, which harmonizes with its subject matter and accords with its general purpose. In re Manigo, 398 S.C. 149, 729 S.E.32 (2012). The purpose of §15-38-15 was to end joint and several liability for *defendants* with less than fifty percent fault. Limiting the meaning of "defendants" to the parties on the verdict form maintains this purpose, as defendants are free to argue they have no fault or less than fifty percent of the fault. The successful defendant will enjoy the protection offered by the statute and avoid joint and several liability.

[4] This principle of statutory interpretation regarding rejected proposed legislation should be known, especially in South Carolina, as *canis non insidiatus,* or "The Dog That Would Not Hunt," a corollary to the Canon of Canine Silence. See Koons Buick Pontiac GMC, Inc. v. Nigh, 543 U.S. 50, 73-74 (2004) (Scalia, J., dissenting) (coining Canon to spoof idea that legislative silence about a subject may be used as evidence of legislative intent). The Canon has roots in Justice Stewart's decision in Harrison v. PPG Industries, Inc., 446 U.S. 578, 592 (1980) ("In ascertaining the meaning of a statute, a court cannot, in the manner of Sherlock Holmes, pursue the theory of the dog that did not bark.").

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In §15-38-15(C)(3), the percentages of fault of the plaintiff, if any, and that of the defendants must add up to one hundred percent. The court is certain the Legislature understood the law of percentages, and it left no percentage for allocation to non-parties. Roschen v. Ward, 279 U.S. 337, 339 (1929) ("[T]here is no canon against using common sense in construing laws as saying what they obviously mean.") (Holmes, J.).

A jury simply cannot apportion fault to anyone other than the defendants at trial. The rule that the Plaintiff alone has the right to determine which tortfeasor to sue is a "firmly entrenched common law principle," Chester v. S.C. Dep. Pub. Safety, 388 S.C. 343, 346, 698 S.E.2d 559 (2010), of which the Legislature was presumptively aware and intentionally chose not to disturb. See Fagnant v. K-Mart Corp., 2013 WL 6901907 (D.S.C. 2013) (reaching same interpretation of section 15-38-15).

G. Jury Conduct

Crane questions the jury's empanelment and deliberations because it alleges that the trial lasted longer than the jury was originally told it would and the jury deliberated less than three hours. When a party alleges irregularities in the jury's deliberations, "[t]he test is whether there is reason to suppose outside influences affected the jury's verdict." Parker v. Evening Post Publ. Co., 317 S.C. 236, 248, 452 S.E.2d 640, 647 (Ct. App. 1994). The court evaluates such a claim based on its "opportunity to view the trial, the character and intelligence of the jurors, and to consider whether the verdict, in light of the evidence, has so little support as to indicate corrupt or improper influence." Id.

There is no evidence the jury's verdict was affected by any improper outside influence. At the end of the second week of trial, the jury was informed the trial would continue into the

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next week. The forelady discussed the matter with the jury members and informed the Court that no jurors had indicated they could not be present for an additional week. The jury was consulted about any scheduling conflicts due to the three extra days of trial, and those were accommodated. One juror expressed concern that two other jurors showed up late for trial one day, but she assured the court that this would not compromise her duties as a juror. In fact, the two she had a complaint about were alternate jurors who did not take part in deliberations.

The jurors were highly attentive throughout the trial and approached their duties studiously. The jury managed its time well, and their efficient deliberations cannot be spun as a signal that some sinister force was at work. See Becker v. Wal-Mart Stores, Inc., 339 S.C. 629, 636, (Ct. App. 2000). The deliberations were not unreasonably brief, and it is quite common for long trials to end with short deliberations, for the human mind can concentrate quite effectively when it has been adequately prepared. Id.; see also Curtis v. Blake, 392 S.C. 494, 504-505 (Ct. App. 2011) (brevity of jury deliberations alone does not suffice as a reason to set aside the verdict); Youmans ex rel. Elmore v. S.C. Dept. of Transp., 380 S.C. 263, 283, 670 S.E.2d 1, 11 (Ct. App. 2008) (noting the trial court should not base a decision on a thirteenth juror new trial motion on the brevity of jury deliberations).

Crane claims a new trial is warranted because the large amount of damages awarded could only be the result of "considerations not reflected by the evidence." It accuses the jury of reaching its verdict as a "result of passion, caprice, prejudice, partiality, corruption or some other improper motives." The Court cannot agree. As noted, the Court observed the jury to be conscientious, thoughtful, and properly focused on the evidence. Many of the jurors were avid note takers. They came from a wide variety of backgrounds, including a forensic technician for

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the Federal Bureau of Investigation, a senior manager for the American Red Cross, a car mechanic, a maintenance worker at a nursing home, a retired Army soldier, a dental hygienist, a self-employed national security investigator, a retired social worker, an office manager, a nurse, a certified medical assistant at the Richland County Jail, and a retail manager. This rather diverse group reached a unanimous verdict for Plaintiffs on all questions.

While the jury did decide on a large award, the Court has no reason to conclude it was the result of any improper motive or outside consideration. Placing a value on pain and suffering is inherently subjective. In the Court's estimation, the members of the jury performed their civic duty admirably throughout the course of the three-week trial.

H. Willful and Wanton Conduct

Crane next argues there was insufficient evidence to support the jury's finding that its conduct was willful, wanton, or reckless, and that the jury's award of punitive damages should therefore be vacated. The record, however, contains ample evidence to support the jury's determination that Crane's conduct warranted punitive damages.

Plaintiffs presented sufficient evidence of Crane's knowledge of asbestos health hazards to raise a fact question for the jury regarding Crane's willful, wanton, and reckless conduct. Plaintiffs' evidence showed that Crane knew or should have known about the dangers of its products. Both categories of evidence are relevant in evaluating whether Crane's conduct was willful, wanton, and reckless, and in assessing punitive damages.

There was evidence that Crane had access to knowledge since the 1930s that asbestos exposure causes fatal disease. Thus, Crane knew of asbestos hazards approximately 50 years before Mr. Garvin's last exposure to asbestos from Crane valves. Crane knew about the

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possibility that asbestos exposure could cause cancer as early as 1957. It knew about the risk of mesothelioma from exposure to asbestos products in the 1960s. It knew that the risk decreased when exposure levels decreased. Crane does not deny that it knew about the health hazards of asbestos exposure in the 1970s, including the risk of cancer and mesothelioma. Crane is aware of the EPA's position that there is no known safe level of exposure to asbestos.

The jury was presented with evidence that Crane continued to sell asbestos-containing materials years after it had actual knowledge of the danger, including up into the 1980s when Mr. Garvin was still working with Crane valves. Crane did not test its products and never warned. Crane did not pass on warnings it received from asbestos gasket suppliers in the 1980s. Dr. Roggli testified that where asbestos products release fibers above background and carry no conspicuous warning, the product is unreasonably dangerous. Crane told the jury that even knowing everything it does now, Crane would not have done anything differently today.

Plaintiffs also presented evidence that the magnitude of the risk is quite significant. The EPA has found that even one year of exposure at current limits of 0.1 f/cc could cause death and disease. 100,000 people die yearly from asbestos-related disease, and Crane is aware of that. Asbestos exposure contributes to 1 of 3 cancer deaths in the occupational setting in the U.S.

Crane argues that its conduct cannot be considered willful, wanton, and reckless because it was consistent with that of other manufacturers in the industry. Crane failed to point to any evidence in support, but even if it had, it would not preclude a finding of willful and wanton

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conduct. Industry standards may be probative, but never dispositive. See Reed v. Tiffin Motor Homes, Inc., 697 F.2d 1192, 1198 (4th Cir. 1982).[5]

I. Punitive Damages

"[P]unitive damages serve at least three important purposes: punishment of the defendant's reckless, willful, wanton, or malicious conduct; deterrence of similar future conduct by the defendant or others; and compensation for the reckless or willful invasion of the plaintiff's private rights." Clark v. Cantrell, 339 S.C. 369, 379, 529 S.E.2d 528, 533 (2000). Those purposes are served by an award of punitive damages in this case.

Although reviewable for compliance with due process, James v. Horace Mann Ins. Co., 371 S.C. 187, 194, 638 S.E.2d 667, 670 (2006), a jury's punitive damages award is "entitled to a strong presumption of validity." TXO Production Corp. v. Alliance Res. Corp., 509 U.S. 443, 453 (1993). In Mitchell v. Fortis Ins. Co., 385 S.C. 570, 686 S.E.2d 176 (2009), the South Carolina Supreme Court set forth three guideposts to be applied in conducting a post-judgment

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[5] The frail idea that one's culpability is lessened by the fellow fault of one's company was memorably rejected long ago by Judge Learned Hand:

> Indeed in most cases reasonable prudence is in fact common prudence; but strictly it is never its measure; a whole calling may have unduly lagged in the adoption of new and available devices. It may never set its own tests, however persuasive be its usages. Courts must in the end say what is required; there are precautions so imperative that even their universal disregard will not excuse their omission.

The T.J. Hooper, 60 F.2d 737, 740 (2d Cir.), cert. denied 287 U.S. 662 (1932); see also Wilson v. Bradlees of New England, Inc., 96 F.3d 552 (1st Cir. 1996) ("Industry standards serve many useful purposes, but we do not think Congress, if squarely asked to address the issue, would say that such a standard should extinguish a common-law claim of design defect. If the Defendant wants to show that they met a prevailing industry standard, fine; but this should not preclude a Plaintiff from showing that industry should have done more under certain conditions.") The jury here was charged on the limited relevance of "industry standards."

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review of a punitive damages award: "(1) the degree of reprehensibility of the defendant's misconduct; (2) the disparity between the actual and potential harm suffered by the plaintiff and the amount of the punitive damages award; and (3) the difference between the punitive damages awarded by the jury and the civil penalties authorized or imposed in comparable cases." Austin v. Stokes-Craven Holding Corp., 387 S.C. 22, 691 S.E.2d 135, 150-151 (2010). These guideposts incorporate the relevant factors set forth in Gamble v. Stevenson, 305 S.C. 104, 406 S.E.2d 350 (1991) and BMW of North America v. Gore, 517 U.S. 559 (1996). See Mitchell, 385 S.C. at 587. The jury was properly charged on these factors, and their findings by clear and convincing evidence, of Crane's willful, wanton and reckless conduct is firmly anchored in the record.

Reprehensibility is the most important consideration, Mitchell, 385 S.C. at 587, for the simple reason that some things are worse than others. A number of factors are relevant to reprehensibility, including whether: "(i) the harm caused was physical as opposed to economic; (ii) the tortious conduct evinced an indifference to or a reckless disregard for the health or safety of others; (iii) the target of the conduct had financial vulnerability; (iv) the conduct involved repeated actions or was an isolated incident; and (v) the harm was the result of intentional malice, trickery, or deceit, rather than mere accident." Id.

Crane continues to deny that Mr. Garvin had exposure to asbestos from Crane valves. This was a matter for the jury, whose finding that Mr. Garvin was exposed to asbestos from Crane valves was supported by the evidence. The jury was entitled to credit the evidence that Mr. Garvin was exposed to asbestos gaskets and packing supplied with Crane's new valves, as well as to replacement parts containing asbestos that Crane's design specified or required. The harm

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caused was physical, and there was evidence of Crane's indifference to the risks posed by the asbestos products it widely distributed.

The jury did not base its award on harm to parties other than the Garvins, as Crane claims. The United States Supreme Court has recognized that "[e]vidence of actual harm to nonparties can help to show that the conduct that harmed the plaintiff also posed a substantial risk of harm to the general public, and so was particularly reprehensible . . . ." Philip Morris USA v. Williams, 549 U.S. 346, 355 (2007). A jury may consequently take into account harm to nonparties in determining reprehensibility. Id. at 357. A jury may not, however, go further and use a punitive damages verdict to punish a defendant for harm caused to nonparties. Id. The jury was specifically instructed that it could not punish for harm to other parties. Such risk of harm was merely a consideration for the jury in evaluating the willful and wanton nature of Crane's conduct.

Also relevant is the ratio between the compensatory damages award, which represents the plaintiff's actual or potential harm, and the punitive damages award. Mitchell, 385 S.C. at 588. "[A] court, when determining the reasonableness of a particular ratio of actual or potential harm to a punitive damages award, may consider: the likelihood that the award will deter the defendant from like conduct; whether the award is reasonably related to the harm likely to result from such conduct; and the defendant's ability to pay." Id. A single-digit ratio is generally thought to comport with due process. Id.

The legislature has capped punitive damages at three times the compensatory award. S.C. Code Ann. § 15-32-530. Such a cap is a public policy judgment that a 3:1 ratio is an inherently reasonable limit on punitive damages. The jury's award was well within this presumptively

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reasonable ratio.

The Court rejects Crane's contention that punitive damages should not be permitted in asbestos litigation. Punitive damages are appropriate against an asbestos manufacturer even if there are other similar claims pending against it. See Greenville v. W.R. Grace & Co., 640 F. Supp. 559, 566 (D.S.C. 1986)(Wilkins, J.), *aff'd on other grounds*, 827 F.2d 975 (4th Cir. 1987). A manufacturer is not insulated from punitive damages simply because it may have caused widespread injury. Id. It was appropriate for the jury to award punitive damages in this case, and the award does not violate due process.

III. Ruling on Motion for New Trial Nisi Remittitur

The jury award against Crane was for $10 million actual damages to Lloyd Garvin, $11 million in punitive damages to Plaintiffs Lloyd and Velda Garvin, and $1 million in compensatory damages to Plaintiff Velda Garvin. I find compelling reasons to grant a new trial nisi remittitur and reduce the jury's compensatory award to Mr. Garvin, as well as the punitive damages award.

A trial court has wide discretion to reduce an excessive verdict by granting a new trial nisi remittitur. The reduced judgment is a suggested settlement figure, which the Plaintiff may accept, or reject and request a new trial. Ruling on a new trial nisi remittitur "requires the court to consider the adequacy of the verdict in light of the evidence presented." Waring v. Johnson, 341 S.C. 248, 256, 533 S.E.2d 906, 910 (Ct. App. 2000). The touchstone for deciding whether to disturb a jury's verdict by granting a nisi motion is whether the award excessively compensates the injured party. Graham v. Whitaker, 282 S.C. 393, 321 S.E.2d 40 (1984). A famous decision by Judge Medina drives home the point: "[t]he very nature of the problem counsels restraint.

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Just as the trial judge is not called upon to say whether the amount is higher than he personally would have awarded, so are we appellate judges not to decide whether we would have set aside the verdict if we were presiding at the trial, but whether the amount is so high that it would be a denial of justice to permit it to stand." Dagnello v. Long Island R. Co., 289 F.2d 797, 806 (2d Cir. 1961).

Mr. Garvin was 74 years old at the time of trial. Although he had a life expectancy of an additional 10.78 years (S.C. Code Ann. § 19-1-150), he has suffered from a number of serious medical conditions in recent years unrelated to his asbestos exposure. Dr. Holstein testified that there were several "things that might affect [Mr. Garvin's] life span" aside from his mesothelioma, including his "history of heart disease, history of adult onset diabetes, history of chronic obstructive pulmonary disease, particularly, the asthmatic component, which can get worse at times." Additionally, there was evidence that before, during, and after Mr. Garvin was diagnosed with mesothelioma and underwent his surgery, he was on a morphine pump to allay pain from prior back surgeries.

The jury was unduly liberal in awarding Mr. Garvin $10 million in compensatory damages, and this award is excessive. This Court finds compelling reasons warrant reduction of the compensatory damages award. As set forth above, Mr. Garvin is 74 years old and suffers from several serious medical conditions wholly unrelated to his diagnosis of testicular mesothelioma and his treatment for that disease. Additionally, Mr. Garvin has been cancer-free since his surgery. Finally, Mr. Garvin's economic damages amount to $149,000.00, and, even if a likely future reoccurrence of cancer is taken into account, those future economic damages are not expected to exceed $150,000 to $300,000, for a total amount of economic damages equaling

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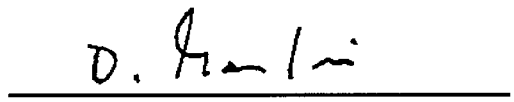
no more than $300,000 to $450,000. Thus, based upon Mr. Garvin's pecuniary damages, the nature of his injury, his treatments, his age, and his life expectancy, compelling reasons justify reducing his actual damages award from $10 million to $1.5 million.

The jury awarded Plaintiffs $11 million in punitive damages. The jury was unduly liberal in awarding this excessive amount. Based on the compelling reasons cited above, the Court grants Crane Co.'s motion for new trial nisi remittitur as to Mr. Garvin's punitive damages award, and reduces the award from $11 million to $3.5 million. The court finds this amount of punitive damages, as analyzed in light of the Mitchell factors discussed above, comports with due process.

Conclusion

The Court **GRANTS** Crane Co.'s motion for new trial nisi remittitur and reduces the compensatory award to Mr. Garvin to $1.5 million, and the punitive damages award to $3.5 million. Mr. Garvin of course may reject the remittitur, and a new trial will be scheduled. The jury's $1 million award to Ms. Garvin for loss of consortium shall stand, as there is no compelling reason to disturb it. Finally, Crane's motions for judgment notwithstanding the verdict and new trial absolute are DENIED.[6]

IT IS SO ORDERED.



D. Garrison Hill
Circuit Judge

November 14, 2014
Greenville, South Carolina

[6] Within 10 days of the date of this order, Plaintiffs shall share with Crane the settlement amounts and agreements received from third parties. The court will then schedule a hearing regarding set off at either party's request.

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STATE OF SOUTH CAROLINA COUNTY OF SPARTANBURG	)))	IN THE COURT OF COMMON PLEAS FOR THE SEVENTH JUDICIAL CIRCUIT
Beverly Dale Jolly and Brenda Rice Jolly, Plaintiffs, v. General Electric Company, et al., Defendants.	)))))))))))	Civil Action No. 2016-CP-42-01592 **DEFENDANTS FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE, LLC'S JOINT MOTION FOR *SET-OFF* PURSUANT TO S.C. CODE § 15-38-50.**

COMES NOW, Defendants Fisher Controls International LLC ("Fisher") and Crosby Valve, LLC ("Crosby"), by and through their attorneys, and hereby move the Court for an order setting off from the jury's verdict of $300,000.00 all money paid or to be paid to the Plaintiffs for Plaintiffs Beverly Dale Jolly and Brenda Rice Jolly's damages allegedly arising from his mesothelioma. For the reasons set forth herein, the Court should enter an order setting off the total judgment against Fisher and Crosby pursuant to S.C. Code Ann § 15-38-50, the Court's equitable powers, and common law.

BACKGROUND

Plaintiffs brought the instant action against sixty-three (63) Defendants, including Fisher and Crosby. Prior to trial, Plaintiffs settled with or dismissed their claims against all of the Defendants with the exception of Fisher and Crosby. The trial against Fisher and Crosby commenced on July 24, 2017 in Spartanburg County. On August 3, 2017, the jury returned a verdict against Fisher and Crosby in the amount of $300,000.[1] Pursuant to S.C. Code Ann. § 15-

[1] The jury found for Plaintiffs against Fisher and Crosby on Plaintiffs' Negligence and Breach of Implied Warranty claims, but found in favor of Fisher and Crosby on the Strict Liability claim. The Jury declined to find that Fisher and Crosby's conduct was willful, wanton, or reckless. The

38-50, Fisher and Crosby are entitled to a setoff reducing the $300,000 judgment by the total amount of money received or to be received by Plaintiffs from any alleged tortfeasor, including but not limited to any settling Defendants in this case.

I. FISHER AND CROSBY ARE ENTITLED TO SET-OFF THE JUDGMENT BY THE TOTAL AMOUNT RECEIVED OR TO BE RECEIVED BY PLAINTIFFS FROM OTHER TORTFEASORS.

A. The Set-off includes the total amount received or to be received by the Plaintiffs from any settling defendants.

Fisher and Crosby are entitled to off-set the judgment amount by the amount of money received or to be received by the Plaintiffs stemming from Mr. Jolly's mesothelioma. Section 15-38-50 of the South Carolina Code provides:

> When a release or a covenant not to sue or not to enforce judgment is given in good faith to one of two or more persons liable in tort for the same injury or the same wrongful death:
>
> (1) it does not discharge any of the other tortfeasors from liability for the injury or wrongful death unless its terms so provide, but it reduces the claim against the others to the extent of any amount stipulated by the release or the covenant, or in the amount of the consideration paid for it, whichever is the greater; and
>
> (2) it discharges the tortfeasor to whom it is given from all liability for contribution to any other tortfeasor.

S.C. Code Ann. § 15-38-50 (2012).

South Carolina courts have found this reduction is mandatory. *See, e.g., Smith v. Widener*, 397 S.C. 468, 471-72, 724 S.E.2d 188, 190 (Ct. App. 2012); *Ellis v. Oliver*, 335 S.C. 106, 515 S.E.2d 268 (Ct. App. 1999); *see also Welch v. Epstein*, 342 S.C. 279, 313, 536 S.E.2d 408, 425-26 (Ct. App. 2000); *Truesdale v. South Carolina Hwy Dep't*, 264 S.C. 221, 213 S.E.2d 740 (1975) (noting that the purpose of allowing setoff credit is to prevent an injured person from

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Jury awarded $200,000 actual damages to Plaintiff Beverly Dale Jolly and $100,000.00 to Brenda Rice Jolly on her loss of consortium claim.

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obtaining a second recovery for the amount of damages sustained which has already been paid to him). Moreover, even prior to the enactment of S.C. Code Ann. § 15-38-50, South Carolina courts have held for over 100 years that trial courts should equitably set off prior settlements against a judgment, to prevent plaintiffs from receiving a double recovery. *See, e.g.*, *Rookard v. Atlantic & Charlotte Air Line Railway Co.*, 89 S.C. 371, 71 S.E. 992, 995 (1911); *Powers v. Temple*, 250 S.C. 149, 156 S.E.2d 759 (1967); *Truesdale v. South Carolina Highway Dep't*, 264 S.C. 221, 234-35, 213 S.E.2d 740, 746 (1975), overruled on other grounds ("[O]ne tort feasor is entitled to credit for the amount paid by another tort feasor for a covenant not to sue. . . . The reason for allowing such a credit is to prevent an injured person from obtaining a second recovery of that part of the amount of damages sustained which has already been paid to him. Or differently stated, it is almost universally held that there can be only one satisfaction for an injury or wrong.").

In *Smith v. Widener*, the court provided the legal framework for setoff in South Carolina:

> There can be only one satisfaction for an injury or wrong. A settlement by a joint tortfeasor reduces the claim against the others to the extent of any amount stipulated by the release or the covenant. Therefore, before entering judgment on a jury verdict, the court must reduce the amount of the verdict to account for any funds previously paid by a settling defendant, so long as the settlement funds were paid to compensate the same plaintiff on a claim for the same injury. When the settlement is for the same injury, the nonsettling defendant's right to a setoff arises by operation of law. Under this circumstance, section 15–38–50 grants the court no discretion in applying a set-off.

397 S.C. at 471–72, 724 S.E.2d at 190 (internal quotations, citations, and brackets omitted). The law in South Carolina is clear. Fisher and Crosby are entitled to have this Court set-off any judgment by the total amount received by or promised to the Plaintiffs from other alleged tortfeasors. *See also Smith v. Tiffany*, 419 S.C. 548, 557, 799 S.E.2d 479, 484 (2017) (noting that "the General Assembly took steps to protect nonsettling defendants by codifying . . .

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the right to offset the value of any settlement received prior to the verdict—a right which arises by operation of law and is not within the discretion of the courts").

This setoff is required regardless of what allocations Plaintiffs make with their settlements proceeds. *See Ellis v. Oliver*, 335 S.C. 106, 515 S.E.2d 268 (Ct. App. 1999); *Vortex Sports & Entm't, Inc. v. Ware*, 378 S.C. 197, 210, 662 S.E.2d 444, 451 (Ct. App. 2008). In *Ellis*, the plaintiff brought negligence causes of action against a hospital and wrongful death against a doctor. 335 S.C. at 112. The plaintiff appealed the trial court's set-off of her award from the doctor with her settlement with the hospital. *Id.* The plaintiff contended because the measure of damages was different for the two causes of action, then two different injuries occurred and no setoff was necessary. *Id.* at 113. The Court of Appeals disagreed, and found that the plaintiff was confusing the concept of damages with the statutory word "injury." The *Ellis* court made clear:

> Injury, as used in the statute, is broad enough to include all damages, including those attributable to both survival and wrongful death causes of action which result from the joint negligence of the various responsible parties.

Id. at 113. Thus, it is not relevant how Plaintiffs allocated the settlement proceeds which they have received. Fisher and Crosby are entitled to set-off the full amount of money received by the Plaintiffs for the injuries allegedly caused by Mr. Jolly's mesothelioma. *See also* Welch, 342 S.C. at 312 ("A nonsettling defendant is entitled to credit for the amount paid by another defendant who settles. The reason for allowing such a credit is to prevent an injured person from obtaining a second recovery of that part of the amount of damages sustained which has already been paid to him.").

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B. The Set-Off includes any and all amounts received or to be received by the Plaintiffs from bankruptcy trusts.

Fisher and Crosby are likewise entitled to a set-off of any and all proceeds received or to be received from bankruptcy trusts associated with Mr. Jolly's occupational exposures to asbestos. As the Court is aware, collections from the various bankruptcy trusts is often a substantial source of recovery for Plaintiffs in asbestos-related litigation. *See, e.g., In re Garlock Sealing Techs.*, LLC, 504 B.R. 71, 83-84 (Bankr. W.D.N.C. 2014) (discussing "bankruptcy wave" in asbestos litigation); *see also* Victor E. Schwartz & Mark A. Behrens, Asbestos Litigation: The "Endless Search for a Solvent Bystander" 23 Widener L.J. 59, 70-71 (2013) (noting that the "'high dose' defendants–makers of friable, amphibole fiber asbestos products–[who] bore primary responsibility . . . . [are] no longer subject to suit" because they filed for bankruptcy and set up trusts to compensate future plaintiffs, and, "[a]s trust proceeds became available to pay claims, the litigation morphed into a two-tiered system of asbestos bankruptcy trust claims and tort claims") (citations omitted). The fact that Plaintiffs apparently chose to wait and file these claims until after the trial against Fisher and Crosby occurred is predictable, but of no consequence. Fisher and Crosby are entitled to a set-off of the full amount of money received by the Plaintiffs for Mr. Jolly's alleged injury, including any funds received from bankruptcy trusts. *See* S.C. CODE ANN. § 15-38-50 (2012); *Reed v. Honeywell Int'l, Inc.*, No. 3022 EDA 2010, 2011 WL 6645694, at *10 (Pa. Super. Ct. Dec. 6, 2011) (finding various bankrupt entities joint tortfeasors for the purpose of offsetting the jury's award); *Lewin v. Am. Exp. Lines, Inc.*, 224 F.R.D. 389, 395-96 (N.D. Ohio 2004) ("The setoff will include any payments that Plaintiffs have received for the asbestos-type injuries to Lewin at issue here. This includes settlements and payments from any bankruptcy trusts."); *Matter of New York City Asbestos Litig. (Dummit v. A.W. Chesterton et al.)*, 36 Misc. 3d 1234(A), 960 N.Y.S.2d 51, 2012 N.Y. Misc. LEXIS 4057,

at * 70-79 (N.Y. Sup. Ct. 2012), *aff'd by Matter of New York City Asbestos Litig.*, 121 A.D.3d 230 960 N.Y.S.2d 51 (N.Y. App. Div. 1st Dep't 2014) (granting defendant's motion for remittitur and ordering the parties to appear at a hearing to determine what the setoff would be after the remittitur, based on what plaintiff had received from settlements with other defendants and from bankruptcy trusts); *Anderson v. Ford Motor Co.*, No. 09-69122, 2010 U.S. Dist. LEXIS 112600 (E.D. Pa. Oct. 20, 2010) (holding "that plaintiffs are not required to disclose their settlement agreements with bankruptcy trusts" early in the discovery process, because "that disclosure of settlements [from bankruptcy trusts] should be deferred until after the entry of the judgment when the court would entertain a defendant's set-off argument") (citations omitted); *Barabin v. Astenjohnson, Inc.*, No. C07-1454RSL, 2010 U.S. Dist. LEXIS 102397, at *7-11 (W.D. Wash. Sept. 13, 2010), rev'd on other grounds (using money received by plaintiffs from bankruptcy trust claims to calculate setoff).

CONCLUSION

Accordingly, for the reasons set forth herein, Fisher and Crosby respectfully request the Court to reduce the amount of judgment against it by the total amounts paid or to be paid by any settling defendant and bankrupt entities.

Respectfully submitted this, the 14th day of August, 2017.

LEATH, BOUCH & SEEKINGS, LLP

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STATE OF SOUTH CAROLINA COUNTY OF SPARTANBURG	)))	IN THE COURT OF COMMON PLEAS FOR THE SEVENTH JUDICIAL CIRCUIT
Beverly Dale Jolly and Brenda Rice Jolly, Plaintiffs, v. General Electric Company, et al., Defendants.	)))))))))))	Civil Action No. 2016-CP-42-01592 **DEFENDANTS FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE, LLC'S JOINT MOTION FOR PRODUCTION OF PLAINTIFFS' SETTLEMENTS AND PAYMENTS WITH ALL THIRD PARTY TORTFEASORS**

COMES NOW, Defendants Fisher Controls International LLC ("Fisher") and Crosby Valve, LLC ("Crosby"), and move the court for an order compelling Plaintiffs to produce competent evidence pertaining to all settlement amounts or payments received now or in the future from any and all third parties arising out of Plaintiffs injuries relating to his mesothelioma. This Motion is made pursuant to Rule 26 of the South Carolina Rules of Civil Procedure, Section 15-38-50 of the South Carolina Code, and the following arguments and legal authorities.

BACKGROUND

Plaintiffs brought the instant action against sixty-three (63) Defendants, including Fisher and Crosby. Prior to trial, Plaintiffs settled with or dismissed their claims against all of the Defendants with the exception of Fisher and Crosby. The trial against Fisher and Crosby commenced on July 24, 2017 in Spartanburg County. On August 3, 2017, the jury returned a verdict against Fisher and Crosby in the amount of $300,000. Specifically, the jury found for Plaintiffs against Fisher and Crosby on Plaintiffs' Negligence and

Breach of Implied Warranty claims, but found in favor of Fisher and Crosby on the Strict Liability claim. The Jury declined to find that Fisher and Crosby's conduct was willful, wanton, or reckless. The Jury awarded $200,000 actual damages to Plaintiff Beverly Dale Jolly and $100,000 to Brenda Rice Jolly on her loss of consortium claim.

Pursuant to S.C. Code Ann. § 15-38-50, Fisher and Crosby are entitled to a setoff reducing the $300,000 verdict by the total amount of money received by Plaintiffs from any alleged tortfeasor, including but not limited to the other settling Defendants in this case or any bankrupt entity. As such, this Court should compel Plaintiffs to produce evidence and permit access to all pertinent setoff information, including access to all settlement amounts and settlement documents that Plaintiffs or their counsel possess related to their claims (and/or potential claims) asserted in this lawsuit against any person or entity, regardless of whether the settlements occurred within the specific context of this lawsuit.

ARGUMENT

There is no question that Fisher and Crosby are entitled to a setoff reducing the Plaintiffs' verdict by the total amount of Plaintiffs' settlements, regardless of the way the settlements were allocated. *See* S.C. CODE ANN. § 15-38-50; *see also Ellis v. Oliver*, 335 S.C. 106, 515 S.E.2d 268 (Ct. App. 1999); *Welch v. Epstein*, 342 S.C. 279, 313, 536 S.E.2d 408, 425-26 (Ct. App. 2000); *Truesdale v. South Carolina Hwy Dep't*, 264 S.C. 221, 213 S.E.2d 740 (1975) (noting that the purpose of allowing setoff credit is to prevent an injured person from obtaining a second recovery for the amount of damages sustained which has already been paid to him). Fisher and Crosby are contemporaneously filing a Motion for Setoff, and hereby incorporate that Motion and its arguments herein. Given

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the requirements set forth in section 15-38-50 of the South Carolina Code, all information and documents related to Plaintiffs' settlements with third parties are relevant and discoverable under South Carolina law, including proceeds from bankruptcy trusts.

I. FISHER AND CROSBY ARE ENTITLED TO DISCLOSURE OF ALL SETTLEMENT INFORMATION AND SETTLEMENT DOCUMENTS BETWEEN PLAINTIFFS AND THIRD PARTIES.

In South Carolina, the discovery of relevant information is very broad. *Samples v. Mitchell*, 329 S.C. 105, 110, 495 S.E.2d 213, 215 (Ct. App. 1997); *Hamm v. S.C. Pub. Serv. Comm'n*, 312 S.C. 238, 241, 439 S.E.2d 852, 853 (1994); *Oncology & Hematology Assocs. of S.C., LLC v. S.C. Dep't of Health & Envtl. Control*, 387 S.C. 380, 387, 692 S.E.2d 920, 924 (2010). Rule 26 provides:

> In General. Parties may obtain discovery regarding *any matter, not privileged, which is relevant to the subject matter involved in the pending action*, whether it relates to the claim or defense of the party seeking discovery or to the claim or defense of any other party, including the existence, description, nature, custody, condition and location of any books, documents, or other tangible things and the identity and location of persons having knowledge of any discoverable matter. It is not ground for objection that the information sought will be inadmissible at the trial if the information sought appears reasonably calculated to lead to the discovery of admissible evidence.

Rule 26(b)(1), SCRCP (emphasis added). The Rule makes clear that relevant information is obtainable regardless of its admissibility. The information pertaining to settlements with third parties is relevant for the purposes of determining the mandatory set-off requirements of S.C. CODE ANN. § 15-38-50. Rule 401 provides that relevant evidence "means evidence having any tendency to make the existence of any fact that is of consequence to the determination of the action more probable or less probable than it would be without the evidence." Rule 401, SCRE. Here, evidence relating to all

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payments from third parties to the Plaintiffs is highly relevant to determining the amount that Fisher and Crosby are entitled to set-off from the jury's verdict.

Moreover, Rule 408 of the South Carolina Rules of Evidence specifically provides that settlement information is discoverable even though the same evidence is not admissible. *See* Rule 408, SCRE ("This rule does not require the exclusion of any evidence otherwise discoverable merely because it is presented in the course of compromise negotiations."). Similarly, it is black letter law that South Carolina does not permit plaintiffs to a double recovery. *See e.g.*, *Rutland v. S.C. Dep't of Transp.*, 400 S.C. 209, 216, 734 S.E.2d 142, 145 (2012); *Welch*, 342 S.C. at 312–13, 536 S.E.2d at 425; *Truesdale v. S.C. Highway Dept.*, 264 S.C. 221, 235, 213 S.E.2d 740, 746 (1975). Here, without full access to the records pertaining to all of the money obtained by Plaintiffs as a result of their alleged injuries, it is impossible for Fisher and Crosby – and this Court – to determine the proper amount to setoff the $300,000 verdict. *Rutland*, 400 S.C. 209, 216, 734 S.E.2d 142, 145; *see also Polston v. Eli Lilly & Co.*, 2010 WL 2926159, at *2 (D.S.C. July 23, 2010) (noting the disclosure of settlement documents is warranted after jury verdict for setoff purposes). Moreover, in order to avoid an inequitable apportionment and setoff, evidence pertaining to Plaintiffs' efforts to obtain additional funds from the various bankruptcy trust must be included. *See id*; *Reed v. Honeywell Int'l, Inc.*, No. 3022 EDA 2010, 2011 WL 6645694, at *10 (Pa. Super. Ct. Dec. 6, 2011) (finding various bankrupt entities joint tortfeasors for the purpose of offsetting the jury's award); *In re Garlock Sealing Techs.*, LLC, 504 B.R. 71, 83-84 (Bankr. W.D.N.C. 2014) (noting plaintiffs' ability to use bankruptcy trusts to manipulate asbestos litigation).

CONCLUSION

For the reasons stated herein, Fisher and Crosby respectfully request the court to require Plaintiffs to produce all pertinent setoff information to Fisher and Crosby, including

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settlement information with other third party defendants and third party bankruptcy trusts, to include future potential claims. Without this information, Fisher and Crosby cannot receive the setoff that section 15-38-50 directs.

Respectfully submitted this, the 14th day of August, 2017.

LEATH, BOUCH & SEEKINGS, LLP

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STATE OF SOUTH CAROLINA	)	
	)	IN THE COURT OF COMMON PLEAS
COUNTY OF SPARTANBURG	)	SEVENTH JUDICIAL CIRCUIT
Beverly Dale Jolly and Brenda Rice Jolly,	)	Civil Action No. 2016-CP-42-01592
Plaintiffs,	)	
vs.	)	**DEFENDANT FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE, LLC'S JOINT MOTION FOR JNOV**
General Electric Company, et al.,	)	
Defendants.	)	

COMES NOW, Defendants Fisher Controls International LLC ("Fisher") and Crosby Valve, LLC ("Crosby"), Pursuant to Rule 50(b) of the South Carolina Rules of Civil Procedure and relevant case law, and respectfully move this Court for a Judgment Notwithstanding the Verdict ("JNOV"). Fisher and Crosby are entitled to a JNOV as to all claims because: (1) the potential hazards posed by asbestos allegedly used in connection with Defendants' products were open and obvious, (2) the potential hazards posed by asbestos allegedly used in connection with Defendants' products were known to Mr. Jolly, (3) Fisher and Crosby are protected from liability by the sophisticated intermediary doctrine as a matter of law [because, among other reasons, Plaintiff's employer knew of the asbestos dangers and warned or should have warned of dangers associated therewith], (4) Plaintiffs failed to show that the Fisher and Crosby products allegedly at issue were in essentially the same condition at the time of exposure or injury to Plaintiff as when they left the hands of the Defendants, (5) Plaintiffs failed to show Fisher and Crosby's alleged failure to warn was the proximate cause of their injuries [because, among other reasons, Plaintiffs put in no evidence that a warning would have been heeded or conduct would have changed based on any particular warning], (6) Plaintiffs failed to present evidence of an

alternative feasible design or meet the required showing under the risk/utility test of *Branham v. Ford Motor Company* because Mr. Jolly's employer ordered the particular products involved based on business need, and Plaintiffs failed to show an alternative feasible alternative product available that would have the requisite utility without undue cost, (7) Plaintiffs' negligence claims fail because they did not show Fisher or Crosby deviated from the standard of care, nor did Plaintiff prove the existence of any standard of care, (8) Defendants are entitled to judgment as a matter of law because they did not manufacture asbestos-containing products at issue, nor was there proof that Defendants sold asbestos-containing products which were proven to have been exposed to Mr. Jolly in a form that could have led to injury, (9) Plaintiffs failed to show Mr. Jolly's exposure to asbestos from the defendants' products was a proximate cause of his mesothelioma [because, among other reasons, Plaintiffs failed to establish specific causation through relevant, reliable expert testimony that would assist jury in reaching a verdict], and (10) Plaintiffs failed to prove causation under the *Henderson/Lohrmann* standard, by failing to make the requisite showing of regularity, proximity, and frequency.

Pursuant to the Court's instruction, this Motion will be further supported by a memorandum of law to be filed on a later date to be determined by the Court. Fisher and Crosby respectfully request that this Court grant their motion for a JNOV on all of Plaintiffs' Claims.

Respectfully submitted,

LEATH, BOUCH & SEEKINGS, LLP

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August 14, 2017

Charleston, South Carolina

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STATE OF SOUTH CAROLINA	)	
	)	IN THE COURT OF COMMON PLEAS
COUNTY OF SPARTANBURG	)	SEVENTH JUDICIAL CIRCUIT
Beverly Dale Jolly and Brenda Rice Jolly,	)	Civil Action No. 2016-CP-42-01592
	)	
Plaintiffs,	)	
	)	
vs.	)	**DEFENDANT FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE, LLC'S MEMORANDUM IN SUPPORT OF THEIR JOINT MOTION FOR JNOV**
	)	
General Electric Company, et al.,	)	
	)	
Defendants.	)	
	)	

COME NOW, Defendants Fisher Controls International LLC ("Fisher") and Crosby Valve, LLC ("Crosby"), pursuant to the South Carolina Rules of Civil Procedure, and respectfully submit this Memorandum in Support of their Joint Motion for Judgment Notwithstanding the Verdict ("JNOV") in the above captioned matter. As set forth more fully below, Fisher and Crosby are entitled to judgment as a matter of law.

BACKGROUND

Plaintiffs brought the instant action against sixty-three (63) Defendants, including Fisher and Crosby. Prior to trial, Plaintiffs settled with or dismissed their claims against all of the Defendants except Fisher and Crosby. The trial against Fisher and Crosby commenced on July 24, 2017 in Spartanburg County. After the close of Plaintiffs' case, Fisher and Crosby moved for a directed verdict. The Court denied the Motion. After the close of all of the evidence, Fisher and Crosby again moved for a directed verdict. The Court denied the Motion.

On August 3, 2017, the jury returned a verdict against Fisher and Crosby in the amount of $300,000. More specifically, the jury found for Plaintiffs against Fisher and Crosby on Plaintiffs' Negligence and Breach of Implied Warranty claims, but found in favor of Fisher and Crosby on

Plaintiffs' Strict Liability claim. The jury declined to find that Fisher and Crosby's conduct was willful, wanton or reckless. The jury awarded $200,000 in actual damages to Plaintiff Beverly Dale Jolly and $100,000 to Brenda Rice Jolly on her loss of consortium claim. The Court ordered the parties to file Post-Trial Motions within ten days pursuant to Rule 50(e) and Rule 59(b) of the South Carolina Rules of Civil Procedure. The parties filed post-trial motions within that time. Included in those motions was the JNOV Motion here at issue, which Fisher and Crosby jointly filed on August 14, 2017.

For the reasons set forth more fully below, the Court should grant these Defendants' Joint Motion for JNOV, because:

(1) Plaintiffs' failure to warn claims (in both negligence and breach of warranty) fail because the potential hazards of asbestos allegedly used in connection with Defendant's products were open and obvious, and additionally because they were known to Mr. Jolly;

(2) Fisher and Crosby are protected from liability on all of Plaintiffs' claims by the sophisticated intermediary doctrine;

(3) Plaintiffs' claims fail as a matter of law because they failed to show that the Fisher and Crosby valves Mr. Jolly encountered were in essentially the same condition at the time of the exposure and injury to Mr. Jolly as when they left the hands of the Defendants;

(4) Plaintiffs' failure to warn claims also fail because they did not show that the Defendants' alleged failure to warn was a proximate cause of their injuries, as Plaintiffs failed to offer any evidence that Mr. Jolly would have heeded a warning, and in fact the uncontroverted evidence demonstrated the opposite;

(5) Plaintiffs failed to meet their burden of proof as to their design defect claims (again, in both negligence and breach of warranty) because they did not present evidence of an alternative, feasible design, or meet the required showing under the risk/utility test set forth in *Branham v. Ford Motor Company*;

(6) Plaintiffs' negligence claims failed as a matter of law because they did not present evidence of the applicable standard of care or evidence that Fisher and Crosby deviated from the applicable standard of care;

(7) Defendants are entitled to judgment as a matter of law because there was no evidence whatsoever showing that they manufactured, sold, or otherwise placed into the stream of

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commerce any of the asbestos-containing products to which Mr. Jolly was allegedly exposed on a regular and frequent basis.

(8) Plaintiffs failed to establish that Mr. Jolly's injury was proximately caused by these Defendants' products because the only specific causation testimony they offered was scientifically unreliable and inconsistent with the law, and it therefore should have been excluded; and

(9) Plaintiffs failed to prove causation under the *Henderson/Lohrmann* standard, because they did not make the requisite showing of regularity, frequency, and proximity.

LEGAL STANDARD

On a motion for a directed verdict or a JNOV, this Court should view the evidence and all reasonable inferences in the light most favorable to the nonmoving party. *See, e.g., RFT Mgmt. Co. v. Tinsley & Adams L.L.P.*, 399 S.C. 322, 331–32, 732 S.E.2d 166, 171 (2012). The Court must deny a motion for a directed verdict or JNOV if the evidence yields more than one reasonable inference or its inference is in doubt. *See Strange v. S.C. Dep't of Highways & Pub. Transp.*, 314 S.C. 427, 429, 445 S.E.2d 439, 440 (1994). However, "if no reasonable jury could have reached the challenged verdict," the Court must grant a motion for JNOV. *Gastineau v. Murphy*, 331 S.C. 565, 568, 503 S.E.2d 712, 713 (1998). Moreover, if the undisputed evidence shows that Plaintiffs' claims are barred as a matter of law, JNOV should properly be granted. *See, e.g., Rogers v. Norfolk S. Corp.*, 356 S.C. 85, 91, 588 S.E.2d 87, 90 n.3 (2003) ("As a practical matter South Carolina's Motion for JNOV and the federal Motion for Judgment as a Matter of Law are the same.").

ARGUMENT

I. PLAINTIFFS FAILED TO MEET THEIR BURDEN OF PROOF ON THEIR FAILURE TO WARN CLAIMS SOUNDING IN NEGLIGENCE AND WARRANTY.

Plaintiffs failed to meet their burden of proof as to several elements of their failure to warn claims. To prove liability under a products liability theory -- regardless of whether the claim sounds in negligence, warranty, or strict liability -- Plaintiffs were required to show: (1) they were injured

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by the product; (2) the injury occurred because the product was in a defective condition, unreasonably dangerous to the user; and (3) that the product at the time of the accident was in essentially the same condition as when it left the hands of the defendant. *Bragg v. Hi-Ranger, Inc.*, 319 S.C. 531, 539, 462 S.E.2d 321, 326 (Ct. App. 1995); *Dema v. Shore Enters., Ltd.*, 312 S.C. 528, 530, 435 S.E.2d 875, 876 (Ct. App. 1993).

In addition, a manufacturer of a product is liable for failure to warn if (1) it knows or has reason to know the product is or is likely to be dangerous for its intended use; (2) it has no reason to believe the user will realize the potential danger; and (3) it fails to exercise reasonable care to inform of its dangerous condition or of the facts which make it likely to be dangerous. *Livingston v. Noland Corp.*, 293 S.C. 521, 525, 362 S.E.2d 16, 18 (1987). Plaintiffs who fail to prove each of these elements cannot recover on a failure to warn claim under any theory of recovery.

A. The potential hazards posed by asbestos allegedly used in connection with Defendants' valves were open and obvious and well known to Plaintiff, as Mr. Jolly admitted during his testimony.

A manufacturer has no duty to warn of an "open and obvious" danger created by its products nor one that the product's users generally recognize. *Moore v. Barony House Rest., LLC*, 382 S.C. 35, 41–42, 674 S.E.2d 500, 504 (Ct. App. 2009) ("[A] product is not defective for failure to warn of an open and obvious danger. . . . A seller is not required to warn of dangers or potential dangers that are generally known and recognized. It follows, then, that a product cannot be deemed either defective or unreasonably dangerous if a danger associated with the product is one that the product's users generally recognize.") (internal citations and quotations omitted). Similarly, the failure to provide a warning is inconsequential where a plaintiff admits he knew of the dangers. *See Rife v. Hitachi Const. Mach. Co.*, 363 S.C. 209, 220, 609 S.E.2d 565, 571 (Ct. App. 2005) (citing *Marchant v. Mitchell Distrib. Co.*, 270 S.C. 29, 37, 240 S.E.2d 511, 514 (1977)); *see also*

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Owings v. Moneynick Oil Mill, 55 S.C. 483, 33 S.E. 511, 512–13 (1899) ("There was not only no testimony in the case tending to show that the plaintiff was ignorant of the danger to which he was exposed in being put to work near the electric wire, but it shows, on the contrary, that the plaintiff was fully aware of the danger, and hence no warning was necessary. The plaintiff's own testimony shows that he was fully aware of the danger of coming in contact with the electric wires . . . . Any warning which might have been given would have been entirely useless, and therefore the defendant neglected no duty in failing to give such warning.").

The undisputed evidence at trial demonstrates that Mr. Jolly knew of the potential hazards of working with asbestos-containing products, and those potential hazards were therefore open and obvious to him and his co-workers at Duke Power Company. Numerous exhibits show Duke was warning its employees about the hazards of asbestos years before Jolly began working at Duke, and it continued to warn its employees during Jolly's tenure.[1] Moreover, Mr. Jolly admitted, based on information Duke conveyed to him, that he always knew asbestos could be hazardous.[2]

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[1] *See, e.g.*, Defendants' Exhibits 134 (1977 Duke Power document detailing safe working practices when working with asbestos); 135 (revising safe working practices for working with asbestos); 136 (1984 Duke Power guidelines on how to protect employees from asbestos); 137 (January 1, 1985 revised safe working practices for asbestos exposure at Duke Power).

[2] *See* 7/25/17 Trial Tr., at 119:13–120:8 ("Q Warning signs shall be provided and displayed at each location where asbestos dust hazards are present did Duke have such signs? A Well -- ***well they warned us.*** I'll put it like this. I couldn't tell you if a piece of dust on the floor came from insulation. Could I not tell you. Could I not tell you -- I couldn't identify it. And I wasn't looking for it. So I wasn't looking for it. ***I mean we know I think asbestos was bad for you*** but I didn't know how bad it was for me at the time. And a lot of other people didn't. But I didn't specifically go around looking down at the floor, could you get killed. You had to watch where you was going and I couldn't tell you -- and we were specifically – in fact, a guy on another crew almost got fired because he took off a piece of installation himself, he removed it. We could not remove it. That wasn't our job. We didn't remove insulation. ***We didn't touch it if we didn't have to. We didn't have to and we didn't touch it so you knew those restrictions were there you know. We didn't -- we tried to avoid the insulation you know.***") (emphasis added); *id.* at 126:10-17 ("Q So is it fair to say in terms of warning that was Duke taking on responsibility to provide warnings for anything on its premise correct? A ***Yes there were warning signs. They had them everywhere. You see them everywhere. I mean everywhere you go*** you wouldn't believe the size of these building.

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Nonetheless, Mr. Jolly continued to work with asbestos-containing products. Even viewing the evidence and all reasonable inferences in the light most favorable to Plaintiffs, a reasonable fact finder could reach only one conclusion: the hazards of asbestos-containing products were open and obvious because Mr. Jolly admitted that he and his co-workers knew of those hazards at all times when Mr. Jolly could have come into contact with these Defendants' products. Thus, to paraphrase *Owings, supra*, "there was not only no testimony in the case tending to show that [Mr. Jolly] was ignorant of the danger to which he was exposed . . . but [the testimony] shows, on the contrary, that [Jolly] was fully aware of the danger, and hence no warning was necessary. . . . Any warning which might have been given would have been entirely useless, and therefore the defendant neglected no duty in failing to give such warning." *Owings*, 55 S.C. 483, 33 S.E. at 512–13. Hence, Fisher and Crosby are entitled to a JNOV on all of Plaintiffs' failure to warn claims.

B. Fisher and Crosby are protected from liability by the sophisticated intermediary doctrine as a matter of law.

The sophisticated intermediary doctrine is based on the principle that, when there is no effective way for a manufacturer to convey a product warning to the ultimate consumer or user,

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Some of them are football fields long and wide you know.") (emphasis added); *id.* at 126:20–127:15 ("Q Duke was a user of all these asbestos products from insulation to asbestos containing gaskets or packing correct? . . . Q Duke used all these products correct? A Yes. Q Insulation gaskets packing? A Yes at one time or the other yes. . . . Q Duke was knowledgeable about the hazards of asbestos correct? A I would say so. ***Q They had a warning after all? A Yes, they did.*** Q They were a sophisticated company? A I would say so yes I think so. ***Q Now when you saw these warning labels and you saw work being done on gaskets did you get a respirator? A Well if they had a sign up they did if they didn't I didn't you know.*** We didn't have to because it wasn't you know we were told when to use the respirators.") (emphasis added); *id.* at 130:14-20 ("Q In any event you were never handed a procedure on how to deal with asbestos in that 80 to 84 period correct? A ***We were told not to take off any insulation you know or be around when it they were taking it out or installing it. We knew that now you know. I knew not to do that. We all knew that.***") (emphasis added).

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the manufacturer should be permitted to rely on downstream suppliers to provide the warning. *See, e.g.*, *Webb v. Special Elec. Co.*, 63 Cal. 4th 167, 186, 370 P.3d 1022, 1034 (2016). More specifically, the sophisticated intermediary doctrine allows a seller to "discharge its duty to warn end users about known or knowable risks in the use of its product" if it (1) "provides adequate warnings to the product's immediate purchaser, ***or*** sells to a sophisticated purchaser that it knows is aware or should be aware of the specific danger," and (2) "reasonably relies on the purchaser to convey appropriate warnings to downstream users who will encounter the product." *Id.* (emphasis added). A manufacturer can satisfy the first prong by warning the intermediary of the hazard. *Id.* at 1035. However, a manufacturer is not required to warn the intermediary if the intermediary is "so knowledgeable about the material supplied that it knew or should have known about the particular danger." *Id.* As to the second prong, courts generally consider three factors: (1) the gravity of the risk posed by the product, (2) the likelihood that the intermediary will warn those downstream, and (3) the feasibility or effectiveness of the manufacturer giving a warning directly to the user. *Id.* at 1036.

The first factor weighs the reasonableness of the manufacturer's conduct against the potential severity of the harm. *Id.* at 1037. The second factor focuses on the reliability of the intermediary. *Id.* The manufacturer's knowledge about the reliability of the intermediary is determined using an objective standard based on what a reasonable manufacturer would have known under the circumstances. *Id.* In an analysis of the second factor, it is significant if the intermediary itself had a legal duty to warn end users about the hazard. *Id.* "In general, 'every person has a right to presume that every other person will perform his duty and obey the law.'" *Id.* (citation omitted). The third factor focuses on what the manufacturer can reasonably accomplish to warn end users of the risks. *Id.*

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Applying the sophisticated intermediary doctrine to this case, Fisher and Crosby cannot be held liable for failing to warn Duke Power employees like Mr. Jolly of the potential hazards of asbestos. Fisher and Crosby sold valves to Duke. It is undisputed that Duke was a sophisticated entity at all relevant times in this case. Documentary evidence showed Duke was knowledgeable about the hazards of asbestos leading up to and during Mr. Jolly's career at its power plants.[3] Further, Mr. Jolly testified that Duke was a sophisticated entity who warned him and his co-workers about the hazards of asbestos from the very beginning of his tenure at Duke.[4] Similarly, Plaintiffs' own expert, Dr. Arthur Frank, agreed that Duke documents indicate that Duke was warning its employees about the hazards of asbestos, consistent with its policies and mandates under OSHA, prior to and during the time that Mr. Jolly worked for Duke.[5] In fact, when specifically asked by Plaintiffs' own counsel, Dr. Frank suggested four things Fisher and Crosby could have done to warn Duke or its employees:

> Q: So the question I have is *what is your opinion Dr. Frank as to what Crosby valve company or Fisher Controls could have told Duke Power in 1979 or 1980 about whether there was a hazard from the regular foreseeable maintenance of valves that they had sold to Duke Power*?
>
> * * *
>
> MR. BOUCH: Same objection I've been making for the record assumes facts not in evidence incomplete hypothetical.
>
> THE COURT: Overruled.
>
> THE WITNESS: *What could have been shared by the companies supplying asbestos containing materials was that their product contained asbestos and that asbestos was known to be hazardous and that the people working with their product should be cautioned about it about using the product about liberating dust and if they were going to do that to do it in a manner that would reduce their exposures.*[6]

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[3] *See* Note 1, *supra*.
[4] *See* Note 2, *supra*.
[5] *See* 7/26/17 Trial Tr., at pp. 160–75.
[6] *Id.* at 108:9 – 109:4 (emphasis added).

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Dr. Frank then admitted that Duke *already knew each of these things* by the time Mr. Jolly arrived to work there.[7] Accordingly, Duke qualifies as a sophisticated buyer "so knowledgeable about the material supplied that it knew or should have known about the particular danger," and Fisher and Crosby satisfied the first prong of the sophisticated intermediary doctrine as a matter of law. *Webb*, 370 P.3d at 1035.

Fisher and Crosby also satisfied the second prong of the sophisticated intermediary doctrine: Duke was a reliable intermediary. *See id.* at 1037. The documentary evidence and admissions by Plaintiffs' own experts show that Duke had a duty to warn (and protect) its employees of the potential hazards of asbestos-containing products pursuant to OSHA regulations, *and it complied with that duty during Mr. Jolly's entire career at Duke.* Fisher and Crosby were entitled to presume that Duke would comply with its obligations. *See id.*

Finally, Fisher and Crosby were not positioned to realistically warn Mr. Jolly about the alleged hazards of asbestos connected to their valves. The entity so positioned was Duke. Fisher and Crosby therefore satisfy the requirements of the sophisticated intermediary doctrine as a matter of law, and they are therefore entitled to a JNOV on all of Plaintiffs' claims.

C. Plaintiffs failed to show that the Fisher and Crosby valves allegedly at issue were in essentially the same condition at the time of Jolly's alleged exposure or injury as when they left the hands of the Defendants.

It is well settled that plaintiffs in a products liability case, regardless of their theory of recovery, must show that the allegedly defective product, at the time of the accident, "was in essentially the same condition as when it left the hands of the defendant." *Madden v. Cox*, 284

[7] *See id.* at 181:3-20 (admitting, after looking at Duke Power documents, that Duke Power ordered and knew it was receiving asbestos gaskets, knew the hazards of asbestos since at least 1964, was under a responsibility to reduce asbestos hazards pursuant to OSHA regulations beginning at least in 1972, and knew how to reduce exposures through various methods).

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S.C. 574, 579, 328 S.E.2d 108, 112 (Ct. App. 1985) (citing W. Prosser, Law of Torts 671-72 (4th ed. 1970); S.C. Code Ann. §§ 15-73-10, 36-2-318, 36-2-715; *Claytor v. General Motors Corp.*, 277 S.C. 259, 286 S.E. 2d 129 (1982); *Logan v. Montgomery Ward & Co., Inc.*, 216 Va. 425, 219 S.E. (2d) 685 (1975)); *see also Graves v. CAS Med. Sys.*, 401 S.C. 63, 79, 735 S.E.2d 650, 658 (2012) ("In any products liability action, a plaintiff must establish three things: (1) he was injured by the product; (2) the product was in essentially the same condition at the time of the accident as it was when it left the hands of the defendant, and (3) the injury occurred because the product 'was in a defective condition unreasonably dangerous to the user.'") (citing *Madden*).

Here, Defendants' valves are made of metal and do not cause disease. Plaintiffs presented no proof that he was exposed to any alleged asbestos materials within the Defendants' valves that were in essentially the same condition as when they left the hands of Defendants. Rather, Mr. Jolly testified that he was exposed to asbestos from flange gaskets that were placed between piping and the Defendants' valves *after* they were sold and sent to Duke.[8] These flange gaskets were placed on those valves by Duke employees long after they left the hands of Fisher and Crosby.[9]

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[8] *See* 7/25/17 Trial Tr. at pp. 73-78; *id.* at 85-90; *id.* at 103-105.

[9] *See* 7/28/17 Trial Tr. at 107:20 – 108:23 ("Now, these things here we've heard these called flanges? A Yes. Q And they're connected to the pipes? A Yes. These flanges are connected to the pipe by virtue of these /HOELTS. Q Now, are there gaskets on the ends of these flanges? A No. There's not on the end of these flanges no. ***Eventually the user would select a gasket and apply against this surface here*** and there would be a similar surface that the pipe would employ. ***Q Did you say Fisher sell these gaskets? A No no these are called line gaskets we were never in the business had nothing to do with line gaskets. Q Did Fisher ever recommend a type of line gasket? A No not really no. Q Did Fisher ever require a line gasket? A No. We didn't impose that upon them.*** I think it's just a general practice again process control experts, they would select even what type of flange on our valve to apply. We could accommodate that and in some cases there are flanges like there that don't even require gaskets they could require a metal part that goes in there. And other cases not uncommon to have actually welds here, a lot of power, a lot of valves in the power industry are actually (welded not) flange they want no possibility of leak so they weld that construction right there to the pipe.") (emphasis added); *see also id.* at pp. 136-138; 7/31/17 Trial Tr. at 65:11-20 ("Q So that's a little different. Is it the position today of Crosby, will Crosby recognize today that being exposed to asbestos that is will I be graduated from an asbestos

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Thus, these Defendants cannot be liable for these products, and the Court should grant judgment in favor of these Defendants on all of Plaintiffs' claims.

D. Plaintiffs failed to show Fisher and Crosby's alleged failure to warn was the proximate cause of their injuries.

For reasons similar to those set forth above, Fisher and Crosby are entitled to a JNOV on Plaintiffs' failure to warn claims because Plaintiffs failed to establish that the Defendants' alleged failure to warn about the hazards of asbestos associated with their valves proximately caused their injuries.

To prove their failure to warn claims, Plaintiffs had to show that a warning given by Fisher and Crosby would have made a difference and prevented the harm. Plaintiffs did not make this required showing. Plaintiffs elicited no evidence regarding 1) what the warning should have contained, 2) where it should have been displayed, 3) who would have seen it, 4) what the person seeing the warning would have done in response to seeing the warning, 5) how that response would have impacted Mr. Jolly's alleged exposure, or 6) whether the response to the warning would have impacted causation (i.e. that if warned, Mr. Jolly would not have contracted mesothelioma).

There is no presumption in South Carolina that a plaintiff would have heeded a warning if it had been given. Instead, "[t]he plaintiff has the burden of showing that a warning would have made a difference in the conduct of the person warned." *Allen v. Long Mfg. NC, Inc.*, 332 S.C. 422, 432, 505 S.E.2d 354, 359 (Ct. App. 1998). If the plaintiff would not have heeded a warning, the failure to warn cannot be the proximate cause of the plaintiff's injuries. *See id.* Mr. Jolly made

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containing gasket that was used on the flange of a valve and exposure occurred via removal that that poses a health risk? A Are we talking about external gaskets. Q Talking about flange face gasket? ***A External gasket. We did not supply those gaskets so we wouldn't warn anybody about those gaskets.***") (emphasis added).

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clear during his testimony that he knew about the hazards of asbestos.[10] Plaintiffs' evidence also proves that Duke Power knew asbestos could be hazardous and repeatedly warned its employees.[11] Despite Duke's warnings and Mr. Jolly's knowledge, Jolly admitted that he continued to work with and around asbestos-containing products and Fisher and Crosby valves.

Plaintiffs submitted no evidence that Mr. Jolly would have heeded a warning had one been given; they did not even attempt to elicit such testimony and for this reason alone their failure to warn-related claims should be dismissed. By contrast, because Mr. Jolly admitted that Duke Power warned him about asbestos, and he failed to take precautions anyway, the only reasonable inference from the evidence in the record is that Mr. Jolly did not heed warnings about asbestos and therefore would not have heeded Defendants' warnings had they been given.

Hence, no reasonable jury could find that Mr. Jolly would have heeded warnings from Fisher and Crosby on their valves. Consequently, as a matter of law, Fisher and Crosby's failure to warn was not the proximate cause of Plaintiffs' injuries. *See Owings*, 33 S.E. at 512–13 (1899) ("Any warning which might have been given would have been entirely useless, and therefore the defendant neglected no duty in failing to give such warning."); *see also, e.g.*, *Billiar v. Minnesota Min. & Mfg. Co.*, 623 F.2d 240, 243 (2d Cir. 1980) ("When the user is fully aware of the nature of the product and its dangers, however, the supplier cannot be held liable for failure to warn him."). Fisher and Crosby are entitled to JNOV on all of Plaintiffs' failure to warn claims.

II. PLAINTIFFS FAILED TO MEET THEIR BURDEN OF PROOF ON THEIR DESIGN DEFECT CLAIMS.

Fisher and Crosby are also entitled to JNOV on Plaintiffs' design defect claims (in both negligence and implied warranty). For a plaintiff to succeed on a design defect claim, "he must

[10] *See* Part I.A, *supra*.
[11] *See id.*

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show that the design of the product caused it to be 'unreasonably dangerous.'" *Branham v. Ford Motor Co.*, 390 S.C. 203, 218, 701 S.E.2d 5, 13 (2010). South Carolina courts apply the risk-utility test in analyzing design defect claims. *Id.* at 220, 701 S.E.2d at 14. The risk-utility test requires a plaintiff asserting design defect claims to prove the availability of a reasonable alternative design. *Id.*; *Holland ex rel. Knox v. Morbark, Inc.*, 407 S.C. 227, 237, 754 S.E.2d 714, 720 (Ct. App. 2014) ("[T]he requirement of proving a reasonable alternative design in a design defect case is mandatory."). A plaintiff must "point[] to a design flaw in the product and show[] how his alternative design would have prevented the product from being unreasonably dangerous." *Graves v. CAS Med. Sys., Inc.*, 401 S.C. 63, 79, 735 S.E.2d 650, 658 (2012) (quoting *Branham*, 390 S.C. at 225, 701 S.E.2d at 16). To prove a reasonable alternative design, a plaintiff must "set forth some evidence of an 'alternative design,' which necessarily include[s] the 'consideration of costs, safety, and functionality associated with the alternative design.'" *Holland*, 407 S.C. at 237–38, 754 S.E.2d at 720 (citation omitted). Plaintiffs must satisfy this requirement to prove a product was in a defective condition unreasonably dangerous to the user; therefore, the following analysis applies to all of Plaintiffs' theories of recovery. *See Bragg*, 319 S.C. at 539, 462 S.E.2d at 326; *Dema*, 312 S.C. at 530, 435 S.E.2d at 876.

Plaintiffs did not put forth any evidence showing that a reasonable alternative design existed during the time that Mr. Jolly allegedly worked around the Defendants' valves. In fact, as to asbestos-containing thermal insulation that may have gone near or around some valves (which are not at issue in this case for purposes of these Defendants' potential liability, as set forth in Section IV, *infra*), Plaintiffs' own expert, Dr. Frank, admitted that asbestos was the *only* insulating

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option available in the hot settings at issue (i.e., applications greater than 1,000 degrees).[12] The evidence at trial showed that Duke ordered the particular products it needed for its nuclear power plant. It was Duke, not the Defendants, who ordered and deployed asbestos products. Plaintiffs presented no evidence that a feasible alternative design existed that Defendants could have supplied, which would have met Duke's needs, under the risk-utility test required by *Branham*, supra. Notably, this requirement for alternative feasible design proof also applies to Plaintiffs' failure to warn claim. *Holland ex rel. Knox v. Morbark, Inc.*, 407 S.C. 227, 237, 754 S.E.2d 714, 720 (Ct. App. 2014).

III. PLAINTIFFS' NEGLIGENCE CLAIMS FAIL BECAUSE THEY DID NOT SHOW FISHER OR CROSBY DEVIATED FROM THE REQUISITE STANDARD OF CARE.

A plaintiff asserting products liability claims under a negligence standard—in addition to the elements described above—bears the burden of demonstrating the defendant failed to exercise due care. *See Bragg*, 319 S.C. at 539, 462 S.E.2d at 326. This requires the plaintiff to present evidence showing the applicable standard of care and that the defendant deviated from the standard of care. *See Sunvillas Homeowners Ass'n, Inc. v. Square D Co.*, 301 S.C. 330, 333, 391 S.E.2d 868, 870 (Ct. App. 1990) ("To establish negligence the plaintiff must prove the defendant failed to exercise due care in some respect."); *see also AT & T Corp. v. Med. Review of N.C., Inc.*, 876 F. Supp. 91, 93 (E.D.N.C. 1995) (providing that the elements of a products liability claim for negligence include "evidence of a standard of care owed by the reasonably prudent person in similar circumstances" and "breach of that standard of care"). Plaintiffs did not present evidence

[12] *See* 7/27/17 Trial Tr. at 147:24 – 148:5 ("Q Do you know the temperature ranges between the [fiberglass insulation] and the asbestos ranges? A Asbestos will go up to higher heat ranges I don't know the exact numbers. Q So if you had something going up to a thousand degrees you couldn't use fiberglass could you? A There's probably a limit a little bit below that.").

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of the applicable standard of care at the time of Mr. Jolly's alleged exposure to asbestos from the Defendants' valves. *Bragg*, 319 S.C. at 548–49, 462 S.E.2d at 331 (stating that, under South Carolina products liability law, "a product must be measured against a standard existing at the time of sale"); *id.* at 548, 462 S.E.2d at 331 ("A manufacturer . . . has no duty to notify previous purchasers of its products about later developed safety devices or to retrofit those products if the products were nondefective under the standards existing at the time of the manufacture or sale."). Therefore, Plaintiffs failed to prove a necessary element of their negligence claims, and Defendants are entitled to JNOV on that specific claim.

Furthermore, regardless of whether Plaintiffs properly presented evidence of the standard of care, they did not prove that Fisher or Crosby deviated from that standard. As explained in the previous section, to prove a negligent design defect, Plaintiffs had to present evidence of a reasonable alternative design. *Branham*, 390 S.C. at 225, 701 S.E.2d at 16; *Holland*, 407 S.C. at 237, 754 S.E.2d at 720. Accordingly, Plaintiffs had to show Fisher and Crosby designed products in an unreasonably dangerous manner despite the existence of a feasible, less dangerous alternative design. *Id.*; *Allen*, 332 S.C. at 426–27, 505 S.E.2d at 356 (providing "[l]iability for negligence requires . . . proof that the manufacturer breached its duty to exercise reasonable care to adopt a safe design"). To prove a negligent failure to warn, Plaintiffs had to show Fisher and Crosby had actual or constructive knowledge of the risk posed by their products. *Webb*, 370 P.3d at 1030. The standard of care is whether a prudent seller of a product who knew of a risk would have acted reasonably in not giving a warning. *Id.* A seller of a product who reasonably does not give a warning is not liable for negligent failure to warn.

Here, as explained in Part II, Plaintiffs did not present evidence of a reasonable alternative design; therefore, they offered no proof Fisher and Crosby deviated from the standard of care as

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to Plaintiffs' design defect claim. As explained in Part I, Fisher and Crosby had no duty to warn Mr. Jolly of the hazards of asbestos. Consequently, Plaintiffs presented no evidence that Fisher and Crosby deviated from the standard of care as to Plaintiffs' negligent failure to warn claims.

Moreover, it was undisputed that any potential exposure to asbestos that Mr. Jolly may possibly have had in connection with Fisher and Crosby valves would not have exceeded applicable permissible exposure limits (e.g., applicable OSHA PELs) – both those in place at the time Mr. Jolly worked around those valves, and the PEL in place today.[13] Compliance with government safety standards—such as OSHA standards—constitutes strong and substantial evidence that a product is not defective. *Gideon v. Johns-Manville Sales Corp.*, 761 F.2d 1129, 1144 (5th Cir. 1985). Plaintiffs failed to prove their negligence claims, and Fisher and Crosby are thus entitled to judgment on those claims.

IV. DEFENDANTS ARE ENTITLED TO JUDGMENT AS A MATTER OF LAW BECAUSE THERE WAS NO EVIDENCE THAT THEY MANUFACTURED, SOLD, OR OTHERWISE PLACED INTO THE STREAM OF COMMERCE ANY OF THE ASBESTOS-CONTAINING PRODUCTS TO WHICH MR. JOLLY WAS ALLEGEDLY EXPOSED.

[13] *See, e.g.*, 7/31/17 Trial Tr. at 248:22 – 249:9 ("Q So the five slides that you look at or five studies you looked at what are the results in regards to those studies as far as the current OSHA permissible exposure limit or PEL of.1 fiber? A ***For the studies which I determined to have appropriate data specific to gaskets and packing that were full shifts so long enough to really understand the full workday, that's right those exposures ranged from nondetectable up to an upper bound of 0.062 fibers per cc as an eight-hour or a full shift time-weighted average and in fact in this case all of those numbers were below the current OSHA PEL. So it would be allowable today in the workplace.***") (emphasis added); *see also id.* at 253:20 – 255:20 (testifying that Mr. Jolly's entire potential working career around Fisher valves, even using the highest numbers possible from reported literature, was "well below" the OSHA PEL); 7/28/17 Trial Tr. at pp. 56-59 (testifying that Fisher complied with OSHA PELs, and testing associated with asbestos-containing components in Fisher valves showed compliance with OSHA PELs).

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Fisher and Crosby are also entitled to JNOV as to all of Plaintiffs' claims, because there was no evidence demonstrating that Mr. Jolly was exposed to asbestos that Fisher or Crosby sold, designed, or placed into the stream of commerce.

Many of the valves Fisher and Crosby manufactured and sold were asbestos-free when they left Fisher and Crosby's hands. Moreover, Plaintiffs presented no evidence that Mr. Jolly worked around *original* asbestos-containing components of a Fisher or Crosby valve nor that he worked around these *internal* components at all. Instead, Mr. Jolly's only testimony alleging exposure to asbestos in connection with Fisher or Crosby valves related to *flange gaskets*, which these Defendants did not sell or recommend.[14] Thus, there is *no evidence* that Mr. Jolly was ever exposed to an asbestos component actually sold by Fisher or Crosby. Instead, Fisher and Crosby sold "bare metal" valves, and Duke later added asbestos-containing (or non-asbestos-containing) flange gaskets (and, in some cases, insulation) to the outside of those valves. However, because Fisher and Crosby did not make, sell, specify, or place into the stream of commerce any such flange gaskets and/or insulation, courts across the country have held that companies in the same position as Fisher and Crosby owed no legal duty to Plaintiffs with respect to, and may not be liable for, those products.

Indeed, an overwhelming majority of courts in recent years have concluded that they will not hold manufacturers responsible for allegedly defective component products supplied by third parties. This trend appears to have begun with the Fourth Circuit, which reached such a holding under South Carolina law, and it has more recently been extended into the asbestos context for companies just like Fisher and Crosby. *See Baughman v. General Motors Corp.*, 780 F.2d 1131,

[14] *See, e.g.*, 7/25/17 Trial Tr. at pp. 75-77 (Mr. Jolly testifying that he was present when others were scraping flange gaskets off Crosby and Fisher valves); *see also* 7/28/17 Trial Tr. at pp. 107-108 (testifying that Fisher never provided or recommended flange gaskets to customers).

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1132–33 (4th Cir. 1986) (applying South Carolina law in holding that a defendant cannot be liable for a defective component part where the defendant manufacturer did not incorporate the defective component part into its finished product and did not place the defective component into the stream of commerce); *see also, e.g.*, *O'Neil v. Crane Co.*, 266 P.3d 987, 991, 1005 (Cal. 2012) (holding pump and valve defendants were not liable for harm caused by asbestos products they did not manufacture or distribute, regardless of the theory of recovery articulated, and stating "California law does not impose a duty to warn about dangers arising entirely from another manufacturer's products, even if it is foreseeable that the products will be used together"); *Simonetta v. Viad Corp.*, 197 P.3d 127 (Wash. 2008). South Carolina has also expressed this requirement in the asbestos context in *Henderson*, in which the Supreme Court held that in order to establish a cause of action for an asbestos injury, a plaintiff must show, *inter alia*, that he or she "was exposed to the [*defendants'*] asbestos containing products." *Henderson v. Allied-Signal, Inc.*, 373 S.C. 179, 185, 644 S.E.2d 724, 727 (2007) (emphasis added) (altered from "Respondents" to "defendants").

Judge Norton, of the District of South Carolina, initially agreed with this notion, as he ruled in late 2014 that a pump manufacturer could not be liable for other companies' asbestos components that were incorporated into its pumps post-sale. *See Sparkman v. A.W. Chesterton Co.*, No. 2:12-cv-02957-DCN, 2014 U.S. Dist. LEXIS 177560 (D.S.C. Dec. 29, 2014) ("*Sparkman I*"). As Judge Norton explained:

> ***The rationale for strict liability under South Carolina product liability law comports with the principles of the bare metal defense.*** Specifically, under *comment c of 402A*, Goulds should not be held liable for products which it did not manufacture, supply or market. See *Restatement (Second) of Torts § 402A cmt. c* (1965). In light of this rationale, and the persuasive weight of authority from other jurisdictions adopting the bare metal defense, ***the South Carolina Supreme Court would likely find a manufacturer is not subject to a duty to warn about potential dangers from exposure to parts of its product which it did not manufacture or otherwise supply. Accordingly, Goulds cannot be liable for exposure to asbestos from products that it did not manufacture or otherwise supply.***

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Id. at *19–20 (emphasis added).

Judge Norton subsequently reversed course on this point based on a South Carolina circuit court's holding in its post-trial ruling in *Garvin v. Agco Corp.*, Case No. 2012-CP-40-6675 (2014) (the "*Garvin* Order"), because there was evidence that the defendant in *Sparkman* "specified the use of asbestos for packing and gaskets in its pumps." *Sparkman v. Goulds Pumps, Inc.*, No. 2:12-cv-02957, 2015 U.S. Dist. LEXIS 19579 (D.S.C. Feb. 19, 2015) ("*Sparkman II*"). However, Fisher and Crosby respectfully submit that Judge Norton's initial holding was correct and the appropriate ruling. Moreover, the *Garvin* Order is not binding upon this Court, and, as set forth below, the rationale for imposing liability under the *Garvin* Order is not present here.

The *Garvin* Order arose from very different facts. In *Garvin*, (1) <u>*every*</u> valve made by the defendant had asbestos, (2) Plaintiff worked on <u>*every*</u> valve made by the Defendant, (3) Defendant, not the site owner, specified the asbestos gaskets and packing, (4) Defendant specified asbestos replacement parts be used in the future and (5) Plaintiff was exposed to causative levels of asbestos from <u>*Defendant's own products*</u>. No such evidence exists here as to any of these five points. In fact, the only products Plaintiffs blamed for Mr. Jolly's disease appear to be asbestos-containing flange gaskets supplied by Duke, which purchased them from third parties and placed them next to Fisher and Crosby valves after the valves left Fisher and Crosby. Unlike in *Garvin*, there is no evidence in this case that Fisher and Crosby specified the use of asbestos-containing components with their valves. Rather, the evidence demonstrates the opposite: *Duke Power engineers* specified which parts they would use in connection with the Defendants' products.

The specification by Duke Power engineers places this case squarely within the analysis in the *O'Neil* case authored by the California Supreme Court. There, the court found the United States Navy was responsible for using asbestos-containing replacement parts in the defendants'

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valves. 266 P.3d at 996. The court found "there was no evidence that defendants' products *required* asbestos-containing gaskets or packing in order to function." *Id.* In fact, "*[a]part from the Navy's specifications*, no evidence showed that the design of defendants' products required the use of asbestos components, and their *mere compatibility* for use with such components is not enough to render them defective." *Id.* (emphasis added). The court further held, "[w]here the intended use of a product *inevitably* creates a hazardous situation, it is reasonable to expect the manufacturer to give warnings. Conversely, where the hazard arises entirely from another product, and the defendant's product does not create or contribute to that hazard, liability is not appropriate." *Id.* at 1004 (emphasis added).

Similarly, in cases decided more recently than *Garvin*, South Carolina federal courts have declined to adopt a rule that defendants are liable whenever the use of asbestos in their products is foreseeable. *See, e.g.*, *Andrews v. Crane Co.*, Case No. 2:13-cv-02055-RMG, D.E. 454 at 16 (D.S.C. June 2, 2015). Instead, the courts have rejected application of the bare metal defense only "where the defendant has specified or recommended the use of another product with its own." *Id.* (finding a defendant could not be liable for insulation used with its pumps because the plaintiff did not put forward evidence that the defendant "recommended or specified asbestos-containing insulation for use with its valves"); *see also Andrews v. Crosby Valve LLC*, Case No. 2:13-cv-02055-RMG, D.E. 418 at 13 (D.S.C. May 26, 2015) ("There is no evidence that Crosby specified, recommended or required the use of these external asbestos gaskets or the external asbestos jacket insulation. Therefore, summary judgment is appropriate.").

Accordingly, because Plaintiffs presented no evidence that Fisher and Crosby specified or required the use of asbestos-containing replacement parts with their valves or that it was inevitable

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that original parts would be replaced with asbestos-containing parts, Fisher and Crosby are entitled to judgment as a matter of law.

V. PLAINTIFFS FAILED TO SHOW MR. JOLLY'S EXPOSURE TO ASBESTOS FROM THE DEFENDANTS' PRODUCTS WAS A PROXIMATE CAUSE OF HIS MESOTHELIOMA.

A. Plaintiffs Have Failed to Establish Specific Causation Through Relevant, Reliable Expert Testimony That Would Assist Jury In Reaching a Verdict.

Regardless of their theory of recovery, Plaintiffs did not show that Mr. Jolly's exposure to asbestos in connection to these Defendants' products proximately caused his mesothelioma. In a case where a medical causal relationship is at issue and not within common knowledge (such as the relationship between asbestos exposure and mesothelioma), Plaintiffs must prove both general and specific causation through expert testimony. *Fisher v. Pelstring*, 817 F. Supp. 2d 791, 814 (D.S.C. 2011) ("Where a medical causal relation issue is not one within the common knowledge of the layman, proximate cause cannot be determined without expert medical testimony.") (citing *In re Bausch & Lomb Inc. Contacts Lens Solution Prod. Liab. Litig.*, 693 F.Supp.2d 515, 518 (D.S.C.2010)). "To establish medical causation in a product liability case, a plaintiff must show both general causation and specific causation." *Id.* "General causation is whether a substance is capable of causing a particular injury or condition in the general population, while specific causation is whether a substance caused a particular individual's injury." *Id.*

The testimony of Plaintiffs' experts failed to prove specific causation because they gave opinions that were unreliable and inadmissible under Rules 702 and 403 of the South Carolina Rules of Evidence. Specifically, Plaintiffs offered the testimony of Dr. Frank and Dr. Maddox, who testified that each and every exposure to asbestos causes mesothelioma and the cumulative effect of Mr. Jolly's "exposures to asbestos contribute[d] to his cumulative exposure that gave Mr.

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Jolly his mesothelioma."[15] Dr. Frank repeatedly emphasized his opinions that every single exposure to asbestos, regardless of the dose, was a cause of Mr. Jolly's mesothelioma, and, because he cannot "rule out" a particular exposure as the cause of the mesothelioma, he must "rule in" every exposure as being the cause.[16] This opinion is clearly unreliable, as several courts have held.

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[15] *See* 7/26/17 Trial Tr. at 65:24–68:24.

[16] *See* 7/25/17 Trial Tr. pp. 41:19–44:5 ("There is one safe level of exposure to asbestos: Zero. It's the only level at which you're absolutely safe. . . . One day of exposure to asbestos, both in animal studies and a few human cases that have been reported, have occurred with a single day of exposure. Now it's unusual and it's rare but it tells you how little exposure it takes to give somebody a mesothelioma. Or a lung cancer. And for mesothelioma, one day has done it. We know a month or less of exposure to asbestos has doubled the risk of lung cancer for example. That's epidemiological data in humans but the animal data, one day and mesothelioma individual cases of one day of exposure."); *id.* at 57:2–58:19 ("Q: And if those products are removed from hand wire brushing electric grinders the gaskets and someone gets mesothelioma some decades later are those exposures an important and substantial part of the cause of that mesothelioma? A: Yes. Q: Okay can you Dr. Frank say there's one exposure to removal of an steel asbestos jacket on one day for half an hour a person gets mesothelioma 30 years later and say you know what that sounds so puny let's just put that to the side in terms causation? . . . THE WITNESS: ***What we have to say at the end of the day if someone gets a mesothelioma is that the cumulative exposure they have had to asbestos from whatever products over whatever time is what gave it to them. . . . Some products contribute more some less but you can't leave any of them out*** because it is a cumulative exposure, the totality of what people were exposed to that gave them their disease so all of them have to be included.") (emphasis added); *id.* at 65:24–68:24 ("Q: Do you have an expert opinion with a reasonable degree of medical certainty as to whether or not that exposure even if it were just one occasion would be a substantial contributing factor to what caused the mesothelioma? A: I do have an opinion. Q: And what is that? A: And ***my opinion is since there is no exposure that can be left out for reasons we discussed a bit earlier that would be a substantial contributing factor. It was part of one's cumulative exposure.*** Q: Dr. Frank, if all of the things I've asked you to assume, if that were the only asbestos exposure, in and of itself, would it be sufficient to cause mesothelioma? A: By that are you saying his one day of exposure or just the exposure over four years to. Q: The one day of exposure standing there doing inspection work on a gasket on a flange face of a valve -- an asbestos containing gasket on a flange face of a valve is being removed with a power tool hand tools and scraper? A: ***Again I have not ever seen such a case but if that was the facts of case given to me I would have to say that that exposure because we know in other settings one day of exposure was sufficient that that was the cause of someone's mesothelioma if they developed one.*** Q: So if that person developed mesothelioma, that exposure in and of itself would be sufficient is that what you're saying? A: Yes. . . . Q: Let me ask you to make all of those same assumptions about exposures and ask you to assume that that is the exposure of Mr. Dale Jolly. Do you have an opinion with a reasonable degree of medical certainty as to whether or not Dale Jolly's mesothelioma was substantially contributed to by exposure to asbestos liberated from asbestos gaskets on the flange face of valves during removal

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In fact, at one point Dr. Frank even admitted that he held this opinion with regard to *a single asbestos exposure* one might receive as a bystander to work being done to the flange of a valve on one day, ***even though he was not aware of such a case ever occurring***.[17] Moreover, as the Honorable Judge David C. Norton held earlier this month in South Carolina federal court, such "cumulative exposure" opinion testimony is directly at odds with Plaintiffs' burden of proof as set forth in *Henderson v. Allied-Signal, Inc.*, 373 S.C. 179, 644 S.E.2d 724 (2007), should have been excluded, and cannot serve as a basis to deny this JNOV motion. . . *See Haskins v. 3M Co.*, Nos. 2:15-cv-02086 & 3:15-cv-02123, 2017 U.S. Dist. LEXIS 113657 (D.S.C. July 21, 2017) (Norton, J.). In fact, another federal court in recent weeks excluded *this very opinion from Dr. Frank. See Rockman v. Union Carbide Corp.*, No.: RDB-16-1169, 2017 U.S. Dist. LEXIS 110181 (D. Md. July 17, 2017). Additionally, neither Dr. Frank nor Dr. Maddox had any foundation to offer testimony (because of the complete absence of any industrial hygiene testimony contradicting that submitted by the defense), neither compared the relative contribution of any two or more exposures, and each lumped in, if not entirely relied upon, exposures to flange gaskets or insulation used next to Crosby or Fisher valves, but for which those companies bear no responsibility as a matter of fact or law.

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using the processes that I described? A: I do have an opinion. Q: And what is that? A: My opinion would be that ***Mr. Jolly had those exposures contribute to his cumulative exposure*** that gave Mr. Jolly his mesothelioma. Q: And Dr. Frank but for those exposures, meaning if you try to take them out, would you be able to say that Mr. Jolly would have still gotten mesothelioma? A: When you say is that ***all of them contributed.*** What occurs when you take them out any one of them is unclear what other exposures may have done without those may I have changed the time or the setting in which you get a mesothelioma. Some of my own research point out that additional exposures increased cell division so if you leave out one exposure or another, maybe this cells don't divide and the diseases show up a bit later but one has to say that whatever exposures one has when one gets disease was contributory.") (emphasis added).

[17] *See id.* at 65:24–68:24.

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The admissibility of expert testimony is generally governed by Rule 702 of the South Carolina Rules of Evidence, which provides that "[i]f scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training or education, may testify thereto in the form of an opinion or otherwise." In determining whether to admit expert testimony under Rule 702, the Court must make three inquiries: (1) whether the subject matter is beyond the ordinary knowledge of the jury, thus requiring an expert to explain the matter to the jury; (2) whether the expert has acquired the requisite knowledge and skill to qualify as an expert in the particular subject matter; and (3) whether the substance of the testimony is reliable. *See Graves v. CAS Medical Systems, Inc.*, 401 S.C. 63, 74, 735 S.E.2d 650, 655 (2012); *Watson v. Ford Motor Co.*, 389 S.C. 434, 446–47, 699 S.E.2d 169, 175–76 (2010). Plaintiffs' experts' "no safe level/every exposure" causation opinions failed to meet the reliability requirement of Rule 702.

If an expert's testimony is scientific in nature, then the trial court must determine its reliability pursuant to the factors set forth in *State v. Council*, 335 S.C. 1, 19, 515 S.E.2d 508, 517 (1999).[18] *Graves*, 401 S.C. at 74, 735 S.E.2d at 655. There can be little doubt that testimony that there is no safe level of exposure to asbestos or that "every exposure" to asbestos causes mesothelioma is scientific in nature; therefore, the Court must look to the following factors:

(1) the publications and peer view of the technique;

(2) prior application of the method to the type of evidence involved in the case;

(3) the quality control procedures used to ensure reliability; and

[18] Although these are sometimes referred to as the *Jones* factors (or the *Jones* standard), from *State v. Jones*, 273 S.C. 723, 259 S.E.2d 120 (1979), the Supreme Court of South Carolina articulated these factors in the *Council* decision.

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(4) the consistency of the method with recognized scientific laws and procedures.

Id.; *Council*, 335 S.C. at 19, 515 S.E.2d at 517.

The "no safe level/every exposure/cumulative exposure" opinion offered by Plaintiffs' experts did not satisfy *any* of the *Council* factors because (1) it is neither published nor peer reviewed; (2) it has been rejected by other courts when plaintiffs have attempted to apply it to the type of evidence involved here; (3) there are no quality control procedures or other methods used to ensure reliability; and (4) the method is wholly inconsistent with recognized scientific laws and procedures. *See Yates v. Ford Motor Co.*, 113 F. Supp. 3d 841, 846 (E.D.N.C.) (rejecting the "each and every exposure" theory as unreliable because it "cannot be tested, has not been published in peer-reviewed works, and has no known error rate"). In fact, one federal court reached this exact conclusion with regard to Dr. Frank's "theory" just before the trial of this case. See *See Rockman v. Union Carbide Corp.*, No.: RDB-16-1169, 2017 U.S. Dist. LEXIS 110181, at *4 (D. Md. July 17, 2017) (excluding Dr. Frank's "each and every" exposure opinion because there was "simply insufficient data to support their theory that any exposure to asbestos, no matter how brief, and regardless of the type of asbestos, should be considered a 'substantial factor' in plaintiff developing mesothelioma."); id. at *11 (finding that the specific causation opinions of plaintiff's experts, including Dr. Frank, were not the "product of reliable principles and methods," as required by Rule 702, nor do they satisfy the *Daubert/Council* factors, including "testability," "peer review," and "general acceptance" within the "relevant scientific community.").

As courts have noted, Dr. Frank's theory is not a "method" at all, but rather an end run around the Plaintiffs' burden of proof. *See Yates*, 113 F. Supp. 3d at 850 (E.D.N.C.) (excluding opinions similar to Dr. Frank's and explaining "given the potential persuasiveness of expert testimony, proffered evidence that has a greater potential to mislead than to enlighten should be

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excluded"). Therefore, this opinion testimony should have been stricken[19] and given no weight in the Court's causation analysis, in line with the courts across the country that have considered and rejected this opinion in differing forms.[20]

In line with this notion, Judge Norton expressed frustration with this opinion in a recent case in South Carolina federal court. *See Haskins v. 3M Co.*, supra. There the court took issue

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[19] In addition to the arguments set forth herein, these Defendants hereby also refer the Court back to and incorporate the arguments and points in their Motion *in Limine* and Motion to Strike this testimony, which were made with the Court prior to and contemporaneously with Plaintiffs' experts offering the above testimony.

[20] *See, e.g.*, *Vedros v. Northrop Grumman Shipbuilding, Inc.*, 119 F.Supp.3d 556, 560 n.2 (E.D. La. 2015) (rejecting this theory and noting that "[t]he theory that Defendants refer to [as the "'Every Exposure theory'"] has other names, including 'No Safe Level of Exposure,' 'Each and Every Exposure,' 'Any Exposure,' and 'Single Fiber.' All theories have as their basis the principle that all exposures to asbestos should be included as a cause of mesothelioma because there is no way to know which exposures caused it and which ones did not."); *Bell v. Foster Wheeler Energy, Corp.*, No. 15-6394, 2016 U.S. Dist. LEXIS 138817, at *11 (E.D. La. Oct. 5, 2016) ("The Court sees no material difference between the 'every exposure' theory and the 'every significant exposure' theory.") (citing *Vedros*); *Krik v. Crane Co.*, 76 F. Supp. 3d 747, 750–51 (N.D. Ill. 2014) ("Any Exposure," "Each and Every Exposure," "Single Fiber," and "no safe level of exposure" are all different "monikers" for the same theory); *Anderson v. Ford Motor Co.*, 950 F. Supp. 2d 1217, 1225 (D. Utah 2013) ("[A] growing number of courts have determined that the ["no safe level/every exposure"] theory is not proper under Daubert and Rule 702."); *Smith v. Ford Motor Co.*, No. 2:08–cv–630, 2013 U.S. Dist. LEXIS 7861, at *6 (D. Utah Jan. 18, 2013) (concluding that the "no safe level/every exposure" theory does not hold up under careful examination, noting that "[i]t is questionable whether it can even properly be called a theory, inasmuch as a theory is commonly described as a coherent collection of general propositions used to describe a conclusion, and while there are some general propositions used by [plaintiff's expert], they fall far short of supporting the legal liability he attempts to reach with them."); *Suoja v. Owens-Illinois, Inc.*, 211 F. Supp. 3d 1196 (W.D. Wis. 2016) (rejecting the "cumulative exposure" theory, also described as the "no safe level" theory or "every exposure contributes" theory, and concluding the opinion was "no different from the 'any exposure' theory" or from the impermissible conclusion that "if there is exposure, then there is causation"); *Comardelle v. Pa. Gen. Ins. Co.*, 76 F. Supp. 3d 628, 634 (E.D. La. 2015) ("Although there may be no known safe level of asbestos exposure, this does not support [plaintiff's expert's] leap to the conclusion that therefore every exposure Comardelle had to asbestos must have been a substantial contributing cause of his mesothelioma. The Court agrees that this 'is not an acceptable approach for a causation expert to take.'"); *Sclafani v. Air & Liquid Sys Corp.*, 14 F. Supp. 3d 1351, 1356 (C.D. Cal. 2014) (noting that the "'every exposure' theory could not be tested and had not been published in any peer-reviewed literature [and] therefore failed to satisfy at least two of the four Daubert criteria").

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with the plaintiff's expert's attempt to "equate causation with 'contribution' to the total cumulative level of exposure" and concluded that the expert's causation opinion, strikingly similar to that presented by Plaintiffs in this case, "would render the substantial causation rule meaningless, as any level of exposure may cause mesothelioma." *Id.* at *21-27; *see also id.* at *17 ("The trouble with [the expert's] opinions is his application of these principles to conclude that, because [plaintiffs'] exposures to the defendants' asbestos containing products were—by definition—part of their respective "cumulative doses," such exposures significantly contributed to their development of mesothelioma.").

Similar to Drs. Frank and Maddox, the plaintiff's expert in *Haskins* never defined "what level of exposure he considers significant and openly admits that he did not even need to know [plaintiff's] actual level of exposure to defendants' products in order to render his opinions." *Id.* at 18. This "leads to the bizarre conclusion that any exposure that carries any chance of causing mesothelioma—however miniscule—constitutes a 'substantial cause,' regardless of the other exposures that may have contributed to the total cumulative exposure." *Id.* at *24. Because the expert's "opinions are premised on his conclusion," the Court determined that "his opinions cannot be used to support a finding of substantial causation." *Id.* at 27. The Defendants are entitled to a JNOV because Plaintiffs failed to offer any reliable specific causation testimony.

This opinion is also inadmissible under Rule 403 of the South Carolina Rules of Evidence, which mandates that testimony be excluded if its probative value is substantially outweighed by the danger of undue prejudice and confusion of the issues. In *Smith v. Ford Motor Co.*, the court held that even if a plaintiff's expert's "no safe level/every exposure" theory were deemed admissible under Rule 702, it would exclude the testimony pursuant to Rule 403 because "the probative value of such unsupported speculation by [the expert] is substantially outweighed by the

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danger of unfair prejudice, as well as being confusing, and presenting a danger of misleading the jury." No. 2:08–cv–630, 2013 U.S. Dist. LEXIS 7861, at *6 (D. Utah Jan. 18, 2013). In reaching this conclusion, the court noted its agreement with "the growing number of published opinions from other courts that have reached a similar result: that the every exposure theory as offered as a basis for legal liability is inadmissible speculation that is devoid of responsible scientific support." *Id.*

Judge Norton agreed with this notion:

> The trouble with Bedrossian's opinions is his application of these principles to conclude that, because [Plaintiffs'] exposures to the defendants' asbestos-containing products were—by definition—part of their respective "cumulative doses," such exposures significantly contributed to their development of mesothelioma. . . . Plaintiffs argue that Bedrossian does not claim that every exposure contributes to the cumulative dose—or, at least, he does not opine that every exposure contributes to the cumulative dose in a way that can be considered causative. Instead, plaintiffs claim that Bedrossian only considers exposures to be causative if they reach "non-trivial," "above background," or "occupational" levels. . . . But Bedrossian never defines what level of exposure he considers significant and openly admits that he did not even need to know [Plaintiffs'] actual level of exposure to defendants' products in order to render his opinions. . . . Instead, it appears that Bedrossian assumes that, because every "occupational" exposure contains exponentially more fibers than any background exposure, every "occupational" exposure significantly contributes to the total cumulative dose. Thus, in Bedrossian's view, whenever the total cumulative dose results in mesothelioma, every "occupational" exposure should be considered causative, no matter how small.
>
> ***Regardless of whether this is sound science, it is inconsistent with the law.*** In a products liability action under maritime law, the plaintiff must show "that (1) he was exposed to the defendant's product, and (2) the product was a substantial factor in causing the injury he suffered." This analysis must be conducted on a defendant-by-defendant basis. "'Minimal exposure' to a defendant's product is insufficient."
>
> . . .
>
> ***Because Bedrossian's opinions evaluate causation in a manner that is inconsistent with the appropriate legal standard, they are essentially irrelevant, and any probative value they may have is easily outweighed by their tendency to confuse or mislead the jury.***

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Haskins, 2017 U.S. Dist. LEXIS 113657 , at *17-21 (emphasis added) (internal citations and footnotes omitted). Consistent with the principles articulated above, applicable law demonstrates that "casual" or "minimal" exposure to a defendant's product, unproven to cause disease, is never sufficient to support a finding of causation against that defendant. *See Henderson*, 373 S.C. at 185, 644 S.E.2d at 727. The any-exposure theory propounded by Dr. Frank and Dr. Maddox during trial is totally inconsistent with that basic principle and should have properly been excluded under Rule 403, in addition to Rule 702. Without these opinions, Plaintiffs presented no evidence of specific causation, and Fisher and Crosby are thus entitled to JNOV.

B. Plaintiffs Failed to Prove Causation Under the *Henderson/Lohrmann* Standard.

Plaintiffs also failed to prove causation pursuant to the "frequency, regularity, and proximity" standard. In *Henderson v. Allied-Signal, Inc.*, 373 S.C. 179, 644 S.E.2d 724 (2007), the South Carolina Supreme Court adopted the following standard as a requirement for a plaintiff to maintain an actionable claim of asbestos exposure:

> In determining whether exposure is actionable, we adopt the "frequency, regularity, and proximity test" set forth in *Lohrmann v. Pittsburgh Corning Corp.*, 782 F.2d 1156, 1162 (4th Cir. 1986): "To support a reasonable inference of substantial causation from circumstantial evidence, there must be evidence of exposure to a ***specific product on a regular basis over some extended period of time in proximity to where the plaintiff actually worked.***"

Id. at 185, 644 S.E.2d 727 (emphasis added). Moreover, after finding the plaintiff had failed to present evidence of regular and frequent exposure to asbestos-containing products in proximity to where the plaintiff worked, the *Henderson* court specifically held "presence in the vicinity of static asbestos" is not actionable exposure. *Id.*

Here, Plaintiffs failed to show Mr. Jolly was exposed to asbestos dust from a "specific [Fisher or Crosby asbestos-containing] product on a regular basis over some extended period of time." *Henderson*, 373 S.C. at 185, 644 S.E.2d at 727. Put another way, the evidence and

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testimony in this case is not sufficient to satisfy the "regular," "frequent," and "proximate" exposure standard required by *Henderson* and its parent case, *Lohrmann*. *See Lohrmann*, 782 F.2d at 1163 (noting that the plaintiff "testified that he was exposed to an asbestos-containing pipe covering called Unibestos on *ten to fifteen occasions of between one and eight hours duration* during the term of his employment," and holding that "[t]he trial court correctly ruled that, viewing the evidence in the light most favorable to the plaintiff, his exposure to Unibestos *was not sufficient* to raise a permissible inference that such exposure was a substantial factor in the development of his asbestosis") (emphasis added); *see also Pace v. Air & Liquid Sys. Corp.*, 642 Fed. Appx. 244 (4th Cir. 2016) (affirming summary judgment under South Carolina law and *Henderson/Lohrmann*, despite the fact that one witness testified that he <u>believed</u> the Plaintiffs worked with the defendant's pumps "a lot," because an examination of the witness's total testimony demonstrated that this statement amounted to pure speculation).

Plaintiffs had to meet the *Henderson/Lohrmann* test with respect to exposure to asbestos-containing products (with the asbestos in a form that can be breathed) placed into the stream of commerce by Defendants. Plaintiffs did not meet this burden. <u>In fact, Plaintiffs offered only conclusory testimony employing words like "regularly" and emphasized Mr. Jolly's alleged exposure to flange gaskets which Defendants did not place in the stream of commerce and for which they have no legal responsibility.</u>[21] Lastly, Plaintiffs may not render this legally required showing irrelevant by presenting alleged expert testimony of "any exposure," addressed above. In fact, the *Henderson/Lohrmann* test operates to preclude such testimony. Plaintiffs would have it the other way around, improperly attempting to preclude the *Henderson/Lohrmann* test through

[21] *See* Dale Jolly testimony at 7/25/17 Trial Tr. pp. 93:17–25: Dr, Frank testimony at *See* 7/26/17 Trial Tr. pp. 62:18-65:5 (expressing opinions as to exposure from flange gaskets).

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the introduction of such testimony. This is impermissible, and the Defendants are entitled to JNOV.

CONCLUSION

For the foregoing reasons, including the arguments, points, and reasons set forth in the Directed Verdict Motions made by these Defendants, which are incorporated herein, Fisher and Crosby respectfully request that this Court grant their motion for JNOV on all of Plaintiffs' Claims.

Respectfully submitted,

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August 23, 2017

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STATE OF SOUTH CAROLINA COUNTY OF SPARTANBURG	)	IN THE COURT OF COMMON PLEAS FOR THE SEVENTH JUDICIAL CIRCUIT
BEVERLY DALE JOLLY and BRENDA RICE JOLLY, Plaintiffs, v. CROSBY VALVE, LLC and FISHER CONTROLS INTERNATIONAL LLC, et al., Defendants.	)	Civil Action No. 2016-CP-42-01592 **DEFENDANTS' BRIEF IN OPPOSITION TO PLAINTIFF'S MOTION FOR NEW TRIAL *NISI ADDITUR***

Defendants Crosby Valve, LLC and Fisher Controls International LLC respectfully submit this brief in response to the Plaintiff's Motion for New Trial *Nisi Additur*.

INTRODUCTION

Plaintiffs brought this action against sixty-three (63) defendants, including Fisher and Crosby. Before trial, Plaintiffs settled with or dismissed every defendant except Fisher and Crosby. A jury trial commenced in Spartanburg County on July 24, 2017. As trial progressed, the Court frequently noted the diligence with which the jury undertook their service. (*See* 7/27/17 TT, pg. 233-4; 7/31/17 TT, p. 209; TT 8/2/17, p 196).

On August 3, 2017, after five (5) hours of deliberation, the jury returned a verdict in which it found in favor of Plaintiffs as to their negligence and breach of implied warranty claims. The jury found in favor of Fisher and Crosby as to Plaintiffs' strict liability claim and further determined that Fisher and Crosby's actions were not willful, wanton or reckless. The jury awarded $200,000 in damages to Plaintiff Beverly Dale Jolly and $100,000 to Brenda Rice Jolly for her loss of consortium claim. Immediately after the verdict was returned, the Court directed

the parties to file post-trial motions within ten days pursuant to Rules 50(e) and 59(b) of the South Carolina Rules of Civil Procedure. (TT 8/3/17, p 211).

The Plaintiffs have filed a Motion for New Trial *Nisi Additur*. The Plaintiffs ask the Court to disregard the jury's verdict and increase the award for Mr. Jolly to include $1 million in medical expenses and $1 million for non-economic damages and increase the award for Mrs. Jolly to $500,000. For the following reasons, Fisher and Crosby respectfully ask that the Court reject Plaintiffs' motion.

ARGUMENT

I. STANDARD OF REVIEW.

When a verdict indicates that the jury was unduly liberal or conservative in its assessment of the damages, the trial court may alter the verdict by granting a new trial *nisi*. *Riley v. Ford Motor Co.*, 414 S.C. 185, 192, 777 S.E.2d 824, 828 (2015). However, courts must give substantial deference to a jury's determination of damages. *Harrison v. Bevilacqua*, 354 S.C. 129, 140, 580 S.E.2d 109 (2003); *Luchok v. Vena*, 391 S.C. 262, 264, 705 S.E.2d 71, 72 (Ct. App. 2010). Accordingly, a trial court cannot grant a motion for new trial *nisi additur* without identifying "compelling reasons" to justify invading the jury's province. *Riley*, 777 S.E.2d at 829; *Luchok,* 391 S.C. at 264. Trial judges are not empowered to simply substitute their judgment for that of the jury. *Graham v. Whitaker*, 218 S.C. 393, 402, 321 S.E.2d 40, 45 (1984). Awards for non-economic damages are particularly within the province of the jury to weigh and determine because such damages are indeterminate in character. *Kalchthaler v. Workman*, 316 S.C. 499, 503, 450 S.E.2d 621, 623 (Ct. App. 1994).

In reviewing a trial court's denial of a motion for a new trial, appellate courts "must consider the testimony and reasonable inferences to be drawn therefrom in the light most

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favorable to the nonmoving party." *Umhoefer v. Bollinger,* 298 S.C. 221, 379 S.E .2d 296 (Ct.App.1989). The United States Supreme Court has similarly remarked on the caution trial judges presented with requests for *additur* should exercise, stating:

> It is not the function of a court to search the record for conflicting circumstantial evidence in order to take the case away from the jury on a theory that the proof gives equal support to inconsistent and uncertain references. The focal point of judicial review is the reasonableness of the particular inference or conclusion drawn by the jury. It is the jury, not the court, which is the fact-finding body. It weighs the contradictory evidence and inferences, judges the credibility of witnesses, receives expert instructions, and draws the ultimate conclusion as to the facts. The very essence of its function is to select from among conflicting inferences and conclusions that which it considers most reasonable. That conclusion, whether it relates to negligence, causation or any other factual matter, cannot be ignored. Courts are not free to reweigh the evidence and set aside the jury verdict merely because the jury could have drawn different inferences or conclusions or because judges feel that other results are more reasonable.

Tennant v. Peoria & P.U. Ry., Co., 321 U.S. 29, 35, 64 S.Ct. 409, 88 L.Ed. 520 (1944).

II. THERE IS NO BASIS TO GRANT *ADDITUR* AS TO ECONOMIC DAMAGES (MEDICAL BILLS).

The case the Plaintiffs presented to the jury as to economic damages (the sole component of which was medical expenses for Dale Jolly's treatment) was noteworthy for what it <u>*did not*</u> include.

- Not one single medical bill was offered into evidence.
- Not one single medical record was offered into evidence.
- No treating physician of Mr. Jolly was called as a witness, even via read-in testimony. This was a glaring omission in light of the fact that Plaintiffs' discovery revealed that <u>five doctors from Spartanburg alone</u> treated Mr. Jolly for his illness and that he was treated at an exceptional hospital, Duke, that is only three hours away from the courthouse. (*See* Plaintiffs' Responses to Defendants'

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Master Set of Interrogatories and Request for Production of Documents, attached hereto as **Exhibit A**).

- Plaintiffs' counsel elected not to ask any Jolly family member about Mr. Jolly's medical expenses, even though both Mrs. Jolly and their daughter testified in addition to Mr. Jolly.

Instead, Plaintiffs opted to delve into economic damages exclusively, and superficially, through Dr. Arthur Frank. Dr. Frank was not one of Mr. Jolly's treating physicians, and he had never even met Mr. Jolly, his family or the treating physicians (7/26/17 TT, p. 126). Dr. Frank said he saw "about" $142,000 in bills, but this was from memory and he never said which bills he had seen, or when or in what form he had seen them. (7/26/17 TT, p. 179). Since Dr. Frank was an expert hired by Plaintiffs' counsel, if he saw medical bills, he obviously would have received them from counsel, and, in turn, the bills must have been readily available.

The jury may well have interpreted the Plaintiffs' failure to submit bills they possessed as an attempt to exaggerate the amount of past medical expenses. Additionally, the jury certainly could have considered the evidence that they were provided about past expenses to be speculative. This was doubly so with respect to future medical expenses in light of the fact Dr. Frank failed to identify any reasonably precise range and had no connection to any facility providing treatment to Mr. Jolly[1].

Further, contrary to what Plaintiffs state in their brief, Dr. Frank did **not** state that Mr. Jolly's medical expenses would be $1,000,000, but only that *some cases* can generate bills of that amount ("Case (sic) even go to a million dollars or more.").[2] (7/26/17 TT, p. 101). Likewise, he

[1] Dr. Frank also never identified what medical records he may have reviewed, only that he reviewed "some" of them. (7/26/17 TT, p. 105).

[2] Plaintiffs also state the $1,000,000 expense sum was "undisputed." (Brief, p. 2). To the contrary, as noted above, Plaintiffs did not prove this sum in the first instance.

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did **not** state that a surgery Mr. Jolly had costs hundreds of thousands of dollars (Plaintiffs' Brief, p. 13), but only that it "*could be.*" (7/26/17 TT, p. 101). These words clearly communicated to the jury that Frank likely had not seen those particular surgical bills. An attuned jury would be well within its rights to consider such statements to be speculative.

Plaintiffs' current motion ignores the fact the jury was not required to simply accept without question the testimony that Plaintiffs presented about the amount of economic damages. To the contrary, a jury is not required to simply accept one party's damage evidence, even if the other side presents no competing evidence. *Steele v. Dillard*, 327 S.C. 340, 343-4, 486 S.E.2d 278 (Ct. App. 1997). Instead, the jury is entitled to consider the quality of the evidence, its source, and factors that weigh on its credibility, for there always remain the questions of probability and credibility. *Id.* Jurors also appropriately resist speculation, which is never an acceptable foundation for a damage award. *See Kiriakides v. School Dist. of Greenville Cnty.*, 382 S.C. 8, 675 S.E.2d 439, 445 (2009) ("An abuse of discretion occurs when the conclusions of the circuit court . . . are based on unsupported factual conclusions."). Indeed, the jurors here were instructed as follows:

> The plaintiff is never entitled to recover speculative damages. That is damages that you arrived at by guesswork. The plaintiff has the burden of proving the existence and the causation and the amount of damages...

(Jury Charges, p. 18; 8/3/17 TT, p. 141). This rule applies not only to an award of future damages, but to any type of damages.. *Baughman v. American Tel. & Tel Co*, 306 S.C. 101, 116, 410 S.E.2d 537, 546 (1991).

This case strongly resembles *Tarrants v. Owens-Corning Fiberglas Corp.*, 225 F.3d 659 (6th Cir. 2000), in which the widow of a man who died of mesothelioma brought a claim against an insulation manufacturer and others. She claimed $52,000 in medical bills, and sought a new

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trial in part because the jury awarded only $10,000 for that item. The Sixth Circuit noted that the plaintiff "failed to produce any written documentation to support her claims for out-of-pocket medical and funeral expenses, but instead relied on her own recollection and expert witness testimony" (as here, she had provided undisclosed documents to the expert). *Id.*, at *2. The Sixth Circuit rejected the plaintiff's arguments, stating that even though the defendant did not present specific evidence to refute her claims, it was her burden as the plaintiff to prove "with some reasonable degree of certainty the extent and amount of such damage" and her failure to present sufficient credible evidence of out-of-pocket damages justified confirmation of the jury verdict. *Id.*

Juries are presumed to follow the instructions they are given by the trial judge. *Conner v. City of Health Forest Acres*, 363 S.C. 460, 471, 611 S.E.2d 905, 910 (2005); *Miranda C. v. Nissan Motor Co. Ltd.*, 402 S.C. 577, 590, 741 S.E.2d 34, 41 (Ct. App. 2013). Thus, it is appropriate to assume the jury here honored the instructions they were given and determined that the case the Plaintiffs presented to them required them to speculate as to economic damages.

The degree to which the jury's award to Mr. Jolly reflects his medical bills is unknown[3], but there is nothing inappropriate about it. The question here is not whether Mr. Jolly incurred medical expenses, but rather whether the jury's answer should be disturbed in light of the Plaintiffs' chosen method to prove this part of their case. The Plaintiffs made the calculated decision not to prove up medical expenses by submitting the medical bills into evidence. Indeed, the Court may recall plaintiffs' counsel did not even begin to address damages in the closing argument until the last 5 to 8 minutes of her hour-plus long closing. By choosing not to submit

[3] The verdict form asked the jury to assess each plaintiff's damage award without division into separate economic and non-economic damages. It is also unknown if the award to Mrs. Jolly included a proportion of the medical bills. The loss of consortium jury instruction, applicable to Mrs. Jolly's claim, informed the jury she was entitled to recover "for any expenses for the care and treatment to her spouse"

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any medical bills, any medical records detailing the procedures Mr. Jolly underwent, call any of the treating physicians, or have any witness speak with any degree of precision about the medical bills, the Plaintiffs forced the jury into guesswork. The award appropriately reflects the jury's refusal to disobey the law they were instructed to follow.

III. THERE IS NO BASIS TO GRANT *ADDITUR* AS TO THE AWARD FOR MR. JOLLY.

This Court should deny Plaintiffs' motion for additur with respect to the award for Mr. Jolly. The jury was patient and attentive for eight days of trial, then deliberated for five hours and awarded a significant sum of money to Mr. Jolly. Mr. Jolly's attorney, Simona Farrise, has publicly commented that she is "very satisfied" with the verdict. (*See* Court Room View Network Email, attached hereto as **Exhibit B**). The award may be characterized as conservative, but it was not inappropriately so, was not unjustified, and it should be affirmed.

As stated previously, additur is appropriate only where an award is unduly conservative, and where "compelling reasons" exist to justify invading the province of the jury [*Riley v. Ford Motor Co.*, 414 S.C. at 192, 777 S.E.2d at 828, 829], and, further, non-economic damage determinations are particularly within the province of the jury. *Kalchthaler*, 316 S.C. at 503, 450 S.E.2d at 623. The word "unduly" means to "an unwarranted degree," "inordinately, or in "an unjustifiable manner." *See Dictionary.com*; *Merriam-Webster*; *Oxford Dictionary*. As the authorities cited at pages 2 and 3 of this brief make clear, a court will not vacate a jury's damage conclusions simply because it believes a different result seems more reasonable or appropriate. To the contrary, jury determinations are entitled to substantial deference. That is particularly true here, where the jurors impressed the Court with the detailed attention they paid to the case and then took 5 hours to consider the verdict questions formulate their answers to it.

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The Plaintiffs begin their argument with a flawed premise; they claim that the jury did not award anything to Mr. Jolly for his pain, suffering or mental anguish. (Brief, pp. 14, 16). This claim violates two guiding requirements: (1) that the evidence and reasonable inferences to be drawn therefrom be considered in the light most favorable to the nonmoving party; *Umhoefer, supra,* 298 S.C. 221, and (2) that the jury was not required to unquestioningly accept Plaintiffs' speculative economic damage evidence. *Steele, supra*, 327 S.C. at 343-4. To the contrary, if there is an "utter lack of documentation to support" an economic damage award, it "is not subject to challenge." *Tarrants,* 225 F.3d at *4. As noted in section II of this brief, Plaintiffs failed to put on convincing evidence of the economic damages. What medical expenses might be incurred for the treatment of mesothelioma is not common knowledge; Plaintiffs made a calculated decision to focus on liability and not submit medical bills or take other approaches that a jury may have found convincing as to economic damages. Additionally, if Plaintiffs wanted to know what sum the jury awarded for economic damages, they could have asked for a separate question on the verdict form concerning such damages. They did not do so. *See, e.g., Smoak v. Liebherr-Am., Inc.*, 281 S.C. 420, 421, 315 S.E.2d 116, 118 (1984) (duty of party to submit special verdict form); *Moore v. Moore*, 360 S.C. 241, 257, 599 S.E.2d 467, 475 (Ct. App. 2004) ("Appellant did not request the trial court to submit a special verdict form to determine whether the actual damages were for lost profit or some other measure. Without a special verdict form, we cannot speculate as to what portion of the award the jury attributed to lost profit as opposed to other tort damages."). Defendants respectfully submit that the verdict award to Mr. Jolly much more likely reflects a six-figure award to Mr. Jolly for non-economic damages than no award at all.[4] There is no basis to claim the award included nothing for non-economic damages.

[4] Plaintiffs cite *Waring v. Johnson*, 341 S.C. 248, 260, 533 S.E.2d 906 (Ct. App. 2000), but there the court could

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The Plaintiffs also base their arguments on the proposition that Mr. Jolly's damage evidence went largely uncontested. However, awards for non-economic damages in personal injury cases are always highly subjective and indeterminate. *See Kalchthaler, supra*, 450 S.E.2d at 623. The award to Mr. Jolly was substantial by any ordinary measure.

Plaintiffs cite *Garvin v. Agco Corp.*, No. 2012-CP-40-6675 (S.C. Ct Comm. Pleas). (Plaintiffs' Br., Ex. 7). They attempt to use the result in *Garvin* as a measure of Mr. Jolly's damages by noting that "Mr. Garvin's total economic damages were no more than $300,000 to $450,000" (Brief, p. 19), but this ignores the fact that the proof of economic damages in that case was far more concrete than here. Mr. Garvin had greater past medical expenses than Dr. Frank *recalled from memory* was the case for Mr. Jolly, and Mr. Garvin's future medical expenses were presented in a fairly concrete range as between $150,000 and $300,000. Certainly Mr. Garvin was awarded more for the non-economic components of his case, but such results happen. No two cases, no two claimants and no two jury panels are the same, and any number of reasons may explain the differing results, including the detail of the testimony provided, the precision and accuracy with which Plaintiffs presented their cases, the degree to which the jury was impressed by the witnesses and other factors. In our case, Mr. Jolly and his family were very pleased with the care they received at Duke, a world-renowned medical facility. (7/25/17 TT, p. 166). Specific factors such as this may also have influenced the result.

There are numerous mesothelioma verdicts in which similar awards for non-economic damages have been made. These include the following:

- *Ford v. Ferro Engineering*, 2014 Jury Verdicts Lexis 4373 (Pa.Com.Pl, 2014) (jury awarded $50,000 for pain and suffering in mesothelioma case).

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easily determine that there was no pain and suffering award because "The jury's award of exactly the amount of Waring's medical expenses, to the penny, is an attempt to reimburse her for those very expenses."

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- *Ihlenfeld v. Ferro Engineering*, 2014 WL 5023563 (Pa.Com.Pl, 2014) (award of $120,000 for pain and suffering where claimant was diagnosed with and eventually died of mesothelioma).
- *Paasch v. Crane Co.*, 2012 WL 4077858 (Pa.Com.Pl. 2012) ($38,000 for pain and suffering and $70,000 for wrongful death in mesothelioma case).

Cases that Plaintiffs cite (Brief, pp. 19-20) are distinguishable. For example, in *Goede v. Aerojet Gen. Corp.*, 143 S.W.3d 14 (Mo. Ct. App. 2004), the plaintiff was a 43-year-old mother of three when she died; at that age her life expectancy was at least 30 years longer than that of Mr. Jolly, so the $2,000,000 pain and suffering award there was not out of proportion to the award here.

The cases of *John Crane, Inc. v. Linkus*, 190 Md. App. 217, 988 A.2d 511 (Md. Ct. App. 2010), *In re New York City Asbestos Litigation (Re D'Ulisse)*, 842 N.Y.S.2d 333 (N.Y. Sup. Ct. 2007) and *Williams v. CSX Transp., Inc.*, 176 N.C.App. 330, 626 S.E.2d 716 (N.C. Ct. App. 2006) simply illustrate the deference that courts in other states have paid to jury verdicts. All of these cases had fairly high awards, but based on the specific facts of those cases, the courts reviewing them did not find the awards to be unduly high, just as the Court here should not find the verdict in this case unduly low.

These cases are also distinguishable. The jury in *Linkus* deliberated for only 34 minutes and multiple jurors were inattentive (*See* 988 A.2d at 527); that time seems inadequate and it pales in comparison to the time the jury in the case at bar deliberated, while, as the Court noted, our jury was extremely attentive. In the *Re D'Ulisse* case, there was a finding of recklessness, which is a factor that tends to drive up compensatory awards. There was also an extremely detailed account of the loss, ranging from plaintiff's pre-disease ability to engage "in such

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activities as baseball, handball, paddle ball and dancing," to radiation treatment that left his skin "so itchy and burnt red so that (plaintiff) couldn't put his clothes on (and caused him to suffer) from severe constipation and rectal bleeding" and other maladies, to "loss of sexual companionship." 842 N.Y.S.2d at 336. As with *Goede*, the plaintiff in *Williams* was far younger, having a 21-year life expectancy. Two of these verdicts also came from extremely pro-plaintiff, high-verdict jurisdictions, Baltimore (*Linkus*) and New York City *(Re D'Ulisse)*. Similarly, *Romano v. Metropolitan Life Ins. Co.*, 221 So.3d 176, 2017 WL2265424 (La. Ct. App. 2017) was from such a jurisdiction, New Orleans. *Romano* is also noteworthy as the plaintiffs appear to have been very precise in their proof, as the court noted the "medical bills totaled over $566,274.00"; additionally plaintiff continued to be in pain two years following his diagnosis.

IV. THERE IS NO BASIS TO GRANT *ADDITUR* AS TO THE AWARD FOR MRS. JOLLY.

As to Mrs. Jolly's damages, the Plaintiffs simply disagree with the sum awarded, and in requesting *additur* present the Court with the most favorable possible view of the evidence relating to her claim, while ignoring evidence or reasonable conclusions to be drawn from the evidence that support the jury's award. The Plaintiffs' motion for *additur* with respect to Mrs. Jolly's claim should be rejected because the $100,000 award was well within reason based on the evidence.

The Plaintiffs' evidentiary presentation at trial focused overwhelmingly on liability issues. In contrast, their presentation specifically relating to Mrs. Jolly's loss of consortium claim was extremely brief. Mr. Jolly himself provided no testimony at all concerning his relationship with his wife; he never even mentioned her. Further, counsel did not elicit many specifics from Mrs. Jolly concerning how her husband's illness affected their relationship or her daily routine. Plaintiffs presented testimony that Mrs. Jolly had a mild heart attack a few weeks

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before trial, but for a number of reasons, the jury was certainly entitled to believe that her heart attack was unrelated to her husband's illness. No physicians testified that her heart attack related to Mr. Jolly's disease or to any stress that Mrs. Jolly may have had because of her husband's disease; most heart attacks are not related to emotional upset, but rather to the slow process of atherosclerosis; and, finally, she did not appear to show any signs of illness from heart disease when she appeared at trial.

The jury was properly instructed concerning the elements of a loss of consortium claim, but Plaintiffs did not submit evidence concerning several of those elements. For instance, there was minimal or no testimony concerning what services Mr. Jolly may have provided in the Jollys' home. An exception was testimony that Mr. Jolly gardened, but he stopped doing so in 2014 (7/25/17 TT, p. 170). This was over one year before he became ill, so a jury was entitled to conclude that he stopped gardening for reasons unrelated to his illness.

Awards for loss of consortium in other asbestos-related cases illustrate that the award here was reasonable. Those cases include the following:

- A California jury awarded $100,000 to the widow of a man who died of mesothelioma in *McCarthy v. AstenJohnson, Inc.*, 2009 WL 577769 (C.D. Cal.).
- A Connecticut jury awarded $100,000 to the widow of a man who died of mesothelioma in *Acquarulo v. The Tile Council of North America,* in 2012. (*See* Case Summary, attached hereto as **Exhibit C**).
- A California jury awarded $100,000 to the widow of a man who was diagnosed with mesothelioma in 2010 and later went to trial. (*Gray v. Highland Stucco* case.) (*See* **Exhibit C**).

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- In *Tarrants, supra*, the Sixth Circuit, in ruling on a request for a new trial based on an award of $5,000 for loss of consortium (and on other grounds as discussed above), stated that since the decedent had been diagnosed with cancer only two months prior to his death and had other ailments, the award was "not so unreasonable as to be subject to challenge." *Id.*, at *4.
- A New Jersey jury awarded $100,000 for loss of consortium to a widow of a man who died of mesothelioma at age 63 in *Schiavo v. Owens-Corning Fiberglas Corp.*, 282 N.J. Super. 362, 660 A.2d 515 (1995).
- *Thalman v. Owens-Corning Fiberglas Corp.*, 290 N.J.Super. 676, 676 A.2d 611 (1996) the jury awarded $168,000 for loss of consortium to a widow of a 68 year old who died of mesothelioma.
- *Paasch v. Crane Co.*, 2012 WL 4077858 (Pa.Com.Pl. 2012) ($15,000 loss of consortium award in mesothelioma case).

Reported verdict awards in non-asbestos cases in South Carolina also illustrate the reasonableness of the award here. These cases include the following:

- *Branham v. Columbia Neurosurgical Associates P.A.* 2002 WL 34935718 (S.C.Com.Pl., Richland County, 2002). In this medical malpractice case, a 57-year-old male suffered a heart attack and stroke with permanent severe neurological injuries, including serious brain stem trauma and paralysis to his entire left side. He was awarded over $1.6 million, while the award for loss of consortium was $27,740.
- *Starnes v. Cundey*, 2013 WL 8282951 (S.C.Com.Pl, Aiken County, 2013): In this medical malpractice case, an adult male sustained burns and scarring to his back

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that required skin grafting. While he was awarded $100,000 in damages, his spouse was awarded $5,000.

The Plaintiffs also suggest that if additur is granted, Mrs. Jolly should be awarded one-half the amount Mr. Jolly was awarded. However, there is no mechanical formula for awarding damages for loss of consortium, as the results discussed above demonstrate. The award to Mrs. Jolly was not an outlier. It was a reasonable award based on the evidence and should not be overturned.

CONCLUSION

The jury here deliberated for five hours, and during that time it asked questions of the Court and demonstrated the great depth at which it considered the issues. The Court noted it was "amazed at the kind of seriousness and attention that you have paid and consideration you have given to both sides." (8/3/17 TT, p. 196). The jury's damage calculation should not be disturbed. For these reasons and those set forth above, Defendants Crosby and Fisher respectfully request that the Court deny the motion for *nisi additur*.

Dated this 6th day of September, 2017.

LEATH, BOUCH & SEEKINGS, LLP

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EXHIBIT C

Connecticut Jury Awards $2,403,339 In Asbestos Case; Industry Group Found Liable

Mealey's(R) Litigation Report: Asbestos

Online ISSN: 2158-9798, Print ISSN: 0742-4647

June 6, 2012

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Body

A Connecticut jury on May 22 held an industry group liable for a man's exposure to asbestos and awarded his surviving family members $2,403,339 (Gail S. Acquarulo, Executrix of the Estate of Hannibal Saldibar, et al. v. Tile Council of North America Inc., f/k/a Tile Council of America, No. CV095024498S, Conn. Super., Fairfield at Bridgeport Dist.; See 2/15/12, Page 12).

(Verdict sheet available 01-120606-012V)

The jury's award consists of $3,339 in economic damages, $1.5 million in noneconomic damages and $100,000 in loss of consortium for Gail Acquarulo.

Judge Dale Radcliffe then awarded $800,000 in punitive damages based on the jury's finding of reckless disregard, sources told Mealey Publications.

Acquarulo filed suit as the executrix of the estate of Hannibal Saldibar in the Fairfield at Bridgeport District Superior Court in April 2009 against numerous defendants whose conduct allegedly exposed him to asbestos, causing his death. Saldibar's exposure allegedly came while he was serving as a petty office in the U.S. Navy from 1943 to 1946 and as a tile setter from 1946 to 1979.

The Tile Council of North America moved for summary judgment, arguing that as a trade association, it did not fall within the Connecticut Products Liability Act's (CPLA) definition of a distributor or retailer.

Liability

Judge Barbara Bellis denied the motion, finding that Acquarulo's allegations that the Tile Council held a patent on mortar, controlled how that product was used and marketed and held itself out as a manufacturer created triable issues.

Judge Bellis noted that the Tile Council provided detailed specifications governing the product, the percentage of asbestos to be used and the grade of asbestos to be used. The Tile Council received fees for licensing the product and issued warnings regarding its use, Judge Bellis said. Further, the product was marketed with the defendant's name prominently displayed, Judge Bellis said.

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Christopher Avallone

EXHIBIT C

Acquarulo called Barry Castleman, Sc.D., an expert in state of the art from Baltimore, Arnold Brody, Ph.D., a pathologist and cell biologist from Raleigh, N.C., and Jerrold Abraham, a pathologist from Syracuse, N.Y. The Tile Council did not call any expert witnesses.

At trial, the Tile Council argued that it was neither a manufacturer nor a supplier of asbestos-containing tile products.

Sources told Mealey Publications that the verdict is believed to be the first in an asbestos case against the Tile Council.

Brian Kenney and Chris Meisenkothen of Early Lucarelli Sweeney & Meisenkothen in New Haven, Conn., represent Acquarulo. Philip J. O'Connor of Gordon, Muir & Foley in Hartford, Conn., and Robert S. Bystrowski of Morrison Mohoney in Hartford represent the Tile Council.

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Christopher Malone

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LEVIN SIMES LLP ATTORNEY WINS $830,000 VERDICT ON BEHALF OF CATERING TRUCK DRIVER EXPOSED TO ASBESTOS

Robert Gray was diagnosed with mesothelioma in May of 2010. Mr. Gray, who was 83 years old at the time of his diagnosis, had been a cook and catering truck operator for much of his working life. At the time of his diagnosis, Mr. Gray had no personal knowledge that he had ever worked with or around asbestos or asbestos-containing products.

Fortunately, Mr. Gray and his wife Norma contacted LEVIN SIMES, LLP. The attorneys and investigators at LEVIN SIMES, LLP immediately went to work reconstructing Mr. Gray's work history. The investigation revealed that between 1972 and 1990, Mr. Gray worked a catering truck route in Van Nuys, CA, which included twice daily visits to the parking lot of a manufacturing plant operated by Highland Stucco & Lime Products, Inc. ("Highland Stucco"). The investigation further revealed that for a portion of that time Highland Stucco was blending raw asbestos and other asbestos-containing raw materials into many of the products it manufactured at the plant.

At trial, Highland Stucco's attorneys argued that because Mr. Gray did not personally handle Highland Stucco's asbestos-containing products and because of his proximity to the manufacturing portion of the plant, Mr. Gray's exposure to asbestos,

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if any, would have been negligible. Highland Stucco also argued that Mr. Gray's disease was most likely "idiopathic" which means without an identifiable cause.

LEVIN SIMES, LLP attorney Sean Worsey tried the case on behalf of Mr. and Mrs. Gray, and presented testimony from a Certified Industrial Hygienist demonstrating that Mr. Gray was likely exposed to significant and unsafe levels asbestos during his twice daily interactions with Highland Stucco's workers. Plaintiffs' counsel also presented medical and scientific evidence demonstrating that even very low levels of asbestos exposure can substantially increase the risk of mesothelioma in susceptible persons.

The Verdict

The jury found that Mr. Gray was exposed to asbestos during his visits to Highland Stucco, and that the exposure was a substantial factor contributing to his mesothelioma. The jury ultimately found Highland Stucco liable for failing to warn Mr. Gray about the hazards of asbestos that were then known or knowable to Highland Stucco through the use of scientific knowledge.

The Award

The jury awarded a total of $830,000 to Mr. and Mrs. Gray, including: $600,000 for Mr. Gray's pain and suffering: $100,000 for Mrs. Gray's loss of consortium; and $130,000 for past and future medical expenses. Highland Stucco was found 100% at fault.

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STATE OF SOUTH CAROLINA **COUNTY OF SPARTANBURG** **BEVERLY DALE JOLLY and BRENDA RICE JOLLY,** **Plaintiffs,** **v.** **GENERAL ELECTRIC COMPANY, et al.** **Defendants.**	)))))))))))))))	**IN THE COURT OF COMMON PLEAS** **FOR THE SEVENTH JUDICIAL CIRCUIT** **C/A No. 2016-CP-42-1592** **PLAINTIFFS' RESPONSE IN OPPOSITION TO DEFENDANTS FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE, LLC'S JOINT MOTION FOR JNOV**

Plaintiffs Beverly Dale Jolly and Brenda Rice Jolly oppose the motion for JNOV filed by Defendant Fisher Controls International LLC ("Fisher") and Defendant Crosby Valve, LLC ("Crosby") (collectively "Defendants"), and would respectfully show the Court that the motion should be denied and that the jury's verdict should be upheld as well grounded in the evidentiary record and South Carolina law.

STANDARD OF REVIEW

In ruling on a JNOV motion, the trial court is required to view the evidence and the inferences that reasonably can be drawn therefrom in the light most favorable to the non-moving party. *Law v. S.C. Dept. of Corrections*, 368 S.C. 424, 434, 629 S.E.2d 642, 648 (2006); *Swicegood v. Lott*, 379 S.C. 346, 351, 665 S.E.2d 211, 213 (Ct. App. 2008); *Israel v. Carolina Bar-B-Que, Inc.*, 292 S.C. 282, 287, 356 S.E.2d 123, 127 (Ct. App. 1987). The court must deny the motion for JNOV when the evidence yields more than one inference or its inference is in doubt. *Id.*; *Steinke v. South Carolina Dep't of Labor, Licensing, & Regulation*, 336 S.C. 373, 386, 520 S.E.2d 142, 148 (1999). The jury's verdict may not be overturned if *any evidence* sustains the factual findings

implicit in its decision. *Welch v. Epstein*, 342 S.C. 279, 300, 536 S.W.2d 408, 419 (Ct. App. 2000); *Smalls v. South Carolina Dep't of Educ.*, 339 S.C. 208, 215, 528 S.E.2d 682, 686 (Ct. App. 2000).

ARGUMENTS AND AUTHORITIES

I. Plaintiffs have met their burden on their failure to warn claims.

A. Jolly did not know that asbestos gaskets and packing were dangerous to his health.

Fisher and Crosby first contend that they had no duty to warn Jolly because the danger was open and obvious. This is contradicted by the fact that Fisher and Crosby take the position, *to this day*, that asbestos gaskets and packing are not dangerous. (**Ex. A**, TT 7/27 at 145-46).[1] Neither Duke, nor Jolly knew that the asbestos gaskets and packing used in Fisher and Crosby valves were dangerous because Fisher and Crosby never informed anyone of that fact.

It is, of course, correct that "'a seller is not required to warn of dangers or potential dangers that are generally known and recognized.'" *Moore v. Barony House Rest., LLC*, 382 S.C. 35, 41–42, 674 S.E.2d 500, 504 (Ct. App. 2009) (quoting *Anderson v. Green Bull, Inc.*, 322 S.C. 268, 270, 471 S.E.2d 708, 710 (Ct. App. 1996). The jury was instructed to this effect in this case. It was told that there is no duty to warn of a danger that is "obvious" or that is "generally known and recognized." (TT 8/3 at 124).[2]

The jury rightly found that Jolly did not know that exposure to asbestos gaskets and packing could cause fatal disease and that Defendants were not relieved of their duty to warn. Defendants rely entirely on Jolly's testimony discussing warnings that Duke provided about asbestos

[1] The citations to the trial transcript, abbreviated "TT," are to the rough transcript. The parties do not yet have the final trial transcript. In some cases, there are gaps and undecipherable words in the rough transcript. When quoting the transcript, Plaintiffs have taken the liberty of inserting in brackets what appears to be the correct testimony.

[2] This transcript is attached to Plaintiffs' Motion for New Trial *Nisi Additur* as Exhibit 6.

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insulation. This is clear from the context as well as Jolly's numerous references to insulation. (TT 7/25 at 119-20, 124-25, 130, 135).[3] Jolly's testimony makes clear that he was cognizant of safety warnings and followed any pertinent warnings about asbestos.

The evidence established that Duke *did not know* that asbestos gaskets were hazardous, and thus was not warning its employees. (**Ex. D**, Pl's Ex. 136). A Duke document from September 1984 instructing employees how to protect themselves from asbestos insulation, states that asbestos used in gasket material is "bonded, which means it produces virtually no dust." (Pl's Ex. 136 at Script, p. 3). Jolly explained that he was only warned by Duke about asbestos hazards that Duke knew about. (TT 7/25 at 142). Jolly's co-worker, David Taylor, testified that when Duke gave its employees information about asbestos hazards, Duke made a distinction between insulation and gaskets. (TT 7/27 at 126). Employees did not know there was a danger from asbestos gaskets until several years after Duke started warning that precautions needed to be taken around asbestos insulation. (TT 7/27 at 127).

Fisher's representative, Ronald Duimstra, contended at trial that, with regard to asbestos components sold with Fisher valves and as replacement parts "there's no health risk and therefore there would be no reason to warn." (TT 7/27 at 146; *see also id.* at 148). Duimstra testified that there was no action for Duke to take with respect to warning about asbestos gaskets and packing:

> Q. But as to the internal components that were asbestos containing and included within the Fisher valve that was made in Iowa and sold to the Duke plants, [] just trying to be clear, is it Fisher's contention that there was no health hazard and **so there was nothing for Duke to do**?
>
> A. Yes that would be correct.

(TT 7/27 at 148-49, emphasis added). Crosby's representative, Robert Martin (**Ex. C**, TT 7/31 at

[3] This transcript is attached to Plaintiffs' Motion for New Trial *Nisi Additur* as Exhibit 2.

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2, 4), similarly testified that "[w]e saw no danger from the gasket and the way we use them." (TT 7/31 at 33).

Not only do Fisher and Crosby contend there was no health hazard from working with asbestos gaskets and packing, they did not always inform Duke that these materials contained asbestos. Some of Fisher's asbestos-containing packing specifications did not describe the material as containing asbestos, and instead referred to it as metallic or plastic. (TT 7/27 at 208-09). Duimstra agreed that a reference to a metal packing ring would not alert people that the product contained asbestos. (TT 7/27 at 210). Duke purchase orders to Fisher stated the Fisher part number and a brief description, but oftentimes in order to determine if the part contained asbestos one would have to reference Fisher's own material specification for that part number. (TT 7/27 at 215-16, 221-22). For example, a gasket described as nitrile did not state it was asbestos in some of the purchase orders, but that was, in fact, an asbestos-containing part. (TT 7/27 at 215-16, 225; **Ex. E**, Pl's Ex. 1012.5). Jolly's co-worker, David Taylor, testified that the only way to know if a gasket had asbestos was whether it was marked as asbestos-containing on the gasket packaging. (TT 7/27 at 115).

It is not possible to conclude, as a matter of law, that asbestos gaskets and packing were obviously dangerous to Jolly when Fisher and Crosby completely deny that they were dangerous at all and did not even consistently notify Duke and its employees that valve gaskets and packing were asbestos-containing. The question of whether Jolly knew about the danger of working around asbestos gaskets and packing was for the jury, which it properly decided in Plaintiffs' favor.

B. Defendants are not protected by the sophisticated intermediary defense.

This Court should decline to recognize the sophisticated intermediary defense in this case. Defendants ask this Court to follow California law, not South Carolina law, regarding the

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sophisticated intermediary defense. The South Carolina Supreme Court has observed that the sophisticated intermediary defense has not been adopted in South Carolina. *See Lawing v. Univar, USA, Inc.*, 415 S.C. 209, 226, 781 S.E.2d 548, 557 (2015), *reh'g denied* (Feb. 12, 2016). The Court explained that no South Carolina court has adopted this defense, and that it was not necessary to do so for the Court to reach its decision in *Lawing*:

> In arguing that the court of appeals erred in affirming the trial court's decision to charge the jury on the sophisticated user defense, the Lawings contend that the sophisticated user defense is not the law of South Carolina. **We agree that prior to the court of appeals' opinion in this case, neither this Court, nor the court of appeals, had explicitly adopted the defense However, we need not formally adopt the doctrine at this time** because . . . the facts of this case do not implicate the sophisticated user defense.

Id. (emphasis added).

Even if the sophisticated intermediary defense was a recognized defense in this State, Defendants have failed to show that it is applicable under the facts of this case. As in *Lawing*, the defense is not applicable here. In *Lawing*, the Court held that the question of "the adequacy of the labeling on the sodium bromate does not require a sophisticated user analysis." *Id.* at 228. There, the employer was aware that sodium bromate was dangerous, but the defendant product supplier had failed to put visible labels on the product indicating that it contained sodium bromate. The situation in this case is similar in that Duke may have known that asbestos was hazardous, but neither Duke nor Jolly knew that friable asbestos was released from Defendants' asbestos gaskets and packing because those products contained no warning to that effect. Defendants took the position that the asbestos was encapsulated in these products, and have not shown that Duke knew any differently. Duke's knowledge was thus not useful to Jolly because of the inadequacy of Defendants' labeling.

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Defendants have failed to meet the elements of the sophisticated intermediary defense in this case. Even under the California authority they cite, this is an affirmative defense for which they have the burden of proof. *Webb v. Special Elec. Co.*, 63 Cal. 4th 167, 187, 370 P.3d 1022, 1034 (2016). Even under *Webb* their burden is high: "Because the sophisticated intermediary doctrine is an affirmative defense, **the supplier bears the burden of proving that it adequately warned the intermediary, or knew the intermediary was aware or should have been aware of the specific hazard, and reasonably relied on the intermediary to transmit warnings.**" *Id.* (emphasis added).

Fisher and Crosby have not shown that they warned Duke; the evidence establishes that Fisher and Crosby provided no warnings to Duke whatsoever. (TT 7/27 at 144; TT 7/31 at 64-65). Fisher and Crosby also failed to show that they knew Duke was aware or should have been aware of the danger from asbestos gaskets. Again, the evidence shows the opposite: Duke distinguished asbestos gaskets as non-hazardous because it was thought they did not release asbestos fibers when disturbed. (Pl's Ex. 136 at Script, p. 3; TT 7/27 at 126). And Fisher and Crosby certainly have not shown that they were relying on Duke to inform its employees of those hazards. They introduced no evidence to this effect.

Finally, this defense does not entitle Defendants to judgment as a matter of law. The jury was instructed on the sophisticated intermediary defense and the elements that Fisher and Crosby were required to prove. (TT 8/3 at 135-36). At a minimum, there were questions for the jury regarding Duke's knowledge about asbestos gaskets and packing and whether Fisher and Crosby were reasonably relying on Duke to pass on any warnings about asbestos gaskets and packing. The record clearly supports the jury's rejection of this defense.

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C. There was not an unanticipated material alteration to Defendant's valves after they left the factory.

Fisher and Crosby contend that they are not liable for Jolly's exposure to asbestos flange gaskets because the flange gaskets "were placed between piping and the Defendants' valves *after* they were sold and sent to Duke." JNOV Motion at 10. Because the flange gaskets were applied to the flange during the installation of the valve at the power plant, Fisher and Crosby argue that the valve was not in the same condition as when they sold it.

Plaintiffs cannot agree that connecting flanged valves to a pipeline in the field could possibly be considered an alteration of the valve. Fisher and Crosby sold valves that were connected to the pipeline with a flange connection that utilized flange gaskets. (TT 7/27 at 187-89; 7/31 at 122-23, 125). A lot of the valves Crosby sold to Duke had flanged connections. (TT 7/25 at 74; TT 7/31 at 164-65). The flange connections on the valve were manufactured by Defendants before they left the factory. (TT 7/31 at 18).

Duimstra explained that the valve was connected to the pipeline via the flange on the valve:

> Q. Can we agree sir that the Fisher basic valve contains these kinds of metal flange faces that are part of what is sold from Fisher to customers?
>
> A. This is a flanged globe style sliding stem valve.
>
> Q. And it has a flange face?
>
> A. Yes.
>
> Q. And unless it is connected to a piping system it really doesn't have any purpose just is [s]itting there correct?
>
> A. No. That's the purpose of a flange. See valves have to be connect to the pipeline and so flanges are one way to connect it to the pipeline.

(TT 7/27 at 187; *see also* TT 7/31 at 18-19).

Moreover, Fisher and Crosby intended that flange gaskets would be used to connect their valves to the pipe line. Duimstra explained:

> Q. And at those two flange faces there would be a gasket to connect it to the piping system correct depend[ing on]what kind of flange it was?
>
> A. Yes if they ordered this type of valve there would be, the customer would have a requirement to purchase and install a gasket on each [side] of that valve to connect to the pipeline. Gasket is basically a means by which two metal surfaces that may have imperfections an[d] it provides a sealing what's called a static seal and it allows the pressure to stay inside, i[t] squeeze[s] that gasket together very tightly and it keeps the pressure from escaping that joint.

(TT 7/27 at 188-89). Martin similarly agreed that "the design was a flange that would have to have a gasket." (TT 7/31 at 123).

Fisher and Crosby knew that asbestos flange gaskets would sometimes be used. (TT 7/31 at 122-23). Defendants' engineers determined what materials the flange gaskets were made of to create the right seal under different conditions. (TT 7/27 at 189). Taylor recalled that the Fisher and Crosby valves present at Duke had flange connections that utilized asbestos flange gaskets. (TT 7/26 at 210-13).

Defendants' alteration argument also fails because Fisher and Crosby did not simply sell valves, they both also sold replacement asbestos flange gaskets to Duke. Taylor testified that all of the valve flange gaskets were supplied by the valve manufacturers. (TT 7/26 at 214-15; TT 7/27 at 104-05). Most of those valve flange gaskets were asbestos gaskets. (TT 7/26 at 202, 210-13). Duke purchase orders corroborate his recollection, as they show asbestos flange gaskets being sold by Fisher. (**Ex. F**, Pl's Ex. 1012.6). The jury could have easily found that Jolly was exposed to asbestos flange gaskets sold directly by Defendants.

Finally, to the extent that the use of a flange gasket on a flanged valve could be considered an "alteration" of the valve, it was an entirely foreseeable and expected modification. "Liability

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may be imposed on a manufacturer or seller notwithstanding subsequent alteration of the product when the alteration could have been anticipated by the manufacturer or seller." *Small v. Pioneer Machinery, Inc.*, 329 S.C. 448, 466, 494 S.E.2d 835, 844 (Ct. App. 1997). Only "if it can be shown that the product was (1) materially altered before it reached the injured user and (2) such alteration could not have been expected by the manufacturer or seller, then the manufacturer or seller is not liable." *Fleming v. Borden, Inc.*, 316 S.C. 452, 457, 450 S.E.2d 589, 592–93 (1994).

Fisher and Crosby cannot possibly show that the "alteration" of their valves by placing a flange gasket in the valve flange was unexpected. The valves could not be placed in service without use of a flange gasket, and, as noted, Fisher and Crosby sold asbestos flange gaskets to Duke. The jury properly credited this evidence and declined to find an unexpected alteration to the valves simply from application of a flange gasket in the field that was called for by Defendants' flanged valve design.

D. Plaintiffs' evidence shows that Defendants' failure to warn was a proximate cause of Jolly's mesothelioma.

Fisher and Crosby next contend that Plaintiffs did not show that Defendants' failure to warn about the dangers of asbestos exposure from their valves was a proximate cause of Jolly's mesothelioma. They contend that there is no evidence that Jolly would have heeded such a warning if it had been given. Fisher and Crosby are mistaken.

Defendants rely on *Allen v. Long Mfg. NC, Inc.*, 332 S.C. 422, 505 S.E.2d 354 (Ct. App. 1998), which does state the basic rule that the failure to warn must be the proximate cause of the plaintiff's injuries. *Allen* makes clear, however, that the issues of proximate cause, and the adequacy of the defendant's warning, are for the jury to decide. *Id.* at 432-33, 505 S.E.2d at 360. In *Allen*, the product manufacturer did warn and the plaintiff did not follow the warning. But

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because there was evidence that the warning was inadequate, that created a fact issue as to whether a different warning would have caused the plaintiff to behave differently. *Id.*

If there was a fact issue for the jury in *Allen*, when the plaintiff failed to follow an inadequate warning, there is certainly one here when the evidence shows that Jolly *followed* every asbestos warning he was given. Jolly testified that when he saw asbestos warnings at Duke, he heeded them and avoided touching asbestos insulation. (TT 7/25 at 120). He did not go into areas that were cordoned off for asbestos abatement. (TT 7/25 at 125). When he was in a zone that required a respirator, he wore a respirator. (TT 7/25 at 121, 127).

The evidence further showed that Jolly checked for any applicable warnings on Fisher and Crosby products:

> Q. Did you ever see any asbestos warnings on replacement gaskets that Crosby sold to Duke or on replacement gaskets that Fisher sold to Duke?
>
> A. No. They weren't there.

(TT 7/25 at 142). Jolly knew there were no warnings from Fisher and Crosby because he checked and they were not there.

The evidence showed, of course, that Fisher and Crosby did not ever warn about the hazards of asbestos on the asbestos-containing products they sold to Duke. (TT 7/25 at 209-10, 218-19; TT 7/27 at 143-44, 232; TT 7/31 at 64-65). Plaintiffs' expert, Dr. Frank, testified that product warning labels were required by OSHA as of 1972 and that Fisher and Crosby's products should have contained asbestos warnings. (TT 7/26 at 110-11, 189).[4]

The jury therefore had every reason to conclude that Jolly would have followed an adequate warning if one had been present on Fisher and Crosby products. Under *Allen*, the mere fact that an

[4] This transcript is attached to Plaintiffs' Motion for New Trial *Nisi Additur* as Exhibit 3.

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adequate warning was missing from the products was sufficient to create a jury question regarding whether an adequate warning would have changed Jolly's behavior. 332 S.C. at 432-33, 505 S.E.2d at 360. This issue was therefore properly left to the jury, and the evidence supported its determination that Fisher and Crosby's failure to warn was a proximate cause of Jolly's mesothelioma.

II. Plaintiffs have met their burden of proof on their design defect claims.

Fisher and Crosby contend that Plaintiffs failed to prove their design defect claims because, they argue, there was no evidence of a reasonable alternative design. While a reasonable alternative design must be shown, this is part of the risk-utility test for design defect that is a matter for the jury to decide. *See Branham v. Ford Motor Co.*, 390 S.C. 203, 219, 701 S.E.2d 5, 13 (2010). The jury weighs the availability of a feasible alternative design, as well as the associated costs, safety, and functionality. *Id.* at 225, 701 S.E.2d at 16.

There was overwhelming evidence that Fisher and Crosby had alternative feasible designs available to them at the time they were selling asbestos gaskets and packing with their valves and as replacement parts.

Duimstra testified that non-asbestos gasket and packing materials were always available:

> Q. So I think sir we agree that the Fisher control valve company at all times had available to sell to its customers packing and gaskets that were nonasbestos containing right?
>
> A. Yes.

(**Ex. B**, TT 7/28 at 141). Martin agreed that "one of the standards at that time would be asbestos but there are other materials they could have used." (TT 7/31 at 122-23).

Duimstra and Martin both testified that there were a variety of non-asbestos gasket materials available, including graphite, metal, and Teflon. (TT 7/28 at 100; TT 7/31 at 125).

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Defendants both sold some valves to Duke that did not contain asbestos materials. (TT 7/31 at 139, 163).

With regard to packing, Duimstra testified that "the most common [selected] by the customer would be Teflon [v-]ring packing and that's been available since the 50s any way and it's extremely good packing material." (TT 7/28 at 100). Duimstra testified at length about the beneficial qualities of Teflon packing:

> Q. Why would you use Teflon as opposed to asbestos?
>
> A. Teflon has properties in a that make the packing much [slipperier]. As you know about Teflon it's a slippery material. If you use it in a packing application it's a great []convenience for packing because it allows that stem that we talked about earlier to move up and down without much friction. And if you don't have friction that [] signal you get from the control room on that valve to move will automatically move as opposed to jerking with a packing that's not quite as slick so it provides the control to be much more accurate and much more responsive. Teflon is a plastic material that has some incredible properties with regard to low friction.

(TT 7/28 at 101). He reiterated that Teflon v-ring packing was available in the 1950s or 60s, was available to Fisher, and could have been sold instead of asbestos packing. (TT 7/28 at 140-41).

Duimstra acknowledged at trial that there was no reason for Jolly to be exposed to asbestos gaskets and packing because of the availability of alternative materials:

> Q. And just to be clear can we agree that it sounds like it's the position of Fisher Controls valve company that since it had nonasbestos products like Teflon available according to you since the 60s there was absolutely no reason that these men had to be exposed to asbestos in Fisher valves because you could have sold something else right?
>
> A. Something else could have been selected but that's kind of a perhaps a legal argument.

(TT 7/28 at 145-46).

The evidence thus shows that there were multiple non-asbestos materials that were available to Fisher and Crosby and that they have could have sold gaskets and packing to Duke

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that were made exclusively out of graphite, metal, or Teflon. Defendants did, in fact, sell gaskets and packing to Duke that were made out of these alternative materials. The use of such materials was entirely feasible and obviously not cost prohibitive. Given Duimstra's admission that "[s]omething else could have been selected," the evidence was more than sufficient to create a fact issue for the jury on reasonable alternative design.

III. The evidence shows Defendants deviated from the standard of care.

Fisher and Crosby are mistaken in their contention that there was a lack of evidence as to the standard of care and their deviation from that standard. A product manufacturer is held to the skill of an expert in its business and to an expert's knowledge of the materials and processes in its industry. *Carolina Home Builders, Inc.*, 259 S.C. 346, 358, 191 S.E.2d 774, 779 (1972); *see also Humphries v. Mack Trucks, Inc.*, 198 F.3d 236 (4th Cir. 1999); § 32-20 Products Liability - Standard of Care - Manufacturer, Anderson, S.C. Requests to Charge - Civil, 32-20. The manufacturer is therefore required to keep abreast and aware of current standards and scientific knowledge in his industry. *Id.* Because the manufacturer is held to the knowledge and skill of an expert, "[t]his is relevant in determining (1) whether the manufacturer knew or should have known the danger, and (2) whether the manufacturer was negligent in failing to communicate this superior knowledge to the user or consumer of its product." *Borel v. Fibreboard Paper Prods. Corp.*, 493 F.2d 1076, 1089-90 (5th Cir. 1973). Industry knowledge may be established through expert testimony. *Reed v. Tiffin Motor Homes, Inc.*, 697 F.2d 1192 (4th Cir. 1982).

Manufacturers have a duty to test and inspect their products. *Nelson v. Coleman Co.*, 249 S.C. 652, 657, 155 S.E.2d 917, 920 (1967). "A manufacturer who incorporates into his product a component made by another has a responsibility to test and inspect such component, and his negligent failure to properly perform such duty renders him liable for injuries proximately caused

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as a consequence." *Duncan v. Ford Motor Co.*, 385 S.C. 119, 133, 682 S.E.2d 877, 884 (Ct. App. 2009). In addition, "[s]afety standards promulgated by government or industry organizations [] are relevant to the standard of care for negligence." *Elledge v. Richland/Lexington Sch. Dist. Five*, 341 S.C. 473, 477–78, 534 S.E.2d 289, 290–91 (Ct. App. 2000), *aff'd,* 352 S.C. 179, 573 S.E.2d 789 (2002) (collecting cases).

Fisher and Crosby failed to meet the standard of care in many respects. They did not keep aware of the health hazards caused by the asbestos products they were selling, they failed to test their products to determine whether they were exempt from OSHA labeling regulations, they failed to comply with OSHA labeling requirements, and, of course, failed to warn about asbestos hazards in any way.

Plaintiff's expert Dr. Frank established that the dangers of asbestos exposure became known in industry around the turn of the century. (TT 7/26 at 86-89). In the early twentieth century there were a number of publicly available publications about the dangers of asbestos exposure. (TT 7/26 at 88-89). In 1930, researchers Meriwether and Price published a paper that not only described the risk of lung diseases from asbestos exposure, but set forth the precautions that could be taken to reduce exposures and reduce the risk. (TT 7/26 at 86). Crosby received such information in the 1930s through its membership in the National Safety Council. (TT 7/31 at 91-92; **Ex. G**, Pl's Ex. 102).

By 1955, it was established that occupational asbestos exposure could cause lung cancer. (TT 7/26 at 89-90). By 1960, it was known that asbestos exposure could cause mesothelioma. (TT 7/26 at 91-93).

In the 1950s, the U.S. Department of Labor established workplace safety regulations for government contractors, called the Walsh-Healey Act, that required employers to take steps to

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control asbestos exposure levels below a certain threshold. (TT 7/26 at 83-85; **Ex. H**, Pl's Ex. 149.) As government contractors, Fisher and Crosby were required to comply with the Walsh-Healey Act. (TT 7/26 at 83-84; 7/31 at 85-86).

Defendants failed to keep abreast of safety information regarding the asbestos products, as required by law. Although Fisher maintained a library at its manufacturing facility and corporate offices, it contained no publications regarding the hazards of asbestos. (TT 7/27 at 149). Crosby did not even maintain a library of health and safety materials. (TT 7/31 at 71, 99). Nor did it have an industrial hygiene or a medical department. (TT 7/31 at 71). Martin admitted that Crosby had done nothing to keep up with the dangers of the asbestos products it was selling:

> Q. I believe we agree that Crosby did nothing to your knowledge to stay abreast of the state of medical and scientific knowledge as it relates to whether and there was a health hazard from being exposed to minimal amounts or any amounts of asbestos?
>
> A. Not to my knowledge.

(TT 7/31 at 99).

By 1980, when Jolly began working around asbestos gaskets and packing associated with Fisher and Crosby valves, Defendants should have known that the only safe level of asbestos exposure is zero. (TT 7/26 at 109).

In Dr. Frank's opinion, Fisher and Crosby should have been warning users about the dangers of asbestos exposure from their valves:

> Q. So the question I have is what is your opinion Dr. Frank as to what Crosby valve company or Fisher Controls could have told Duke Power in 1979 or 1980 about whether there was a hazard from the regular foreseeable maintenance of valves that they had sold to Duke Power?
>
> THE WITNESS: What could have been shared by the companies supplying asbestos containing materials was that their product contained asbestos and that asbestoswas known to be hazardous and that the people working with their product

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> should be cautioned about it aboutusing the product about liberating dust and if they were going to do that to do it in a manner that would reduce their exposures.

(TT 7/26 at 108-09, objections omitted).

In 1972, OSHA promulgated asbestos regulations, some of which applied to product manufacturers. (TT 7/26 at 81, 110-11; **Ex. I**, Pl's Ex. 155). As of that time, product manufacturers were required to place warning labels on any asbestos-containing products. (TT 7/26 at 81-82, 110-11). The OSHA asbestos regulations applied to Fisher and Crosby as sellers of asbestos-containing products. (TT 7/26 at 83).

The only exemption from OSHA's warning label requirement was if the manufacturer could demonstrate that the asbestos in the product was encapsulated and would not be released during the normal use of the product. (TT 7/26 at 111). In order to determine if a product releases asbestos dust during normal use, there must be tests conducted under normal working conditions to measure any fiber release from the product. (TT 7/26 at 112).

Fisher and Crosby claim that the asbestos fibers in gaskets and packing was not friable, but they have no way of knowing that because they never measured the fiber release levels from the removal of asbestos gaskets and packing. Fisher never conducted a single field test to determine whether maintenance on Fisher valves released asbestos fibers into the air. (TT 7/27 at 146-47).[5] Neither did Crosby. (TT 7/31 at 13, 33-34, 40-41, 90). Crosby's representative went so far as to maintain that he was "not aware of any requirement to do that." (TT 7/31 at 13).[6] In the absence

[5] Fisher waited until the mid-1980s to conduct testing in its own facilities to determine the asbestos exposure levels experienced by its own employees. (TT 7/27 at 147-48, 153; **Ex. J**, Pl's Ex. 1725, Interrogatory 21). It claims to have waited until that time "[b]ecause of the fact that there was no known safety hazard in that respect." (TT 7/27 at 147-48).

[6] Crosby also failed to test the gaskets and packing it was selling to determine the type of asbestos fiber used or the percentage of asbestos contained in the products. (TT 7/31 at 31-34).

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of fiber release testing, a manufacturer cannot honestly say that its product does not release asbestos fibers. (TT 7/26 at 112).

Dr. Frank explained that asbestos fibers did not remain encapsulated during the ordinary wear and tear on the gaskets:

> Q. What is your opinion, Dr. Frank, as to whether or not asbestos containing gaskets are encapsulated product?
>
> A. Encapsulated means that the asbestos is bound up with other materials. A new gasket when you put it in is an encapsulate material. There are varying amounts of asbestos and they are held together with other materials, rubberized materials, whatever. So they go in as something that's encapsulated. But during the normal use of that product they deteriorate. Most asbestos products will deteriorate. Your car brake for example if it had asbestos in it you know isn't giving up a lot of fibers as you're working with it though it does have some. But as the dust comes off the brake more and more fibers are being liberated an[d] there's what happens with these gaskets they don't stay [in] their original state and so the removal and then using [hand tools] and electric equipment to [remove them] certainly will create dust liberating individual fibers that are no longer encapsulated.

(TT 7/26 at 109-10).

Replacement asbestos gaskets sold by Fisher and Crosby should have included an OSHA-approved asbestos warning label. (TT 7/26 at 189). In addition to a warning label, Fisher and Crosby could have provided material safety data sheets ("MSD sheet") with information about the product composition, hazards, and recommended precautions. (TT 7/26 at 188-89). That was not an OSHA requirement until 1986, but would have been a good prudent business practice. (TT 7/26 at 194). If Fisher and Crosby had provided MSD sheets to Duke, that would have corrected Duke's misconception, as stated in its asbestos policy, that gaskets do not release asbestos fibers. (TT 7/26 at 191-92).

Even though Fisher was supposed to behave with the knowledge of an expert, Fisher did not even think about discontinuing its sales of asbestos-containing gaskets and packing until its

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customers demanded it. (**Ex. K**, Pl.'s Ex. 1008.01).

Finally, Defendants are mistaken in their argument that Jolly's exposure to asbestos from their valves did not exceed OSHA permissible exposure limits (PEL). The evidence in this case was that Jolly was exposed to visible dust from Defendants' gaskets and packing. Dr. Frank testified that "if you see dust from an asbestos product you're dealing with levels that are generally quite high." (TT 7/26 at 113). In fact, Jolly's exposures to asbestos from gaskets were many orders of magnitude above background levels. (TT 7/26 at 76-77).

The evidence therefore establishes the standard of care and that Fisher and Crosby failed to meet it. Even though they were charged with the knowledge of experts, and were supposed to keep up with scientific developments with regard to the products they were selling, they made no effort to do so. Even though they were required under OSHA to determine whether their products released asbestos fibers, they made no effort to do so. Even though they were required by OSHA to include a warning label on their asbestos products, they completely failed to do so. Had Fisher and Crosby actually tested their products, they would have learned that the gaskets and packing they were selling were friable, released high levels of asbestos, and should have included a caution label. The evidence was thus sufficient for the jury to conclude that Fisher and Crosby violated the standard of care.

IV. Fisher and Crosby bear liability for their asbestos-containing valves.

A. Fisher and Crosby sold asbestos components to Duke facilities.

Fisher and Crosby contend that they are entitled to summary judgment because they did not manufacture or provide the asbestos materials to which Jolly was exposed. This assertion is completely contradicted by the evidence that they were selling asbestos gaskets and packing to

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Duke with some of their valves, and that they also supplied replacement internal gaskets, flange gaskets, and packing.

Under South Carolina law, "[a] manufacturer or assembler who incorporates a defective component part into its finished product and places the finished product into the stream of commerce is liable for injuries caused by a defect in the component part." *Baughman v. General Motors Corp.*, 780 F.2d 1131, 1132 (4th Cir. 1986). In fact, "[a] manufacturer who incorporates into his product a component made by another has a responsibility to test and inspect such component, and his negligent failure to properly perform such duty renders him liable for injuries proximately caused as a consequence." *Duncan*, 385 S.C. at 133, 682 S.E.2d at 884. "The fact that the manufacturer or assembler did not actually manufacture the component part is irrelevant, as it has a duty to test and inspect the component before incorporating it into its product." *Baughman*, 780 F.2d at 1132.

1. Crosby Sales

Crosby valves have incorporated asbestos components since the 1930s. (TT 7/31 at 26). Internal asbestos gaskets were installed by Crosby at the factory. (TT 7/31 at 22). Some Crosby valves contained asbestos components until 1991, but thereafter asbestos replacement parts may still have been available. (TT 7/31 at 39). Crosby provided asbestos replacement gaskets. (TT 7/31 at 41).

Crosby sold a lot of valves to the Duke plants where Jolly worked—66 to Oconee, 72 to McGuire, and 18 to Catawba. (TT 7/31 at 143-150; Pl's Ex. 6395). Some Crosby valves sold to Duke contained asbestos gaskets. (TT 7/31 at 157). Crosby sold asbestos-containing replacement gaskets and packing to Duke. (TT 7/31 at 151, 155; **Ex. L**, Pl's Ex. 6364.04). The material specification for the gasket part card relevant to Duke sales showed that an asbestos gasket made

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by Garlock was supplied by Crosby. (TT 7/31 at 74-77; **Ex. M**, Pl's Ex. 6395, p. 39). This asbestos gasket was used in valve number 67261 at Duke. (TT 7/31 at 78). This gasket specification was for an asbestos-containing gasket until 1989. (TT 7/31 at 79). Other records show that asbestos gaskets were specified by Crosby until 1989. (TT 7/31 at 167).

David Taylor testified that the replacement asbestos flange gaskets came from the valve manufacturers. (TT 7/26 at 214-15; TT 7/27 at 104-05).

2. Fisher Sales

Depending on Fisher's design, its valves came with a number of internal gaskets, including bonnet gaskets, cage gaskets, and seat ring gaskets, as well as packing. (TT 7/27 189-91). The gasket and packing material was selected according to Fisher specifications and installed at its manufacturing facility in Iowa. (TT 7/27 at 190, 208).

Fisher sold valves to Duke that contained asbestos gaskets and packing. (TT 7/27 at 158). Fisher material specification 17A2 was for asbestos gasket material composed of 80 to 85% asbestos fiber. (TT 7/27 at 166-67). Fisher used a material specification number for its asbestos gaskets so that its customers could order replacement gaskets that were the exact same asbestos gaskets that had been supplied with the valve. (TT 7/27 at 167, 169). Every time the 17A2 specification was used in invoices and purchase orders through 1988, the gaskets would have contained 85% asbestos. (TT 7/27 at 168). Fisher also had a specification number its customers could use to order asbestos packing from Fisher. (TT 7/27 at 177, 179, 192; **Ex. N**, Pl's Ex. 1008.1964; **Ex. O**, Pl's Ex. 948 at 27-28).

Purchase orders confirm the presence of Fisher valves at Duke and the sale of replacement asbestos gaskets, including asbestos flange gaskets, to Duke. (Pl's Ex. 1012). Duke purchase orders indicate that Fisher was selling replacement asbestos gaskets, specification 17A2, to Duke

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facilities. (TT 7/27 at 168-69; Pl's Ex. 1008.1964). In 1982, Fisher sold replacement asbestos gaskets, specification 17A2, to the Oconee nuclear station. (TT 7/27 at 169; **Ex. P**, Pl's Ex. 1012.5).

In 1982, Fisher sold several different asbestos gaskets to Oconee, including bonnet gaskets, gage gaskets, adaptor gaskets, and seat ring gaskets for Fisher valves. (Pl's Ex. 1012.5). Fisher sold more asbestos gaskets to Oconee in 1985. (**Ex. Q**, Pl's Ex. 1012.7).

David Taylor testified that the replacement asbestos flange gaskets came from the valve manufacturers. (TT 7/26 at 214-15; TT 7/27 at 104-05). Purchase orders confirm the sale of flange gaskets from Fisher. Fisher sold asbestos gaskets for a "flanged fitting" on the "Fisher Vee-Ball Valve U Type 10 inch." (Pl's Ex. 1012.6; TT 7/26 at 215-16).

Given the extensive sales of asbestos gaskets and packing from both Crosby and Fisher to Duke, the jury could infer that Jolly was exposed to asbestos materials supplied directly by Defendants. The jury could have easily concluded that Jolly was exposed to asbestos gaskets supplied by Fisher and Crosby with their valves and as replacement parts.

B. Fisher and Crosby are liable for all foreseeable harm caused by their valves.

Fisher and Crosby once again raise the "bare metal" defense that has already been rejected by this Court. A manufacturer's duty to warn extends to foreseeable dangers resulting from the anticipated and intended use of its products. *See* Ralph King Anderson, Jr., South Carolina Requests to Charge - Civil § 32-27 (2d ed. 2009); *Spruill v. Boyle-Midway, Inc.*, 308 F.2d 79, 84 (4th Cir. 1962); *Claytor v. General Motors Corp.*, 277 S.C. 259, 286 S.E.2d 129 (1982); *Carolina Home Builders, Inc. v. Armstrong Furnace Co.*, 259 S.C. 346, 359-60, 191 S.E.2d 774, 870 (S.C. 1972); *Mickle v. Blackmon*, 252 S.C. 202, 233, 166 S.E.2d 173, 187 (S.C. 1969). A manufacturer is "'expected to anticipate the environment which is normal for the use of his product and . . . must

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anticipate the reasonably foreseeable risks of the use of his product in such environment.'" *Mickle*, 252 S.C. at 233 (quoting *Spruill*, 308 F.2d at 83-84). "Liability may be imposed on a manufacturer or seller notwithstanding subsequent alteration of the product when the alteration could have been anticipated by the manufacturer or seller." *Small v. Pioneer Machinery, Inc.*, 329 S.C. 448, 466, 494 S.E.2d 835, 844 (Ct. App. 1997).

This Court's decision in *Garvin* definitely resolved the liability of asbestos equipment manufacturers for the reasonably foreseeable use of replacement asbestos gaskets and packing. When a "manufacturer recommends specifies, or requires that asbestos gaskets and packing be replaced with like materials," liability may be imposed for replacement gaskets and packing because the product remains in essentially the same condition as when it was sold. *Garvin v. Agco Corp.*, No. 2012-CP-40-6675, at 18 (S.C. Ct. Comm. Pleas Nov. 14, 2014).[7] This Court reasoned that, "Crane's liability arises because its own valves caused injury as manufactured, supplied, and designed by Crane with asbestos gaskets and packing. When the asbestos gaskets and packing were replaced with the same materials that came with the valve, it was not a 'substantial change' in the condition of the product." *Id.* at 16-17.

The Court rejected the "bare metal defense" on the basis that Crane Co. valves were not sold as mere bare metal, but with asbestos materials that needed to be replaced. When asbestos replacement parts are recommended, specified, or required, the manufacturer cannot reasonably claim that its product somehow ceases to be harmful when such replacements are used. The Court reasoned that, "[t]o say Crane was no longer part of the 'chain of distribution' when the original gaskets and packing wore out on its still-functioning product would be artificial, if not silly." *Id.* at 13-14. The evidence showed that the asbestos gaskets and packing used as replacements were

[7] This Order is attached to Plaintiffs' Motion for New Trial *Nisi Additur* as Exhibit 7.

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no different than that originally supplied by Crane Co., that Crane Co. knew those parts would have to be replaced, and knew that exposure to asbestos was hazardous. *Id.* at 13. The Court determined that Crane Co. "occupied the best position in the chain of distribution to warn consumers of those risks." *Id.* This conclusion is supported by the comments to the Restatement of Torts (Second) § 402A, incorporated by reference by S.C. Code Ann. § 15-73-30, which espouses the policy that "'the consumer of such products is entitled to the maximum of protection . . . and the proper persons to afford it are those who market the products.'" *See id.* at 14 (quoting Comment c to the Restatement of Torts (Second) § 402A).

Here, the evidence similarly shows that Fisher and Crosby sold their valves with asbestos components, knew that the gaskets and packing would have to be changed according to the valve design, and intended that asbestos replacements be used. Fisher engineers selected the components and built the valves for the particular application requested. (TT 7/27 at 197). Similarly, the internal components of Crosby valves were installed by Crosby pursuant to the valve design of Crosby engineers. (TT 7/31 at 22).

As set forth above, both Fisher and Crosby had material specifications that called for asbestos gaskets and/or packing to be used. (TT 7/27 at 166-69; Pl's Ex. 1008.1964; TT 7/28 at 84-86; TT 7/31 at 74-79, 167; Pl's Ex. 6395, p. 39). Jolly recalled that the manuals for Fisher and Crosby valves specified the type of gaskets that must be used. (TT 7/25 at 78).

Crosby, in fact, recommended that asbestos gaskets be used as replacement parts:

> Q. Okay. So if you looked at this, let's say Crosby Valve model JA-6, per the Crosby design it would be your expectation if you found it in the field that the original design would have called for an asbestos gasket somewhere in the body of that valve?
>
> A. Correct.

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Q. And if the valve had to be maintained while it was in service, then and if they followed the instructions of Crosby in the instruction manual, the replacement parts for that valve would -- for that particular part wherever it exists in the belly would also be an asbestos containing –

A. In that time frame yes.

(TT 7/31 at 30-31).

Fisher could decide whether or not to utilize asbestos-containing materials for the parts it supplied with its valves and as replacement parts. (TT 7/27 at 225). In 1986, for example, it changed the material composition of its packing to substitute non-asbestos materials. (TT 7/27 at 226-27). After that substitution, Duke would order the same Fisher part number and receive a part with a different material. (TT 7/27 at 227-28).

In addition to specifying asbestos gaskets, Fisher recommended that the gasket should be replaced whenever the valve is opened:

Q. Fisher recommends that any time a valve is disassembled for any reason, that the gasket should be changed, even if there's no repair right?

A. If you disassembly a valve, take the bonnet in the case of a sliding stem valve, take the bonnet apart, then yeah you need to inspect that gasket and the gasket surface and most likely to have replace that gasket because it's already been sealed once and created that seal. So yeah in those occasions where you have to disassembly for some maintenance reason I would guess yes.

Q. Well, if it's disassembled merely for inspection not necessarily for maintenance it needs to be re[p]laced isn't that true?

A. I would say most of the time yes but I'm not going to say that would be the practice in every case because I don't have that experience.

Q. Okay. I'm going to ask you to turn over to page 40 of the student guide. There is a section entitled assembly considerations special procedures reuse of parts gasket considerations. Do you see that?

A. Yes.

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Q. And under this reuse of parts here Fisher says generally speaking all soft parts should be replaced whenever the control valve has been disassembled. Packing seals piston rings and gaskets are very inexpensive compared to the cost of bringing a process down and reworking a repair. First of all did I read that correctly?

A. Yes. And it lines up perfectly with what my prior answer was.

Q. And so it is Fisher's recommendation that whenever a valve is disassembled, the gaskets and the packing should be replaced?

A. No. It says generally speaking. Just like I said earlier.

(TT 7/28 at 38-39). Crosby was similarly aware that the internal gaskets in its valves would have to be removed and replaced during routine maintenance of its valves. (TT 7/31 at 41).

Defendants also designed their valves with flanged connections, as set forth above. (TT 7/27 at 187-89; 7/31 at 18, 122-23, 125). A lot of the valves Fisher and Crosby sold to Duke had flanged connections. (TT 7/31 at 164-65). Because of that design, flange gaskets had to be used to connect their flanged valves to the pipeline. (TT 7/27 at 187-89; *see also* TT 7/31 at 18-19, 123). Fisher and Crosby knew that asbestos flange gaskets would sometimes be used. (TT 7/31 at 122-23). The Fisher and Crosby valves at Duke did, in fact, require asbestos flange gaskets. (TT 7/26 at 210-13).

This evidence that Fisher and Crosby could anticipate that asbestos components would be used in their valves, and that asbestos flange gaskets would be used with their flanged valves, raised questions of fact for the jury regarding Defendants' breach of their duty to warn of the foreseeable dangers resulting from the design of Defendants' valves. When Jolly or his co-workers used replacement asbestos gaskets and packing in Fisher and Crosby valves, the valves remained unchanged from Defendants' original design. The evidence was therefore sufficient for the jury to conclude that Fisher and Crosby were negligent in failing to warn Jolly of the asbestos hazards associated with their valves.

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V. Jolly's mesothelioma was caused by his exposure to asbestos from Defendants' valves.

A. Plaintiffs' experts' testimony was relevant and reliable.

Fisher and Crosby contend, as they did before trial, that the causation testimony of Plaintiffs' medical experts, Dr. Arthur Frank and Dr. John Maddox, is unreliable and insufficient to establish substantial factor causation. In the interest of brevity, Plaintiffs adopt and incorporate by reference their Consolidated Response to Defendants' Motion in Limine to Exclude the Opinion that "Every Exposure" Causes Disease ("Consolidated Response"), and the exhibits attached thereto.

Defendants once again ask this Court to preclude testimony from Plaintiffs' causation experts that "each and every exposure" caused Jolly's mesothelioma. Contrary to Defendants' mischaracterization, Plaintiffs' experts do not hold the opinion that "any exposure" or "every exposure" or "every asbestos fiber" causes mesothelioma generally or caused Jolly's mesothelioma. As set forth below, they instead testified that Jolly's mesothelioma was caused by his cumulative asbestos exposure, and that given the specific facts of his exposure to visible asbestos dust from Fisher and Crosby valves regularly for four years, these exposures were a substantial factor in causing Jolly's disease. Dr. Frank's and Dr. Maddox's

1. Standard of Review

The admissibility of expert testimony is governed by South Carolina Rule of Evidence 702. That rule provides that "[i]f scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise." S.C. R. Evid. 702.

Courts evaluating the admissibility of scientific expert evidence "must find the evidence will assist the trier of fact, the expert witness is qualified, and the underlying science is reliable." *State v. Council*, 335 S.C. 1, 20, 515 S.E.2d 508 (S.C. 1999). The reliability of scientific evidence is evaluated based on several factors, including: (1) the publications and peer review of the technique; (2) prior application of the method to the type of evidence involved in the case; (3) the quality control procedures used to ensure reliability; and (4) the consistency of the method with recognized scientific laws and procedures. *State v. White*, 382 S.C. 265, 274, 676 S.E.2d 684 (S.C. 2009) (citing *Council*, 335 S.C. at 19).

"[C]ross-examination, presentation of contrary evidence, and careful instruction on the burden of proof" are the ordinary means to attack an opposing expert. *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 596 (1993). Courts should not exercise their gatekeeping responsibility by excluding expert testimony that falls within the range of matters on which reasonable experts can disagree. *See Milward v. Acuity Specialty Prods. Group, Inc.*, 639 F.3d 11, 22 (1st Cir. 2011). While the trial court may look at the reliability of the expert's methodology, it is for the jury to determine the soundness of the facts underlying the expert's opinion and the correctness of the expert's conclusions. *See id.* 22.

2. Dr. Frank and Dr. Maddox have a reliable scientific basis for their opinions.

Contrary to Defendants' assertions, Plaintiffs' experts' opinions regarding the cumulative nature of asbestos diseases and the lack of any safe threshold of exposure are not "unreliable," but are generally accepted medical facts. The scientific evidence establishing these basic medical facts is set forth in Plaintiffs' Consolidated Response. In addition, at trial, both Dr. Frank and Dr. Maddox testified that mesothelioma can be caused by brief or low level cumulative exposures. (TT 7/26 at 41-43, 52-53; TT 7/31 at 179-80, 184-85).

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Plaintiffs' experts were entitled to rely on these basic medical facts in reaching their opinion in this case. That does not mean that they concluded that "each and every exposure" that Jolly had was a *substantial factor* in causing his disease. This distinction was recently explained by the Pennsylvania Supreme Court in an opinion upholding the admissibility of Dr. Frank's causation opinions. *See Rost v. Ford Motor Co.*, 151 A.3d 1032, 1045-46 (Pa. 2016). The court found his testimony to be entirely compatible with the substantial factor causation standard:

> We must agree with the Rosts that Ford has confused or conflated the "irrefutable scientific fact" that every exposure cumulatively contributes to the total dose (which in turn increases the likelihood of disease), with the legal question under Pennsylvania law as to whether particular exposures to asbestos are "substantial factors" in causing the disease. It was certainly not this Court's intention, in either Gregg or Betz, to preclude expert witnesses from informing juries about certain fundamental scientific facts necessary to a clear understanding of the causation process for mesothelioma, even if those facts do not themselves establish legal (substantial factor) causation.

Rost v. Ford Motor Co., 151 A.3d 1032, 1045-46 (Pa. 2016).

The Eleventh Circuit has recently held the same. In *Bobo v. Tennessee Valley Auth.*, 855 F.3d 1294 (11th Cir. Apr. 26, 2017), the court noted that expert testimony that "there is no evidence that there is a threshold level of exposure below which there is zero risk of mesothelioma," and that "all 'significant' exposures to asbestos 'contribute to cause mesothelioma,'" is not the same thing as saying that each and every exposure is causative. *Id.* The expert's causation opinion was admissible because it was based on the exposure facts in the case and was supported by scientific literature, including the Helsinki Criteria. *Id.*

Judge Robreno of the federal asbestos MDL has similarly found that a "cumulative exposure" opinion is not the same as the "each and every exposure" opinion. The court agreed with the plaintiff that the "cumulative exposure" opinion is different "in substance and by definition" from the "any exposure" opinion. *Mortimer v. A.O. Smith Corp.*, 2015 WL 12533103,

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at *8 (E.D. Pa. Oct. 23, 2015). The court therefore rejected the defendant's argument that the experts' opinions were inadmissible on grounds that they are the same as the "any exposure" opinion. *Id.*

As recognized by these other distinguished courts, which have all admitted the causation testimony of Dr. Frank and/or Dr. Maddox, it is not proper to evaluate the experts' medical opinions with reference to only one narrow part of the basis for the opinion. Here, in reaching their opinions Dr. Frank and Dr. Maddox both relied on their many years of experience in the area of asbestos-related diseases, as well as a broad range of evidence including epidemiology and other scientific literature, the dose-response relationship, the science regarding the low levels of exposure that can cause mesothelioma, the exposure levels documented from working with gaskets and packing in the manner described by Jolly and Taylor, and the facts surrounding Jolly's exposure to visible dust from Defendants' valves. (TT 7/26 at 19-95; 7/31 at 169-77, 179-80, 184-85). Defendants' attack on their opinions as "unreliable" has absolutely no merit.

3. Dr. Frank and Dr. Maddox based their specific causation opinions on the exposure evidence in this case.

The specific causation opinions of Drs. Frank and Maddox were firmly grounded in the exposure evidence presented at trial. First, Dr. Frank testified that he had reviewed Jolly's medical records, the deposition transcripts of Jolly and his co-workers and other case-specific materials:

Q. You have reviewed some materials specifically for the Beverly Dale Jolly case correct?

A. Yes. That's the file I have here.

Q. Can you tell the jury what you have looked at?

A. Well I have looked at some information about his exposures, I've looked at medical records, I've looked at deposition testimony of Mr. Jolly and some others that have been involved in this case, I've look at some legal filings and documents as well.

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* * *

Q. And Mr. Jolly's particular matter here can you tell the jury what testimony you have had a chance to review?

A. I've read several of his own transcripts where he described what it was like at workplaces and that he was standing there watching as this work was being done.

(TT 7/26 at 58-60).

He also described that, based on Jolly's testimony, the exposures he had from working with Fisher and Crosby valves were high:

Q. Let's take asbestos gaskets used on flanges as it relates to valves. Have those type of gaskets as a product been studied and data published about the levels and amount of asbestos that is emitted when they are removed using hand tools and power tools?

A. Yes.

Q. And have you reviewed that body of literature as well?

A. I have.

Q. Okay.

A. Clearly it's above zero.

Q. Let me ask you. Is it a little exposure or a significant substantial amount when an asbestos containing gasket that's been in place is removed using a hand wire brush like you would use on a barbecue grill or even an electric powered grinder to clean a surface?

A. You can get significant levels when you use those kinds of tools creating individual fibers in that material. It may have start out as a solid material but individual fibers are released.

(TT 7/26 at 51).

Jolly's exposures were many orders of magnitude above background levels. (TT 7/26 at 76-77). The level of asbestos in the ambient air in urban areas is .00001 f/cc, and in rural areas it's

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.000001 f/cc. (TT 7/26 at 76). Dr. Frank explained that Jolly's exposures were millions of times higher than that:

> Q. Okay, so now that we have an idea of what background or ambient levels are, how does the exposure levels to asbestos that you have opined in your expert view for Mr. Jolly from the removal of asbestos containing gaskets as we've been discussing it compare?
>
> A. Workplace levels even as a bystander that Mr. Jolly would have had would have been many many orders of magnitude greater than that. We're probably dealing with single or even double digits for short periods of time compared to all those zeros you will you get to a one.
>
> Q. So we have a court report are the so we want to be clear. What does single or double digits?
>
> A. Single would be one to nine double ten and above triple a hundred and [above but] we're not dealing with those kind of levels here [I] wouldn't [ex]pect. But clearly with at least single digits for the time that gaskets is being ground out that contains a considerable amount of asbestos.
>
> Q. So my math is not too good which is why I'm a lawyer. It looks like to me though one fiber per cc compared to .000001 fiber per cc is millions of times higher than exposures we have as background?
>
> A. Five orders of magnitude or six orders of [magnitude], that's millions of times. Hundreds of millions.

(TT 7/26 at 76-77). Dr. Frank also took into account the fact that asbestos gaskets contain up to 90% asbestos. (TT 7/26 at 51-52).

In stating his causation opinion, Dr. Frank relied on a summary of the exposure facts proven to the jury:

> Q. Okay. Dr. Frank, let me ask you sort of another hypothetical which is that Mr. Jolly worked as a mechanical inspector from 1980 [to] 1984, th[en] ask you to assume that it was part of his frequent and normal job responsibilities to be on outages where on a consistent and regular bas[i]s gaskets would be removed from the flange face of valves using wire brushing tools and scrapers and that Mr. Jolly's proximity would be ten feet or less from that work that he could see the dust that he breathed the dust and that this was part of the regular and consistent work that he did during this 80 to 84 time frame. Do you have that in mind?

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A. Yes.

Q. What is your expert opinion with a reasonable degree of medical certainty as to whether or not that exposure would be a substantial contributing factor to the development of his mesothelioma?

THE WITNESS: My opinion would be that those exposures were a significant contributing cause to his mesothelioma.

(TT 7/26 at 72-73).

Dr. Frank clarified that Jolly's exposures were quite extensive, and not in the category of an isolated or short-term exposure:

Q. What is your opinion as to whether or not the sort of one day, one gasket circumstance that we were hypothetically discussing, is that after you have reviewed the materials what Mr. Jolly had?

A. From reading the materials, my understanding is in that four-year period we are not dealing with one day of exposure. We are dealing with man who had a particular job title that regularly but him in situations where gasket after gasket was something that he reviewed being taken out and new ones being put in.

(TT 7/26 at 71).

Dr. Maddox similarly based his specific causation opinions on a summary of the exposure facts in this case:

Q. Okay Dr. Maddox. Based on your knowledge and experience and the factors you use to evaluate causation, I'd like to give you some hypotheticals. Would that be okay?

A. Yes, ma'am.

Q. I want to you assume the following facts first about duration, that the exposure to Mr. Jolly was the over three to 4 years in the early 1980s okay?

A. Yes, ma'am.

Q. Assume that the exposures came from asbestos containing gaskets and packing used in some but not all of the valves at the power plant during

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outages and shutdowns at several plants which happen as a regular part of his job. Are you with me?

A. Yes, ma'am.

Q. All right. Assume that the exposure were not direct[] but as an inspect[or] he was close enough to see the dust often working one to 2 feet from where the work was done?

A. Yes, ma'am.

Q. Okay. And assume that this is an exposure that resulted in visible dust from brushing grinders where the jury has heard that the exposures were in the hundreds or thousands to hundred of thousand to millions of times higher than background okay?

A. Yes, ma'am.

Q. And we spoke to an occupational medicine already but I want to focus on epidemiology more than industrial hygiene. Given those assumptions that I've given you is this exposure as compared to low dose exposures in epidemiology significant and substantial?

A. Yes, ma'am. I believe that would constitute a significant exposure to asbestos because they were real exposures that were witnessed, they were high enough to provide visible dust, they were repetitive and they were within a reasonable latency period.

Q. If this person came in with mesothelioma and this was the only exposure as I've described to you, would it by itself be the cause of the disease?

A. I believe that if the mesothelioma, if that was the only known exposure to asbestos then the mesothelioma, then that would be considered a causation of the malignant mesothelioma. That does not exclude any other possible recognized or unrecognized causes but I believe that the exposure would be sufficient to deem that causative.

Q. Okay Dr. Maddox now I want to you assume that Mr. Jolly had multiple exposure like that like I've described to you from Fisher valves and Crosby valves in addition to several other companies' equipment, will you assume that?

A Yes, ma'am.

Q. In a situation where the exposure to Fisher valves as I've described above is one of several exposures, can you tell me if that exposure, given the facts

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about dose, frequency, proximity and frequency was a substantial contributing cause in Mr. Jolly's development of mesothelioma?

A. Yes, ma'am. I believe that that the exposures as described would be significant contributor to the diagnosis and development of malignant mesothelioma.

Q. And would that also be true of the valve at issue came from Crosby?

A. Yes.

(TT 7/31 at 180-82).

Dr. Maddox also addressed the issue of very low exposures and explained that low exposures carry only a low risk of disease:

Q. The jury has heard some testimony that there is in the literature and I believe you have expressed it in the past, that exposure of even one day to asbestos could be significant enough to cause mesothelioma correct?

A. Well there have been a few case reports, reports and individual cases that are in the medical literature where extremely short exposures of a day or a week or a month have been the only known exposures in some malignant mesotheliomas however would I strongly state at that there is a dose response approximate the people with larger the cumulative exposures have a higher rate of mesothelioma and the people with very smart exposures have a very low rate of malignant mesothelioma.

(TT 7/31 at 194-95).

The trial testimony of Dr. Frank and Dr. Maddox thus demonstrates that their causation opinions are based on the record of Jolly's repeated exposures to asbestos from Fisher and Crosby valves during four years at Duke, as well as the scientific literature and other evidence mentioned above. In this way, the methodology and approach of Dr. Frank and Dr. Maddox is exactly the same as that approved by Judge Hill in *Garvin v. Agco Corp.*. There, Judge Hill rejected a challenge to the admissibility of Dr. Maddox's causation testimony. No. 2012-CP-40-6675 (S.C. Ct. Comm. Pleas Nov. 14, 2014). The Court noted that Dr. Maddox's "specific causation opinions are based on fact witness testimony regarding the frequency, duration, and proximity of Mr.

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Garvin's exposure, Mr. Garvin's medical and pathology materials, Crane-specific documents, studies of the fiber release from asbestos gaskets and packing, and the peer-reviewed medical literature regarding the causation of mesothelioma." *Id.* at 22. Judge Hill concluded that Dr. Maddox's testimony was reliable and helpful to the trier of fact. *Id.*

Plaintiffs urge this Court to similarly hold that the causation testimony of Dr. Frank and Dr. Maddox is supported by the scientific literature as well as the facts of this case, and that their testimony was relevant and helpful to the jury.

B. Jolly's exposure was more than sufficient to satisfy the *Henderson* causation standard.

The *Henderson* exposure standard is met here. A plaintiff in an asbestos case may defeat summary judgment with evidence of "actionable exposure" to a defendant's asbestos product. *Henderson v. Allied Signal, Inc.,* 373 S.C. 179, 185, 644 S.E.2d 724, 727 (2007). To determine whether exposure is actionable, South Carolina courts apply the "frequency, regularity and proximity" factors set forth in *Lohrmann v. Pittsburgh Corning Corp.*, 782 F.2d 1156 (4th Cir. 1986). *Id.* Therefore, "'[t]o support a reasonable inference of substantial causation from circumstantial evidence, there must be evidence of exposure to a specific product on a regular basis over some extended period of time in proximity to where the plaintiff actually worked.'" *Id.* (quoting *Lohrmann*, 782 F.2d at 1162).

The evidence showed that when Jolly was a mechanical inspector at Duke from 1980 to 1984, he was repeatedly exposed to asbestos gaskets and packing from Fisher and Crosby valves. (TT 7/25 at 61-63, 72-76, 84-94). During that time period, Jolly worked at three of Duke's nuclear power plants: Oconee, McGuire, and Catawba. (TT 7/25 at 66-67). Jolly's job put him alongside mechanics that were changing asbestos gaskets and/or asbestos packing on valves, particularly during outages at the plant, and he recalled that the use of grinders and brushes created dust. (TT

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7/25 at 74-76, 84-94). He recalled that there were a lot of Fisher and Crosby valves present at those plants. (TT 7/25 at 72-73, 90-91). At time, engineers from Fisher or Crosby were present to assist with the maintenance of the valves. (TT 7/25 at 107).

Exposure testimony was also provided by Jolly's co-worker, David Taylor, who worked at the same Duke nuclear plants where Jolly was stationed, particularly Oconee. (TT 7/25 at 198-201). As a pipefitter, it was Taylor's job to connect and disconnect valves and pumps to and from pipe systems. ((TT 7/25 at 201). He explained that he bolted valves to pipe flanges, and that there was a gasket between the pipe and the valve to seal the connection. (TT 7/25 at 201). He had to remove valve gaskets about once a week. (TT 7/25 at 204). He removed asbestos gaskets from valve flanges with a scraper or wire brush, which created a lot of dust. (TT 7/25 at 202, 204-05). Jolly was nearby when Taylor removed asbestos flange gaskets. (TT 7/25 at 220). Other workers removed the internal gaskets and packing from valves in a similar manner that created dust. (TT 7/25 at 207-09).

Taylor recalled both Fisher and Crosby valves at Duke. (TT 7/25 at 210). The Fisher and Crosby valves had flanged connections that utilized asbestos gaskets. (TT 7/25 at 210). Fisher and Crosby valves also had internal asbestos gaskets and packing. (TT 7/25 at 210-13). The original and replacement asbestos valve gaskets came from the valve manufacturer. (TT 7/25 at 214-15).

In addition, Plaintiff notes that Crosby admitted at trial that there is a health risk from the removal of asbestos gaskets and packing from its valves. Martin testified as follows:

> Q. Is it Crosby's position today that if a person is exposed to asbestos because of inhaling asbestos that's liberated from an asbestos gasket that came from the flange face of the valve, that -- that expose causes a health risk?
>
> A. That's correct it does.
> Q. It does?
>
> A. Yeah.

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Q. And can we agree that it is Crosby's position or awareness today that if a person is exposed to as[bestos] that comes from the removal of asbestos containing packing used in a valve, that if that exposure occurs that that too poses a health risk?

A. That would seem reasonable yes.

(TT 7/31 at 65-66).

Finally, the causation evidence at trial established that at least some of the gaskets and packing sold by Defendants contained crocidolite, or Blue African asbestos. In 1980, Fisher's own air monitoring tested a Blue crocidolite spiral wound gasket. (**Ex. R**, Pl's Ex. 1008.10670; **Ex. S**, Pl's Ex. 1008.10665). Fisher also had a specification for crocidolite packing. (TT 7/27 at 201-02, 206; **Ex. T**, Pl's Ex. 2006.037). Defense expert Dr. Oury testified that gaskets and packing were known to contain crocidolite. (TT 8/2 at 188-89; **Ex. U**, Crosby Ex. 533).

It is significant that Jolly was exposed to crocidolite asbestos from Defendants' products because crocidolite is known to be by far the most potent type of commercial asbestos fiber. (TT 8/1 at 114;[8] TT 8/2 at 179).[9] Even Defense expert Dr. Crapo admitted that a "very low" level of exposure to crocidolite can cause mesothelioma. (TT 8/1 at 114). Dr. Oury testified that crocidolite asbestos fibers would remain in an exposed person's lungs for the rest of their life. (TT 8/2 at 179).

The jury could thus have concluded not only that Jolly had frequent, regular, and proximate exposure to asbestos gaskets and packing from Fisher and Crosby valves, but that these exposures were to the most potent form of asbestos fiber. This evidence is more than sufficient to support the jury's determination that Jolly's mesothelioma was caused by his exposure to asbestos products sold by Defendants and for which they are responsible.

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[8] This transcript is attached to Plaintiffs' Motion for *Nisi Additur* as Exhibit 4.
[9] This transcript is attached to Plaintiffs' Motion for *Nisi Additur* as Exhibit 5.

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CONCLUSION

For the foregoing reasons, Plaintiffs respectfully ask the Court to deny Defendants Fisher and Crosby's Joint JNOV Motion in all respects.

Respectfully submitted,

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CERTIFICATE OF SERVICE

I hereby certify that a true and correct copy of the above and foregoing instrument was e-filed with the Spartanburg County court's e-filing system, and thereby served electronically on all counsel of record, this 6th day of September 2017.

/s/ Jonathan M. Holder
Jonathan M. Holder
Attorney for Plaintiffs

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STATE OF SOUTH CAROLINA **COUNTY OF SPARTANBURG** **BEVERLY DALE JOLLY and BRENDA RICE JOLLY,** **Plaintiffs,** **v.** **GENERAL ELECTRIC COMPANY, et al.** **Defendants.**	))))))))))))))	**IN THE COURT OF COMMON PLEAS FOR THE SEVENTH JUDICIAL CIRCUIT** **C/A No. 2016-CP-42-1592** **PLAINTIFFS' RESPONSE IN OPPOSITION TO DEFENDANTS FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE, LLC'S JOINT MOTION FOR PRODUCTION OF PLAINTIFFS' SETTLEMENTS AND PAYMENTS WITH ALL THIRD-PARTY TORTFEASORS**

Plaintiffs Beverly Dale Jolly and Brenda Rice Jolly oppose the motion for production of Plaintiffs' settlements and payments with third-party tortfeasors filed by Fisher Controls International LLC and Crosby Valve, LLC ("Defendants") and would respectfully show the Court that this motion should be denied. Plaintiffs alternatively request that this Court enter a protective order to prevent the discovery of these confidential documents.

ARGUMENTS AND AUTHORITIES

I. Defendants have not demonstrated the relevance of the settlement documents beyond the information Plaintiffs have already provided.

Defendants argue that they are entitled to disclosure of Plaintiffs' confidential settlement documents. Defendants claim that the documents are "relevant" to set-off, but have not articulated any reason for compelling disclosure of the documents in this case. The only matter of relevance is the amount of the settlements.[1] This information has been disclosed. There can be no reason for examining the releases with other parties.

[1] Plaintiffs discuss the total settlement amount received in their response to Defendants' Joint Motion for Set-Off pursuant to S.C. Code § 15-38-50.

Defendants make the argument that "full access to the records pertaining to all of the money obtained by Plaintiffs as a result of their alleged injuries" is vital to the determination of the proper amount of setoff. However, the settlement documents, contrary to Defendants' contention, will not aid this endeavor, as settlement documents do not contain any allocation provisions. Further, because the settlement proceeds include funds for claims not tried to verdict, this Court has the discretion to allocate and reallocate the funds. This procedure does not require the disclosure of confidential settlement documents.

II. The Court should enter a protective order to prevent discovery of Plaintiffs' confidential settlement documents.

The Court should enter a protective order providing that Plaintiffs' settlement agreements with other defendants are not discoverable. By their terms, these are confidential documents not permitted to be disclosed to outside parties. In most cases, the agreements state that part of the consideration for the releases is that Plaintiffs agree to treat the terms of the settlements as confidential.

Rule 26(c), SCRCP, provides that a protective order may be issued for good cause shown: "[u]pon motion by a party or by the person from whom discovery is sought, and for good cause shown, the court in which the action is pending . . . may make any order which justice requires to protect a party or person from annoyance, embarrassment, oppression, or undue burden by expense . . . ." Good cause is shown by describing "a particularized harm which will result if the challenged discovery is had." *Hamm v. S.C. Pub. Serv. Comm'n*, 312 S.C. 238, 241, 439 S.E.2d 852, 854 (1994).

Here, the particularized harm is that Plaintiffs will be violating the confidentiality provisions of the releases if they produce these documents to Defendants, and that future settlements, even with these Defendants, will be discouraged. Settlements are, of course, generally

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favored under the law. The South Carolina Supreme Court "encourages such compromise agreements because they avoid costly litigation and delay to an injured party." *Poston v. Barnes*, 294 S.C. 261, 264, 363 S.E.2d 888, 890 (1987); *Rock Smith Chevrolet, Inc. v. Smith*, 309 S.C. 91, 93, 419 S.E.2d 841, 842 (Ct. App. 1992) ("It has long been the policy of the court to encourage settlement in lieu of litigation, and courts have usually enforced settlement agreements.").

The confidential nature of Plaintiffs' releases is an integral part of the inducement—for both sides—to settle the case and avoid further costly litigation. "Parties are typically permitted, and often encouraged, to reach private settlements." *Baker v. Dolgencorp, Inc.*, 818 F. Supp. 2d 940, 942-943 (E.D. Va. 2011). "In many cases, private or confidential settlements provide parties with incentives to reach amicable resolutions, especially where one party fears that publicity of a settlement could potentially encourage additional litigation." *Id.* "An early and amicable settlement is certainly a desirable outcome, and for that reason courts regularly enforce confidentiality provisions in settlement agreements." *Stephens v. County of Albemarle*, 422 F. Supp. 2d 640, 644 (W.D. Va. 2006).

If the confidentiality provisions of settlement agreements are disregarded, parties will be less likely to settle claims in the future or may do so on terms less favorable to plaintiffs. "Defendants in particular are reluctant to disclose the terms of settlement lest those terms encourage others to sue." *Jessup v. Luther*, 277 F.3d 926, 928 (7th Cir. 2002).

Plaintiffs are obligated to protect the confidential nature of their settlement agreements with other defendants. In order to do so, Plaintiffs have disclosed the amount of the settlements in an aggregate manner that maintains the confidentiality of each individual settlement. Defendants have not explained how this disclosure is insufficient, or provided any rationale for disregarding the confidentiality provisions of Plaintiffs' private contracts with other defendants.

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Similarly, Rule 408 of the South Carolina Rules of Evidence does not require Plaintiffs to disclose confidential settlement agreements. Rule 408 reads:

> Evidence of (1) furnishing or offering or promising to furnish, or (2) accepting or offering or promising to accept, a valuable consideration in compromising or attempting to compromise a claim which was disputed as to either validity or amount, is not admissible to prove liability for or invalidity of the claim or its amount. Evidence of conduct or statements made in compromise negotiations is likewise not admissible. This rule does not require the exclusion of any evidence otherwise discoverable merely because it is presented in the course of compromise negotiations. This rule also does not require exclusion when the evidence is offered for another purpose, such as proving bias or prejudice of a witness, negativing a contention of undue delay, or proving an effort to obstruct a criminal investigation or prosecution.

Nothing in Rule 408 supports Defendants' contention that the confidential settlement agreements between Plaintiffs and other defendants are discoverable. The rule is clear that it does not convert discoverable information into undiscoverable information. However, Defendants cannot demonstrate that the confidential settlement agreements reflecting settlements between Plaintiffs and other defendants are discoverable. Consequently, Defendants' request for this information should be denied.

Plaintiffs' settlement agreements should be protected from disclosure. In the alternative, if the Court believes that examination of the releases is warranted, Plaintiffs request that this be conducted by the Court through in camera inspection in lieu of disclosure to Defendants should the Court need to review the agreements to ensure that the releases were given in good faith.

Finally, the aggregate amount provided to the Defendants includes the amounts paid by bankruptcy trusts, if any, in this matter to date. Defendants, without any authority, appear to seek to include amounts which "might" be paid at some future date by a bankruptcy trust. There is no South Carolina authority to support that position and, to the extent that this is what the Defendants seek, Plaintiffs respectfully suggest that that relief should also be denied.

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Therefore, for the reasons set forth herein, Plaintiffs respectfully request that Defendants' motion for production of Plaintiffs' confidential settlement documents be denied.

CONCLUSION

Accordingly, for the reasons set forth herein, Plaintiffs request that Defendants' motion for production of Plaintiffs' settlement agreements with other defendants be denied and the Court to enter a protective order preventing the discovery of these confidential documents.

Respectfully submitted,

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STATE OF SOUTH CAROLINA **COUNTY OF SPARTANBURG** **BEVERLY DALE JOLLY and BRENDA RICE JOLLY,** **Plaintiffs,** **v.** **GENERAL ELECTRIC COMPANY, et al.** **Defendants.**	)))))))))))))))	**IN THE COURT OF COMMON PLEAS FOR THE SEVENTH JUDICIAL CIRCUIT** **C/A No. 2016-CP-42-1592** **PLAINTIFFS' RESPONSE IN OPPOSITION TO DEFENDANTS FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE, LLC'S JOINT MOTION FOR SET-OFF PURSUANT TO S.C. CODE § 15-38-50**

Plaintiffs Beverly Dale Jolly and Brenda Rice Jolly oppose the motion for set-off pursuant to S.C. Code § 15-38-50 filed by Defendants Fisher Controls International LLC and Crosby Valve, LLC ("Defendants") and would respectfully show the Court that this motion should be denied. Alternatively, Plaintiffs request that the Court hold a ruling on this motion in abeyance until this Court rules on Plaintiffs' Motion for New Trial Nisi Additur and that this Court approve the allocation of settlement funds as made by Plaintiffs.

ARGUMENTS AND AUTHORITIES

I. Defendants' Motion for Set-Off Should be Considered After Plaintiffs' Motion for Additur

Plaintiffs agree, generally, that Defendants are entitled to a set-off in the amount of Plaintiffs' settlements with other defendants in this case. S.C. Code Ann. § 15-38-50 provides that, [w]hen a release . . . is given in good faith to one of two or more persons liable in tort for the same injury or the same wrongful death: (1) . . . it reduces the claim against the others to the extent of any amount stipulated by the release or the covenant, or in the amount of the consideration paid for it, whichever is the greater . . . ." S.C. Code Ann. § 15-38-50.

In this case, Plaintiffs went to trial against two defendants, Fisher and Crosby. The jury, contradictory to undisputed evidence, awarded only $300,000 in compensatory damages to the Jolly. The jury did not consider punitive damages because it determined that Defendants' conduct was not willful, wanton, or reckless. Shortly after the jury rendered its verdict, Plaintiffs filed their motion for *additur*, requesting that the Court, based on the uncontroverted evidence presented during trial, increase the award to at least $2.5 million. [1]

Defendants correctly assert that Plaintiffs have received settlement money from other defendants in this case. In total from all settlements, Plaintiffs received a total of $2,220,000. While Plaintiffs generally agree that Defendants are entitled to a set-off in this matter, the amount of the set-off should not be considered by this Court until after this Court rules on Plaintiffs' motion for *additur*.

II. Defendants are not entitled to setoff as a matter of law and the allocation of the settlement funds will dictate the setoff amount.

Defendants imply that their right to set-off arises as a matter of law and states that the allocation of the settlement funds is irrelevant. Both of these contentions, however, are without merit. In *Ellis v. Oliver*, 335 S.C. 106, 112-13, 515 S.E.2d 268, 271-72 (Ct. App. 1999), the Court of Appeals held that "when a prior settlement involves compensation for the same injury for which the jury awarded damages, the right to setoff arises as an operation of law." *Smith v. Widener*, 397 S.C. 468, 473, 724 S.E.2d 188, 191 (Ct. App. 2012). However, when the settlement involves compensation for an injury different from the one tried to verdict, there is no setoff as a matter of law. *Hawkins v. Pathology Assocs. of Greenville, P.A.*, 330 S.C. 92, 114-15, 498 S.E.2d 395, 407 (Ct. App. 1998). Further, when the settlement "is argued to involve two claims, one of which

[1] Plaintiffs refer the Court to their arguments in favor of *additur* as detailed in Plaintiffs' Motion for New Trial *Nisi Additur*.

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involves the same injury as the claim tried to verdict and one of which does not, the circuit court must make the factual determination of how to allocate the settlement between" the claims. *Smith*, 397 S.C. at 473, 724 S.E.2d at 191.

The claims tried to verdict in this matter were personal injury claims on Mr. Jolly's behalf and loss of consortium on Mrs. Jolly's behalf. On the other hand, the settlements received by Plaintiffs in this matter include compensation for claims not tried to verdict; specifically, the release of future claims such as wrongful death. As such, Defendants' entitlement to a setoff does not arise as a matter of law, and this Court must make the determination of how to allocate the settlement. After this allocation is made, the setoff amount can be determined.[2]

Further, in contrast to Defendants' contention, the allocation of the settlement amount is relevant to this issue because Defendants are only entitled to "credit for the amount paid by another defendant **who settles for the same cause of action**." *Rutland v. S.C. Dep't of Transp.*, 400 S.C. 209, 216, 734 S.E.2d 142, 145 (2012) (emphasis added). Defendants argue, essentially, that, based on the Court of Appeals' holding in *Ellis, supra*, there is no difference between the claims that were tried to verdict and the claims for which Plaintiffs received settlement prior to trial. The Court of Appeals later acknowledged the conflict created by the *Ellis* holding. In reference to the requirement that the trial court determine the allocation of the settlement funds when the settlement includes payment for claims tried to verdict and claims that were not, the *Smith* court stated:

> This is what the circuit court did in *Rutland v. S.C. Dep't of Transp.*, 390 S.C. 78, 700 S.E.2d 451 (Ct. App. 2010), cert. granted, (Oct. 19, 2011).[3] This court affirmed the *Rutland* trial court's determination that the settlement that the settlement was

[2]As of the date of this response, the settlements received by Plaintiffs have been internally allocated as follows: 1/3 to Mr. Jolly's personal injury claims, 1/3 to Mrs. Jolly's loss of consortium claims, and 1/3 to the release of future claims.

[3] The Court of Appeals' holding was appealed to the Supreme Court of South Carolina in *Rutland v. S.C. Dep't of Transp.*, 400 S.C. 209, 734 S.E.2d 142 (2012). On this issue, the Supreme Court affirmed the trial court's equitable reallocation of the settlement received by the plaintiff.

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> not properly allocated between the different claims of wrongful death and survival because there was no evidence of conscious pain and suffering. 390 S.C. at 85–86, 700 S.E.2d at 455. In this respect, *Rutland* conflicts with *Ellis*. In *Ellis,* the court of appeals stated that wrongful death and survival are the same claim for the same injury. 335 S.C. at 112–13, 515 S.E.2d at 272. The conflict between *Rutland* and *Ellis* is resolved by reference to *Bennett v. Spartanburg Railway, Gas & Electric Co.,* 97 S.C. 27, 81 S.E. 189 (1914), in which the supreme court held that wrongful death and survival actions are different claims for different injuries. 97 S.C. at 29–30, 81 S.E. at 189–90. The court stated: "Necessarily, therefore, there must be separate verdicts and separate judgments, and hence there should be separate actions." 97 S.C. at 31, 81 S.E. at 190.

Smith, 397 S.C. at 481, ftn, 1, 724 S.E.2d at 195, ftn, 1.

Ellis is not controlling in this matter. The personal injury and loss of consortium claims are separate and distinguishable from the potential wrongful death claim. Because this potential claim is different, factually and legally, from the claims tried to verdict in this matter, the amount that Plaintiffs received as compensation for the release of future claims, such as wrongful death, should not be considered by this Court when determining the setoff amount. Instead, in accordance with *Rutland* and *Smith*, only the amounts received by Plaintiffs in settlement for the personal injury and loss of consortium claims should be considered by this Court when determining the setoff amount.

Additionally, the internal allocation of the settlement funds received by Plaintiffs is reasonable as required by South Carolina precedent. *See Riley v. Ford Motor Co.*, 414 S.C. 185, 196-97, 777 S.E.2d 824, 830-31 (2015). *See also In re Wells*, 43 S.C. 477, 21 S.E. 334, 337 (1895) (finding the party seeking departure from the application of standard set-off rules bears the burden of proof and must be "prepared to justify such [reallocation] as fair, bona fide, and just," particularly where "there is an executed contract between [the parties], which is not contested as between them but which is sought to be invalidated by third parties"). In *Rile*y, the Supreme Court noted that "[s]ettling parties are naturally going to allocate settlement proceeds in a manner that

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serves their best interests. That fact alone is insufficient to justify . . . reapportionment for the sole purpose of benefitting [non-settling defendants]." *Riley*, 414 S.C. at 197, 777 S.E. 2d at 831.

Defendants have failed to present any evidence that Plaintiffs' internal allocation of the settlement proceeds is unreasonable. In fact, Defendants only argue that the allocation of funds is irrelevant in an attempt to influence the set-off amount to their benefit. However, as recognized by this state's Supreme Court, this reasoning is insufficient. As such, given the totality of the circumstances and the lack of evidence to demonstrate that Plaintiffs' internal allocation of settlement funds is unreasonable, this Court should find said allocation reasonable and approve the allocation as made by Plaintiffs.

CONCLUSION

Accordingly, for the reasons set forth herein, Plaintiffs request that this Court deny Defendants' Joint Motion for Set-Off, approve the allocation of settlement funds as made by Plaintiffs, or alternatively, hold in abeyance a ruling on Defendants' motion until after the Court enters a ruling on Plaintiffs' Motion for New Trial *Nisi Additur*.

Respectfully submitted,

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CERTIFICATE OF SERVICE

I hereby certify that a true and correct copy of the above and foregoing instrument was e-filed with the Spartanburg County court's e-filing system, and thereby served electronically on all counsel of record, this 6th day of September 2017.

/s/ *Jonathan M. Holder*
Jonathan M. Holder
Attorney for Plaintiff

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STATE OF SOUTH CAROLINA **COUNTY OF SPARTANBURG** **BEVERLY DALE JOLLY and BRENDA RICE JOLLY,** **Plaintiffs,** **v.** **GENERAL ELECTRIC COMPANY, et al.** **Defendants.**	)))))))))))))))	**IN THE COURT OF COMMON PLEAS FOR THE SEVENTH JUDICIAL CIRCUIT** **C/A No. 2016-CP-42-1592** **PLAINTIFFS' REPLY IN SUPPORT OF THEIR MOTION FOR NEW TRIAL *NISI ADDITUR***

Plaintiffs submit this Reply in support of their Motion for New Trial *Nisi Additur*.

As the Court is aware, the jury awarded total damages of only $300,000, including only $200,000 to Mr. Jolly for all the past and future damages arising from his mesothelioma diagnosis and expected death. Plaintiffs have asked for an increase in the award because this amount is inadequate to cover his damages that were proven at trial. The jury either awarded nothing for Mr. Jolly's medical expenses, or nothing for his past and future pain and suffering. It is not proper for the jury to award medical expenses but no pain and suffering damages. *Waring v. Johnson*, 31 S.C. 248, 260, 533 S.E.2d 906, 912 (2000). And it is not proper for the jury to award less in medical damages than was proven by the evidence. *See Haselden v. Davis*, 353 S.C. 481, 484, 579 S.E.2d 293, 295 (2003) ("A plaintiff in a personal injury action seeking damages for the cost of medical services provided to him as a result of a tortfeasor's wrongdoing is entitled to recover the reasonable value of those medical services, not necessarily the amount paid."). Viewed from any angle, the amount awarded by the jury is too low to fully compensate Mr. Jolly.

Fisher and Crosby argue that, with regard to Mr. Jolly's medical expenses, the jury did not have to credit Dr. Frank's testimony regarding the amount of his treatment. While the jury is of

course the judge of the credibility of the witnesses, the jury cannot find that Defendants caused Mr. Jolly's cancer, necessitating extensive medical care and invasive surgery, and then decline to credit the uncontested evidence regarding the value of that medical care. In considering whether to grant a new trial, this Court may take into consideration the fact that the medical evidence was uncontested. *See Lane v. Gilbert Const. Co.*, 383 S.C. 590, 598-99, 681 S.E.2d 879, 883 (2009).

Defendants also completely ignore the future aspect of Mr. Jolly's damages. It is the duty of the jury to estimate, as best it can, the future damages which are reasonably certain to be accrued by the plaintiff. *Pearson v. Bridges*, 344 S.C. 366, 373, 544 S.E.2d 617, 620 (2001); *Haltiwanger v. Barr*, 258 S.C. 27, 29, 186 S.E.2d 819, 820 (1972). Not only did Dr. Frank testify about that Mr. Jolly would continue to require medical care until his death, and that his medical needs and physical pain would increase as his disease progressed, but defense expert Dr. Crapo completely agreed. (TT 7/26 at 101-02; TT 8/1 at 151-53). There is also the matter of Mr. Jolly's life expectancy of more than 11 more years, which, according to experts on both sides of the case, will be cut short prematurely because of his mesothelioma. (TT 7/26 at 99; TT 8/2 at 190).

The purpose of a new trial *nisi additur* is to provide a remedy in exactly this situation, where the jury finds the defendant liable for the plaintiff's injury but does not award a sufficient amount in damages. "The trial judge *alone* has the power to grant a new trial *nisi* when he finds the amount of the verdict to be merely inadequate or excessive." *O'Neal v. Bowles*, 314 S.C. 525, 527, 431 S.E.2d 555, 556 (1993). "Absent an abuse of discretion, the trial court's grant of a motion for new trial *nisi additur* will not be reversed on appeal." *Waring v. Johnson*, 341 S.C. 248, 258, 533 S.E.2d 906, 911 (Ct. App. 2000).

Once the jury found that Defendants' negligence and breach of warranty caused Mr. Jolly to develop cancer, they were required to award adequate damages according to the evidence

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presented. In light of the evidence that Mr. Jolly has suffered for two years from an incurable and terrible disease, that he will die about 11 years prematurely, and that his medical situation will deteriorate before his death, the jury's damages award was inadequate. Plaintiffs therefore ask the Court to adjust the jury's verdict to adequately compensate Mr. and Mrs. Jolly by granting a new trial *nisi additur*.

Respectfully submitted,

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STATE OF SOUTH CAROLINA	)	IN THE COURT OF COMMON PLEAS
	)	FOR THE SEVENTH JUDICIAL CIRCUIT
COUNTY OF SPARTANBURG	)	
Beverly Dale Jolly and Brenda Rice Jolly,	)	Civil Action No. 2016-CP-42-01592
	)	
Plaintiffs,	)	**DEFENDANTS FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE, LLC'S JOINT RULE 59(e) MOTION TO ALTER OR AMEND**
	)	
v.	)	
	)	
General Electric Company, et al.,	)	
	)	
Defendants.	)	
	)	
	)	

COMES NOW, Defendants Fisher Controls International LLC ("Fisher") and Crosby Valve, LLC ("Crosby"), by and through their attorneys, and hereby move the Court pursuant to Rule 59(e) of the South Carolina Rules of Civil Procedure to reconsider its un-dated Order filed September 25, 2017 denying Defendants' Motion to Quash Plaintiffs' Trial Subpoenas.

BACKGROUND

Plaintiffs brought the instant action against sixty-three (63) Defendants, including Fisher and Crosby. Prior to trial, Plaintiffs settled with or dismissed their claims against all of the Defendants with the exception of Fisher and Crosby. The trial commenced on July 24, 2017 and the jury rendered a verdict on August 3, 2017.

Prior to trial, Plaintiffs' counsel, by way of letter dated July 11, 2017 mailed two trial Subpoenas to Fisher and Crosby's trial counsel's office in Charleston, South Carolina via FedEx delivery. (*See* Trial Subpoenas, attached hereto as **Exhibit A**). The Subpoenas commanded

Fisher and Crosby to appear at trial. Fisher and Crosby are out-of-state corporations and the subpoenas were unknowingly[1] - received by the undersigned counsel on July 12, 2017.

Fisher and Crosby filed Motions to Quash the subpoenas on July 20, 2017, and a hearing on the Motion took place on July 21, 2017. (*See* July 21, 2017 Hearing Transcript, attached hereto as **Exhibit B**). The Court orally denied the Motions at the hearing and again at trial. The Court commanded Fisher and Crosby, over their objections, to produce their Rule 30(b)(6) corporate witnesses to testify at trial in Plaintiff's case-in-chief. On September 25, 2017, the Court entered an undated formal order denying the Motions to Quash. Because the Order fails to address certain issues previously raised, Fisher and Crosby now move the Court pursuant to Rule 59(e) of the South Carolina Rules of Civil Procedure to alter or amend the Order. The Court should reconsider its ruling for all of the reasons set forth in Fisher and Crosby's previous arguments, including the following:

1. **The mailing of a subpoena on an out-of-state defendant corporation's trial counsel via FedEx delivery does not constitute personal service pursuant to the Rules.**

The Court should reconsider its Order denying the Motions to Quash the Trial Subpoenas because sending a sealed FedEx package to the out-of-state corporation's trial counsel is not personal service as required by the South Carolina Rules of Civil Procedure. *See* Rule 4 and 45, SCRCP. Rather, service of a subpoena on an out-of-state corporation must be done in accordance with Rule 45, SCRCP. That Rule provides for provides for service only in "the same manner prescribed for service of the a summons and complaint in Rule 4(d) or (j)." *See* Rule 45(b)(1), SCRCP. Neither was accomplished here. The Plaintiffs did not serve the Subpoenas in the same

[1] As stated at the July 21, 2017 Hearing, the undersigned counsel happened to be in the lobby of the law firm at that time the FedEx delivery occurred on July 12, 2017. The undersigned was not aware of the contents of this particular package at the time they were delivered by FedEx.

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manner as a Summons and Complaint as contemplated in Rule 4(d). To do so, they would have had to serve the Defendants' registered agent, and they did not. Indeed, these Defendants do not have registered agents in South Carolina. Nor did the Plaintiff serve the Subpoenas in the manner prescribed for in Rule 4(j), SCRCP. That Rule is not applicable here. Rule 4(j) contemplates when no other "proof" of service is required, not the validity of the service itself. "Rule 4(j) establishes no new procedure for service of process; rather, it is but a recognition of the long standing practice that acknowledgement or acceptance of service is equivalent to personal service." *Langley v. Graham*, 322 S.C. 428, 431–32, 472 S.E.2d 259, 261 (Ct. App. 1996).

Importantly, the undersigned counsel did not "acknowledge" service of these subpoenas as contemplated by Rule 4(j), SCRCP. On the contrary, the undersigned did not know the contents of what was inside the sealed FedEx package and it was therefore impossible for him to "acknowledge" that he was accepting service of a subpoena. This is not what Rule 4(j) contemplates. The Plaintiffs did not have written acknowledgment from the undersigned counsel that he would accept service of the subpoenas. The subpoenas were not served in compliance with the Rules and this court should reconsider is denial of the Defendants' Motion to Quash.

2. The Court's exercise of personal jurisdiction does not give it subpoena power over out-of-state parties.

South Carolina courts cannot compel an out-of-state party to appear at trial, regardless of whether the court has properly exercised personal jurisdiction over the party. Personal jurisdiction contemplates the court's authority to entertain a particular action and to render a judgment against a defendant. *See, e.g., Boan v. Jacobs*, 296 S.C. 419, 421, 373 S.E.2d 697, 698 (Ct. App. 1988). Personal jurisdiction, however, does not give the court power to compel the out-of-state party's attendance at trial. *See Vaught v. Nationwide Mut. Ins. Co.*, 250 S.C. 65, 74, 156 S.E.2d 627, 632 (1967) ("a subpoena issued to an out of state witness would have been legally

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ineffective to compel the attendance of such witness at a trial in this State."); *Johnson v. Land O'Lakes, Inc.*, D.C. Iowa 1998, 181 F.R.D. 388 ("The fact that a nonparty witness is an officer of a party does not extend the trial court's subpoena power beyond the 100-mile limit.")

There exists no legal requirement that a civil defendant attend trial, much less a civil defendant that is beyond the reach of the court's subpoena power. *See id; see also* Rule 32, SCRCP (providing the deposition of a witness, *whether a party or not*, may be used for any purpose if the court finds the witness is out of State) (emphasis added). There are no exceptions in Rule 45, SCRCP that allow a South Carolina court to Compel non-officer employees of an out-of state corporation to testify within South Carolina, in trial or otherwise. As such, the Court should reconsider its ruling and grant the Defendants' Motion to Quash.

Respectfully submitted this, the 2nd day of October, 2017.

LEATH, BOUCH & SEEKINGS, LLP

By: /s/ Yancey McLeod

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DAVID RAMSAY HOUSE
c 1740

LEATH, BOUCH & SEEKINGS, LLP
COMMERCIAL LITIGATION • CONSTRUCTION • ENVIRONMENTAL

October 20, 2017

VIA REGULAR MAIL
The Honorable Jean H. Toal
Chief Justice, South Carolina Supreme Court (Ret.)
1231 Gervais Street
Columbia, SC 29201

Re: *Beverly Dale Jolly and Brenda Rice Jolly v. General Electric Company, et al.* - 2016-CP-42-1592

Dear Chief Justice Toal:

Enclosed please find a courtesy copy of the following pleading:

- **OBJECTIONS OF FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE, LLC TO PROPOSED ORDER DRAFTED BY PLAINTIFFS AND SUPPLEMENTAL MEMORANDUM IN SUPPORT OF THEIR MOTIONS AND IN OPPOSITION TO NEW TRIAL *NISI* MOTION.**
- **EXHIBIT A – PLAINTIFF'S PROPOSED ORDER**

These objections were filed with the Clerk's office this morning.

With best regards I am,

Very truly yours,

LEATH, BOUCH & SEEKINGS, LLP

Yancey A. McLeod, III

Enclosures: As stated

Cc: Theile McVey, Esq.
Jonathan Holder

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STATE OF SOUTH CAROLINA COUNTY OF SPARTANBURG	)	IN THE COURT OF COMMON PLEAS FOR THE SEVENTH JUDICIAL CIRCUIT
BEVERLY DALE JOLLY and BRENDA RICE JOLLY, Plaintiffs, v. CROSBY VALVE, LLC and FISHER CONTROLS INTERNATIONAL LLC, et al., Defendants.	)	Civil Action No. 2016-CP-42-01592 **OBJECTIONS OF FISHER CONTROLS INTERNATIONAL LLC AND CROSBY VALVE, LLC TO PROPOSED ORDER DRAFTED BY PLAINTIFFS AND SUPPLEMENTAL MEMORANDUM IN SUPPORT OF THEIR MOTIONS AND IN OPPOSITION TO NEW TRIAL *NISI* MOTION**

Defendants Fisher Controls International LLC ("Fisher") and Crosby Valve, LLC ("Crosby") respectfully submit the following objections to the "Order Denying Defendants' Post-Trial Motions and Granting Plaintiffs' Motion for a New Trial *Nisi Additur*" prepared by counsel for the Plaintiffs and submitted to the Court on October 6, 2017. (*See* Plaintiff's Proposed Order, attached hereto as Exhibit 1). At the outset, the Defendants incorporate by reference all arguments and objections set forth in their pre-trial, trial and post-trial briefs and related argument to the Court at each pre-trial, trial and post-trial hearing. The following are additional objections based on the form or content of the proposed order. Any Order entered by this Court should state that the Court has considered all arguments and grounds, written and oral, in ruling on the pending motions.

First, the proposed order sets forth numerous facts and conclusions as though they were findings made by the jury; apart from questions specifically answered by the jury, there were no factual findings made by the jury and it is inappropriate to state or infer otherwise.

THE STATE OF SOUTH CAROLINA
In The Court of Appeals

APPEAL FROM SPARTANBURG COUNTY
Court of Common Pleas

Jean H. Toal, Circuit Court Judge

Appellate Case No. 2017-002611
Case No. 2016-CP-42-1592

Beverly Dale Jolly and Brenda Rice Jolly, ... Respondents,

v.

General Electric Company, et al., ... Defendants,

Of whom Fisher Controls International LLC and Crosby Valve, LLC are the..... Appellants.

CERTIFICATE OF COUNSEL

The undersigned certifies that the Record on Appeal contains all material proposed to be included by any of the parties and not any other material.

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By: _____

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August 14, 2018