

IN THE SUPREME COURT OF OHIO

MARK SCHWARTZ, *et al.*, : Supreme Court Case No. 2016-1372
: :
Appellees, : On Appeal from the
: Court of Appeals,
Cuyahoga County : Eighth Appellate District
: :
v. : Court of Appeals No. CA-15-103377
: :
HONEYWELL INTERNATIONAL, INC., : :
: :
Appellant. :

**AMICUS CURIAE BRIEF OF COALITION FOR LITIGATION JUSTICE, INC.
IN SUPPORT OF APPELLANT HONEYWELL INTERNATIONAL, INC.**

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INTEREST OF AMICUS CURIAE

The Coalition for Litigation Justice, Inc. (“Coalition”) is a nonprofit association formed by insurers in 2000 to address and improve the litigation environment for asbestos and other toxic tort claims.¹ The Coalition files *amicus curiae* briefs in important cases that may have a significant impact on the asbestos litigation environment. The Coalition has a substantial interest in ensuring that Ohio’s asbestos litigation environment is fair and reflects sound science and public policy. The Coalition supports Honeywell International, Inc.’s position and requests reversal of the opinions below admitting the discredited and unscientific *cumulative exposure* testimony presented by plaintiff’s experts.

STATEMENT OF FACTS

The Coalition adopts the Honeywell’s statement of facts.

INTRODUCTION

The appellate court allowed plaintiff’s experts to testify as to causation without identifying or demonstrating that decedent Ms. Schwartz had a sufficient amount of exposure to cause the disease in question. Dr. Bedrossian, like other testifying plaintiff experts in asbestos litigation, calls this theory the “cumulative exposure” approach. That approach is founded on the notion that even very small exposures to asbestos accumulate with other exposures in the lung and thus must be considered part of the overall cause – much like a match thrown into a forest

¹ The Coalition includes Century Indemnity Company; San Francisco Reinsurance Company; Great American Insurance Company; Resolute Management, Inc., a third-party administrator for numerous insurers; and TIG Insurance Company. The Coalition, often in conjunction with other *amici*, has filed *amicus* briefs in similar appeals in many jurisdictions. Those courts include the highest courts of Virginia, Pennsylvania (three times), Texas (twice), New York, Michigan, Maryland, Georgia, and California; multiple intermediate appellate courts in those states; and the U.S. Court of Appeals for the Ninth Circuit.

fire “contributes” to the fire, or a bucket of water thrown into the ocean “contributes” to the ocean. This testimony creates a form of strict liability in asbestos litigation in which defendants are held liable for mere exposure to their products.

The forest fire and ocean analogies, however – which plaintiff experts like Dr. Bedrossian often use – quite nicely illustrate how frail and unreliable the cumulative exposure theory really is. The “cumulative exposure” approach to causation is not based on rational science or any scientifically reliable methodology. It is instead a highly speculative litigation tool used by testifying plaintiff experts to capture even trivial amounts of exposure from any particular product and make that exposure the focus of a trial. This “every exposure counts” approach has by and large been *rejected* by a great many courts, including the Sixth Circuit Court of Appeals repeatedly. The Coalition urges the Court to review these well-reasoned opinions, described in detail in Section II below. Those opinions highlight the errors made by the Eighth District court in its analysis. The decision below should be reversed.

This case in particular illustrates the unbalanced effect of the cumulative exposure theory. Plaintiff’s experts claim (falsely) that they are not contending that each exposure is a cause (knowing such testimony makes them appear unreasonable). And yet in this case only five or six brake jobs were deemed sufficient, even though Ms. Schwartz apparently was never around her father when he was conducting these repairs. Without question, Dr. Bedrossian would have considered even a single brake job enough, despite the existence of more than twenty epidemiology studies that have never documented increased mesothelioma even in lifelong vehicle mechanics.

As the Appellant’s brief below demonstrates, the cumulative exposure testimony permitted by the Eighth District court ignores the scientific principle of dose and misuses the

notion of cumulative exposure in a way neither real life nor science could support. The “cumulative exposure” label these experts are currently using for this “everything is causative” testimony – founded on the refusal to estimate the actual dose – is in fact nothing more than an attempt to rename the old and widely rejected “each and every exposure” theory to avoid yet more rejections. The Eighth District court fell into the trap of accepting the experts’ statements at face value, without any analysis of the actual underlying science – the classic *ipse dixit* error criticized so pointedly in the United States Supreme Court’s *Joiner v. General Elec. Co.*, 522 U.S. 136, 146-47 (1997), decision.

Asbestos litigation must have a stronger foundation than this, or the litigation will become grossly unbalanced, favoring a featherweight burden of proof for plaintiffs. The Coalition supports reversal to ensure that asbestos litigation in Ohio remains grounded in sound science and not speculation.

ARGUMENT

I. “Cumulative Exposure” Testimony Improperly Assumes All Exposures Are Causative In Lieu of Demonstrating an Actual Causative Dose.

The scientific principle of “dose” is the foundation of all modern toxicology and toxic tort case law. Yet in asbestos litigation these experts ignore any obligation to demonstrate anything other than an exposure to any amount of visible dust. A methodology like this does not provide helpful testimony for the jury – the jury is left to figure out whether a workplace exposure is sufficiently consequential to be a “substantial factor” under Ohio law, with no guidance from these experts. Nor could such testimony survive in other toxic tort contexts.²

² Almost all courts require a competent dose assessment to prove causation in non-asbestos contexts. *See, e.g., Nelson v. Tennessee Gas Pipeline Co.*, 243 F.3d 244, 252-53 (6th Cir. 2001) (groundwater contamination); *Pluck v. BP Oil Pipeline Co.*, 640 F.3d 671 (6th Cir. 2011) (Footnote continued on next page)

This Court should reject the cumulative exposure/every exposure approach, as have the Sixth and Ninth Circuit federal courts of appeal and appellate and trial courts in Texas, Georgia, Virginia, Florida, Illinois, Louisiana, Pennsylvania, Utah, Washington, Wisconsin, and elsewhere.

A. A Scientific Quantification of Dose Is Essential in Any Toxic Tort Case.

The most important step in any toxic tort case is to distinguish between inconsequential exposures that are not likely the cause of any disease, and those that are extensive enough to cause actual harm. “[W]here a plaintiff relies on proof of exposure to establish that a product was a substantial factor in causing injury, the plaintiff must show a high enough level of exposure that an inference that the asbestos was a substantial factor in the injury is more than conjectural.” *Moeller v. Garlock Sealing Tech., Inc.*, 660 F.3d 950, 955 (6th Cir. 2011). Here, however, the experts’ testimony instead lumps all exposures into one basket – cumulative and causative. As demonstrated so well by the facts here – a case involving take-home exposure following just six brake jobs – no asbestos exposure is too small for Drs. Bedrossian and Guth to speculate that it is causative.

Dr. Bedrossian and other “cumulative exposure” experts are ignoring the most important principle of toxicology – the dose makes the poison. Or put another way, no substance is toxic to the human body unless the dose is sufficient.³ The fundamental *dose* requirement is set forth in the Federal Judicial Center’s *Reference Manual on Scientific Evidence, Reference Guide on Toxicology* 403 (2d ed. 2000), and even more concretely in one of the best medical descriptions

(benzene); *Wintz by and through Wintz v. Northrop Corp.*, 110 F.3d 508, 513 (7th Cir. 1997) (bromide in photographic materials); *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1106 (8th Cir. 1996) (formaldehyde); *Allen v. Pa. Eng’g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996) (ethylene oxide in hospital).

of the application of toxicology to litigation, by Dr. David Eaton of the University of Washington. As Professor Eaton's article explains: "***Dose is the single most important factor*** to consider in evaluating whether an alleged exposure caused a specific adverse effect."⁴

Asbestos, like any toxin, requires some level of overall dose to produce disease. The human body has many mechanisms for defending against minor exposures, both for asbestos and for other toxins.⁵ The lung can defend itself against a whole array of small, daily exposures that at much higher levels might well cause harm. Disease results when those exposures reach a level that overwhelms our defenses, called the "threshold" point. Aspirin, alcohol, sunlight, even known poisons like arsenic are only poisonous if the dose is high enough to make them so. At lower doses, they are either harmless or, in some instances, beneficial.

As Professor Eaton notes, this dose principle holds true for carcinogens like asbestos just as much as it does for any other toxin:

Most chemicals that have been identified to have "cancer-causing" potential (carcinogens) do so only ***following long-term, repeated exposure for many years. Single exposures or even repeated exposures for relatively short periods of time (e.g., weeks or months) generally have little effect*** on the risk of cancer, unless the exposure was remarkably high and associated with other toxic effects.

³ See Federal Judicial Center, *Reference Manual on Scientific Evidence* 403 (3d ed. 2011) (the "fundamental tenet" of toxicology).

⁴ David L. Eaton, *Scientific Judgment and Toxic Torts – A Primer In Toxicology For Judges and Lawyers*, 12 J.L. & Pol'y 5, 11 (2003) (emphasis added). Numerous courts have looked to the Eaton article in recent years to apply the dose principle and reject various forms of the any exposure theory. See, e.g., *Watkins v. Affinia Grp.*, 54 N.E.3d 174, 179 (Ohio Ct. App. 2016); *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233 (11th Cir. 2005); *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765 (Tex. 2007); *Adams v. Cooper Indus., Inc.*, 2012 WL 2339741 (E.D. Ky. June 19, 2012); *Henrickson v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142 (E.D. Wash. 2009).

⁵ See Eaton, *supra*, at 32 (describing some of the body's protective mechanisms).

Eaton, *supra*, at 9 (emphasis added). Airplane flight crews and passengers receive doses of radiation above background at high elevations, but scientists do not ascribe cancer to those flights.⁶ Foods often contain low levels of natural carcinogens not known to cause any harm. Science has cleared even a lifetime of such “exposures” through the use of epidemiology studies that have found no link between such typical low-level exposures and cancer. This finding is true even when the substance is without question a carcinogen at high doses.⁷ To claim that every such exposure is “cumulative” with other, much higher exposures, and therefore a contributing cause of disease, is nonsensical and not found in the published literature. That claim is also contrary to established toxic-tort causation law which traditionally distinguishes substantial from insubstantial factors in causation analysis.

Yet this is the methodology the experts in this case relied on to focus on the highly speculative secondary exposure of decedent to her father’s six brake jobs. It is not even obvious that her disease was asbestos induced at all.⁸ One of these two, widely different exposures is a

⁶ See Health Physics Soc’y, *Radiation Exposure During Commercial Airline Flights* (2014); Health Physics Soc’y, *Airport Screening Fact Sheet* (2011) (compiling studies).

⁷ Epidemiology is universally recognized as the “most desirable evidence” for assessing causation in the science of toxicology. Michael Green, *Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of the Agent Orange and Bendectin Litigation*, 86 Nw. U. L. Rev. 643, 646 (1992).

⁸ The vast majority of peritoneal mesothelioma found in women today is believed to be induced by transcription errors in the human body itself and not by asbestos exposures. See Paul Sugarbaker et al., *A Review of Peritoneal Mesothelioma at the Washington Cancer Institute*, 12 *Surgical Oncology Clinics of N. Am.* 605, 606 (2003) (data did not support a relationship between peritoneal mesothelioma in women and asbestos exposure); Bertram Price & Adam Ware, *Time Trend of Mesothelioma Incidence in the United States and Projection of Future Cases: An Update Based on SEER Data for 1973 through 2005*, 39 *Critical Reviews in Toxicology* 576, 576, 585 (2009) (“female peritoneal mesothelioma often occurs without exposure to asbestos”); John Goldblum & William Hart, *Localized and Diffuse Mesotheliomas of the Genital Tract and Peritoneum in Women*, 19 *Am. J. Surgical Pathology* 1124, 1136 (1995) (17 of 19 women with peritoneal mesothelioma had “no known asbestos exposure”).

likely or possible cause, and the other simply is not. It is nonsensical, irrational, and unscientific to lump them together.

B. Plaintiff's Experts Are Distorting the Concept of Cumulative Exposures to Incorporate Non-Causative Exposures in Their Testimony.

Experts like Dr. Bedrossian are grossly misusing the concept of “cumulative” exposures to capture speculative and inconsequential exposures in asbestos litigation. Mesothelioma is sometimes described as a “cumulative” disease, in the sense that fibers can build up over a lifetime of asbestos-related jobs. Not every such exposure, however, is sufficient to overwhelm the body’s defenses – lifelong insulators did not get their disease from conducting a backyard brake job. *See, e.g., Martin v. Cincinnati Gas & Elec. Co.*, 561 F.3d 439, 443 (6th Cir. 2009) (“Plaintiff also argues that, because mesothelioma is a progressive disease, any exposure is a substantial cause. ***This argument would make every incidental exposure to asbestos a substantial factor.***”) (emphasis added); *Stallings v. Georgia-Pacific Corp.*, 2017 WL 87023, at *2 (6th Cir. Jan. 10, 2017) (testimony that “*any* further exposure” from gaskets, beyond plaintiff’s four years of heavy Navy exposure, would have been a substantial factor cannot satisfy that causation standard) (emphasis in original); *Juni v. A.O. Smith Water Prods.*, 48 N.Y.S.3d 365, 368 (N.Y. App. Div. 2017) (hereinafter “*Juni Appellate Division*”) (rejecting cumulative exposure theory as irreconcilable with required quantification of exposure).

In reality, humans are regularly exposed to low levels of asbestos and other cancer-causing materials, either naturally-occurring or from limited occupational exposures, without incurring disease.⁹ Those exposures no more “cumulate” to cause disease than a bucket of water

⁹ *See* Radon and Cancer, National Cancer Inst., at <http://www.cancer.gov/about-cancer/causes-prevention/risk/substances/radon/radon-fact-sheet>; Eaton, *supra*, at 25, 29 (discussing accumulation of dioxin in the human body).

contributes to the ocean. *See Moeller*, 660 F.3d at 955 (claiming gasket exposures caused mesothelioma is “akin to saying that one who pours a bucket of water into the ocean has substantially contributed to the ocean’s volume.”). Plaintiffs’ experts themselves illustrate this point by nonsensically *excluding* background exposure to asbestos, which virtually everyone in the United States experiences. There is no difference between background and product-generated asbestos fibers, and these background exposures contribute many millions of fibers to the lungs over the years. Over a lifetime, background exposures likely contributed far more asbestos fibers to decedent’s lungs than merely being in the house of someone who did six brake jobs in the garage. Plaintiff’s experts have not demonstrated otherwise through a competent assessment of decedent’s in-home dose, substituting instead their unproven claim that these brake jobs released “hundreds of thousands or millions of fibers.” Even based on this claim, the experts cannot distinguish her exposures to brake-induced fibers from the many millions of background fibers she would have also cumulatively collected during her life. The experts thus conveniently ascribe to the “cumulative” theory only when it suits them – to pursue litigation targets such as Honeywell here. They are not applying a rational, consistent or scientific methodology.

The reality is that these experts have not bothered to perform a causation analysis properly. *See Eaton, supra*. A real causation analysis starts with epidemiology studies documenting disease in populations with known or estimated exposure levels to a particular substance. The experts must then perform some scientific assessment or estimation of plaintiff’s exposure to show those exposures reached the levels documented in these studies. The “every exposure” theorists, including those who are now calling their testimony “cumulative exposure,” instead testify to trial hypotheticals describing plaintiff’s claimed exposures, and then simply

declare all of them sufficient collectively to cause mesothelioma. There is no dose assessment at all, there is no attempt to distinguish non-causative, trivial workplace exposures, and there is no comparison of any individual product exposure to studies showing such exposures cause disease. If the experts undertook such an analysis here, the exposures of decedent would be so small as to fall far below background levels and nowhere near the levels of similar exposures demonstrated to cause disease in epidemiology studies.¹⁰ Plaintiff's experts never proved otherwise. This testimony involves speculation, not science, and it is clear legal error to admit such testimony as sufficient to prove causation.

C. The Take-Home Aspect of Plaintiff's Exposures Creates Even More Necessity for a Scientific Dose Assessment.

Plaintiff's experts are stretching credulity beyond the breaking point by claiming that decedent's disease was caused, not by exposure to the brake jobs in the garage, but by whatever miniscule portion of that exposure came into the house with her father. Take-home exposures would logically, and as demonstrated by studies, be far lower than the exposures from the work activity itself – much of the breathing zone fibers never attach to clothing; more of it falls off

¹⁰ The vast majority of studies documenting mesothelioma in workers involve the much more potent forms of asbestos crocidolite or amphiboles, not the chrysotile used in brakes. Chrysotile has induced mesothelioma only in studies involving extremely high exposures (miners, textile workers). As discussed herein, studies of brake workers have almost universally found no increased mesothelioma from such work.

To illustrate the limited degree of decedent's likely exposures, one survey of brake-related work documented an average exposure of 0.04 fibers/cc on an 8-hr time weighted average, the methodology OSHA uses to protect workers in the workplace. Dennis Paustenbach et al., *An Evaluation of the Historical Exposures of Mechanics to Asbestos in Brake Dust*, 18 Applied Occup. & Envtl. 786-804 (2003) (Exh. 17) (average lifetime mechanic exposures calculated at 0.04 f/cc or less). OSHA has established an *acceptable* exposure to asbestos of 0.1 f/cc, every day for a 45-year work life. Mr. Weber's six days of brake jobs would have reached nowhere near this level, and his daughter's exposures, if any, would likely not have even been distinguishable from background in the home.

the clothing as the worker moves around; and much of the remaining fiber goes into the washing machine with the clothes.

As a result, the medical literature documents take-home disease only in highly unusual circumstances involving heavy workplace and home exposure situations – “asbestos miners, asbestos factory workers, shipyard/dock workers, textile workers, furnace/engine boiler room workers, railway carriage workers, pipefitters, and insulators.”¹¹ Even in those settings, however, it was very difficult to produce a case of take-home mesothelioma. Newhouse, for instance, in her 1965 study found only nine such spouses across the entire population served by the London Hospital, at a time when asbestos factories with uncontrolled exposures were common.¹² A later study of all mesotheliomas in New York found only ten spouses with apparent take-home disease in an entire decade following years of heavy asbestos usage and exposure.¹³

The historical trend line for mesothelioma, attached as Exhibit A, demonstrates that while mesotheliomas for men rose dramatically starting in the 1970s (based on exposures mostly starting in the 1930s), the line for women stayed almost flat. Thus, women have incurred far less mesothelioma than men, even though a great many of the men in the above chart came

¹¹ Ellen Donovan et al., *Evaluation of Take Home (Para-Occupational) Exposure to Asbestos and Disease: A Review of the Literature*, 42 *Critical Reviews in Toxicology* 703, 716 (2012) (focus on “household contacts of miners, insulators, and workers in shipyards and other industrial locations containing historically high airborne asbestos concentrations and the potential for substantial clothing contamination”; citing studies); Emily Goswami et al., *Domestic Asbestos Exposure: A Review of Epidemiologic and Exposure Data*, 10 *Int’l J. Env’tl. Res. Pub. Health* 5629 (2013) (citing studies).

¹² See Murial Newhouse & Hilda Thompson, *Mesothelioma of the Pluera and Peritoneum Following Exposure to Asbestos in the London Area*, 22 *British J. Indus. Med.* 261 (1965).

¹³ See Nicholas Vianna & Adele Polan, *Non-Occupational Exposure to Asbestos and Malignant Mesothelioma in Females*, 1 *Lancet* 1061, 1062 (1978).

“home,” with clothes laden with asbestos fibers, to clothes-washing spouses in their households. If the claims such as the one in this case were true – that a mere six brake jobs would produce a take-home mesothelioma – then the trend line for woman would match that of men.

There is no scientific basis for the notion that mesotheliomas, particularly peritoneal mesotheliomas, are the result of minimal or trivial in-home asbestos exposures that were likely indistinguishable from background. Dr. Bedrossian could not cite to any literature or studies supporting a conclusion that essentially unmeasurable levels of in-home asbestos exposure the decedent might have experienced on only six occasions would cause a take-home case of mesothelioma. Dose remains the critical missing feature of the experts’ analysis, and its absence is only more pronounced given the speculative nature of the take-home exposure claims in this case.

II. The “Cumulative Exposure” Theory Is the Same Theory Excluded in Many Cases, with a Name Change Attempting to Avoid Court Exclusion.

The Court should not be distracted by the claim that the “cumulative exposure” theory is somehow meaningfully different from the widely-rejected “each and every exposure” theory, which these experts are now disavowing. Plaintiff expert causation theories based on a lack of any dose assessment have undergone a number of name changes over the years to attempt to avoid the growing number of court exclusions. The latest change is to “cumulative exposures,” but the testimony is fundamentally based on the same methodology.

These same experts initially called their testimony – that every cumulative workplace exposure counts – the “single fiber” theory. They routinely testified that a single fiber of asbestos could cause disease, so this plaintiff’s exposures were sufficient. When defendants filed motions challenging this version of the “every exposure” theory, courts began to exclude it. *See, e.g., Bartel v. John Crane, Inc.*, 316 F. Supp. 2d 603 (N.D. Ohio 2004), *aff’d sub nom. Lindstrom*

v. A-C Prod. Liab. Trust, 424 F.3d 488 (6th Cir. 2005) (“not sufficient to assert, as did Plaintiff’s expert Dr. Frank, that even one asbestos fiber that got into Lindstrom’s lungs could have caused his mesothelioma...”).

After these early rejections, plaintiffs’ experts learned not to testify based on the obviously questionable *single fiber* theory. Instead, these experts changed their nomenclature and began to testify that “each and every exposure above background is a substantial factor in causing mesothelioma.” In describing this version of the theory, the experts routinely told juries that it is the “cumulative” effect of all asbestos exposures over a lifetime that cause disease, so each and every workplace exposure must count. Just like the single fiber theory, however, the each and every exposure methodology is similarly grounded on a failure to assess the dose, a refusal to eliminate any identified low-dose exposures, and a lack of scientific or logical support for the opinion that anything above background is causative.

The shift in language did not help – courts continued to exclude “each and every” exposure testimony much as they did the “single fiber” theory. Between 2006 and 2010, the courts rejecting “each and every exposure” testimony included the Sixth Circuit Court of Appeals (twice), the Pennsylvania Supreme Court and two Pennsylvania trial courts, the Texas Supreme Court and two intermediate courts of appeal, and several state courts in Washington and Delaware.¹⁴ By the end of 2013, more than twenty courts had issued opinions criticizing and

¹⁴ See *In re Toxic Substances Cases*, 2006 WL 2404008 (Pa. Ct. Com. Pl. Aug. 17, 2006), *aff’d sub nom. Betz v. Pneumo Abex, LLC*, 44 A.3d 27 (Pa. 2012); *Gregg v. V-J Auto Parts Co.*, 943 A.2d 216 (Pa. 2007); *Flores*, 232 S.W.3d at 765; *Georgia-Pacific Corp. v. Stephens*, 239 S.W.3d 304 (Tex. Ct. App. 2007); *Anderson v. Asbestos Corp.*, No. 05-2-04551-5SEA (Wash. Super. Ct. Oct. 31, 2006); *In re W.R. Grace & Co.*, 355 B.R. 464 (D. Del. 2006); *Martin*, 561 F.3d at 439; *Free v. Ametek*, 2008 WL 728387 (Wash. Super. Ct. Feb. 28, 2008); *In re Asbestos Litig. (Certain Asbestos Friction Cases Involving Chrysler LLC)*, 2008 WL 4600385 (Footnote continued on next page)

rejecting dose-irrelevant causation theories as the basis for expert testimony or as insufficient evidence in asbestos litigation. The new courts adding their opinions included a Georgia intermediate appellate court, the Supreme Court of Virginia, the federal court for the District of Columbia, another Texas appellate court, the Sixth Circuit yet again, a Mississippi trial court, and two federal judges in Utah.¹⁵

The string of opinions did not end there. In the last three years, at least fifteen more courts have added their opinions rejecting “any exposure,” “cumulative exposure,” and similar forms of testimony. Those opinions include two from the Ninth Circuit Court of Appeals, one rejecting such testimony outright and the other reversing an \$11 million verdict in part because the judge did not critically examine the theory; another Sixth Circuit opinion; the Georgia Supreme Court; the Texas Supreme Court for the second time; an Ohio intermediate appellate court; eight federal court decisions from five different states; and most recently the New York intermediate court of appeals.¹⁶

(Pa. Ct. Com. Pl. Sept. 24, 2008); *Smith v. Kelly-Moore Paint Co., Inc.*, 307 S.W.3d 829 (Tex. Ct. App. 2010).

¹⁵ See *Butler v. Union Carbide Corp.*, 712 S.E.2d 537 (Ga. Ct. App. 2011); *Ford Motor Co. v. Boomer*, 736 S.E.2d 724 (Va. 2013); *Wannall v. Honeywell Int’l, Inc.*, 292 F.R.D. 26 (D. D.C. 2013), *aff’d*, 775 F.3d 425 (D.C. Cir. 2014); *Moeller*, 660 F.3d at 950-55; *Smith v. Ford Motor Co.*, 2013 WL 214378 (D. Utah Jan. 18, 2013); *Anderson v. Ford Motor Co.*, 950 F. Supp. 2d 1217 (D. Utah 2013).

¹⁶ See *McIndoe v. Huntington Ingalls Inc.*, 817 F.3d 1170 (9th Cir. 2016); *Estate of Barabin v. AstenJohnson, Inc.*, 740 F.3d 457 (9th Cir.), *cert. denied*, 135 S. Ct. 55 (2014); *Stallings*, 2017 WL 87023; *Scapa Dryer Fabrics v. Knight*, 788 S.E.2d 421 (Ga. 2016); *Georgia-Pacific Corp. v. Bostic*, 439 S.W.3d 332 (Tex. 2014); *Watkins*, 54 N.E.3d at 174; *Comardelle v. Pa. Gen. Ins. Co.*, 76 F. Supp. 3d 628 (E.D. La. 2015); *Sclafani v. Air & Liquid Sys. Corp.*, 2013 WL 2477077 (C.D. Cal. May 9, 2013); *Smith*, 2013 WL 214378, at *1; *Yates v. Ford Motor Co.*, 113 F. Supp. 3d 841 (E.D. N.C. 2015); *Vedros v. Northrup Grumman Shipbuilding, Inc.*, 119 F. Supp. 3d 556 (E.D. La. 2015); *Davidson v. Georgia Pacific LLC*, 2014 WL 3510268 (W.D. La. July 14, 2014), *vacated and remanded on other grounds*, 819 F.3d 758 (5th Cir. 2016); *Crane Co. v. DeLisle*, (Footnote continued on next page)

The plaintiff experts have *not* responded, as they should have, by fundamentally changing their approach to include a scientific dose assessment and epidemiological proof of causation – the science needed to prove any toxic tort case. Instead, they are attempting to change the language used yet again without changing the actual methodology. Around 2013, the experts who were getting excluded began to claim that they were *not* testifying that “each and every exposure” was a cause, but only that “*this* plaintiff’s exposures cumulatively over his lifetime (but not background) were the cause.”¹⁷ Dr. Bedrossian appears to have joined the new chorus.

The “cumulative exposure” theory rests on the same principle as the “each and every exposure” theory – any asserted exposure by plaintiff to any particular product is a cause of disease. *See Suoja v. Owens-Illinois, Inc.*, 211 F. Supp. 3d 1196, 1207 (W.D. Wis. 2016) (expert’s “cumulative exposure” opinion “is no different from the ‘any exposure’ theory”). The newer “cumulative” form of testimony still does not utilize any dose assessment; identifies no studies proving causation from low levels of exposure; and captures each and every exposure just as surely as the old “each and every exposure” theory did.¹⁸ Causation testimony based on take-

206 So. 3d 94 (Fla. Ct. App. 2016); *Suoja v. Owens-Illinois, Inc.*, 211 F. Supp. 3d 1196 (W.D. Wis. 2016); *Juni Appellate Division*, 48 N.Y.S.3d at 365.

¹⁷ *See, e.g., Juni v. A.O. Smith Water Prods.*, 11 N.Y.S.3d 416, 464 (N.Y. Sup. Ct. 2015) (hereinafter “*Juni Trial Court*”).

¹⁸ The experts now typically testify based on a hypothetical set of facts provided by plaintiff’s counsel and then declare that those exposures are sufficient cumulatively to cause disease. The hypothetical is meaningless, however, because the cumulative exposure experts only need to hear that there was some exposure to unquantified dust. The details are irrelevant, and the extent of exposure in the hypothetical is irrelevant. Thus, the claim that cumulative exposure testimony differs from each and every because it relies on the actual exposures of the plaintiff is false – the experts never identify a set of exposure facts which would result in the opposite opinion, that the exposure is not enough. Each and every still rules.

home exposure from only six brake jobs, while ignoring the heavy amphibole exposure as the actual cause, is clear evidence of a similar “each and every” result.

Several courts have recognized that this shift in language is nothing more than a litigation tactic, not a genuinely different theory. Those courts have rejected *cumulative exposure* testimony as unscientific and not grounded in any dose assessment. *See, e.g., id.; Juni Trial Court*, 11 N.Y.S.3d at 436-438; *Juni Appellate Division*, 48 N.Y.S.3d at 368-69; *Yates v. Ford Motor Co.*, 113 F. Supp. 3d 841, 846 (E.D. N.C. 2015). These opinions took on the slightly revised cumulative version of the testimony and rejected it anyway based on its continuing lack of logical coherence and scientific support. The Eighth District court’s misplaced belief that the cumulative exposure theory is different does not justify a different outcome for the “cumulative exposure” theory presented here.

III. The Appellate Court Did Not Conduct an Adequate Inquiry Into the Propositions Relied Upon by Plaintiff’s Experts.

The Eighth District panel fell into the same trap a number of other courts did in reviewing “every exposure” or “cumulative exposure” testimony – the panel repeatedly referred to statements made by the experts themselves as support for the reliability of their own testimony under Rule 702. Yet not a single time did the court actually examine the basis for those statements and determine if they were in fact credible and derived from a scientific methodology. Virtually every court that has admitted “every exposure” forms of testimony has made the same error – accepting the *ipse dixit* of the expert to self-quality the expert’s reliability.¹⁹ This is a

¹⁹ *See, e.g., Neureuther v. Atlas Copco Compressors, L.L.C.*, 2015 WL 4978448, at *4 (S.D. Ill. Aug. 20, 2015) (citing only to expert’s own statements before finding “nothing invalid” about the testimony); *Waite v. All Acquisition Corp.*, 194 F. Supp. 3d 1298, 1314-17 (S.D. Fla. 2016) (repeated references to Dr. Frank’s own testimony); *Davis v. Honeywell Int’l Inc.*, 245 Cal. App. 4th 477, 487 (2016) (citing only to Dr. Strauchen’s own explanation).

Wizard of Oz situation – if the court declines to pull back the curtain, the Wizard continues his charade.

Opinions such as *Schwartz* that allow this testimony are replete with references merely to the expert’s own declarations. Over *forty times* in the *Schwartz* opinion, the Eighth District panel simply restated the expert’s testimony by noting that the expert “testified,” “opined,” “found,” “discussed,” “considered,” or “stated” certain opinions.²⁰ And yet not once did the panel proceed to review the claimed supportive literature or in any other way investigate or critique any of those statements.

Ohio law requires more. In a case almost the exact opposite of the *Schwartz* opinion, a different Eighth District panel reversed a trial verdict because the court did “not independently examine and evaluate the reliability of” the experts’ each and every/cumulative exposure testimony. *Watkins v. Affinia Grp.*, 54 N.E.3d 174, 181 (Ohio Ct. App. 2016). That appellate court would not permit the experts to assume or guess at exposure levels but required the very proof the experts here failed to adduce – “it is not adequate to simply establish that ‘some’ exposure occurred.” *Id.* (quoting *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 773 (Tex. 2007) and *Eaton, supra*, at 39); *see also Crane Co. v. DeLisle*, 206 So. 3d 94, 101 (Fla. Ct. App. 2016) (court’s gatekeeping function requires more than “taking the expert’s word for it.”) (citing *United States v. Frazier*, 387 F.3d 1244, 1265 (11th Cir. 2004)). *Amicus* suggests that the Court review the *Yates* and two *Juni* decisions as excellent examples of what happens when a court actually checks the experts’ claims by review the studies and materials plaintiffs claimed were supportive – and found they were not.

²⁰ *Schwartz v. Honeywell Int’l., Inc.*, 66 N.E.3d 118, 125-128 (Ohio Ct. App. 2016).

Many of the *ipse-dixit* statements made by Drs. Bedrossian and Guth and accepted at face value by the Eighth District have been examined by other courts and found to be illogical and lacking in scientific support. A few examples will illustrate:

Circular logic causation reasoning: Dr. Bedrossian “testified that mesothelioma is a ‘sentinel event’²¹... and that when you diagnose mesothelioma, it is considered caused by asbestos until proven otherwise.” *Schwartz v. Honeywell Int’l, Inc.*, 66 N.E.3d 118, 125 (Ohio Ct. App. 2016). The panel cited no scientific support for this proposition, and Dr. Bedrossian in fact has it backwards. Many mesotheliomas, particularly in women, are not caused by asbestos at all, and thus medical professionals must document a sufficient exposure to asbestos to diagnose the mesothelioma as asbestos-induced. Dr. Bedrossian’s “sentinel” methodology is a form of circular logic reasoning – asbestos exposure must be the cause of the disease because the plaintiff was exposed to asbestos and has the disease. Several courts have criticized this reasoning as unsupported. *See Yates*, 113 F. Supp. 3d at 856 (testimony that “all exposures” were proven “in their accumulation to cause diffuse malignant mesothelioma” was “entirely circular and conclusory and will not assist the jury”); *Butler v. Union Carbide Corp.*, 712 S.E.2d 537, 551 (Ga. Ct. App. 2011) (“It is improper for an expert to presume that the plaintiff ‘must have somehow been exposed to a high enough dose to exceed the threshold (necessary to cause the illness), thereby justifying his initial diagnosis.’ This is *circular reasoning*.”) (emphasis in original, citation omitted). The Eighth District panel improperly accepted Dr. Bedrossian’s similar circular logic without analysis.

²¹ Heavy amphibole asbestos exposure is a “sentinel” event for pleural mesothelioma in men, but as noted above, chrysotile exposure in women has not been associated with peritoneal mesothelioma and thus could not possibly be considered a sentinel event for that exposure and disease.

“*Very low exposures*” *cause mesothelioma*: This statement has become a mantra for these experts and is repeated often by courts that allow them to testify. *See Schwartz*, 66 N.E.3d at 125. The testimony often contends that “one day of exposure” has been shown to be sufficient to cause mesothelioma,²² or sometimes refers to the statement found in regulatory documents that “there is no known safe dose of asbestos.”²³ These statements (1) do *not* draw any distinction based on the widely different potencies of different fiber types, but lump them all together; (2) do *not* identify an actual dose from these “very low” exposures and show that plaintiff had such exposures; and (3) do *not* refer to epidemiology studies documenting disease at such levels.²⁴

The “no safe dose” and “very low exposure” propositions in this case are intended to whitewash over the lack of scientific studies documenting an increased incidence of mesothelioma in *any* population of workers engaged in minimal work with chrysotile-containing products such as brakes, or even more pertinent, from minor take-home exposures from such work. Yet this is the actual causation principle at issue in this case.

²² To support this proposition, the experts often cite to nothing – if pressed, they can only identify sporadic *case reports* of persons engaged in extremely intense, short term exposure activities to far more potent forms of asbestos such as crocidolite (brakes contain chrysotile, a much less potent form). Case reports are notoriously unreliable for deriving causation because they utilize no control group for comparison.

²³ Regulatory and health agencies such as IARC do not, of course, apply a tort causation standard to their determinations, and thus many courts have held that such prophylactic statements as these cannot independently support court causation testimony. *See Yates*, 113 F. Supp. 3d at 847 (citing cases distinguishing regulatory pronouncements of “no safe dose” from causation standard).

²⁴ Nor do the studies cited for the proposition of very low dose causation support the entirely different notion that *all* cumulative exposure must be considered causative. *See Crane Co.*, 206 So. 3d at 104.

A proper and reliable causation methodology would identify cohorts of workers who did a similar number of brake jobs and show that such work causes an increase of disease among family members of that worker. There is no such study.²⁵ In fact, even lifetime brake work does not cause an increase of mesothelioma among *the mechanics themselves*.²⁶ The likelihood that such work would cause mesothelioma from only six jobs, or from the even more limited in-house exposures from six jobs in the garage, is zero. There is certainly no such proof, and the experts here are guessing. The New York *Juni* appellate opinion properly rejected reliance on the “low dose” mantra – even if there is a “medical consensus” that low doses of asbestos exposure cause mesothelioma, “we do not agree that the existence of any such consensus entitles a particular plaintiff to be awarded judgment against a particular defendant by merely establishing some exposure to a product containing any amount of asbestos.” *Juni Appellate Division*, 48 N.Y.S.3d at 237; *see also Free v. Amatek*, 2008 WL 728387 (Wash. Super. Ct. Feb. 28, 2008) (any exposure theory is a mere “hypothesis”).

Visible dust surrogate for a dose assessment: The two experts here rely on testimony that “dust” was created by the brake jobs, and that any sign of visible dust means that the exposures were far above OSHA standards. The Eighth District panel again simply accepted this proposition without any examination. In fact, this testimony has no scientific basis. A number of courts have heavily criticized the experts’ reliance on testimony of “visible dust” in lieu of a

²⁵ *See, e.g., Juni Trial Court*, 11 N.Y.S.3d at 433 (expert cited to no study supporting notion that friction product work causes mesothelioma, even in lifetime brake workers).

²⁶ *See Juni Appellate Division*, 48 N.Y.S.3d at 235 (faulting plaintiff expert for ignoring that 21 out of 22 epidemiology studies of brake workers and mechanics did *not* find any association with mesothelioma); *Yates*, 113 F. Supp. 3d at 859 (referencing 30 epidemiology studies “which find no association between brake work and mesothelioma”). The definitive medical study is David H. Garabrant et al., *Mesothelioma Among Motor Vehicle Mechanics: An Updated Review and Meta-Analysis*, 60 Ann. Occup. Hyg. 8 (2016).

professional assessment of exposure and dose. *See, e.g., Juni Appellate Division*, 48 N.Y.S.3d at 370; *Juni Trial Court*, 11 N.Y.S.3d at 435 (extended critique of reliance on “dust” as exposure surrogate); *Yates*, 113 F. Supp. 3d at 853 (extended discussion rejecting “dust” approach to causation in asbestos cases). It is, of course, extremely easy for a plaintiff to claim that “dust” was created by some activity – many activities create some amount of dust, including ordinary housekeeping work. But dust from an asbestos-related product could well contain no or very little asbestos, and even if it has asbestos, the actual, breathable exposures must be measured by a professional industrial hygienist in a comparable setting, using OSHA-approved techniques, before assuming any exposure occurred or declaring it to be significant.

More critically, the very activity engaged in by decedent’s father – brake repair involving asbestos-containing brakes – was tested many times and the results published in peer-reviewed journals. The results, even for workers performing brake jobs all day long, are frequently well below even today’s OSHA standard.²⁷ A reference to untested and unquantified asbestos in dust is a highly unscientific and unreliable means of determine the degree of exposure in any situation, especially when published articles document only minimal exposures from that activity.

Ongoing household exposures to fiber drift: The panel allowed Dr. Guth to escape any scrutiny for his speculative testimony that the house would have been “contaminated” with asbestos fibers; that the father’s clothes would have transferred their fibers to all the other household clothing; and that his handful of brake jobs would have “continued to expose her for literally years.” Dr. Guth does not even explain how these clothes – washed and dried after

commingling and before anyone wore them – could have contaminated anyone after such a process. He cited to no studies showing that households contain any measurable asbestos fibers after clothes-washing, and certainly not from such minimal exposures as here. It is inconceivable that such testimony could support a verdict and not have drawn even a minimal evaluation by the Eighth District.

Frequency and duration as requirements of causation testimony: A demonstration of a sufficient frequency and duration of exposure is one of the central requirements for proof of asbestos causation in Ohio. R.C. 2307.96. The panel, however, allowed these experts to testify without any demonstration of sufficiency. The experts merely identified the frequency – six times – and the duration – six clothes-washing experiences. There was no showing that such minimal exposures constituted *enough* frequency and *enough* duration to support a verdict. *See Juni Trial Court*, 11 N.Y.S.3d 416, 432, 436 (exposure testimony must demonstrate the *sufficiency* of the exposure and not just its existence). The panel’s single-sentence conclusion, focusing only the experts’ mere identification of exposure events, is telling: “Plaintiffs introduced evidence of the manner, proximity, frequency, and regularity of Schwartz’s exposure to Bendix brakes.” Apparently the panel would have accepted one incident as causative as long as the experts talked about the one incident. The lack of any documentation of a sufficient frequency and duration does not comport with Ohio law or toxic tort law generally, or fundamental toxicological principles.

²⁷ Plaintiff’s claim of high exposures is further weakened because Mr. Webber did not even engage in one of the activities plaintiffs’ experts often claim produces the highest level of exposures - blowing out brake dust with compressed air.

Quantifying the exposures: The Eighth District panel, along with several other courts that have allowed *every exposure* testimony, allowed these experts to escape the requirement of dose on the asserted ground that no one measured the decedent's actual exposures and thus it is impossible to quantify them. There is a large canyon, however, between having no specific measurements of every individual plaintiff's exposures and not even trying to assess or estimate any quantum of exposure at all. As noted above, multiple studies have documented the degree of exposure from brake work, and that work typically produces exposures well below today's OSHA standard. A competent industrial hygienist could readily estimate the range of exposures from Mr. Webber's removal of brakes with no compressed air blowing of brake dust by utilizing studies of similar practices.

For this reason, the better-reasoned opinions require at least some competent effort to identify the range or degree of exposure. The argument that it is not possible to "precisely quantify" plaintiff's exposures is a red herring. Some quantification is required, and is neither difficult nor impossible to develop. *See Moeller*, 660 F.3d at 955 (plaintiff presented "no evidence quantifying" the exposures); *Stallings*, 2017 WL 87023, at *2 (failure of two experts to "quantify the extent of Mr. Stallings' exposure"); *Juni Appellate Division*, 48 N.Y.S.3d at 370 (plaintiffs must present at least "some quantification or means of assessing the amount, duration, and frequency of exposure").

CONCLUSION

Rulings such as the Eighth District panel's opinion would place Ohio at the extreme end of states that require minimal proof from plaintiffs in asbestos cases. Any balance in proving exposure and causation would disappear, as plaintiffs could take any case to a jury with mere testimony of "dust" being present. There is no basis for creating an absolute liability regime in

Ohio that ignores the dose needed to induce a disease. This Court should reverse the ruling below and the trial verdict, and in the process announce clearer and more balanced standards for asbestos causation than those adopted by the Eighth District panel.

Respectfully submitted,

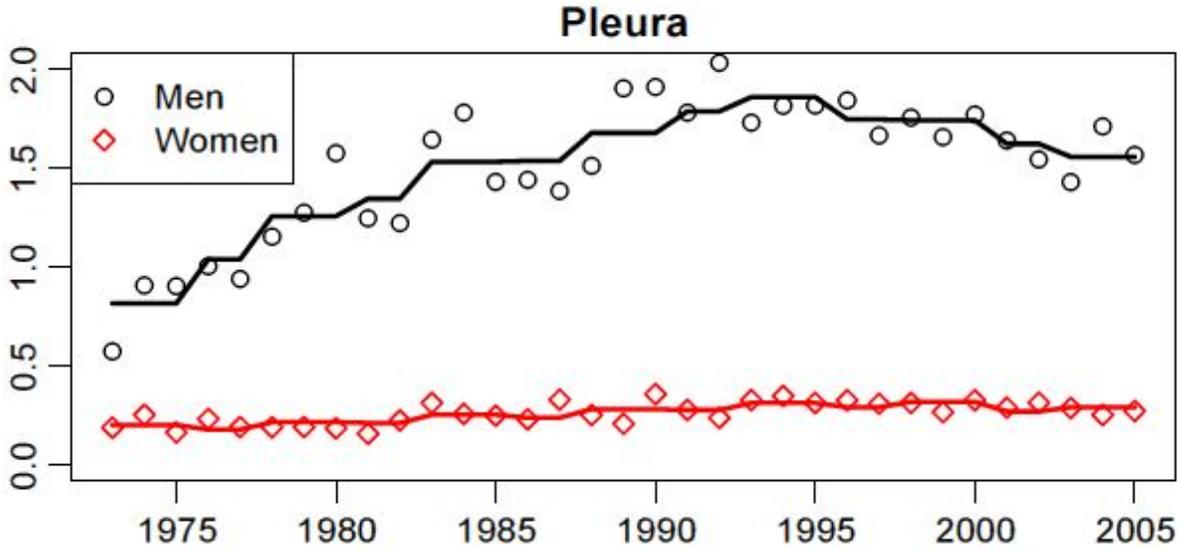
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Dated: June 16, 2017

EXHIBIT A



Mesothelioma incidence in men (top line) and women (bottom line). Taken from S. Moolgavkar et al., Pleural and Peritoneal Mesotheliomas in SEER: Age Effects and Temporal Trends, 20 Cancer Causes Control 935, 939 fig. 1 (2009).

CERTIFICATE OF SERVICE

I certify that I served two copies of the foregoing upon counsel by depositing a copy in a first-class postage-prepaid envelope into a depository under the exclusive care and custody of the U.S. Postal Service on June 16, 2017, addressed to the following:

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