

IN THE COURT OF APPEALS OF MARYLAND

No. 102, September Term, 2012

GEORGIA-PACIFIC, LLC,

Petitioner,

v.

JOCELYN A. FARRAR,

Respondent.

On Appeal from the Court of Special Appeals of Maryland
No. 751, September Term, 2010

**AMICI CURIAE BRIEF OF COALITION FOR LITIGATION JUSTICE, INC.,
CHAMBER OF COMMERCE OF THE UNITED STATES OF AMERICA,
NATIONAL ASSOCIATION OF MANUFACTURERS, AMERICAN
INSURANCE ASSOCIATION, PROPERTY CASUALTY INSURERS
ASSOCIATION OF AMERICA, AMERICAN PETROLEUM INSTITUTE,
AMERICAN CHEMISTRY COUNCIL, ALLIANCE OF AUTOMOBILE
MANUFACTURERS, AND NFIB SMALL BUSINESS LEGAL CENTER
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QUESTIONS PRESENTED

1. Does this Court's multi-factor test for determining when duties enforceable in tort law are owed to third parties apply to product liability claims and, if so, do product manufacturers have a legal duty to warn household residents of the risks of exposure to toxic materials carried home by workers who did not use the product in question but were exposed indirectly as bystanders?
2. What is the minimum threshold of sufficient evidence that satisfies [the *Eagle-Picher Indus., Inc. v. Balbos*, 326 Md. 179, 604 A.2d 445 (1992)] "frequency, proximity, and regularity" test for substantial-factor causation?

INTEREST OF AMICI CURIAE

*Amici*¹ are organizations whose members are named as defendants in asbestos cases and their insurers. This appeal, and the appeal in *Dixon v. Ford Motor Co.*, No. 82, September Term 2012, set for oral argument the same day as this case, present this Court with important opportunities to provide significant guidance to trial courts that are wrestling with complex exposure and causation issues in asbestos cases. Consequently, *amici* have a significant interest in the issues before this Court.

STATEMENT OF THE CASE

Amici adopt Petitioner Georgia-Pacific, LLC's Statement of the Case.

STATEMENT OF FACTS

Amici adopt Petitioner Georgia-Pacific, LLC's Statement of Facts as relevant to *amici*'s argument here.

¹ None of the parties or their counsel, or anyone other than the *amici*, their members, or their counsel, authored this brief in whole or in part or made a monetary contribution intended to fund the brief's preparation or submission.

SUMMARY OF THE ARGUMENT

Amici believe that the process of deciding whether an asbestos case gets to a jury in Maryland has, in some instances, gone off track. The result is a body of case law in which cases are going to trial on the thinnest of exposure evidence. The foundation for these decisions is the combination of the discredited *any exposure* theory² and the 1980s “frequency, regularity, proximity” causation standard set forth in *Lohrmann v. Pittsburgh Corning Corp.*, 782 F.2d 1156 (4th Cir. 1986) (interpreting Maryland law) (“*Lohrmann*”) and adopted by this Court in *Eagle-Picher Industries, Inc. v. Balbos*, 326 Md. 179, 604 A.2d 445 (1992) (“*Balbos*”). The *Lohrmann/Balbos* test was developed in the context of a different kind of asbestos litigation. That process needs to be redirected by this Court in its opinions in *Dixon* and here to adequately reflect the tenets of asbestos science and causation.

First, both *Dixon* and *Farrar* illustrate the inappropriate use of the *any exposure* theory by plaintiffs’ counsel and their experts bent on coercing settlements from minimal dose defendants. *Amici* filed a brief in *Dixon* advising the Court of the unscientific nature of the *any exposure* theory and the necessity of declaring it unacceptable in Maryland toxic tort cases. The experts in *Farrar* likewise disdained to develop *any* estimate of the dose Ms. Farrar may have received from bystander or “passer-by” joint compound exposures of her grandfather, instead declaring any exposure “above background” to be sufficient to cause disease.

Nearly thirty courts around the country have rejected *any exposure* testimony as a speculative and unproven hypothesis that is inconsistent with the scientific principle of dose – including very recently this Court in the context of fear of cancer and medical monitoring claims from alleged MTBE and benzene exposures. See *Exxon Mobil Corp.*

² Plaintiffs’ experts who support this theory opine that any occupational or product-related exposure to asbestos fibers above or different from “background” exposures is a substantial contributing factor to the ultimate disease, without regard to assessing dosage. See Mark A. Behrens & William L. Anderson, *The “Any Exposure” Theory: An Unsound Basis for Asbestos Causation and Expert Testimony*, 37 Sw. U. L. Rev. 479 (2008).

v. Albright, 2013 WL 673738, *32-33 (Md. Feb. 26, 2013); *Exxon Mobil Corp. v. Ford*, 2013 WL 673710, *15 (Md. Feb. 26, 2013).

Furthermore, Plaintiffs' experts in *Farrar*, Drs. Arthur Frank and John Maddox, are among the most frequently criticized experts in these opinions – their *any exposure* testimony has been rejected at least ten times.³ The causation proof and expert testimony in Maryland asbestos cases must return to ordinary toxicology and tort principles and identify a dose that is causative. Anything less will only exacerbate the never-ending asbestos litigation crisis and deprive defendants of any fair allocation of the burden of proof.

Second, the *Farrar* case illustrates why the *Lohrmann/Balbos* “frequency, regularity, proximity” test no longer functions adequately as a limiting device in today’s asbestos litigation. That standard helped correct causation-evidence abuses in the 1980s by using frequency, regularity, and proximity as a marker for substantial factor causation. At that time, most cases were about insulation exposures in large facilities like the Sparrows Point steel mill in Maryland. See *Reiter v. Pneumo Abex, LLC*, 417 Md. 57, 8 A.3d 725 (2010). Today’s asbestos litigation is very different – the types of exposures at issue are far lower, the fiber type at issue (usually chrysotile) is far less potent than insulation, and the products are typically encapsulated or otherwise unlikely to give off

³ See *Moeller v. Garlock Sealing Technologies, Inc.*, 660 F.3d 950 (6th Cir. 2011) (Dr. Frank); *Bartel v. John Crane, Inc.*, 316 F. Supp. 2d 603, 611 (N.D. Ohio 2004) (Dr. Frank), *aff'd sub nom. Lindstrom v. A-C Prod. Liab. Trust*, 424 F.3d 488 (6th Cir. 2005); *Ford Motor Co. v. Boomer*, 736 S.E.2d 724 (Va. 2013) (Dr. Maddox) (declining to address *any exposure* theory directly but requiring plaintiff’s experts to “opine as to what level of exposure is sufficient to cause mesothelioma, and whether the levels of exposure at issue in this case were sufficient”); *Betz v. Pneumo Abex, LLC*, 44 A.3d 27 (Pa. 2012) (Dr. Maddox); *Gregg v. V-J Auto Parts Co.*, 943 A.2d 216 (Pa. 2007) (Dr. Frank); *Butler v. Union Carbide Corp.*, 712 S.E.2d 537 (Ga. Ct. App. 2011) (Dr. Maddox); *Smith v. Kelly-Moore Paint Co., Inc.*, 307 S.W.3d 829 (Tex. Ct. App.-Ft. Worth 2010) (Dr. Maddox); *Daly v. Arvinmeritor, Inc.*, 2009 WL 4662280 (Fla. Cir. Ct. Broward County Nov. 30, 2009) (Dr. Frank); *In re Asbestos Litig. (Certain Asbestos Friction Cases Involving Chrysler LLC)*, 2008 WL 4600385 (Pa. Ct. Com. Pl. Phila. County Sept. 24, 2008) (Dr. Frank); *Basile v. American Honda Motor Co., Inc.*, 2007 WL 712049 (Pa. Ct. Com. Pl. Indiana County Feb. 22, 2007) (Dr. Maddox).

any significant respirable fibers. The *Lohrmann/Balbos* standard is not capable of dealing with the extreme differences in dose and toxicity involved in these minimal exposure situations. Maryland needs to revise its standard (like the highest courts of Texas and Pennsylvania have done) to account for dose and potency, bringing judicial decisions into line with asbestos science.

Third, *Farrar* is the latest in a series of Maryland asbestos cases in which judges are being forced to take on the role that plaintiffs' experts should fulfill but have abdicated. The experts in these cases should be performing a competent scientific assessment of causation. In any other context, a causation assessment would require (1) an estimate of the range of dose likely received by the plaintiff from each product or activity; (2) an adjustment in the required dose to account for the fiber potency involved; and (3) a comparison of the plaintiff's dose to epidemiology studies of similarly exposed populations to show that these exposures actually cause disease. This testimony would help the court and jury understand which asbestos-related exposures are causative and which are not. These experts have done none of this. Instead, they come into court claiming only that there was "visible dust" in the environment and the plaintiff breathed some of it. With that declaration, they leave it entirely to the jury and judge to figure out exactly which of those exposures might serve as an actual cause of asbestos disease.⁴

Judges in cases like *Reiter, supra*; *John Crane, Inc. v. Linkus*, 190 Md. App. 217, 988 A.2d 511 (2010); *Scapa Dryer Fabrics, Inc. v. Saville*, 418 Md. 496, 16 A.3d 159 (2011); and now *Farrar* and *Dixon* are being forced to try to figure out whether once a week is "frequent" enough, whether ten exposure incidents is "regular" enough, and whether fifteen feet away is "proximate" enough. *This is the job of plaintiffs' experts and causation science, not judges.* Judges should assess what the experts do, not do their job for them. *Amici* respectfully request that the Court require a competent causation

⁴ The role of experts in this line of Maryland cases has become so pointless that the Court of Special Appeals recently held that plaintiffs can prove their case without any industrial hygiene or dose expert testimony at all. See *John Crane, Inc. v. Linkus*, 190 Md. App. 217, 988 A.2d 511 (2010).

determination from these experts – not fanciful reliance on “dust” and “increased risk” – and extricate Maryland’s trial and appellate judges from making these scientific determinations in the first instance.

Amici discuss the key tenets of asbestos science and toxicology that should be guiding Maryland court decisions, but have been gradually left by the wayside in the course of the last twenty years of decisions. Those tenets are applied widely in science itself and in other toxic tort litigation, and they have recently formed the core of corrective decisions by the Supreme Courts of Pennsylvania, Virginia, and Texas. Those decisions have helped restore order and fairness to asbestos litigation in those states. The same principles need to be returned to their rightful place in Maryland asbestos litigation, particularly since the principle of dose played such an important role in this Court’s very recent decisions in *Albright*, 2013 WL 673738, *32-33, and *Ford*, 2013 WL 673710, *15.

Amici request that the Court correct the course of asbestos litigation in Maryland by reiterating that: (1) asbestos litigation does not require its own set of special causation rules; (2) the *any exposure* theory will not serve as a basis for causation or expert testimony; and (3) plaintiffs and their experts must prove their causation case through competent industrial hygiene and epidemiology testimony and not assume it using guesswork about “visible dust,” unquantified “increased risk,” and causation opinions based on “exposures above background.”

ARGUMENT

I. THE *ANY EXPOSURE* THEORY – THE LYNCHPIN BEHIND THE RULINGS IN *FARRAR* AND *DIXON* – IS UNSCIENTIFIC AND CANNOT SUPPORT THE VERDICT

The plaintiff’s experts in *Farrar* – much as a similar asbestos plaintiffs’ expert (Dr. Laura Welch) did in *Dixon* – relied on the litigation-based and discredited *any exposure* theory to support their causation opinions. Under this theory, the actual dose received by the plaintiff is irrelevant – any exposure in the workplace, no matter how limited, is declared causative or substantial. This theory is pure speculation and inconsistent with asbestos science. It is a device used to shift the burden of proof

inappropriately to defendants and undercut any notion of substantiality in the *substantial factor* causation standard. The Court should reject its use in *Farrar* as well as in *Dixon*.

The *amici* here filed a brief in *Dixon* that addresses the flaws in the *any exposure* theory and its pernicious effect on asbestos litigation. See *Amici Curiae* Brief of Coalition for Litigation Justice, Inc. et al., *Dixon v. Ford Motor Co.*, No. 82, Sept. Term 2012 (Md. filed Feb. 22, 2013) (hereinafter “CLJ *Dixon* Brief”). *Amici* refer the Court to that brief and will not repeat those arguments here. Suffice it to say that nearly thirty courts have rejected the *any exposure* theory in the last few years.

Farrar arises in a different procedural posture than *Dixon* – sufficiency of the trial evidence rather than expert admissibility – but that does not change the end result. Whether the court is testing an expert under *Daubert/Frye* or assessing the sufficiency of causation evidence to go to a jury, the *any exposure* theory is not a suitable basis for causation for an asbestos or other toxic tort case. Bad science is bad science, and it cannot support either expert testimony or a jury’s causation findings.

By any standards, the judgment against Georgia-Pacific in *Farrar* is based on extremely weak exposure evidence involving drywall work. Mr. Hentgen, Ms. Farrar’s grandfather, probably did take home a significant amount of fibers that infused his granddaughter’s lungs – but they emanated from twenty-two years of his intense and direct work as a professional insulator, not from a handful of times Ms. Farrar may have washed his clothes containing unquantified amounts of drywall fiber. [E. 712-13, 903-16, 921-23, 1160; T1452]. Extensive work with insulation is one of the paradigm workplace settings that can cause take-home disease – studies have only demonstrated such cases occurring in the context of heavy workplace exposures to amphibole fibers.⁵

⁵ U.S. Department of Health and Human Services Public Health Service Centers for Disease Control and Prevention, Nat’l Inst. for Occupational Safety & Health, Report to Congress on Workers’ Home Contamination Study Conducted Under the Workers’ Family Protection Act (29 U.S.C. 671a) 6 (1995) (“The occupations associated with asbestos-related disease in family members are those where workers were exposed to asbestos dust during: construction and renovation; prospecting and mining; manufacturing textiles, tiles, boilers, and ovens; shipbuilding and associated trades;

But the jury did not find that the suppliers of Mr. Hentgen's insulation were responsible for Ms. Farrar's disease. That is because those suppliers are bankrupt and were not a part of the trial. Instead, relying on the testimony of plaintiff's experts, the jury blamed Ms. Farrar's disease on her very limited and speculative contact with drywall fibers. Her grandfather never sanded drywall, never swept up drywall residue, and only on a few occasions was even within four to five feet of this drywall work. His granddaughter only washed his clothes for a few months, and she shared the once-a-week washing duty with other members of the household.⁶ If Mr. Hentgen took home any fibers from the drywall work, they would have been dwarfed in the much larger quantity of insulation fibers on his clothes – a molehill (if that) compared to a mountain.

The drywall product and fibers were also not the same potency as the insulation Mr. Hentgen worked with every day, since drywall contained only *chrysotile*, a fiber type much less potent than the amphibole fibers frequently found in insulation. [E.377-78]. Furthermore, the percentage of asbestos in drywall and joint compound at issue here (1.5 to 5%) is much lower than the percentage in insulation. [E. 778-79, 1031]. Cohorts of workers exposed to chrysotile fibers have almost never experienced an increased incidence of mesothelioma, and those that do have only a very few cases resulting from extremely high exposures – upwards of 100 f/cc. *See CLJ Dixon* Brief at 14-15. That amount is more than 1,000 times today's "acceptable exposure" standard for asbestos as established by the Occupational Safety and Health Administration (OSHA) and far above anything Ms. Farrar could have experienced. *See* 29 C.F.R. § 1910.1001(c) (0.1 f/cc 8-hr TWA standard). Even more telling, take-home epidemiology studies have only identified

certain railroad shop trades; welding; insulation; use and manufacture of asbestos products such as cords, seals, and plates; and renovation and demolition projects within the construction industry.”).

⁶ Family members washed Mr. Hentgen's clothes once a week, and he was around the drywall work for a period of six to seven months, for a maximum of 28-30, five to ten minute exposures. Ms. Farrar testified that she shared this duty with four other family members, so the actual number of exposures for her is far less even than that. [E. 712, 765-67]. Her short-term exposures are below even the longer-term exposures of a similar frequency rejected in *Lohrmann* as inconsequential. *See Lohrmann*, 782 F.2d at 1162-63.

an excess of mesothelioma in populations in which the workers were heavily exposed to amphiboles, not to chrysotile. Put succinctly, as even Maryland courts have acknowledged, not all asbestos is the same, and not all products are the same. See *Linkus*, 190 Md. App. at 224-25, 988 A.2d at 516; *Gideon v. Johns-Manville Sales Corp.*, 761 F.2d 1129, 1145 (5th Cir. 1985) (“all asbestos products cannot be lumped together in determining their dangerousness”). Yet plaintiffs’ experts continue to ignore these differences and opine that all exposures are equally causative.

Given the dramatic differences in Ms. Farrar’s take-home insulation exposures and her very limited (if any) take-home drywall exposures, one would think the causation experts would make at least some effort to identify what her dose of drywall fibers would be and discuss why that dose would be considered causative in light of the extensive insulation fibers she probably inhaled. These experts *did no such thing*. Instead, the experts assumed that any exposures above background or ambient levels are causative of disease, then opined that Ms. Farrar’s exposures from drywall would have been “more than background.” [E. 392-93, 437-38, 453-63, 477- 80, 615-23, 646, 795-97, 812-15].⁷

These experts are relying for their opinions on the infamous and discredited *any exposure* theory – usually stated as “each and every exposure above background is a substantial factor in causing mesothelioma.” In recent years, challenges to the

⁷ Both Drs. Frank and Maddox testified that Ms. Farrar’s exposures were “more than background” – and were thus causative. *Georgia-Pacific, LLC v. Farrar*, 207 Md. App. 520, 531, 53 A.3d 424, 431 (2012). Even this declaration is unsupported by any attempt to assess what her dose actually was and/or to compare it to known “background” level exposures in, e.g., urban environments. The opinion is a mere guess. Likewise, the experts’ testimony that Ms. Farrar had a lung asbestos burden “above background” is no better. “Above background” is not the standard for causing disease, and these experts produced no studies demonstrating such a thing. Her lung tissue revealed an excess of crocidolite and tremolite, neither of which is found in the drywall at issue here (the insulation Mr. Hentgen worked with or near contained crocidolite, [E. 377-78, 627, 642-43, 9013-16, 921-33]), and did not find evidence of the type of short chrysotile fibers used in that drywall.

unscientific nature of this theory have resulted in many courts rejecting it outright.⁸ These are not minor opinions – they include the Supreme Courts of Pennsylvania and Texas (and arguably Virginia), plus the United States Court of Appeals for the Sixth Circuit (three times) and numerous federal district courts and state trial and appellate courts. *See* CLJ *Dixon* Brief at 19-20 n.20.

Plaintiffs have even attempted to export the *any exposure* theory into non-asbestos litigation in recent years, where it has been almost uniformly rejected. For example, this Court very recently rejected such testimony as insufficient to support fear of cancer and medical monitoring claims from alleged MTBE and benzene exposures. *See Exxon Albright*, 2013 WL 673738; *Ford*, 2013 WL 673710. In *Albright*, this Court explained that “[f]or a fear to be objectively reasonable, it must be based on more than mere exposure to a chemical or contaminant of concern.” 2013 WL 673738, *20. The Court went on to explain why generalized, *any exposure* theory testimony was insufficient:

[I]n order . . . to recover damages for medical monitoring, *[plaintiff]* must present expert testimony quantifying his or her risk of developing a latent disease. Specifically, the expert must indicate a particularized, significantly-increased risk of developing a disease in comparison to the general public. Here, in support of their medical monitoring claims, *[plaintiffs]* presented testimony by medical experts Dr. Kathleen Burns and Dr. Nachman Bratbaur. *Dr. Burns testified, in effect, that if an individual is exposed to MTBE or benzene in any dosage or amount, he or she incurs an additional risk of developing cancer. Similarly, Dr. Brautbar opined that plaintiffs exposed to MTBE or benzene contamination in groundwater at greater levels than that to which they would otherwise be exposed through everyday activities possessed a significantly increased risk of developing cancer. . . .*

Such testimony is insufficient to establish that [plaintiffs] had a significantly increased risk of developing cancer as a result of their alleged exposure to MTBE and benzene. Neither Dr. Brautbar nor Dr. Burns attempted to

⁸ *See* CLJ *Dixon* Brief at 19-20 n.20; William L. Anderson et al., *The “Any Exposure” Theory Round II: Court Review of Minimal Exposure Expert Testimony in Asbestos and Toxic Tort Litigation Since 2008*, 22 Kan. J.L. & Pub. Pol’y 1 (2012); Behrens & Anderson, *supra*; Victor E. Schwartz, *A Letter to the Nation’s Trial Judges: Asbestos Litigation, Major Progress Made Over the Past Decade and Hurdles You Can Vault in the Next*, 36 Am. J. of Trial Advoc. 1 (2012).

quantify any individual [plaintiff's] increased risk of developing cancer. Rather, because Dr. Brautbar and Dr. Burns testified under the assumption that any exposure to MTBE or benzene is unacceptable from a public health standpoint and increases the risk of developing cancer, they offered no [plaintiff]-specific testimony. *The level of generalization in this regard presented at trial is insufficient* to establish that the remaining [plaintiffs] suffered a significantly increased risk of developing cancer as a result of their exposure to MTBE and/or benzene as a consequence of the Exxon leak. Accordingly, we reverse.

Id. at *32-33 (emphasis added).

The New York Court of Appeals rejected an attempt by two experts to claim that gas station exposure to benzene in gasoline caused leukemia, without conducting any assessment of plaintiff's actual dose. *See Parker v. Mobil Oil Corp.*, 857 N.E.2d 1114 (N.Y. 2006). Plaintiff's asbestos expert Dr. David Egilman tried to assert the theory in a popcorn diacetyl case, but a federal district judge issued a 32-page opinion eviscerating Dr. Egilman's approach and excluding his testimony. *See Newkirk v. ConAgra Foods, Inc.*, 727 F. Supp. 2d 1006 (E.D. Wash. 2010), *aff'd*, 438 Fed. Appx. 607 (9th Cir. 2011). Another common asbestos expert, Dr. Carl Brodtkin, tried to assert the theory in a low-dose benzene case, with the same result – rejection by a federal judge. *See Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142 (E.D. Wash. 2009). A series of experts in a Kentucky federal court case tried to testify to causation from PCBs, TCE, and dioxins without assessing the dose; the district court judge excluded their opinions and dismissed the case. *See Adams v. Cooper Indus., Inc.*, 2007 WL 2219212 (E.D. Ky. July 30, 2007), *reconsideration denied*, 2008 WL 339714 (E.D. Ky. Feb. 5, 2008). Another common plaintiff expert, James Dahlgren, attempted to link benzene with cancers in an Ohio case, but the Sixth Circuit Court of Appeals rejected that testimony because he failed to establish plaintiffs' dose. *See Pluck v. BP Oil Pipeline Co.*, 640 F.3d 671 (6th Cir. 2011). Likewise, the theory is now also being rejected in the arena – asbestos litigation – in which it began.

Amici urge this Court to follow the lead of so many other courts and reject *any exposure* testimony. The theory is completely discredited at this point and is so extreme

as to make it an inconceivable basis for toxic tort causation. See David L. Eaton, *Scientific Judgment and Toxic Torts – A Primer In Toxicology For Judges and Lawyers*, 12 J.L. & Pol’y 5, 13, 16 (2003) (carcinogens require multiple, long-term exposures; “there is some dose below which even repeated, long term exposure would not cause an effect in any individual”).

Maryland’s “substantial factor” test would not mean much if “each and every” exposure were deemed by experts to be substantial or causative. In the same way, Maryland’s “frequency, regularity, and proximity” limit – even though it is a relatively low bar for causation – would disappear completely if *any exposure* testimony (including infrequent and irregular exposures) sufficed to support causation. Judges can and should act to eliminate this kind of testimony at the expert stage, or failing that on directed verdict or similar motions when it becomes clear the expert does not intend to produce a scientifically competent dose assessment. *Farrar* is such a case.

If the Court is not inclined to adopt the proposals in the next two sections of this brief, the flawed *any exposure* testimony in *Farrar* is nevertheless sufficient by itself to warrant reversing the Court of Special Appeals and dismiss the case. When plaintiffs’ experts are not able or willing to assess and discuss Ms. Farrar’s actual dose, the case should not proceed to trial.

II. THE LOHRMANN STANDARD NEEDS UPDATING TO ACCOMMODATE DOSE AND POTENCY

Since the 1992 *Balbos* case, Maryland courts have utilized the *Lohrmann/Balbos* approach to asbestos disease causation testimony to establish causation in an asbestos case. For reasons explained below, this approach needs updating to ensure that expert causation evidence is scientifically based and that such testimony properly distinguishes between real and speculative causation. *Amici* urge the court to modify the *Lohrmann/Balbos* approach – much as the Court adopted *Lohrmann* two decades ago – to

correct the overly-permissive approach to causation creeping into today's Maryland asbestos litigation.⁹

A. The Context and Need for *Lohrmann* in the 1980s

At the time the Fourth Circuit issued the *Lohrmann* decision (1986) interpreting Maryland law, asbestos litigation focused heavily on workers in large facilities who worked directly with or in very close proximity to insulation on a daily basis. In Maryland, for instance, the massive Sparrows Point steel facility has produced many such cases and several appellate decisions on asbestos causation. Plaintiffs at the time wanted the courts to adopt a rule whereby they would only need to prove that a defendant's product was somewhere in the plant. Some of these plants, however, were enormous – the size of many football fields. The *Lohrmann* court correctly recognized that such a rule would be an abuse of Maryland's substantial factor causation standard by assigning liability to defendants without any evidence that a plaintiff actually or even likely breathed in any significant fibers from that defendant's product. 782 F.2d at 1162.¹⁰

The federal court in *Lohrmann* devised a marker or substitute for “substantial factor” by placing a floor on evidence that could go to the jury. That floor became the “frequency, regularity, and proximity” test regularly used since 1992 in Maryland cases. At the time, this standard was a much-needed corrective measure intended to keep out speculative causation testimony based on “fiber drift” – the fictional notion that fibers

⁹ Georgia-Pacific has pointed out in its briefs that *Lohrmann* and *Balbos* also do not eliminate the necessity that each exposure be sufficient by itself to cause disease. *Amici* agree with this position – and *Balbos* is clear. See 326 Md. at 208, 604 A.2d 459 (“[I]f two causes occur to bring about an event, and ***either one of them, operating alone, would have been sufficient to cause the identical result***, some test of causation other than ‘but for’ is needed.”) (emphasis added) The Court of Special Appeals omitted this language in holding otherwise, and misinterpreted a later section of *Balbos*, which merely stated that two equally causative defendants could not point to the other as the sole cause. See *Farrar*, 207 Md. App. at 547-48, 53 A.3d at 440.

¹⁰ In *Lohrmann*, even the plaintiff expert agreed that an exposure of thirty days in these steel mills was “insignificant as a causal factor.” 782 F.2d at 1162-63. This testimony illustrates how the *any exposure* theory is a relatively recent litigation invention designed to expand the scope of defendants brought into these cases.

anywhere in a large facility would “drift” to expose workers at another place in the building. *See Linkus*, 190 Md. App. at 224-25, 988 A.2d at 516. Maryland judges have done their best since then to try to figure out how much frequency, regularity, and proximity is required to send a case to the jury.

B. Lohrmann Is Not Sufficient for Today’s Low-Dose Litigation

Lohrmann had value at the time and in the context in which it was adopted. Today, however, it no longer works as a meaningful brake on speculative causation testimony. The Court should use this case to correct that situation.

1. The Nature of Asbestos Litigation Has Changed Dramatically Due to the Incorporation of Low-Dose Exposures

Today’s litigation is very different from the old insulator steel plant cases. Faced with the bankruptcy of almost all insulation manufacturers, plaintiffs today sue a much larger range of defendants – over 10,000 at last count – essentially any company whose products or buildings contained any amount of asbestos in any form.¹¹ Some of the cases presented to courts today beg credulity – dental technicians who worked with “dental tape”; shipyard supervisors (exposed to enormous amounts of insulation) who “walked by” an engine under repair and thus allegedly were exposed to gaskets; warehouse or auto shop employees who did nothing more than handle brake pads; salesmen who merely walked through a repair area or manufacturing setting; and backyard mechanics performing only a single brake job.¹² These are the kinds of cases filling today’s dockets.

2. Lohrmann Cannot Account for Dose and Potency, Both Critical Elements in Low-Dose Cases

The science regarding low-dose cases is very different than the science this Court faced in 1992 when the *Balbos* Court adopted the *Lohrmann* test in the context of large-

¹¹ In 1980, there were about 300 defendants in asbestos litigation, but today there are more than 10,000. The expansion to so many defendants, most of whom make or used products bound in resins and highly unlikely to cause any meaningful exposure, is largely attributable to the *any exposure* theory. *See* Mark A. Behrens, *What’s New in Asbestos Litigation?*, 28 Rev. Litig. 501 (2009).

¹² *See* Behrens & Anderson, *supra*, at 487-88 nn.50-51.

scale insulation exposures. Unlike insulation, many newer cases involve products that were encapsulated or otherwise sealed or bound; it is very difficult to generate measurable asbestos exposures from them. Studies have shown, for instance, that the exposures received from replacing automotive brakes would have amounted to less than half of today's OSHA standard.¹³ Likewise, replacing gaskets on engines results in, at most, only very minor exposures, in part because of the limited time needed to remove a gasket.¹⁴ These studies stand in sharp contrast to those of the old "dusty trades" – mining work, insulation application and removal, asbestos factory and shipyard work – that produced the vast majority of asbestos disease. Exposures from that work were often orders of magnitude higher than today's OSHA standard and far higher than the exposures alleged in most of today's litigation.

The most critical distinction between these two worlds of asbestos cases is the factor of dose. Dose arises from consistent and sustained exposures of a sufficient intensity and quantity over time. *See Eaton, supra*, at 10. Unless the exposure is not only regular, frequent, and proximate, but also of sufficient intensity and duration to produce a significant dose, it cannot be considered causative without engaging in pure speculation. *See id.* at 39. In toxicological terms, dose is "the single most important factor to consider in evaluating whether an alleged exposure caused a specific adverse effect." *Id.* at 11. An insulator, or person working the bulk of his career close to an insulator, may well receive a significant dose from years of such work. A worker who removes a few gaskets a year, however, regardless of how regular, frequent, and proximate that work is, will receive at most a dose that is inconsequential in toxicological terms and could not be distinguished from mere background exposures. The exposed person's lungs would be

¹³ *See* Dennis Paustenbach et al., *An Evaluation of the Historical Exposures of Mechanics to Asbestos in Brake Dust*, 18 *Applied Occupational & Env'tl. Hygiene* 786 (2003); Amy K. Madl et al., *Airborne Asbestos Concentrations Associated with Heavy Equipment Brake Removal*, 53 *Annals Occupational Hygiene* 839 (2009).

¹⁴ *See* Amy K. Madl et al., *Exposure to Airborne Asbestos During Removal and Installation of Gaskets and Packings: A Review of Published and Unpublished Studies*, 10 *J. Toxicology & Env'tl. Health* 259 (2007).

indistinguishable from the lungs of persons with no workplace asbestos exposure. The dose of the insulation worker is the primary feature in causing that person's disease. Based on current scientific evidence (e.g., the epidemiology studies showing that most cohorts of chrysotile-exposed workers experience no increased mesothelioma, and none at all among low-level exposed populations), the dose of the gasket worker would almost certainly not cause disease at all. The *Lohrmann/Balbos* test is incapable of distinguishing between these two workers and would send both cases to the jury.

Maryland cases have treated very low intensity exposures, like cutting of rope seal (*Linkus*) or scraping of wet dryer felt (*Scapa Dryer Fabrics*), the same as high intensity insulation cases. Under the approach used in *Farrar* by the Court of Special Appeals, cutting a few small asbestos ropes or removing a few gaskets would send a defendant to trial just as quickly and easily in Maryland as the old steel mill insulators and crafts. These low-dose, low-potency cases survive appellate review only because the courts applied the *Lohrmann* approach formulaically, and did not consider the vastly different science of causation involved in the type and use of these products. This is not an acceptable way to handle causation.

Dose is not the only element missing from *Lohrmann/Balbos* – the test does not include any assessment of product potency difference. Unlike insulation, the vast majority of low-exposure products are made of a different material – chrysotile. Chrysotile has different mineral ingredients, is made up of different chemicals, and has a different physical structure that makes it much less rigid. As a result, the human body readily breaks down and removes many chrysotile fibers.¹⁵ The half life of chrysotile is mere months (meaning half of any given dose is gone within a few months), whereas the

¹⁵ See Report on the Peer Consultation Workshop to Discuss a Proposed Protocol to Assess Asbestos-Related Risk, U.S. EPA, May 30, 2003, at viii (“The panelists unanimously agreed that the available epidemiology studies provide compelling evidence that the carcinogenic potency of amphibole fibers is two orders of magnitude greater than that for chrysotile fibers.”); Christine Rake et al., *Occupational, Domestic and Environmental Mesothelioma Risks in the British Population: A Case Control Study*, 100 *Brit. J. Cancer* 1175, 1182 (2009) (“The mesothelioma risk caused by amosite (brown asbestos) is two orders of magnitude greater than that by chrysotile (white asbestos)”).

amphiboles in insulation remain in the body for many years. Only a few studies have found mesothelioma in workers exposed to chrysotile, and those studies identified only small numbers of such cases and associated with very high exposures – up to 100 f/cc. No epidemiology study has ever demonstrated an increased incidence of mesothelioma from the kind of extremely low doses of chrysotile produced by most of the products involved in the current asbestos litigation. *See CLJ Dixon* Brief at 23-26.

3. **The Lohrmann Standard Needs Modification to Incorporate a True Dose and Causation Assessment and Bring Maryland Cases Back Into Line with Good Scientific Practices**

Somewhere in the course of Maryland asbestos litigation the distinctions so critical to low-dose science disappeared and have not played a role in determining which cases go to trial.¹⁶ They are simply not part of the analysis under Maryland law today. Any reasonable assessment of a far less potent material would require a far greater dose of that material before attributing causation to it. *Farrar* illustrates how plaintiffs' experts do exactly the opposite – the experts often agree that chrysotile is less potent, but then make no attempt to establish or compare doses and instead ascribe the same causation to minimal chrysotile fibers on Mr. Hentgen's clothing that they do to the extensive amount of insulation fibers that must have been on the same clothing. Rationally, the reverse is true – Ms. Farrar's dose of chrysotile from Mr. Hentgen's clothing should have been much *higher* (not lower) than the insulation fibers to play any role in her disease.

In fact, applied literally, the *Lorhmann/Balbos* standard would require courts to send mere *background* cases to the jury (assuming there were anyone to sue for background exposures). Virtually every person in the country has been exposed to background asbestos fibers (mostly chrysotile) every day, all day, for an entire lifetime, and in close proximity. There is no exposure that has been more regular, frequent, and

¹⁶ The original *Lohrman/Balbos* standard also included a *fourth* requirement – the nature of the product. If courts today were effectively including this requirement, it should compel the dismissal of many cases involving products that contained bound asbestos, lower potency asbestos, or minimal asbestos exposure.

proximate than background exposures. Yet even plaintiff experts agree these exposures do not contribute to disease, presumably because the composite *dose* from those exposures is so low. This point illustrates how *Lohrmann* becomes unworkable in the context of low dose, low intensity, and low potency exposures. Many regular and frequent workplace or product exposures nevertheless produce only a very low lifetime *dose* of fibers. Since dose is the critical element, and mere exposure is not (no matter how frequent or regular), Maryland law today is out of line with the most fundamental principles of toxicology by using *Lorhmann/Balbos* to send frequent, regular and proximate *but low dose and low potency* exposures to the jury.

Principles of potency and intensity must be reintroduced to eliminate the unfairness of, as well as the unscientific basis for, sending minimal dose defendants to trial over and over again. The Court clearly understands the need to keep out some alleged exposure cases while allowing others to proceed – it attempted to do exactly this in *Reiter v. Pneumo Abex, LLC*, 417 Md. 57, 8 A.3d 725 (2010), by parsing the individual exposures of several plaintiffs, yet was constrained by the rigid *Lorhmann/Balbos* approach.

Other courts have also recognized and addressed the inadequacies of the *Lohrmann* approach in today's litigation. The Texas Supreme Court recognized the limitations of *Lohrmann* in *Borg-Warner Corp. v. Flores* 232 S.W.3d 765 (Tex. 2007). The court adopted a "*Lohrmann-plus*" standard, holding that implicit in the *Lohrmann* test "must be a requirement that asbestos fibers were released in an amount sufficient to cause" the plaintiff's disease. *Id.* at 772. The court explained, "proof of mere frequency, regularity, and proximity is necessary but not sufficient, as it provides none of the quantitative information necessary to support causation..." *Id.* The plaintiff in *Borg-Warner* was a lifetime automotive mechanic, who handled brake pads on a regular, frequent, and proximate basis. The Texas justices recognized that his dose and its potential to cause disease could in fact be very low, given the nature of those bonded products. *See id.* at 771.

The Pennsylvania Supreme Court in *Betz v. Pneumo Abex, LLC*, 44 A.3d 27, 56 (Pa. 2012), implicitly added the elements of dose and potency to that state's *Lohrmann*-based approach by criticizing Dr. Maddox for failing to apply any of the three factors he testified were important – potency, intensity and duration.

Virginia is not a *Lohrmann* state, but the Virginia Supreme Court similarly recognized this problem by requiring the experts in *Ford Motor Co. v. Boomer*, 736 S.E.2d 724 (Va. 2013), to “opine as to what level of exposure is sufficient to cause mesothelioma, and whether the levels of exposure at issue in this case were sufficient.” *Id.* at 733.

These and other courts have begun to catch up with the vastly spreading net of asbestos litigation and put rules in place – much as this Court did in 1992 in *Balbos* – to change an inappropriately low bar to causation that only encourages the filing of frivolous cases. The Maryland *Lohrmann/Balbos* standard should be modified to require plaintiff experts to prove not just a regular, frequent, and proximate exposure, but one that also is of sufficient intensity and overall lifetime dose, taking into account the type of product and potency of fiber involved, to produce asbestos disease. The “yardstick” against which those exposures must be compared is competent epidemiology studies of similarly exposed cohorts – including to the same fiber type – that show such exposures are capable of causing mesothelioma or other asbestos diseases at those exposure levels. This is not a novel or difficult test – it is the same standard that is applied in toxic tort litigation all over the country.

Asbestos cases have gone far off the track in recent years and with special rules and practices applied (like *Lohrmann* and the “any exposure above background” approach) that would astound ordinary toxic tort lawyers. This Court should join federal and state supreme courts that have acted recently to correct this abusive situation.

III. PLAINTIFF EXPERTS SHOULD BE REQUIRED TO CONDUCT CAUSATION ASSESSMENTS

In addition to the problem with *Lohrmann/Balbos*, this case illustrates another serious problem with asbestos litigation in Maryland – the state’s judges are struggling to determine the degree of evidence necessary to allow a case to go to trial under *Lohrmann/Balbos* because plaintiffs’ experts have abandoned any efforts to help judges distinguish between causative and non-causative exposures. Reading these opinions, it is apparent that judges are being forced to perform what is essentially a scientific assessment of causation. Judges have been put in this position because *the plaintiffs’ experts are not doing their jobs correctly*. They use shortcuts – “visible dust,” “any exposure above background,” and an unquantified “increased risk” – to avoid the real work required to prove causation in a low dose case. Because these experts have not performed a proper causation assessment, the *Lohrmann/Balbos* standard (coupled with the *any exposure* theory) has turned into a box that traps judges in the role of determining, in the first instance, how much exposure is enough, rather than reviewing expert determinations on this point. This case provides an opportunity to fix this situation.

A. Recent Maryland Opinions Reflect How Plaintiff Experts Have Left the Role of Establishing Causation to Judges and Juries

Several opinions issued by the Court of Special Appeals or this Court over the last decade illustrate how the task of determining causation has shifted from the experts to the judge and jury. Early on, plaintiff experts themselves were eliminating certain workplace exposures, which allowed the courts at least to review those determinations under *Lohrmann* rather than perform this task themselves. In *Lohrmann*, for instance, even the plaintiff expert agreed that an exposure of thirty days in these steel mills was “insignificant as a causal factor.” 782 F.2d at 1162-63. Even so, trial and appellate judges wrestled with how to apply the *Lohrmann* standard in specific fact situations. The standard only requires “frequency, regularity, and proximity” without any definitions of what those terms really mean in a workplace. Thus, in the *Lorhmann* case itself, the

federal court had to determine whether exposure to insulation on ten or fifteen occasions, over a one- to eight-hour duration, was sufficient; the held it was *not*. See *id.* at 1163.

The pattern of the experts abandoning their role of establishing causation expanded in *Balbos*, a case involving exposures to asbestos fibers in manufacturing cement pipes and from ripping out insulation. Plaintiff's medical expert testified that "all" exposures were causative (the now infamous *any exposure* theory), thus giving the court no help in determining whether the plaintiff's specific work activity was sufficient to cause disease. This Court was forced to engage in a detailed analysis of the size of the facilities and likelihood of exposure based on factors such as distance and frequency – a task the experts themselves should have performed – to decide whether the two plaintiffs in *Balbos* met the *Lohrmann* standard and could go to the jury.

In three more recent cases, the exercise has grown ever more attenuated as the alleged exposure scenarios became more and more miniscule. In *Reiter v. Pneumo Abex, LLC*, 417 Md. 57, 8 A.3d 725 (2010), involving a series of plaintiffs who alleged exposures merely from being around brakes on cranes twenty-five feet over their heads, this Court again faced a total absence of any testimony from plaintiff experts distinguishing between causative and non-causative exposures. Instead, the experts relied on the *any exposure* theory and the *Lohrmann* approach to testify that exposures to crane brake dust of any amount were enough. As a result, the Court had to perform the job of comparing which workers were close enough, for long enough, and on enough occasions, to support causation. The Court unfortunately had no real science to work with (because the experts gave them none) and instead had to rely on its own notions of how close to an overhead crane brake should someone be to anticipate asbestos disease (e.g., is "in the vicinity" enough?). In *Scapa Dryer Fabrics, Inc. v. Saville*, 418 Md. 496, 16 A.3d 159 (2011), this Court once again had to determine whether plaintiff's limited work with dryer felts was enough (under *Lohrmann*) to suffice. Plaintiff's experts claimed the smallest amount of contact was sufficient, so it was up to the Court to try to distinguish causative from non-causative exposures.

The opinion in *John Crane, Inc. v. Linkus*, 190 Md. App. 217, 988 A.2d 511 (2010), perhaps represents the ultimate in experts abdicating their role, and in this instance the court did likewise. Working with expert testimony that “dust” in any amount from an asbestos-containing product causes disease, the Court of Special Appeals agreed that the mere cutting of small ropes was sufficient to support causation. This court accepted *any exposure* testimony at face value, and itself basically abandoned any attempt to distinguish causative and non-causative exposures. The court held that any product emitting asbestos-containing dust would send a case to the jury, with or without expert industrial hygiene testimony that the dust had enough asbestos in it to be truly hazardous.¹⁷

This string of decisions does not utilize scientific principles or reflect how scientists do their work. It exists only because experts abdicated their roles by relying on the *any exposure* theory and shortcuts such as “dust” or “more than background” assertions.

B. The Experts Should Perform the Causation Analysis and Distinguish Causative Exposures from Inconsequential Ones

Science requires more than what these experts are doing. Competent industrial hygiene principles require an assessment of the dose of an individual rather than guesswork on what might be in dust. *See Eaton, supra*, at 39 (“The individual must have been exposed to a sufficient amount of the substance in question to elicit the health effect in question.”). For forty years and more, industrial hygienists have conducted such measurements, of thousands of occupations, and determined whether those occupations satisfied the existing medical and regulatory standards for safety. The entire framework

¹⁷ Lacking any real scientific basis for distinguishing causative exposures from those that are not, the courts usually end up resorting to a comparison of exposures in one case to another. The courts measure each new case against prior case exposure scenarios and declare them sufficiently “like” or “unlike” those prior cases to support a trial or verdict. *See Scapa Dryer Fabrics*, 418 Md. at 510, 16 A.3d at 167; *Reiter*, 417 Md. at 72, 8 A.3d at 734; *Linkus*, 190 Md. App. at 240-41, 988 A.2d at 525. Nowhere in this series of cases have the courts actually examined whether any of these exposures are known to cause asbestos disease.

of the federal workplace safety system, managed by OSHA, is founded on this approach. In 1972, OSHA passed the first asbestos regulations, under which exposures were believed to be “safe” if they were below the regulatory standard of the time, five fibers per cubic centimeter, on an 8-hour daily average (later dropped to two fibers per cc in 1976). *See* 37 Fed. Reg. 11318 (June 7, 1972). The literature is filled with articles in which researchers published the results of these measurements, including regarding asbestos-related work, in part so that others can then rely on them to determine whether comparable workplace situations represent acceptable exposures. OSHA has never deemed “any exposure” to asbestos to be unsafe, and it does not require, even today, a zero exposure or “below ambient” atmosphere.¹⁸ *See* 29 C.F.R. § 1910.1001(c) (establishing 0.1 f/cc 8-hour time-weighted average limit).

Plaintiff experts try to avoid their obligation to assess the dose by claiming that no one measured the plaintiff’s atmosphere at the time of his alleged exposure, so it is impossible today to know what his dose was. This specious argument unfortunately carried the day in the *Linkus* case. *See* 190 Md. App. at 226-28, 988 A.2d at 517-18. It is, of course, highly unlikely that any individual plaintiff’s exposure would have been the subject of contemporaneous measurement – employers are not required, even under OSHA, to constantly monitor all exposures or all employees but only situations where the exposures might be anticipated to exceed the OSHA standard of the time. Most of the exposures alleged in today’s litigation do not exceed even today’s standard much less the higher standards of earlier decades. Today, researchers in industrial hygiene and other disciplines routinely deal with this situation by reviewing comparable exposure situations in studies of similar occupations or work activities, or by performing or reviewing simulations or recreations of those exposure scenarios. As one illustration, literature reviews and actual exposure studies of vehicle mechanics working with asbestos parts document an exposure range around 0.04 fibers/cc (less than half of today’s OSHA

¹⁸ Today’s OSHA standard, enacted in 1994, is 0.1 fibers per cc 8-hr TWA, meaning a worker today can be exposed to up to this amount on a daily, 8-hour average, over the course of his or her 40-year working career without anticipating asbestos disease.

standard) during actual brake-related work.¹⁹ It is unlikely a brake mechanic would greatly exceed that amount of exposure, even if no one measured that particular mechanic's atmosphere at the time. If the plaintiff provides enough information, an industrial hygienist or toxicologist can recreate the likely range of dose by multiplying the number of brake jobs by the likely exposure and computing the lifetime dose from that activity.

What would scientists do with such a number once the industrial hygienists derive it? They would compare it to occupations and workplace activities known to cause asbestos disease to see if the dose is comparable. *See Eaton, supra*, at 2 (fundamental concepts of toxicology and epidemiology continue to serve as the foundation for establishing causation in toxic tort claims); David E. Bernstein, *Getting to Causation in Toxic Tort Cases*, 74 *Brook. L. Rev.* 51 (2008) ("The plaintiff must initially show that the *level of the toxin* he was exposed to *can* cause the illness he contracted. Here, epidemiology ... becomes vitally important, as many courts have emphasized.") (emphasis in original, citations omitted). It would not be appropriate to use amphibole and insulation epidemiology studies because brakes contain only chrysotile, a very different and less potent substance. The brake mechanic's dose would be compared to studies of chrysotile-exposed cohorts. Those cohorts show disease only at extremely high exposures, and in very different occupations (primarily mining and textile manufacturing plants). *See CLJ Dixon* Brief at 14-15. Other studies looking specifically at brake mechanics have never found an increase in disease from those jobs, even performed over a lifetime – they have the same degree of mesothelioma as farmers, office workers, and others who have no known contact with asbestos in the workplace. *See id.* at 15-17 (discussion of epidemiology studies). Thus, a scientific causation assessment would begin with a dose assessment; take into account the particular product and potency of the fiber type; and identify similarly exposed populations to see if disease occurs at an

¹⁹ *See Paustenbach, supra* n.14.

increased rate in that population. Plaintiff experts in *Farrar*, and in fact in all of these cases, failed to perform any of these three steps.

If the assessment described above is performed properly, plaintiffs whose workplace exposures may well be responsible for their disease will have their day in court. But those who are in litigation only because *any exposure* experts have tied them to infinite causation would not be permitted to congest the Maryland court system and burden American industry with frivolous asbestos cases. The *Lohrmann/Balbos* standard is not enough by itself to protect against this outcome, particularly when coupled with *any exposure* testimony. Under the *Lohrmann/Balbos* approach, in the new low-dose cases, Maryland judges are trying to decide whether ten exposures a year is “frequent” or regular enough, or whether a worker has to be within ten feet versus twenty feet for the exposure to be proximate enough. *This is the job of experts*. In all sorts of fields, in all sorts of published articles, and in all sorts of exposure circumstances, experts regularly identify the dose, potency, and likelihood of disease.

C. The Experts Should Not Be Permitted to Resort to Shortcuts Like Mere “Dust” in the Workplace

Competent expert testimony also does not resort to inadequate substitutes for a true exposure and causation assessment like seeing “visible dust” or identifying exposures “above ambient.” These shortcuts are neither logical nor scientific and should no longer form the basis for appellate court opinions in this state. First and foremost is the notion that mere “dust” is enough to cause asbestos disease. Several recent opinions have held that as long as someone saw “dust” from the plaintiff’s work activity, that is enough exposure to support a case. *See, e.g., Linkus*, 190 Md. App. at 235-38, 988 A.2d at 522-24; *Scapa Dryer Fabrics*, 418 Md. 496, 506-07, 16 A.3d 159, 165; *Farrar*, 207 Md. App. at 551-52, 53 A.3d at 443. The experts claim that any such “dust” would contain thousands or millions of asbestos fibers, without citing to any comparable measurements of the relevant work activity to support that claim.

The Court should be asking some hard questions about this testimony and not merely accepting these experts’ substitution of unquantified “dust” in lieu of a dose

assessment. The notion of mere “dust” is so vague that almost any testimony about any product would support causation under such a standard. Sweeping a house causes dust. Combing a dog causes dust. Pouring Cheerios causes dust, as evident when doing so under a bright light. When a plaintiff or co-worker says “I saw dust,” that testimony is meaningless without some standards placed on it. Removing a gasket might produce miniscule dust, but that does not make gasket work dangerous. The minimal dust produced by removing a gasket (or cutting a rope, as in *Linkus*) is nothing like the “dusty trades” work in mining, asbestos manufacturing and shipyards where workers could not see each other for the dust in the room.

In addition, most “dust” in a workplace is just that – dust. Dust by itself does not cause asbestos disease. Most dust, including dust in ordinary air, has some amount of hazardous material in it, including ambient asbestos, and still is not considered hazardous by OSHA or anyone else. To be hazardous, dust must have hazardous substances in it *at a level that would cause harm*. So the question is not whether someone claims there was “dust” produced, or whether there was some asbestos in the dust, but whether there was sufficient asbestos in the dust at least to exceed a health standard, and more appropriately for a causation opinion, whether there was enough to produce asbestos disease.²⁰ An opinion on this point is not possible without a competent industrial hygiene assessment, using similar studies of similar fiber types, activities, intensities, and durations. The ultimate question is whether the lifetime dose associated with a particular product and fiber type could serve, on its own, as a cause of the disease. If not, that product or exposure cannot serve as a substantial factor in causation. *See Balbos*, 326 Md. at 208,

²⁰ The experts’ claim that visible dust would have thousands or millions of asbestos fibers in it, and thus must be harmful, is also useless scientifically for several reasons. The quantity of fibers needs to be compared to a health study to answer the question whether that level causes disease. Running out large-sounding numbers like this is a scare tactic – ordinary people with no direct asbestos exposures have millions or billions of background fibers in their lungs, which is not associated with asbestos disease. In addition, the experts fail to take account of vast differences in asbestos in dust based on the amount of asbestos in the original product, the type of activity, other sources of dust, ventilation, etc.

604 A.2d at 459 (citing W. Page Keeton et al., *Prosser & Keeton on the Law of Torts* § 41 at 266 (5th ed. 1984)). Claiming a person was exposed to unquantified and ambiguous “dust” comes nowhere near answering that question.

Likewise, the shortcut notion that even the smallest exposure “increases the risk” of disease is not a substitute for a true causation assessment. That notion may or may not be correct – it is essentially untestable. Either way, experts in asbestos litigation should not be allowed to premise a causation opinion on “increased risk.” Risk alone is not a cause of disease – it is a regulatory term that is not useful in determining courtroom causation. *See Eaton, supra*, at 34-40 (discussing differences between regulatory “risk” determinations and courtroom causation). In addition, these experts utterly fail to quantify the supposed increased risk, meaning that they will declare a risk of the smallest proportions, approaching zero, to suffice for causation. In other contexts, courts have rejected this miniscule “risk” approach as insufficient, often because the risks identified are so low as to be unimportant. *See Mann v. CSX Transp. Corp.*, 2009 WL 3766056, *5 (N.D. Ohio Nov. 10, 2009), *aff’d*, 656 F.3d 359 (6th Cir. 2011). As one court stated in an asbestos case:

The EPA’s range for acceptable risk of 10⁻⁴ [one in ten thousand] to 10⁻⁶ [one in a million] can also be expressed as 0.01 percent to 0.0001 percent. As a frame of reference, the risk of being struck by lightning is 0.002 percent; the risk of dying from a bicycle accident is 0.019 percent; fire 0.084 percent; drowning 0.11 percent; food poisoning 0.12 percent; homicide 0.45 percent; car accident 1 percent; alcohol 1.1 percent; stroke 14 percent; heart disease 18 percent.

In re W.R. Grace & Co., 355 B.R. 462, 492 (Bankr. D. Del. 2006) (citation omitted), *appeal denied*, 2007 WL 1074094 (D. Del. Mar. 26, 2007). The Court should reject “risk”-based opinions as a substitute for asbestos causation testimony.²¹

²¹ For this reason, *Amici* believe the Court of Special Appeals in *Dixon* did not need to bring the concept of “risk” into a causation assessment in an asbestos or toxic tort case. Causation is determined by the steps outlined above. If “increased risk” is introduced, the courts will end up where they are under *Lohrmann* today, trying to decide how much of an increased risk is sufficient for causation.

D. Maryland Law Needs to Restore Judges to the Role of Evaluating Expert Causation Testimony and Not Performing that Role for the Experts

Until *Dixon*, Maryland seemed to be moving toward a standard under which a case would go to a jury if the plaintiff worked with any kind of asbestos product on more than a few occasions. How many occasions is something the courts themselves were being forced to determine. This is why the *Dixon* Court of Special Appeals opinion appeared as a breath of fresh air. That panel recognized how cases like *Dixon* have gone off track and tried to create some more realistic causation rules to keep at least some restraints on asbestos litigation. But in *Farrar*, a different panel totally dismissed *Dixon*, on the ground that the experts in *Farrar* did not use the word “substantial” in their testimony and thus avoided taking the legal issue from the jury (as if that is all that *Dixon* held). See 207 Md. App. at 558 n.5, 53 A.3d at 446 n.5. According to the *Farrar* court, as long as the experts utter the magic word “contribute” (and do not say the forbidden word “substantial”), every case will go to a jury, no matter how small the exposures.

The Maryland courts need more than this – they need a system based on science. Unless this Court acts (approving *Dixon*, rejecting *Farrar*, and setting forth better rules), plaintiffs will henceforth identify any asbestos-containing product and will claim they saw “dust” from working with it, and their experts will simply claim any visible dust is above background and all exposures above background cause disease. No scientific literature supports this approach – it is purely a litigation construct. And it is a construct that forces judges to make the hard decisions as to which exposures were inconsequential.

If the Court will compel plaintiffs’ experts to do their jobs correctly, Maryland trial and appellate judges can then step back into their appropriate role as *Frye* gatekeepers and evidence evaluators. Judges can review the assessments of these experts to see if those opinions would be generally acceptable under *Frye*, reliable enough for testimony under Maryland’s expert evidentiary rules, and competent enough to support real (i.e., not speculative) causation. This is not a difficult standard – experts in all other fields of tort law are required to do this. The trial judges of Maryland need more help

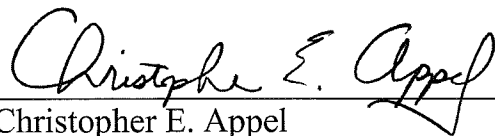
than *Lohrmann/Balbos* can give them, and they need more help than these experts are currently giving them. It is critical that Maryland cases begin to rely on actual science, as presented by competent expert testimony, rather than the shortcuts used by experts who refuse to do the hard work their jobs require.

Amici understand the difficulty of correcting course in the path of a long-running litigation like asbestos. But this Court put such a correction in place in *Balbos* years ago, and Maryland's sister courts in Pennsylvania and Virginia have recently corrected abuses in asbestos litigation by requiring a competent dose and causation assessment. Maryland should do the same.

CONCLUSION

Amici asked the Court in the pending *Dixon* appeal to take the first step in fixing serious problems with asbestos litigation in Maryland – eliminating the *any exposure* theory. In *Farrar*, the Court should go further in elucidating the kind of evidence these experts must present to survive *Frye* scrutiny and a sufficiency of evidence review. A modification of the *Lohrmann/Balbos* standard, coupled with a return to real causation testimony and evidence by plaintiff experts, will contribute greatly to restoring a scientific basis and proper approach to asbestos litigation in Maryland.

Respectfully submitted,



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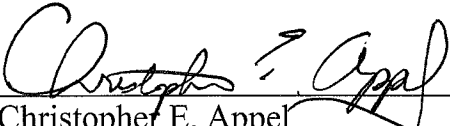
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Dated: February 27, 2013

STATEMENT OF RULE 8-504 COMPLIANCE

Pursuant to Rule 8-504(a)(8), I certify that the foregoing brief is in Times New Roman font with a 13-point typeface.


Christopher E. Appel

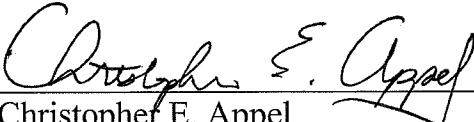
CERTIFICATE OF SERVICE

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