How Much Is Enough?
A Judicial Roadmap to Low Dose Causation Testimony in Asbestos and Tort Litigation

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Introduction

Toxic tort and product liability litigation today are seeing an increasing wave of cases involving minimal and speculative exposure claims.¹ Judges are often confronted with low-exposure litigation because of the declining population of individuals with pre-1970s exposure levels sufficient to be considered hazardous.² The current wave of these low-


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¹ Joseph Sanders, The “Every Exposure” Cases and the Beginning of the Asbestos Endgame, 88 TUL. L. REV. 1153, 1183 (2014) (“Many of the plaintiffs in the preceding cases have been exposed to lower doses of asbestos than was typically the case in the past.”); Mark A. Behrens, What’s New in Asbestos Litigation?, 28 REV. LITIG. 501, 528 (2009) (noting “an increasing number of plaintiffs are bringing claims for de minimis or remote exposures”); see also In re Garlock Sealing Techs., LLC., 504 B.R. 71, 73, 75 (Bankr. W.D.N.C. 2014) (“It is clear that Garlock’s products resulted in a relatively low exposure to asbestos to a limited population and that its legal responsibility for causing mesothelioma is relatively de minimis . . . . Garlock’s products exposed people to only a low-dose of a relatively less potent chrysotile asbestos and almost always in the context where they were exposed to much higher doses of more potent amphibole asbestos.”).

exposure cases encompasses exposures that even a decade ago would have been considered too speculative or de minimis to consider for litigation. The problem is most acute in asbestos litigation, where restrictions by the Occupational Safety and Health Administration (OSHA) largely eliminated disease-producing asbestos exposures in the early to mid-1970s, but the litigation nevertheless shows no signs of slowing down. These low-dose cases should presumably face a major hurdle since the scientific evidence virtually never supports the claim that such minimal exposures in fact produce disease or injury. The exposures are simply too low to be of medical consequence, or at least to produce statistically significant increases of the disease in studied populations. The asbestos plaintiffs’ legal counsel and experts solved this problem some years ago by adopting a unique form of causation testimony—most widely known as either the “every exposure” or any exposure theory—


3 See Behrens & Anderson, supra note 2, at 483 (“In the typical tort case, such a showing would require not only proof of exposure to the defendant’s product, but also exposure to enough of a dose of the defendant’s product to actually cause disease.”).

4 See, e.g., Press Release, A.M. Best Co., Inc., Best’s Special Report: Asbestos Claims Payments Show No Sign of Slowing Despite Drop in Incurred Losses (Nov. 30, 2017), http://news.ambest.com/presscontent.aspx?refnum=25973&altsrc=9 (noting an increase in the estimate for new ultimate asbestos losses and citing a variety of factors, including an increase in secondary exposures and an increase in life expectancy as claimants live into their cancers); see also OSHA, Final Rule, Occupational Exposure to Asbestos, 29 C.F.R. pts. 1910, 1915 & 1926 (Aug. 10, 1994) (stating that the Occupational Safety and Health Act of 1970 imposed health-based regulatory requirements on the U.S. industry broadly); OCC. SAFETY & HEALTH ADMIN., DEP’T OF LABOR, OSHA FACTSHEET: ASBESTOS (2014) (articulating that OSHA established workplace exposure limitations, known as permissible exposure limits (PEL) for several critical substances, including asbestos); Karen Selby, Mesothelioma Incidence & Trends, THE MESOTHELIOMA CTR., (July 6, 2018), https://www.asbestos.com/mesothelioma/incidence (“Doctors diagnose approximately 3,000 new cases of mesothelioma each year in the U.S.”).

5 See cases discussed infra in Part III.

6 See, e.g., Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 857 (E.D.N.C. 2015) (noting that “some studies report adverse health effects at ‘brief’ or ‘low level’ exposures, but without details as to what kind of exposures meet these criteria”).

7 For an overview of the every exposure theory and closely-related forms of testimony, the best sources are several articles on the subject, including three by the authors of this Article. See Behrens, supra note 1, at 528-30; William L. Anderson et

The phrase every exposure as used in this Article encompasses a number of various articulation of theories of causation that do not account for the dose involved but assume each exposure or contact with an identifiable asbestos-containing product is causative. The theory has gone under names such as “single fiber,” “any exposure,”
that ignores dose entirely and assumes that all identifiable exposures are causative.\(^8\) The *every exposure* theory, and its close cousin the *cumulative exposure* theory, both effectively eliminate the “substantial” part of a substantial factor causation requirement and shift the burden of proof to defendants for any identifiable workplace or home exposure.\(^9\) Experts who rely on *every exposure*-type testimony have in recent years attempted, with some success, to export this causation theory to non-asbestos litigation.\(^10\) If allowed to proceed, expert testimony in these

\(^8\) Sanders, *supra* note 1, at 1183 (“[I]t is not surprising that plaintiffs’ lawyers have argued for the ‘any exposure’ standard. Plaintiffs are inclined to adopt this course when they have cases where there is poor evidence on the nature of the individual’s exposure.”). For examples of *every exposure* testimony, see Krik v. Exxon Mobil Corp., 870 F.3d 669, 672 (7th Cir. 2017) (“[T]he defendants . . . s[ought] to exclude Dr. Arthur Frank and other witnesses from testifying about a theory of causation often referred to as ‘each and every exposure theory,’ ‘any exposure theory,’ ‘the single fiber theory,’ or ‘no safe level of exposure theory’ among others. These theories posit that any exposure to asbestos fibers whatsoever, regardless of the amount of fibers or length of exposure constitutes an underlying cause of injury to the exposed individual.” (footnote omitted)); Comardelle v. Pa. Gen. Ins. Co., 76 F. Supp. 3d 628, 632 (E.D. La. 2015) (providing examples of expert witnesses asserting the *every exposure* theory); *In re Garlock Sealing Techs.* LLC., 504 B.R. 71, 80 (Bankr. W.D.N.C. 2014) (Doctors Brodkin and Welch “both testified that any documented occupational exposure to chrysotile—regardless of how minimal—was sufficient to attribute it as a cause of mesothelioma.”).

\(^9\) See, e.g., Krik, 870 F.3d at 677 (“[T]he cumulative exposure theory does not rely upon any particular dose or exposure to asbestos, but rather all exposures contribute to a cumulative dose. . . . [b]ut such a theory of liability would render the substantial-factor test essentially meaningless.” (citations omitted) (internal quotation marks omitted)); McIndoe v. Huntington Ingalls, Inc., 817 F.3d 1170, 1177-78 (9th Cir. 2016) (“Because the [*every exposure* theory] would undermine the substantial factor standard and, in turn, significantly broaden asbestos liability based on fleeting or insignificant encounters with a defendant’s product, we, too, reject it.”); 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.”); Lindstrom v. A-C Prod. Liab. Trust, 424 F.3d 488, 493 (6th Cir. 2005) (noting that, if the *every exposure* theory was accepted, it would eliminate any need for the substantial factor test).

\(^10\) See, e.g., Pluck v. BP Oil Pipeline Co., 640 F.3d 671, 675 (6th Cir. 2011) (benzene); Henricksen v. ConocoPhillips Co., 605 F. Supp. 2d 1142, 1165-66 (E.D.
cases can produce major jury verdicts from only minimal and unquantified exposures.11

Many courts have understood that causation testimony without a dose assessment is scientifically invalid and inadmissible.12 Others, however, have struggled with the debate between the plaintiff and defense experts, and the difficulty of determining how much exposure is enough.13 Given the ubiquity of human exposures to minor or even trace amounts of potentially hazardous materials in almost every aspect of our lives,14 and
the relative ease with which plaintiffs can testify to mere “exposure” to such materials, low-exposure litigation\(^\text{15}\) is likely to be with us for a long time.

Thus, it is critical that judges and appellate courts understand how the scientific community approaches these issues and how that approach should provide the foundation for court assessment of causation testimony. Without a reasonable degree of rigor in the judicial review process, these cases turn into an invitation to file claims with trivial and speculative exposures, albeit involving severe and sometimes fatal diseases such as cancers.\(^\text{16}\) That mix is a recipe for sympathetic but not well-grounded jury verdicts, and unwarranted serial litigation that can easily and inappropriately drive useful products off the market and bankrupt responsible corporate defendants.\(^\text{17}\)

This Article provides judges and litigants a road map to the current state of the case law and legal battle over the viability of low-dose eating too much sugar causes heart disease or skipping breakfast causes diabetes.” (citations omitted)).

\(^{15}\) For purposes of this Article, “low-exposure” is used to refer to exposures that have been prevalent following the advent of stricter environmental and workplace regulations brought on by the conception of the Environmental Protection Agency (EPA) in 1970. EPA History, U.S. ENVTL. PROT. AGENCY, https://www.epa.gov/history (last visited Nov. 14, 2018). Many asserted exposures in today’s litigation are in the range of or below known health standards, and often within the range of background for such substances. See Behrens, supra note 1, at 528-29 (stating the any exposure theory allows plaintiffs to sue defendants with no minimum exposure requirement). Even exposures above health standards are often not an actual cause of disease because regulatory agencies apply a large “margin of safety” before developing those standards. See Sanders, supra note 1, at 1171-72 (“[The court] noted that the experts could not point to studies that demonstrated that asbestos exposure above the background level of asbestos in the ambient air cause[d] mesothelioma.”) (citing Anderson v. Ford Motor Co., 950 F. Supp. 2d 1217, 1224-25 (D. Utah 2013)). Thus, in reality plaintiffs should have to demonstrate causation even for exposures that exceed a health standard, and frequently those exposures would not be sufficient to trigger disease in studied populations.

\(^{16}\) See In re Garlock Sealing Techs., LLC., 504 B.R. 71, 73 (Bankr. W.D.N.C. 2014) (discussing Garlock’s responsibility for current and future mesothelioma claims); Behrens, supra note 1, at 528-29 (noting the high volume of “claims for de minimis or remote exposures” and, more specifically, a case in which a plaintiff who “developed acute myeloid leukemia (AML) from level benzene exposures in gasoline” did not present sufficient evidence of substantial exposure to benzene to satisfy causation).

\(^{17}\) See In re Garlock, 504 B.R. at 73 (describing how the defendant company went bankrupt by defending and settling low-exposure claims for which it had de minimis legal responsibility).
litigation utilizing the *every exposure* theory or other theories of causation that do not assess or prove a causative dose. The focus of the Article is on asbestos litigation. But the principles apply to other types of cases, and examples of those cases are included. In particular, the Article attempts to identify the ideological and other differences that seem to underlie the decisions by some courts in recent years to permit *every exposure* experts to testify when so many other courts have rejected similar testimony in thorough and well-reasoned opinions. The dividing line is not the science—the experts themselves often admit that they are making assumptions where data does not exist. Instead, it appears that some courts are simply discounting the level of review required for these science-based cases. Rather than seriously examining the experts’ own self-serving statements about causation and the scientific evidence, some courts are succumbing to the temptation to simply “let the experts testify.” Courts should and must do better.

The Article begins in Part I with an overview of *every exposure* and similar theories and how they have impacted today’s litigation. Part II then discusses how medical science assesses the actual risk from exposures to potentially hazardous substances and distinguishes causative exposures from those that are likely not. In Part III, the Article follows this discussion with a survey of how courts have addressed *every exposure* and similar testimony, with a particular focus on the errors committed by the handful of courts that have allowed these experts to testify. Finally, Part IV provides a road map for courts and litigants to

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18 See, e.g., King v. Burlington N. Santa Fe Ry. Co., 762 N.W.2d 24, 32 (Neb. 2009) (allowing an expert to testify that exposure to benzene increased the risk of multiple myeloma without establishing that a minimum quantity of exposure was needed before a risk existed).

19 See, e.g., Lindstrom v. A-C Prod. Liab. Tr., 424 F.3d 488, 492 (6th Cir. 2005) (quoting Stark v. Armstrong World Indus., Inc., 21 F. App’x 371, 376 (6th Cir.2001)) (holding testimony of minimal exposure to be insufficient because there must be a great enough quantity of exposure to show that the defendant’s product was a substantial factor in the plaintiff’s injury); Scapa Dryer Fabrics, Inc. v. Knight, 788 S.E.2d 421, 425 (Ga. 2016) (rejecting any exposure testimony for failure to satisfy “the legal standard for causation”); Gregg v. V-J Auto Parts, Co., 943 A.2d 216, 226-27 (Pa. 2007) (stating that every minimal exposure does not constitute a substantial factor of causation).

20 See cases discussed *infra* notes 46-52.

21 See examples discussed *infra* Part III, Section C and accompanying notes 303-23.
assess a low-exposure case under either *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 22 *Frye v. United States*, 23 their state-court variations, 24 or a sufficiency of the evidence review.

The question “how much is enough?” is a difficult one for both science and courts. 25 Yet there is a clear scientific and legal methodology available to separate scientifically baseless cases from those supported by credible science. 26 The established approach—a competent dose assessment followed by demonstrated disease in studies of similarly exposed populations—is neither a mystery nor too difficult for plaintiff’s burden of proof. 27 This accepted methodology, when applied, would assist courts in managing their burdensome asbestos and toxic tort and product exposure dockets while maintaining a path for medically deserving plaintiffs to recover. 28


23 293 F. 1013 (D.C. Cir. 1923) (superseded by statute, the Federal Rules of Evidence, as recognized in Daubert, 509 U.S. at 580-91).

24 See, e.g., Lohrmann v. Pittsburgh Corning Corp., 782 F.2d 1156, 1163 (4th Cir. 1986) (holding “that the trial judge was correct . . . [in applying a] frequency, regularity and proximity test . . . [when] determining whether the inferences raised by the testimony were within the range of reasonable probability so as to connect a defendant’s product to the plaintiff’s disease process”).

25 See infra Part III and accompanying notes 202-16.

26 See Allen v. Pa. Eng’g Corp., 102 F.3d 194, 199 (5th Cir. 1996) (citing Wright v. Willamette Indus., 91 F.3d 1105, 1107 (8th Cir. 1996)) (“Scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities are minimal facts necessary . . . in a toxic tort case.”); Bernard D. Goldstein & Mary Sue Henifin, Reference Guide on Toxicology, in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 633, 636 (3d ed. 2011) (stating a central tenet of toxicology is that the dose of exposure is the factor that makes any substance toxic and dangerous).

27 See Goldstein & Henifin, supra note 26, at 638, 657 (“Dose is a central concept in the field of toxicology” and “[e]xposure assessment . . . can provide the information needed by [an] expert toxicologist to opine on the likelihood that a specific exposure was responsible for an adverse outcome.”).

28 See Bernard D. Goldstein, Toxic Torts: The Devil Is in the Dose, 16 J.L. & POL’Y 551, 587 (2008) (“Unfortunately, as judges attempt to simplify complex issues related to causality, there are too many instances in which relatively simple and straightforward scientific understanding concerning dose is being discarded or obfuscated.”); see also infra text accompanying notes 285-99 discussing the gatekeeping role of the courts.
I. The *Every Exposure* Theory and Other Plaintiff Approaches to Low-Exposure Cases in Today’s Asbestos and Other Toxic Tort Litigation

To understand the problems courts encounter with low-dose litigation, it is helpful first to grasp how the *every exposure* and similar asbestos causation theories have developed and are applied by the plaintiff’s experts in that litigation.\(^{29}\) The medical conundrum of how to identify the exposures that produce asbestos disease has at times resulted in bad law and opportunities for expanding expert testimony into low-dose scenarios, despite the lack of science supporting the testimony.\(^{30}\)

A. Asbestos Disease and Causation Attribution

Asbestos cancers such as mesothelioma, like other latent diseases induced by exposures to other carcinogens, do not readily provide a biological basis for causation attribution.\(^{31}\) The cause of a fatal coma, for instance, may be readily discernible if the victim is holding an empty bottle of aspirin and has extremely high blood levels of salicylates.\(^{32}\) If only the issue was as simple for asbestos-related diseases. Certain types and doses of carcinogens, coupled with the failure of the body’s defenses

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\(^{29}\) See Richard C. Beaulieu, *The Every-Exposure Theory Reviewed*, 11 MASS TORTS LITIG. 13, 13 (2013) (“To meet their burden of establishing that a particular exposure had a causal relationship with the injury in mesothelioma cases, plaintiffs’ experts have in recent years adopted the position that any and every exposure to asbestos is causative.”).

\(^{30}\) Id. at 15-16 (discussing how courts have criticized the *every exposure* theory because it “does not provide the basis for a scientific or reliable methodology for developing a causation opinion”).

\(^{31}\) See Rutherford v. Owens-Illinois, Inc., 941 P.2d 1203, 1207 (Cal. 1997) (“Proof of causation in [asbestos-related cancers] will always present inherent practical difficulties, given the long latency period of asbestos-related disease[s], and the occupational settings that commonly exposed the worker to multiple forms and brands of asbestos products with varying degrees of toxicity.”).

\(^{32}\) “Salicylates are chemicals . . . [that] are a major ingredient in aspirin and other pain-relieving medications.” [*What Is a Salicylate Allergy?*, WEBMD, https://www.webmd.com/allergies/salicylate-allergy* (last visited Nov. 16, 2018)].
against cancerous cells, can produce a tumor through a series of mutations in a given cell.\textsuperscript{33} The tumor, if unchecked, can become cancerous; in the case of asbestos, the tumor can lead to mesothelioma, a disease known since the middle of the last century to be associated with heavy industrial or mining asbestos exposures.\textsuperscript{34} But doctors cannot merely dissect the tumor and flag the specific chemicals or fibers and their sources that produced the tumor.\textsuperscript{35} That direct link is not available because the original cell or cells that produced the tumor are long gone, and the recently-grown tumor tissue typically would not contain the chemicals or fibers in any event.\textsuperscript{36}

At the point a patient has mesothelioma, it seems the most medical science can determine from the patient’s physical condition is (1) the diagnosis of the disease, and (2) that the person’s lung at the time of disease contains asbestos fibers.\textsuperscript{37} Biological assessment cannot even determine that the cancer was produced by asbestos fibers—tumors occur for many reasons or no discernable reason at all.\textsuperscript{38} There also appears


\textsuperscript{35} See Pavlisko & Sporn, supra note 34, at 104 (“The mechanism whereby asbestos induces mesothelioma is not completely understood.”).

\textsuperscript{36} See Rutherford, 941 P.2d at 1219-20 (discussing the inability of plaintiffs to trace an asbestos-induced cancer to a specific fiber or exposure); Dixon v. Ford Motor Co., 47 A.3d 1038, 1045-50 (Md. Ct. Spec. App. 2012) (analyzing the impact of “probabilistic causation” on asbestos causation proof), rev’d on other grounds, Dixon v. Ford Motor Co., 70 A.3d 328 (Md. 2013)).

\textsuperscript{37} See Davis v. Honeywell Int’l, Inc., 199 Cal. Rptr. 3d 583, 588, (Cal. Ct. App. 2016) (recognizing that the decedent died as a result of mesothelioma and stating that “the fibers stay in the lungs for a long time”).

\textsuperscript{38} See Smith v. Ford Motor Co., No. 2:08-cv-630, 2013 WL 214378, at *4 (D. Utah, Jan. 18, 2013) (addressing plaintiff expert’s testimony that “it is not possible to specifically identify an individual fiber from the individual’s occupational, non-occupa-
to be no way at present to trace any specific fibers in the lung back to an
actual source of exposure. Thus, without any biological means of identify-
ing the source of the exposure that caused the disease, scientists must ressort to studies of worker populations—epidemiology—to determine
what types of exposures are most likely to cause mesothelioma. The
process by which scientists attribute causation under these circumstances
is described more fully below in Part II.

As several courts have recognized, causation for asbestos disease
thus becomes an issue of probabilities—how likely is it that an asbestos
exposure is the reason this person incurred mesothelioma? Logic would
reason that the likelihood is high for workers subjected to the “dusty
trades” over long periods. Yet it is seemingly difficult for epidemiology
to say “never,” resulting in uncertainty in regard to exposures that are too
low to produce excess disease in epidemiology studies.

**B. The Every Exposure Theory’s Reliance on the Absence of Evidence**

Plaintiffs’ testifying experts developed the *every exposure* theory to
address their inability to point to the fiber type or quantity that actually


40 See, e.g., Lineaweaver v. Plant Insulation Co., 37 Cal. Rptr. 2d 902, 907 (Ct. App. 1995) (“The probability that an individual asbestos supplier is responsible for plaintiff’s injury may also be decreased by the nature of the particular product.”).

41 See id. at 908 (describing a “return to probabilities” for a fair determination of causation and liability in asbestos cases).

caused the plaintiff’s disease. The every exposure theorists use this lack of evidence to conclude that every exposure must be counted; since they do not know which fiber caused the mesothelioma, they must assume that all inhaled fibers played a part. One such expert testified to the following: “[E]ach and every exposure to asbestos . . ., no matter how de minimis, ‘is a substantial contribution to the cumulative total. . . . Either it’s zero or it’s substantial; there is no such thing as not substantial.” The every exposure experts testify that asbestos is a dose-response disease, meaning that the risk of disease increases or decreases with dose. The experts then rely on government publications stating that because studies have not documented a safe level of asbestos, it is best to conclude that there is in fact no safe dose of asbestos and all exposures are causative. Drawing again from this absence of informa-

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43 See McIndoe v. Huntington Ingalls Inc., 817 F.3d 1170, 1176-77 (9th Cir. 2016) (“McIndoe’s heirs appear to have introduced Dr. Raybin’s testimony and his ‘every exposure’ theory of asbestos causation to reject the substantial-factor test as a whole.”).

44 Krik v. Exxon Mobil Corp., 870 F.3d 669, 677 (7th Cir. 2017) ("[T]he principle behind the ‘each and every exposure’ theory . . . [is] that it is impossible to determine which particular exposure to carcinogens, if any, caused an illness."); Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 846 (E.D.N.C. 2015) ("[B]ecause science has failed to establish that any specific dosage of asbestos causes injury, every exposure to asbestos should be considered a cause of injury."); Yates, 113 F. Supp. at 857 ("Many of these studies are based on the absence of evidence of a “threshold” level of asbestos exposure, rather than affirmative evidence of any particular hazardous level of exposure."); Vedros v. Northrop Grumman Shipbuilding, Inc., 119 F. Supp. 3d 556, 565 (E.D. La. 2015) (noting the doctor’s view that “[e]very incidence of asbestos exposure is causative” and concluding that the doctor’s “specific causation opinions are an unreliable product of the ‘every exposure above background theory’ and must be excluded”); Anderson v. Ford Motor Co., 950 F. Supp. 2d 1217, 1218 (D. Utah 2013) ("The every exposure theory is based on the opposite: a lack of facts and data." (quoting Smith v. Ford Motor Co., No. 2:08-cv-630, 2013 WL 214378, at *2 (D. Utah Jan. 18, 2013))); Bostic v. Georgia-Pacific Corp., 439 S.W.3d 332, 340 (Tex. 2014) ("The any exposure theory effectively accepts that a failure of science to determine the maximum safe dose of a toxin necessarily means that every exposure, regardless of amount, is a substantial factor in causing the plaintiff’s illness. This approach negates the plaintiff’s burden to prove causation by a preponderance of the evidence.").

45 Krik, 870 F.3d at 674-75 (citation omitted).


47 Courts have routinely rejected the experts’ reliance on government pronouncements because of the differences between regulatory protection goals and courtroom causation determinations. See, e.g., In re Garlock Sealing Techs., LLC, 504 B.R. 71, 81-82 (W.D.N.C. 2014) ("The court finds no probative value to the statements of safety
tion, these experts conclude that the risk of disease exists down to zero for workplace or other identifiable exposures.48 One court succinctly described the every exposure theory: “[T]he precise amount of exposure is ‘not really relevant’ because ‘at levels above background regardless of the source or however it happened,’ all asbestos exposures cause disease.”49

The “no safe dose” approach to causation, however, directly contradicts the dose-response principle on which these experts rely.50 The principle not only acknowledges that risk increases with dose, but also recognizes that as doses decrease the risk at some point becomes nonexistent or at least medically meaningless.51 The “no safe dose” assertion also violates the most fundamental principle of toxicology—the importance of establishing a causative dose.52 These experts nevertheless assert, without any apparent limitation, that the only way a jury can identify the exposures that produced the disease is to consider every exposure to be equally a part of the cause.53

and regulatory agencies or to the warnings contained in Garlock’s own Materials Safety Data Sheets. Such statements simply involve something quite different than the issues involved here. Many, if not all, safety and regulatory bodies have issued statements, policies or regulations regarding asbestos exposure. But, these cannot be probative on the issue of causation because of the differences in the way courts and regulatory authorities assess risk.”); Lindstrom v. A-C Prod. Liab. Trust, 424 F.3d 488, 493 (6th Cir. 2005) (“[The plaintiff’s expert] opines that there is no safe level of asbestos exposure, and that every exposure to asbestos, however slight, was a substantial factor in causing Lindstrom’s disease. If an opinion such as [the plaintiff’s expert’s] would be sufficient for plaintiff to meet his burden, the Sixth Circuit’s ‘substantial factor’ test would be meaningless.”); see also Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 847 (E.D.N.C. 2015) (rejecting reliance on EPA and other agency statements regarding “no safe dose” because regulatory evaluations are not consistent with the court’s causation standards and rely on risk rather than actual causation evaluations).

48 _Yates_, 113 F. Supp. 3d at 848.
50 _Krik_, 870 F.3d at 674-75.
51 See id. at 674-75 (noting that “lung cancer is dosage dependent,” which means risk “depends on the length of time of exposure and the amount of exposure”).
52 See, e.g., id. at 675 (“[T]he ‘any exposure’ theory ignored fundamental principles of toxicology that illnesses like cancer are dose dependent.”); see also infra Part II (offering a general discussion of dose in science and medicine).
53 See, e.g., _Betz v. Pneumo Abex LLC_, 44 A.3d 27, 34 (Pa. 2012) (“Dr. Maddox frequently indicated that each and every exposure ‘should be considered,’ ‘contributes to’ and ‘increase[s] the risk of’ asbestos-related diseases.”).
To support this clearly extreme position on causation, the every exposure experts also typically assert (incorrectly) that mesothelioma is a “sentinel” disease associated only with asbestos exposure. For example, some experts incorrectly conclude that all of the plaintiff’s identifiable exposures to asbestos-containing material must be considered cumulative and a cause of the disease, whether the exposure occurred in the workplace or not. For these experts, the only relevance of the actual dose is that the higher the dose, the more risk it created. But once a plaintiff has mesothelioma, these experts claim that the risk for this person, from whatever dose and whatever exposures are identified, was 100% because they have incurred the disease from those known asbestos exposures.

In contrast to the every exposure experts’ refusal to identify a causative dose, industrial hygienists routinely develop information on the actual dose experienced by a given worker for many types of substances. For

54 See, e.g., Bagley v. Adel Wiggins Grp., 171 A.3d 432, 436-38, 445 (Conn. 2017) (including testimony that mesothelioma is a sentinel disease strongly related to asbestos exposure); Gen. Motors Corp. v. Grenier, 981 A.2d 531, 537 (Del. 2009) (including testimony by Dr. Richard Lemen that mesothelioma is a sentinel disease related to asbestos exposures); Robertson v. Doug Ashby Bldg. Materials, Inc., 168 So. 3d 556, 570 (La. Ct. App. 2014) (noting an expert’s opinion “that the causal relationship between exposure to asbestos dust and the development of mesothelioma is firmly established in the scientific literature that it is ‘accepted as scientific fact’”). A “sentinel” disease is one very closely associated with a specific exposure to the point that the disease almost documents that the exposure occurred. See Peter Rabinowitz et al., Human and Animal Sentinels for Shared Health Risks, 45 VETERINÆRA ITALIANA 23, 24 (2009). Mesothelioma, however, has other known causes, including radiation treatment and the human body’s own propensity to produce cancers. See, e.g., Mary Jane Teta et al., Therapeutic Radiation for Lymphoma: Risk of Malignant Mesothelioma, 109 CANCER RADIOTherapy & MESOTHELIOMA 1432 (2007) (“[R]adiotherapy is a cause of mesothelioma.”). Thus, it is incumbent on an expert not to assume that every case of mesothelioma is asbestos-induced, but to document sufficient exposure before attributing mesothelioma to asbestos.

55 See Smith v. Ford Motor Co., No. 2:08-cv-630, 2013 WL 214378, at *3 (D. Utah Jan. 18, 2013) (“Dr. Hammar seeks to base his causation opinion not on the thin reed that he cannot rule any exposure out, but on the opposite: he rules all exposures ‘in,’ boldly stating that Mr. Smith’s mesothelioma ‘was caused by his total and cumulative exposure to asbestos, with all exposures and all products playing a contributing role.’”).


57 See, e.g., Betz, 44 A.3d at 34.

58 The American Industrial Hygiene Association, the leading national organization for this profession, has published a guide specifically for the purpose of assisting in
asbestos, a lifetime dose is typically expressed in terms of “fibers per cubic centimeter years.” As one example, a worker exposed to today’s OSHA asbestos standard of 0.1 fibers per cubic centimeter averaged over an eight-hour work period, for a forty-five-year lifetime of such work, would incur a lifetime dose of 4.5 fibers per cubic centimeter year. OSHA, even today, considers such an exposure to be “acceptable,” and historical dose reconstructions where actual measurements of workers do not exist. See American Industrial Hygiene Association, “Guideline on Occupational Exposure Reconstruction” (Nov. 2008); Kim E. Anderson, Building the Case for Dose Reconstruction in Toxic-Tort Litigation, 34 WESTLAW J. ASBESTOS 1, (2012); 42 C.F.R. § 82.0 (2000) (establishing procedures for reconstructing dose from historical radiation exposures).


OSHA considers a worker having a 0.1 f/cc (fiber/cubic centimeter) exposure, on an eight-hour time weighted average for an entire year, to have a 0.1 f/cc year exposure. See 29 C.F.R. §1910.1001(c)(1) (2012) (“The employer shall ensure that no employee is exposed to an airborne concentration of asbestos in excess of .1 fiber per cubic centimeter of air as an eight (8)-hour time-weighted average (TWA) . . . .”). Running out the math, ten years of continuous exposure to that level would produce 1.0 f/cc years, and forty-five years would thus produce 4.5 f/cc years of exposure. The concept is very similar to “pack-years” of cigarette smoking history, under which a person smoking one pack of cigarettes every day for one year has a one pack-year smoking history. See Dictionary of Cancer Terms: Pack Year, NAT’L CANCER INST., https://www.cancer.gov/publications/dictionaries/cancer-terms/def/pack-year (last visited Oct. 14, 2018) (describing how to calculate “the amount a person has smoked over a long period of time”).

29 C.F.R. §1910.1001(c)(1) (2012). Similarly, the EPA in the United States has established a “re-entry” level of exposure suitable for schoolchildren to come back to school after a school building has undergone asbestos removal. That re-entry figure is 0.01 f/cc. 40 C.F.R. § 763.90(h)(5) (2012). The every exposure experts nevertheless would consider a single or few workplace exposures in excess of 0.01 f/cc to be part of the cause of actual disease.

By 1986, OSHA had reverted to establishing its cancer-related Permissible Exposure Limits (PELs) through a risk model under which the agency set the limits based on keeping risks within a certain goal (e.g., 1 in 10,000 theoretical deaths). John Martonek et al., The History of OSHA’s Asbestos Rule Making and Some Distinctive Approaches that They Introduced for Regulating Occupational Exposure to Toxic Substances, 62 AM. INDUS. HYGIENE ASSOC. J. 208, 213, 214 (2001). In the 1986 preamble, OSHA estimated, under this risk model, that the 0.2 f/cc year PEL established in that rulemaking could still result in a theoretical 7815 deaths for workers exposed to the new standard for forty-five years. Id. at 214. That figure is entirely
appears to have not identified actual (as opposed to theoretical) cases of mesothelioma or other asbestos disease occurring below this lifetime level. By developing a quantified dose, or at least an estimated range for a plaintiff, experts can give the court and jury a way to understand how the exposure compares to health standards or other criteria.\textsuperscript{62}  

The \textit{every exposure} experts, however, do not need, nor do they utilize, any form of dose assessment to reach a causation conclusion.\textsuperscript{63} They frequently disavow, explicitly, any need for a dose estimate or even any knowledge of the extent of exposure because they opine that even the smallest workplace, or other identifiable exposures, are “cumulative” and thus must be counted, regardless of amount.\textsuperscript{64} These experts often use analogies to convey their opinions, including the analogy that “every drop of water contributes to filling a glass.”\textsuperscript{65} They sometimes refer to expos-
ures being “excessive,” “significant,” or the like, but these claims are not backed up by any quantification. In the end, the only distinguishing factor required is that the exposures come from an identifiable product or premises event and be above or different from background or ambient exposures. The exclusion of background exposures is important because background levels of asbestos exposure, which everyone experiences at varying levels, have never been identified as a cause of asbestos disease. Thus, the experts’ exclusion of background is a tacit admission that the dose matters—if a lifetime of background exposures is not enough, how much exposure is required? Instead of making that critical determination, the experts simply draw a line and claim that non-background exposures are causative, regardless of dose. That line does not have a scientific basis, but it does have a litigative basis. The exclusion of background exposures eliminates the only source that cannot be sued and leaves all the other sources (those traceable to a product manufacturer or premises owner) in the litigation subject to a jury verdict. For this reason, some courts have recognized that the every exposure theory is in fact a litigation-driven theory and not a scientific one.

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66 Bernstein, supra note 61, at 56-57, 68. But see Moeller, 660 F.3d at 955 (holding the plaintiff’s failure to quantify exposure to asbestos insufficient to infer Garlock gaskets were a substantial cause of the plaintiff’s mesothelioma).

67 See Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 847-48 (E.D.N.C. 2015) (defining “background exposures” throughout the case as ever-present level of asbestos fibers floating in the air in every city, many rural areas, and nearly the entire modern world).

68 See, e.g., McIndoe v. Huntington Ingalls Inc., 817 F.3d 1170, 1177 (9th Cir. 2016) (“Dr. Raybin did not speak to the severity of McIndoe’s own asbestos exposure beyond the basic assertion that such exposure was significantly above ambient asbestos levels.”); Yates, 113 F. Supp. 3d at 846-48 (“[B]ackground levels vary widely between communities. . . . [T]here is no standard or universality to background. Background can change by the year, by the month, by the date, by the type of work, by the local circumstances and other issues.” (citation omitted)).

69 See, e.g., Anderson v. Ford Motor Co., 950 F. Supp. 2d 1217, 1221, 1224 (D. Utah 2013) (The plaintiff’s expert agreed that “air samples have been used to see the incidence in a background population from nonexposed individuals, and this has never been shown to have a significant increase in the risk for mesothelioma in these background populations.”).

70 Anderson, 950 F. Supp. 2d at 1220, 1222-23.

71 See Yates, 113 F. Supp. 3d at 863 (granting defendants’ motions in regard to the “each and every exposure theory,” but denying the motions as to all other theories).

72 See, e.g., Butler v. Union Carbide, Corp., 712 S.E.2d 537, 540, 545 (Ga. Ct. App. 2011) (upholding the trial court’s ruling that “Dr. Maddox was the ‘quintessential
C. The Impact of Every Exposure Testimony in Expanding Low-Dose Litigation

The effect of the every exposure theory on the expansion of asbestos litigation is significant. The original asbestos litigation typically pitted a worker in an old asbestos industry job, who had incurred mesothelioma or impairing asbestosis, against defendants who produced the type of asbestos or product associated with the worker’s job.\textsuperscript{73} Many of the exposures in these occupations involved insulation or spray-fireproofing containing long, rigid, and often toxic amphibole fibers, rather than the more common, but far less toxic, chrysotile form of fiber.\textsuperscript{74} The paradigm settings for asbestos disease are: occupations such as shipbuilders and Navy personnel working around heavy amphibole asbestos exposures on World War II ships; insulators blowing large clouds of free amphibole or mixed fibers; and asbestos factory workers exposed to “snowstorms” of raw asbestos.\textsuperscript{75}

In part because of the press of many such cases on their dockets and the complexities of proving causation for asbestos disease, some courts began to relax many of the ordinary litigation standards of proof to accommodate these claims.\textsuperscript{76} This “looseness” extended to causation

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\textsuperscript{73} See Stephen J. Carroll et al., Asbestos Litigation, RAND INST. FOR CIV. JUST. 76-77 (2005), http://www.rand.org/pubs/monographs/2005/RAND_MG162.pdf (discussing the recent trend in asbestos-related claims brought by plaintiffs who had not worked in “traditional” industries, such as textile workers).

\textsuperscript{74} Pavlisko & Sporn, supra note 34, at 108; see also In re Garlock Sealing Techs., LLC, 504 B.R. 71, 75 (W.D.N.C. 2014) (“[I]t is clear under any scenario that chrysotile is far less toxic than other forms of asbestos.”).

\textsuperscript{75} See Deborah Hensler et al., Asbestos Litigation in the U.S.: A New Look at an Old Issue, RAND INST. FOR CIV. JUST. 1, 11, 20 (2001), http://www.rand.org/pubs/documented_briefings/2005/DB362.0.pdf (identifying industries in which workers are at risk of asbestos exposure); Behrens, supra note 1, at 528 n.136 (citing Rosenthal v. Unarco Indus., Inc., 297 S.E.2d 638, 640 (S.C. 1982)) (stating that Rosenthal “was one of many pending South Carolina asbestos cases involving plaintiffs who were ‘industrial insulators, shipyard workers, or factory workers’”).

\textsuperscript{76} See, e.g., Victor E. Schwartz et al., A Letter to the Nation’s Trial Judges: How the Focus on Efficiency Is Hurting You and Innocent Victims in Asbestos Liability Cases, 24 AM. J. TRIAL ADVOC. 247, 250-51 (2000) (“The focus on efficiency” [resulting from large numbers of asbestos cases] has been a magnet for new and
requirements.\textsuperscript{77} Courts began to permit plaintiffs to demonstrate that they were merely exposed to a defendant’s product rather than require proof that any one product or premises exposure was significant enough to cause a plaintiff’s disease.\textsuperscript{78} To illustrate, a plaintiff can show mere exposure via having worked in a known asbestos job or industry.\textsuperscript{79}

However, most asbestos cases today involve numerous types of exposures including but not limited to removing a few gaskets, using “dental tape” during dentistry work, removing the cloth insulation on electrical wires, walking by a brake or engine repair, handling boxes of brake pads, and performing a few brake jobs in the back yard. Cases revolving around these instances, and many more unnamed instances, have been the subject of cases, trials, and jury verdicts.\textsuperscript{80} The every exposure theory appears to now encompass almost any product or part that had asbestos in it and almost any alleged direct, walk by, or take-home contact with such a product.\textsuperscript{81} “\[P\]rior to 1980, about 950 asbestos unwarranted cases.”); Richard O. Faulk, \textit{Dispelling the Myths of Asbestos Litigation: Solutions for Common Law Courts}, 44 S. TEX. L. REV. 945, 968-69 (2003) (noting that the Federal Employers Liability Act has relaxed “the traditional concept of proximate cause and replaced it with a featherweight causation rule”).

\textsuperscript{77} See Steven D. Wasserman et al., \textit{Asbestos Litigation in California: Can It Change for the Better?}, 34 PEPP. L. REV. 883, 896-97 (2007) (discussing the relaxation of California’s “substantial factor” standard and the resulting effect).

\textsuperscript{78} Id. at 897, 899.

\textsuperscript{79} Id. at 887-99.


\textsuperscript{81} “Walk-by” cases involve plaintiffs who never handle, work with, or work in proximity to an asbestos-containing product but are merely present in areas containing asbestos materials, usually for short durations such as walking through an area. \textit{See, e.g., Northrop Grumman Sys. Corp. v. Britt}, 241 So. 3d 208, 211 (Fla. Dist. Ct. App. 2017), \textit{review dismissed}, 2018 WL 2411176 (Fla. 2018) (stating that the plaintiff visited “commercial and industrial facilities” for his employment and was allegedly exposed to (and inhaled) asbestos fibers while on the premises of the Northrop facility). “Take-home” cases involve a worker who claims exposure in a workplace or other setting “taking home” fibers that cling to clothing and exposing a spouse or others in the home through clothes-washing or other contact with the clothing. \textit{See William L. Anderson, The Unwarranted Basis for Today’s Asbestos “Take-Home” Cases}, 39 AM. J. TRIAL ADVOC. 107, 108 (2015) (“[T]ake-home cases, also known as household
cases were filed in the federal district courts.”82 Today, however, there are over 10,000 filed asbestos cases, which is an expansion made possible in large part due to the every exposure theory. The following are examples of actual cases brought in the last few years that show the degree to which the litigation has extended far beyond the dusty trades and insulation world and into a realm in which no exposure is too small.

• An every/cumulative exposure expert, Dr. Carlos Bedrossian, claimed that a woman’s mesothelioma was caused by her father’s “five to ten brake jobs in the garage of the family home,” even though she was not present during those jobs and claimed she was exposed only when he came back into the house.84 The expert contended that these unmeasured and unquantified exposures were significant, which to him meant above background.85 The one million dollar jury verdict was upheld by the Ohio intermediate court but reversed by the Ohio Supreme Court, which rejected the cumulative exposure approach as inconsistent with Ohio law.86

• Plaintiffs’ expert Dr. Arthur Frank used the every exposure theory (which he tried to change to a cumulative exposure approach mid-


83 See A Synthesis of Asbestos Disclosures From Form 10-Ks Insights, TOWERS WATSON 1, 1 (Apr. 2010), https://www.towerswatson.com/en-US/Insights/IC-Types/Survey-Research-Results/2010/04/A-Synthesis-of-Asbestos-Disclosures-From-Form-10-Ks-2010 (“The 2000-2003 time period was characterized by recruitment of tens of thousands of unimpaired claimants through mass screenings, leading to a dramatic increase in claims . . . .”).

84 Schwartz, 102 N.E.3d at 479.

85 Id.

86 Id. at 478, 483-84.
stream) to ignore a plaintiff’s thirty-year smoking history and years of heavy insulation work in the Navy, and instead blame plaintiff’s lung cancer on a two-week job working with heaters that were supposedly shielded by asbestos insulation. The case resulted in a defense verdict that was upheld by the United States Court of Appeals for the Seventh Circuit.

• Dr. Frank again relied on his cumulative exposure theory in a Wisconsin federal court case to attribute plaintiff’s disease on only a one-month exposure to insulation work, during the course of a 40-year history of such work. Dr. Frank eschewed any need for a dose assessment from the one-month experience, instead concluding merely that “if there is exposure, then there is causation.” The trial judge rejected this testimony as part of a bench trial.

• Dr. Frank and another every exposure expert, Dr. Jerrold Abraham, opined that a lawyer’s mesothelioma was caused by nothing more than three remodeling jobs in his living quarters, even though he did none of the work himself and was mostly in a back room studying for the bar exam. A federal court in Maryland rejected this testimony under both Daubert and Maryland’s substantial factor causation standard.

• In a Utah federal court trial, Dr. Samuel Hammar contended that a plaintiff’s mere six brake jobs were sufficient to cause mesothelioma, again with no attempt whatsoever to assess the dose from such limited work and compare it to epidemiology studies of automotive mechanics. The trial court excluded his testimony.

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87 Krik v. Exxon Mobil Corp., 870 F.3d 669, 671-72 (7th Cir. 2017).
88 Id. at 672.
90 Id. at 1207.
91 Id. at 1208-09.
93 Daubert v. Merrell Dow Pharms., Inc., 509 U.S. 579, 585-98 (creating the “Daubert standard,” in which a scientific methodology is determined as admissible by meeting a series of factors).
94 Rockman, 266 F. Supp. 3d at 850-52.
96 Id. at *5.
• Dr. Frank testified that infrequent work in the vicinity of asbestos-covered steam pipes, with no further information on a potential for exposure, was sufficient to cause asbestosis, a lung disease that by wide agreement requires a very heavy dose over time. The court allowed the testimony, but the jury found for the defendant.

• A plaintiff claimed take-home exposure from merely being at the home of her husband’s father, who was a construction worker. The plaintiff had no direct contact with asbestos-containing products. The defendant’s motion for summary judgment was denied, implying the court found that a reasonable jury could conclude the plaintiff was exposed to fibers brought home by the father.

• A Florida appellate court upheld a $9 million verdict based on nothing more than plaintiff’s job as a benefits advisor in which he visited locations in a factory. The most he could claim was that he “saw” maintenance activities on insulation-covered pipes. Plaintiff’s expert offered no assessment of his dose, if any, and instead merely claimed that plaintiff’s exposure was “significant,” a claim that the court accepted without challenge.

Outside of asbestos litigation, courts have faced a number of instances in which the plaintiffs’ testifying experts, while not necessarily stating the every exposure theory as explicitly as in asbestos cases, have nevertheless tried to link causation to limited exposures with no quantification or effort to identify a causative dose. Examples include the following.

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98 Id. at 603-604, 611.
100 Id.
101 Id.
103 Id. at 211.
104 Id. at 214-15.
105 See, e.g., Chapman v. Procter & Gamble Distrib., LLC, 766 F.3d 1296, 1307 (11th Cir. 2014) (noting the general causation experts were unable to “determine how much Fixodent must be used for how long to increase the risk of a copper-deficiency, or for how long a copper-deficiency must persist before an individual is at an increased
Experts in a New York Court of Appeals case tried, unsuccess-
fully, to assert that a limited number of exposures of a gas station
worker to benzene in gasoline caused his acute myeloid leukemia
(AML), relying on loose descriptors such as “extensive” rather than
any quantification that related the exposures to those of factory
workers known to incur disease.106

Similar testimony in a Nebraska case produced the opposite result,
as the Nebraska Supreme Court reversed the trial court and allowed
an expert to testify that an unquantified amount of benzene in diesel
fumes could support a jury verdict.107 The expert testified that no
“minimum exposure level had to be reached before there was a
risk.”108

The Eleventh Circuit Court of Appeals rejected four experts’
attentions to link trace amounts of zinc exposure, from eight years’
usage of dental cream, with plaintiff’s spinal cord disorder.109 The
court held that the experts did not demonstrate a causative dose, in part
because “neither . . . [the] experts ‘nor the articles on which they rely’
determine[d] how much Fixodent must be used for how long to
increase the risk of a copper-deficiency.”110

A plaintiff in a Vermont case claimed that a manufacturing plant
utilizing benzene caused his cancer from exposure when he played

risk” (quoting In re Denture Cream Prods. Liab. Litig., 795 F. Supp. 2d 1345, 1352
(Va. 2011) (“[P]laintiff’s suspicion that his cancer was caused by exposure to benzene
on the Goodyear ballfield when he was a teenager is purely speculative. As plaintiff’s
own expert acknowledged, there is no way to know whether any benzene-containing
product actually contaminated the ballfield.”); King v. Burlington N. Santa Fe Ry. Co.,
762 N.W.2d 24, 32 (Neb. 2009) (noting that an expert witness “rejected the idea that
a minimum exposure level had to be reached before there was a risk”); Parker v. Mobil
Oil Corp., 857 N.E.2d 1114, 1122 (N.Y. 2006) (“[The expert] reported that Parker was
‘frequently’ exposed to ‘excessive’ amounts of gasoline and had ‘extensive exposures
... in both liquid and vapor form,’ which—even given that an expert is not required
to pinpoint exposure with complete precision—cannot be characterized as a scientific
expression of Parker’s exposure level.”).

106 Parker, 857 N.E.2d at 1121-22.
107 King, 762 N.W.2d at 31, 51.
108 Id. at 32.
109 Chapman, 766 F.3d at 1299, 1315-16.
110 Id. at 1307 (quoting In re Denture Cream Prods. Liab. Litig., 795 F. Supp. 2d
1345, 1351 (S.D. Fla. 2011)).
baseball as a teenager on a nearby field.\textsuperscript{111} The expert made no attempt to quantify the dose, and the Vermont Supreme Court rejected the testimony.\textsuperscript{112}

The experience in California, in particular, illustrates the ability of the every exposure theory to expand litigation. In the 1990s, in the state’s two most important asbestos causation opinions—Lineaweaver v. Plant Insulation Co.\textsuperscript{113} and Rutherford v. Owens-Illinois, Inc.\textsuperscript{114}—the courts rejected the notion that plaintiffs could shift the burden of proof for causation to defendants by showing mere exposure.\textsuperscript{115} Instead, plaintiffs were required to meet the state’s substantial factor causation standard.\textsuperscript{116} The holding in Lineaweaver applied to asbestosis, a heavy dose disease, but Rutherford extended that holding to mesothelioma cases. Much of the reasoning of these cases demonstrates that every exposure testimony should not be sufficient in California:

- The court did not accept the plaintiffs’ argument that cases should proceed on mere exposure simply because the specific causative fibers could not be identified.\textsuperscript{117} To do so would impose an undue burden and a form of absolute, “market-share liability” on defendants.\textsuperscript{118}
- The court understood, rightly, that such an approach would, in fact, shift the burden of proof of causation back to defendants before plaintiffs had proven all facets of causation—both exposure and sufficient dose to contribute to causation. [The] jury instruction shifting the burden of proof to [the] defendant[] on the element of causation [was] . . . unnecessary and incorrect under settled statewide principles of tort law.\textsuperscript{119}

\textsuperscript{112} Id. at 1275 (“[C]ourts are reluctant to admit causation testimony based on a differential diagnosis where the proffered expert possesses only weak circumstantial evidence that some exposure occurred and makes no effort to scientifically evaluate or roughly estimate the degree of exposure or dosage.” (quoting Plourde v. Gladstone, 190 F. Supp. 2d 708, 722 (D. Vt. 2002))).
\textsuperscript{113} 37 Cal. Rptr. 2d 902 (Cal. Dist. Ct. App. 1995).
\textsuperscript{114} 941 P.2d 1203 (Cal. 1997).
\textsuperscript{115} Rutherford, 941 P.2d at 1206; Lineaweaver, 31 Cal. Rptr. 2d at 904, 907-08.
\textsuperscript{116} Rutherford, 941 P.2d at 1206-07; Lineaweaver, 31 Cal. Rptr. 2d at 905.
\textsuperscript{117} Rutherford, 941 P.2d at 1218-19.
\textsuperscript{118} Id. at 1217, 1219.
\textsuperscript{119} Id. at 1206.
The court acknowledged that not all asbestos exposures are causative and did so in multiple passages. This language cannot be squared with the every exposure theory under which all exposures in the workplace are considered a cumulative part of the cause.

The court acknowledged that multiple sources of asbestos could be part of a cumulative cause, but the court never relaxed the barrier for each such exposure—namely to “demonstrate to a reasonable medical probability that a product or products supplied by the defendant, to which [the plaintiff] became exposed, were a substantial factor in causing his disease or risk of injuries.”

The court in Rutherford addressed the concept of increased risk in its opinion. It could be argued that appellate courts in California have latched onto that language and ignored everything else in Rutherford. The result is some of the most extreme every exposure opinions in the country, under which an expert is apparently allowed to opine to any level of non-background exposure as causative. It appears that no state appellate court has yet identified a level of plaintiff-alleged exposure that the California courts would consider so trivial or de minimis to be insufficient to support a jury verdict.

120 See, e.g., id. at 1218 (“[T]he question arises whether the risk of cancer created by a plaintiff’s exposure to a particular asbestos-containing product was significant enough to be considered a legal cause of disease.”) (emphasis added); see also id. at 1220 (stating that the burden is on the plaintiff to prove “which exposures . . . contributed significantly enough . . . to be considered ‘substantial factors’”).

121 Id. at 1206-07 (emphasis added).

122 Rutherford, 941 P.2d at 1209 (“Medical testimony was also presented to establish that the plaintiffs’ asbestos-related disease was ‘dose-related’—i.e., that the risk of developing asbestos-related cancer increased as the total occupational dose of inhaled asbestos fibers increased.”).

123 See, e.g., Jones v. John Crane, Inc., 35 Cal. Rptr. 3d 144, 151 (Cal. Ct. App. 2005) (“[A] level of [workplace] exposure that is the equivalent of that to which one might be exposed in the ambient air over a lifetime is not necessarily insignificant . . . . The mere fact that comparable levels could be found in ambient air does not render the exposure ‘negligible or theoretical.’” (quoting Rutherford, 941 P.2d at 1203)); see generally Wasserman et al., supra note 77, at 894 (Rutherford’s “‘substantial factor’ test . . . has been much quoted, interpreted, and misapplied to the point that any exposure to asbestos, however insubstantial, seems to be sufficient for a plaintiff to defeat summary judgment.”).

124 California federal courts, in contrast, have often excluded any form of testimony that did not include a dose assessment and proof of sufficiency of exposure. See, e.g., McIndoe v. Huntington Ingalls, Inc., 817 F.3d 1170, 1177 (9th Cir. 2016) (The
As the opinions in several of the above examples demonstrate, the *every exposure* theory is the vehicle that permits these trivial or minimal exposure cases to get to a jury.\(^{125}\) Without it, plaintiffs would have to prove a quantified level of dose (or at least an estimated range), consistent with occupations known through epidemiology studies to have caused asbestos disease—just like any other defendant in any other toxic tort litigation.\(^{126}\)

As discussed in more detail in Part III, since 2005\(^ {127}\) a large number of courts across the country have addressed the *every exposure* theory or similar forms of testimony that eschew any dose estimate. The vast majority hold such testimony to be insufficient scientifically and as evidence.\(^ {128}\) As the Seventh Circuit found in 2017, “more than thirty other federal courts and state courts have held that this cumulative/*any

plaintiff’s expert “generally did not make distinctions between the overall dose of asbestos [the plaintiff] breathed aboard the ships and that portion of such exposure which could be attributed to the shipbuilders’ materials.”); Estate of Barabin v. AstenJohnson, Inc., 740 F.3d 457, 467 (9th Cir. 2014) (finding that the lower court had erroneously admitted expert testimony which relied on the *every exposure* theory and failed the *Daubert* standard); Sclafani v. Air & Liquid Sys. Corp., No. 2:12-cv-3013-svw-pjw, 2013 WL 2477077, at *4-*5 (C.D. Cal. May 9, 2013) (stating Plaintiff’s expert did not establish the use of reliable scientific techniques in his analysis of the cause of the decedent’s mesothelioma, relying instead on the “substantial factor” analysis).

\(^ {125}\) See, e.g., McIndoe, 817 F.3d at 1177; Estate of Barabin, 740 F.3d at 464; Sclafani, 2013 WL 2477077, at *4.

\(^ {126}\) Behrens & Anderson, supra note 2, at 483 (“In the typical tort case, [the plaintiff must show] not only proof of exposure to the defendant’s product, but also exposure to enough of a dose of the defendant’s product to actually cause disease.”); id. at 490 (“It is exceedingly difficult, however, to establish with certainty the level at which asbestos exposures do not cause mesothelioma.”).

\(^ {127}\) In 2008 the author joined with Mark Behrens to write the first published law review article on the *each and every exposure* theory of causation. See generally Behrens & Anderson, supra note 2, at 479. At the time of the first article, only a handful of courts had addressed the admissibility of the *every exposure* theory or even its scientific or logical validity. Since the article appeared, however, more than forty courts all over the country have written opinions on the admissibility or sufficiency of this theory, mostly—but not always—in the asbestos context. Two follow-up articles document the progression of the case law on this subject. See Anderson et al., *Round II*, supra note 7; Anderson & Tuckley, supra note 7.

\(^ {128}\) See, e.g., Moeller v. Garlock Sealing Techs., 660 F.3d 950, 954-55 (6th Cir. 2011) (rejecting the plaintiff’s argument that substantial exposure should be admissible).
exposure’ theory is not reliable.” 120 Those courts include seven opinions from within three federal circuits (Sixth, Seventh, and Ninth); eight state supreme or highest court opinions (Georgia, Florida, Ohio, New York (twice), Pennsylvania (twice), Texas (twice), and Virginia); and numerous opinions from federal district courts and state intermediate appellate courts. 132 California’s Supreme Court has three times rejected...
requests to review every exposure opinions in that state. In recent years, a growing number of courts have ignored the wave of cases rejecting every exposure testimony and instead have allowed the experts to testify—at least in part—despite the experts’ refusal to determine whether the plaintiff had an identifiable dose sufficient to cause disease.

Given this thirteen-year history, it would not be an exaggeration to call the every exposure theory one of the most important scientific legal issues in the history of asbestos litigation. Some decisions have dramatically shifted the landscape of asbestos lawsuits. The two Texas Supreme Court decisions in Borg-Warner Corp. v. Flores and Bostic v. Georgia-Pacific Corp., for example (along with legislative tort reform laws) greatly reduced the scope of asbestos litigation in that state—plaintiff


See, e.g., Neureuther v. Atlas Copco Compressors, L.L.C., No. 13-cv-1327-SMY-SCW, 2015 WL 4978448, at *4 (S.D. Ill. Aug. 20, 2015) (finding that the expert’s testimony passed the Daubert test and should not be excluded); Davis v. Honeywell Int’l Inc., 199 Cal. Rptr. 3d 583, 588 (Cal. Ct. App. 2016) (holding that the trial court did not abuse its discretion by allowing plaintiff’s medical expert to testify regarding the every exposure theory); Scapa Dryer Fabrics, Inc., 788 S.E.2d at 427 (holding that the court’s exclusion of expert testimony required that the lower court’s decision be reversed); Rost v. Ford Motor Co., 151 A.3d 1032, 1047 (Pa. 2016) (holding that physician’s testimony regarding every exposure was inadmissible); Payne v. CSX Transp., Inc., 467 S.W.3d 413, 457 (Tenn. 2015) (holding that a qualified expert can testify without having to first establish the exposure amount); King v. Burlington N. Santa Fe Ry. Co., 762 N.W.2d 24, 51 (Neb. 2009) (finding that the district court improperly excluded expert testimony concerning causation). Some of these opinions have been reversed or modified on appeal, and the viability of the Illinois decisions, particularly the federal decision, is highly doubtful after the Seventh Circuit’s rejection of cumulative exposure testimony in Krik.

232 S.W.3d 765 (Tex. 2007).

439 S.W.3d 332 (Tex. 2014).

See Bostic, 439 S.W.3d at 360 (finding that “dose matters, and this requirement applies to mesothelioma cases”); Flores, 232 S.W.3d at 773 (“[S]ome exposure
law firms in Texas resorted to opening offices in other, more favorable states.\textsuperscript{138} Louisiana’s federal courts have also rejected low-exposure asbestos litigation in the state’s federal courts with a series of five decisions rejecting \textit{every exposure} testimony.\textsuperscript{139} These decisions have made removal to federal court in Louisiana nearly case-dispositive.

Both plaintiffs and defendants are devoting enormous resources and briefing prowess in attacking or defending the \textit{every exposure} theory as the appeals move around the country state-by-state and court-by-court.\textsuperscript{140} If the \textit{every exposure} theory continues to make its way into other tort litigation, the theory’s impact could be much broader than just asbestos litigation. Asbestos litigation is no longer about “dusty trades” workers who incurred large exposures and disease. The litigation has morphed into an endless “elephantine mass” of lawsuits,\textsuperscript{141} based largely on a theory one court called a “fiction.”\textsuperscript{142} Courts need to engage in a careful and thoughtful narrowing of the ability of these experts to testify in order

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\item \textsuperscript{138} See, \textit{e.g.}, Cortney Fielding, \textit{Plaintiff’s Lawyers Turn to L.A. Courts for Asbestos Litigation}, \textit{DAILY J. CORP.} (Los Angeles), Feb. 27, 2009, at 1 (discussing move of law firms to California).
\item \textsuperscript{139} See Schindler v. Dravo Basic Materials Co., No. 17-13013, 2019 WL 446567, at *6 (E.D. La. Feb. 5, 2019) (excluding expert reliance on “each and every” exposure methodology because it was not based on the specific facts and circumstances of the plaintiff’s exposures, and in effect eliminates the specific causation requirement in toxic tort cases); Bell v. Foster Wheeler Energy Corp., No. 15-6394, 2017 WL 876983, at *3 (E.D. La. Mar. 6, 2017) (finding that testimony regarding “significant exposures” are only allowed where “significant” means only “statistically significant” in the sense that exposure at a certain level for a certain duration can cause $x$ in $y$ number of people too develop mesothelioma”); Vedros v. Northrup Grumman Shipbuilding, Inc., 119 F. Supp. 3d 556, 563 (E.D. La. 2015) (“The Court finds no meaningful distinction between the ‘every exposure’ theory and an ‘every exposure above background’ theory.”); Comardelle v. Pa. Gen. Ins. Co., 76 F. Supp. 3d 628, 635 (E.D. La. 2015) (“[T]he Court concludes that Dr. Hammar’s specific-causation opinions . . . are an unreliable product of the ‘every exposure theory’ and must be excluded.”); Davidson v. Georgia Pacific L.L.C., No. 12-1463, 2014 WL 3510268, at *6 (W.D. La. July 14, 2014) (“This court finds that the ‘every exposure’ theory conflicts with the ‘substantial factor’ test of causation that applies under Louisiana law.”).
\item \textsuperscript{140} \textit{Cf.} Behrens & Anderson, \textit{supra} note 2, at 480-81 (reviewing court decisions regarding asbestos litigation from across the country).
\item \textsuperscript{141} Ortiz v. Fibreboard Corp., 527 U.S. 815, 821 (1999).
\item \textsuperscript{142} Gregg v. V-J Auto Parts, Co., 943 A.2d 216, 226-27 (Pa. 2007).
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to bring these dose-less toxic tort cases in line with a rational and science-based jurisprudence as applied in most toxic tort and product litigation.

II. The Role of Science in Distinguishing Between Causative Exposures and Those Unlikely to Produce Disease

Scientists, for many years, have used a well-recognized process to determine whether exposures are responsible for injury or diseases occurring many years later. The leading article on determining causation in the toxic tort causation is the publication by Dr. David Eaton of the University of Washington entitled Scientific Judgment and Toxic Torts—A Primer in Toxicology for Judges and Lawyers. Numerous courts have cited Eaton’s article to articulate the bases for their dose-related decisions. Much of this section is drawn from Eaton’s article and related publications as well as the court cases applying those principles.

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


144 Id.

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.”

The role of the dose is 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. The fundamental dose requirement is set forth in the Federal Judicial Center’s *Reference Manual on Scientific Evidence,* and even more concretely in Dr. Eaton’s article, “[d]ose is the single most important factor to consider in evaluating whether an alleged exposure caused a specific adverse effect.”

Dose, in turn, is more than just an “exposure” to a substance—the dose results from the extent of exposure, how often it is repeated, and the duration of each exposure over time.

Asbestos, along with most other toxins, requires some level of overall dose to produce disease. The human body has many mechanisms for

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147 This formulation is the famous one established in the 1600s by the “father of toxicology,” Paracelsus. See Goldstein, supra note 28, at 553 (“Toxicologists accept Paracelsus—a 16th century alchemist and a bit of a charlatan—as their ancestor and credit him with the first law of toxicology—that the dose makes the poison.”).

148 See Green et al., supra note 39, at 603 (discussing the “fundamental tenet” of toxicology and instances where “there is a safe dose below which an agent does not cause any toxic effect”).

149 Id. at 636 (“[A]ll chemical agents are intrinsically hazardous—whether they cause harm is only a question of dose.”).

150 Eaton, supra note 143, at 11.

151 See In re N.Y. City Asbestos Litig. (Juni I), 11 N.Y.S.3d 416, 435 (N.Y. Sup. Ct. 2015) (involving an expert who failed to address the “amount, duration, or frequency” of the plaintiff’s exposure and did not “even minimally quantify” those exposures), appeal dismissed, (Juni II), 148 A.D. 3d 233 (N.Y. App. Div. 2017), aff’d sub nom. (Juni III), 116 N.E.3d 75 (N.Y. 2018); Betz v. Pneumo Abex, L.L.C., 44 A.3d 27, 56 (Pa. 2012) (involving experts who acknowledged the importance of potency, intensity, and duration of exposure but failed to account for those in any exposure opinion).

152 See, e.g., Betz, 44 A.3d at 53 (recognizing “the considerable tension between the any-exposure opinion and the axiom, which is manifested in myriad ways both in science and in daily human experience, that the dose makes the poison”).
defending against minor exposures, both for asbestos and for other toxins.\textsuperscript{153} Thus, based on those processes, the lung, like other organs, can presumably defend itself against a whole array of small, daily exposures that at much higher levels might well cause harm.\textsuperscript{154} Disease results when those exposures reach a level that overwhelms our defenses, called the “threshold” point.\textsuperscript{155} Aspirin, alcohol, sunlight, and even arsenic are only poisonous if the dose is high enough to make them poisonous.\textsuperscript{156} At lower doses, they are either harmless or, in some instances, they are even beneficial.\textsuperscript{157} As many courts have held, “[s]cientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiff’s burden in a toxic tort case.”\textsuperscript{158}

As Dr. 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.\textsuperscript{159}

\textsuperscript{153} See Eaton, supra note 143, at 32 (describing some of the body’s protective mechanisms against damage to DNA caused by reactive oxygen species).

\textsuperscript{154} Id.

\textsuperscript{155} See generally CURTIS D. KLAASSEN, CASARETT & DOULL’S TOXICOLOGY: THE BASIC SCIENCE OF POISONS 21 (7th ed. 2008) (“[T]he minimally effective dose of any chemical that evokes a stated all-or-none response is called the threshold dose.”).

\textsuperscript{156} Id. at 13-14, 20-21 (explaining the frequency and duration of exposure to a substance affects how harmful the substance can be to the body).

\textsuperscript{157} Id. at 20-21 (Evidence suggests that low doses of exposure to certain toxic substances could potentially be beneficial to the body. For example, low doses of alcohol consumption reduce risk of coronary heart disease and stroke.).

\textsuperscript{158} Allen v. Pa. Eng’g Corp., 102 F.3d 194, 199 (5th Cir. 1996) (emphasis added); see also Zellars v. NexTech Northeast, L.L.C., 895 F. Supp. 2d 734, 742 (E.D. Va. 2012) (“Ruling in” exposure to a particular substance as a possible cause of a patient’s medical condition requires (1) a reliable determination of the level of exposure necessary to cause the condition and (2) a reliable determination that the patient was exposed to the substance at this level.”).

\textsuperscript{159} See Eaton, supra note 143, at 13 (explaining that the effect of a chemical carcinogen depends heavily on the dose or exposure to that carcinogen).
Airplane flight crews and passengers receive doses of radiation above background at high elevations, but scientists do not ascribe cancer to those flights. Foods often contain natural carcinogens at low levels 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: “based upon existing exposure data, the great majority of individual naturally occurring and synthetic chemicals in the diet appears to be present at levels below which any significant adverse biologic effect is likely, and so low that they are unlikely to pose an appreciable cancer risk.”

Thus, low exposures are not necessarily harmful even when the substance is unquestionably a harmful carcinogen at high doses. To claim that every such exposure is “cumulative” with other, much higher exposures, and therefore a contributing cause of disease, is a nonsensical and irrational proposition not found in the published literature and entirely inconsistent with our daily lives. That claim is also contrary to established toxic-tort causation law which traditionally distinguishes substantial from insubstantial factors in causation analysis.

In addition, it is logically understood that millions of people—in fact, virtually everyone alive today—have experienced low levels of “background” or ambient exposure to asbestos. The reason for this is because the fibers are ubiquitous in the environment and found as a naturally

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161 See Bruce N. Ames, Letter to the Editor, Pesticides, Risk, and Applesauce, 244 Sci., May 19, 1989, at 755 (discussing studies of natural toxins in foods that are carcinogenic in animal studies, including the following foods: anise, apples, bananas, basil, broccoli, brussel sprouts, cabbage, cantaloupe, carrots, cauliflower, celery, cinnamon, cloves, cocoa, grapefruit juice, honeydew melon, horseradish, kale, mushrooms, mustard, nutmeg, orange juice, parsley, parsnips, peaches, pineapples, radishes, tarragon, and turnips).


163 Id.

164 See, e.g., Martin v. Cincinnati Gas & Elec. Co., 561 F.3d 439, 443 (6th Cir. 2009) (stating that if every exposure is a substantial exposure then the substantial factor test is meaningless).
occurring substance in many areas of the country. Like asbestos, other carcinogens frequently accumulate in the body and, thus, build up over time, but still may not cause cancer if the built-up levels are not high enough. Much like naturally occurring levels of radon, dioxins, radiation, and other carcinogenic materials, even millions of fibers of these “background” exposures incurred over a lifetime have substantial scientific uncertainty as to whether they have been shown to cause cancer.

Given the reality that at least some degree of dose is necessary before attributing causation to an exposure in asbestos and other contexts, scientists must answer the critical question of how much is enough? They do so by conducting or evaluating existing exposure studies of similar populations, from which they can determine whether those exposures reached an overall dose level comparable to those found to cause disease in epidemiology studies of the same substance and similar exposure circumstances. Accordingly, expert testimony on carcinogens should require a reasonable assessment of the likely range of dose received by the worker and a determination as to whether this dose is comparable to amounts known, and not speculated, to cause disease.


166 See, e.g., Eaton, supra note 143, at 29 (discussing accumulation of dioxin in the human body).

167 See, e.g., id. at 29-30 (discussing the extent of background exposures incurred over a lifetime).

168 See Wright v. Willamette Indus. Inc., 91 F.3d 1105, 1107 (8th Cir. 1996) (explaining there must be minimal evidence in which the factfinder in a case can reasonably conclude that the plaintiff was exposed to certain levels of asbestos as to cause the harm she suffered).

169 McClain v. Metabolife Int’l, Inc., 401 F.3d 1233, 1241 (11th Cir. 2005) (“In toxic tort cases, ‘[s]cientific knowledge of the harmful level of exposure to a chemical, plus knowledge that plaintiff was exposed to such quantities are minimal facts necessary to sustain plaintiff’s burden . . . .’”) (alteration in original) (quoting Allen v. Pennsylvania Eng’g Corp., 102 F.3d 194, 199 (5th Cir. 1996)); Wright, 91 F.3d at 1106-07 (requiring plaintiffs to demonstrate not just some exposure, but “evidence from which a reasonable person could conclude that a defendant’s emission has probably caused a particular plaintiff the kind of harm of which he or she complains before there can be a recovery”).

170 See Wright, 91 F.3d at 1106 (stating that, in order to carry burden of proof, “a plaintiff in a toxic tort case must prove the levels of exposure that are hazardous to
The science behind this is not simple, but the requirement of a dose assessment is as basic as it gets—no one would conclude that taking aspirin caused someone’s death without first at least asking the question how many aspirin are involved.

III. Court Rulings Rejecting Every Exposure Testimony and the Erroneous Basis for Recent Contradictory Opinions

Court review of the every exposure approach started in earnest around 2005 with decisions issued by the United States Court of Appeals for the Sixth Circuit and a trial court in Pennsylvania. Both courts rejected different versions of every exposure testimony. Considering that the every exposure approach to causation had spread virtually unchecked across the asbestos litigation docket before 2005, the cascade of decisions rejecting every exposure between these two 2005 decisions and today’s docket of over forty such decisions is particularly important. Previous articles on this subject provide a case-by-case review of these opinions. Thus, in this section the authors focus on the grounds and reasons for those decisions. Additionally, this section explores the fundamental flaws in the more recent decisions allowing some form of every exposure human beings generally as well as the plaintiff’s actual level of exposure to the defendant’s toxic substance before he or she may recover); see also Butler v. Union Carbide, 712 S.E.2d 537, 550 (Ga. Ct. App. 2011) (Smith & Dillard, JJ., concurring) (quoting Wright, 91 F.3d at 1106 (8th Cir. 1996)) (stating a plaintiff must demonstrate exposure levels and that the levels were hazardous before recovery is permitted).

171 Behrens & Anderson, supra note 2, at 495-96.


173 See Sanders, supra note 1, at 1183-84 (“The ‘every exposure’ theory has enjoyed some success in the past, but . . . today even cases in which the expert did not advocate an ‘any exposure’ standard have been labeled ‘any exposure’ cases and resulted in a defense victory.”).

174 See Behrens, supra note 1, at 529-31 (briefing cases in which courts rejected the every exposure theory); Sanders, supra note 1, at 1176-78 (describing courts’ rationales for rejecting the every exposure theory).
testimony to proceed or by applying a “frequency, regularity, and proximity” of the alleged exposures.175

A. The Reasons Courts Are Rejecting Every Exposure Testimony

The every exposure opinion is not based on scientific process or evidence, and as a result, courts have articulated a myriad of reasons for rejecting its use in the courtroom.176 Some of the more common rejections of the testimony focus on federal or state rules of evidence as typically applied under Daubert v. Merrell Dow Pharmaceuticals, Inc. or Frye v. United States and the scientific unreliability of the theory.177 Other rejections address the notion that every exposure testimony cannot support a finding of “substantial” factor causation to justify a jury verdict.178 However, the rationales supporting rejection of this testimony do not differ significantly even though the discussion is couched in the language of Daubert or Frye in one context and in sufficiency of the evidence language in the other. As the discussion below demonstrates, the flaws in this approach to causation testimony cut across whatever form of review is applied.

1. The Every Exposure Theory’s Failure to Consider Dose

As outlined in Section II above, the central inquiry in a case involving disease caused by toxic exposure is whether the dose of the material is

175 Lohrmann v. Pittsburgh Corning Corp., 782 F.2d 1156, 1163 (4th Cir. 1986).
176 See Sanders, supra note 1, at 1179.
177 See, e.g., Krik v. Exxon Mobil Corp., 870 F.3d 669, 675 (7th Cir. 2017) (stating the plaintiff “failed to bear the burden of demonstrating that [the expert’s] theory would satisfy the minimal requirements of Federal Rule of Evidence 702 and Daubert”); Davidson v. Georgia Pacific L.L.C., No. 12-1463, 2014 WL 3510268, at *5 (W.D. La. July 14, 2014) (“The ‘every exposure’ theory is not testable, and consequently cannot have an error rate, thus failing to satisfy two Daubert factors.”).
178 See, e.g., Martin v. Cincinnati Gas & Elec. Co., 561 F.3d 439, 444 (6th Cir. 2009) (“Plaintiff did not proffer evidence that supports a reasonable inference of exposure from GM products, much less that GM products were a substantial factor in causing Mr. Martin’s mesothelioma.”).
capable of causing the disease. Plaintiffs’ experts routinely testify that asbestos-related diseases are dose-responsive diseases, while also claiming that every exposure regardless of dose is a substantial contributing factor in causing disease. These two concepts are irreconcilable. The inconsistency is a significant reason why courts are rejecting the use of this expert testimony. Virtually every decision on every exposure testimony has criticized these experts for substituting unquantified speculation about the level of exposure (or outright refusal to consider it) in lieu of a proper quantification of the dose involved.

179 See discussion supra Section II.

180 See Sanders, supra note 1, at 1179 (noting that plaintiffs’ experts have claimed “some exposure was a ‘substantial factor’ in causing the plaintiff’s illness”).

181 See id. at 1179-80 (explaining that courts have rejected the every exposure theory when experts “proclaim[] some exposure to be a ‘substantial factor’ in causing the plaintiff’s illness” and when the plaintiffs did not provide “more specific dose evidence”).

182 See, e.g., Stallings v. Georgia-Pacific Corp., 675 F. App’x 548, 551 (6th Cir. 2017) (finding the experts could not quantify the extent of exposure); McIndoe v. Huntington Ingalls Inc., 817 F.3d 1170, 1176-77 (9th Cir. 2016) (“[E]ven if McIndoe was around asbestos dust several times, his heirs presented no evidence regarding the amount of exposure to dust from originally installed asbestos, or critically, the duration of such exposure during any of these incidents. Without such facts, McIndoe’s heirs can only speculate as to the actual extent of his exposure to asbestos from the shipbuilder’s materials.”); Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 862 (E.D.N.C. 2015) (acknowledging an expert failed to establish “any quantitative or qualitative level of reference for exposures”); Comardelle v. Pa. Gen. Ins. Co., 76 F. Supp. 3d 628, 634 (E.D. La. 2015) (referencing the unreliable nature of the expert’s opinion concerning the level of exposure); Wannall v. Honeywell Int’l, Inc., 292 F.R.D. 26, 42 (D.D.C. 2013) (stating that plaintiff’s experts failed to meet the exposure threshold required by Virginia law); In re W.R. Grace & Co., 355 B.R. 462, 493-94 (Bankr. D. Del. 2006) (acknowledging an expert’s opinions must be accepted because the experts presented by opposing counsel based their opinions based on assumptions); Scapa Dryer Fabrics, Inc. v. Knight, 788 S.E. 2d 421, 426 (Ga. 2016) (referencing an expert’s opinion that the amount of exposure to an individual and was not relevant to causation was not helpful to the jury); Butler v. Union Carbide Corp., 712 S.E.2d 537, 541-42 (Ga. App. 2011) (noting that the expert’s methodology was unreliable); In re N.Y. City Asbestos Litig. (Juni II), 148 A.D.3d 233, 239-40 (N.Y. App. Div. 2017) (stating the broad conclusions of an expert were legally insufficient to find the defendant liable, aff’d sub nom. (Juni III), 116 N.E.3d 75 (N.Y. 2018); Howard v. A.W. Chesterton Co., 78 A.3d 605, 608 (Pa. 2013) (holding that an “individualized assessment” of plaintiffs’ exposure history is required in asbestos cases); Bostic v. Georgia-Pacific Corp., 439 S.W.3d 332, 339 (Tex. 2014) (holding that acceptance of the any exposure theory would impose strict liability on individual’s and contradict the plaintiff’s own expert); Borg-Warner Corp. v. Flores, 232 S.W.3d 765, 773 (Tex. 2007) (rejecting the any exposure theory of an expert based on the fact that every individual
Outside of the asbestos context, many courts, including several federal appellate courts, have repeatedly held that the plaintiff must offer proof of an actual toxic dose to prevail in a tort case. The requirement of dose outside of asbestos litigation is so well entrenched at this point, including in the Reference Manual on Scientific Evidence, that it is surprising that asbestos litigation escaped this examination for so long. For a defense attorney used to addressing expert testimony in dioxin, PCBs, benzene, pharmaceutical and other common product and toxic tort litigation, the asbestos every exposure theory seems obvious and inherently suspect. The judicial movement against every exposure theory can be viewed as largely an attempt to bring asbestos litigation

would be exposed under this theory); Smith v. Kelly-Moore Paint Co., 307 S.W.3d 829, 839 (Tex. App. 2010) (“[E]vidence does not support a minimum threshold dose . . . that would increase one’s risk of developing mesothelioma.”); Ford Motor Co. v. Boomer, 736 S.E. 2d 724, 733 (Va. 2013) (acknowledging experts must state their opinion on the threshold of required level of exposure).

See, e.g., In re Bextra & Celebrex Mktg. Sales Prac. & Prod. Liab. Litig., 524 F. Supp. 2d 1166, 1174 (N.D. Cal. 2007) (denying motion to exclude expert testimony and noting studies did not show that the dose at issue could cause the alleged injury and finding “that dose matters”); see also McClain v. Metabolife Int’l, Inc., 401 F.3d 1233, 1241 (11th Cir. 2005) (“In toxic tort cases, ‘[s]cientific knowledge of the harmful level of exposure to a chemical, plus knowledge that [the] plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiff’s burden . . . .’” (quoting Allen v. Pa. Eng’g Corp., 102 F.3d 194, 199 (5th Cir. 1996)); Nelson v. Tenn. Gas Pipeline Co., 243 F.3d 244, 252 (6th Cir. 2001) (upholding exclusion of expert witness who “made no attempt to determine what amount of PCB exposure” the plaintiff received); Mitchell v. Gencorp. Inc., 165 F.3d 778, 781 (10th Cir. 1999) (“[A] plaintiff must demonstrate ‘the levels of exposure that are hazardous to human beings generally as well as the plaintiff’s actual level of exposure to the defendant’s toxic substance before he or she may recover.’” (quoting Wright v. Willamette Indus. Inc., 91 F.3d 1105, 1106 (8th Cir. 1996)); Moore v. Ashland Chem. Inc., 151 F.3d 269, 278 (5th Cir. 1998) (“Because he had no accurate information on the level of Moore’s exposure to the fumes, Dr. Jenkins necessarily had no support for the theory that the level of chemicals to which Moore was exposed caused [reactive airways dysfunction syndrome].”); Abuan v. Gen. Elec. Co., 3 F.3d 329, 333 (9th Cir. 1993) (“In cases claiming personal injury from exposure to toxic substances, it is essential that the plaintiff demonstrate that she was, in fact, exposed to harmful levels of such substances” (quoting Maddy v. Vulcan Materials Co., 737 F. Supp. 1528, 1533 (D. Kan. 1990))).


185 See Dugas v. 3M Co., No. 3:14-cv-1096-J-39JBT, 2016 WL 7246096, at *2-3 (M.D. Fla. Jan. 11, 2016) (defining the every exposure theory as “the greater one’s exposure to asbestos the more likely that person will develop an asbestos related disease”).
back into the world of well-accepted scientific principles and court requirements, including the fundamental requirement of a dose assessment.\textsuperscript{186}

2. The Speculative and Litigation Basis for Every Exposure Testimony

Many courts have reacted to the speculative approach used by the every exposure experts by holding that the testimony is improperly

\textsuperscript{186} See, e.g., Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 849-50 (E.D.N.C. 2015) (applying general dose principles from other Fourth Circuit cases to asbestos “special exposure” opinion).

For dose requirements in other contexts, see, for example, Zellers v. NexTech Ne., L.L.C., 533 F. App’x 192, 198 (4th Cir. 2013) (affirming exclusion of expert for failure to demonstrate plaintiff’s actual level of exposure); Roche v. Lincoln Prop. Co., 278 F. Supp. 2d 744, 754 (E.D. Va. 2003) (excluding an expert upon finding that he “lack[ed] any knowledge of the levels of exposure to mold required to manifest any symptoms”); Cavallo v. Star Enter., 892 F. Supp. 756, 772 (E.D. Va. 1995) (excluding an opinion when the expert “could cite no studies or published literature to support adverse effects from that level of exposure to [the specific toxic agent]” (emphasis added)), aff’d in part, rev’d in part, 100 F.3d 1150 (4th Cir. 1996); Butler v. Union Carbide Corp., 712 S.E.2d 537, 550 (Ga. Ct. App. 2011) (stating that the plaintiff carries the burden of demonstrating that his exposure level to the toxic substance was hazardous); In re N.Y. City Asbestos Litig. (Juni I), 11 N.Y.S.3d 416, 432 (N.Y. Sup. Ct. 2015) (applying a standard that requires a consideration that the dose is sufficient to cause disease), appeal dismissed, (Juni II), 148 A.D. 3d 233 (N.Y., App. Div. 2017), aff’d sub nom. (Juni III), 116 N.E.3d 75 (N.Y. 2018); Borg-Warner Corp. v. Flores, 232 S.W.3d 765, 771-72 (Tex. 2007) (“[A]bsent any evidence of dose, the jury could not evaluate the quantity of respirable asbestos to which [plaintiff] might have been exposed or whether those amounts were sufficient to cause [disease].”).

Plaintiffs routinely contend that it is not possible to quantify the dose of a plaintiff whose exposures occurred years ago and were not specifically measured by defendants at that time. The argument that it is not possible to “precisely quantify” plaintiff’s exposures is a red herring. Some quantification is required, and it is neither difficult nor impossible to develop at least a range of an estimated dose. Precision is not required, but some effort to quantify is. See Stallings v. Georgia-Pacific Corp., 675 F. App’x 548, 551 (6th Cir. 2017) (noting the failure of two experts to “quantify the extent of Mr. Stallings’ exposure”); Moeller v. Garlock Sealing Techs., L.L.C., 660 F.3d 950, 955 (6th Cir. 2011) (finding the plaintiff presented “no evidence quantifying” the exposures); In re N.Y. City Asbestos Litig. (Juni II), 148 A.D. 3d 233, 239 (N.Y. App. Div. 2017) (stating plaintiffs must present at least “some quantification of the amount, duration, and frequency of exposure”), aff’d sub nom. (Juni III), 116 N.E.3d 75 (N.Y. 2018); Flores, 232 S.W.3d at 771-72 (recognizing the potential for proof difficulties and stating plaintiff’s experts must still provide some quantification of the asbestos exposure).
founded on the lack of evidence instead of on actual evidence of causation. The every exposure theory is derived from an entirely untested and untestable assumption—that any exposure to asbestos, regardless of dose, contributes to the development of mesothelioma. When pressed, as described in the cases discussed below, the experts are seemingly forced to admit that there is no epidemiology supporting their position regarding low exposures. Essentially, they assume the very issue that should be proven—that minimal exposures produce cancers like mesothelioma because the exposure occurred and plaintiff has mesothelioma.

Starting with Free v. Ametek, courts began to reject every exposure testimony as a speculative and unproven hypothesis:

Conventional wisdom is that there is no safe level of exposure to asbestos. A more accurate statement of conventional wisdom, however, would be that there is no known safe level of exposure, just as there is no known threshold level for causation of asbestos-related disease. Dr. Hammar’s hypothesis, therefore, while persuasive in lay, “common sense” terms, is not supported by replicable, scientific methodology. . . . [T]he assumption that every exposure to asbestos over a life’s work history, even every exposure greater than 0.1 fibers/cc yr, is a substantial factor contributing to development of an asbestos-related disease, is not a scientifically proved proposition that is

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187 See, e.g., Butler, 712 S.E.2d at 551 (“The claim that there is no known safe level of exposure does not mean that none exists.”).

188 See, e.g., Krik v. Exxon Mobil Corp., 870 F.3d 669, 675 (7th Cir. 2017) (“Experts . . . had not presented any individualized analysis of the level of asbestos exposure, had provided only generalized citations to scientific literature with no indication that they were authorities upon which the experts would rely, did not identify any peer-reviewed scientific journal adopting this theory, did not cite any medical studies or discuss an error rate.”) (citing Krik v. Crane Co., 76 F. Supp. 3d 747, 754 (N.D. Ill. 2014)); Vedros v. Northrup Grumman Shipbuilding, Inc., 119 F. Supp. 3d 556, 564 (E.D. La. 2015) (finding plaintiffs did not show the “‘above background’ theory is testable, published in peer-reviewed works, or has any error rate”); Yates, 113 F. Supp. 3d at 862 (finding the complainants did not show that expert testimony and evidence espousing this theory had the sufficient support of facts or data, was testable, was published in peer-reviewed works, or had any error rate); Bostic v. Georgia-Pacific Corp., 439 S.W.3d 332, 358 (Tex. 2014) (“None of the peer-reviewed scientific studies on which Plaintiffs’ experts relied found a statistically significant link between mesothelioma and occasional exposure to joint compounds comparable to [plaintiff’s] exposure.”).

generally accepted in the field of epidemiology, pulmonary pathology, or any other field relevant to this case.\textsuperscript{190}

A Georgia appellate court in 2011 further examined the underpinnings of every exposure testimony, finding that the opinions of plaintiffs’ expert Dr. John Maddox were “at most, scientifically-grounded speculation: an untested and potentially untestable hypothesis” which did not pass Daubert muster.\textsuperscript{191} Unproven hypotheses should not form the basis for courtroom expert testimony.\textsuperscript{192}

Instead of a scientific foundation, every exposure testimony is derived from litigation and intended to provide the broadest support for ongoing asbestos litigation regardless of the decreasing validity of exposure testimony.\textsuperscript{193} The only exposures typically excluded from the causation opinion are “background” exposures, which could not in any event serve as a basis to sue someone.\textsuperscript{194} In contrast, virtually any identifiable contact with an asbestos product, even if remote and secondary, is included and thus companies making those products are routinely sued.\textsuperscript{195}

The litigation basis of expert testimony was one of the key concerns of the Daubert Court, which feared that speculative testimony not found in published literature could cause litigation to move well outside the paths of demonstrated associations in the science itself.\textsuperscript{196} The Daubert opinion thus included a requirement that the methodology behind the

\textsuperscript{190} Free, 2008 WL 728387.


\textsuperscript{192} See Sanderson v. Int’l Flavors & Fragrances, Inc., 950 F. Supp. 981, 1003 (C.D. Cal. 1996) (“When a plaintiff can’t prove her case with reliable scientific evidence, she can’t prove her case.”).

\textsuperscript{193} See Butler, 712 S.E.2d at 552 (holding expert witness testimony was “scientifically-grounded speculation”).

\textsuperscript{194} See id. at 538-40.

\textsuperscript{195} See id. at 540.

testimony be found in peer-reviewed, published literature. The every exposure and cumulative exposure experts run afoul of this concern. As several courts have noted, no studies in the peer-reviewed literature state as scientific fact that every “cumulative” occupational exposure to asbestos—no matter how brief or small—must be considered a cause of mesothelioma. Any such statement would run contrary to the established principles of cancer causation as set out in the article by Dr. Eaton and the Reference Manual on Scientific Evidence discussed above. Furthermore, none of the experts who routinely provide such opinions

197 Id.

198 Sclafani v. Air & Liquid Sys. Corp., No. 2:12-cv-3013-SVW-PJW, 2013 WL 2477077, at *5 (C.D. Cal. May 9, 2013) (noting the plaintiff’s expert conceded that the every exposure theory had not been published in any peer-review literature); Vedros v. Northrup Grumman Shipbuilding, Inc., 119 F. Supp. 3d 556, 564 (E.D. La. 2015) (finding the plaintiffs did not show that the “above background” theory is testable, published in peer-reviewed works, or has any error rate” as required by Daubert); Rockman v. Union Carbide Corp., 266 F. Supp. 3d 839, 848 (D. Md. 2017) (“Plaintiffs’ experts . . . have failed to support their specific causation opinions with ‘sufficient facts or data’ or a ‘testable,’ ‘peer-reviewed’ theory that is ‘generally accepted’ within the scientific community.”).

Plaintiffs’ experts often cite to the “Helsinki Criteria” for the proposition that cumulative low doses of exposure can cause disease. A. Tossavainen, Consensus Report, Asbestos, Asbestosis, and Cancer: The Helsinki Criteria for Diagnosis and Attribution, 23 SCAND. J. WORK ENV’T & HEALTH 311, 314 (1997). However, the Helsinki Criteria requires that a past exposure to asbestos be “significant” before mesothelioma can be attributed to the asbestos exposure. Id. Thus, it implies that a certain level has been established at which the asbestos exposure attains “significance.” See, e.g., Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 861-62 (E.D.N.C. 2015) (noting Dr. Eugene Mark inappropriately relied on the Helsinki Criteria for his causation opinion). The Seventh Circuit Krik decision contains a strong rejection of an attempt to have the Helsinki Criteria admitted as evidence. Krik v. Exxon Mobil Corp., 870 F.3d 669, 678 (7th Cir. 2017) (citing other decisions rejecting this document).

Several plaintiff experts have cited to a particular “article.” See Laura S. Welch, Asbestos Exposure Causes Mesothelioma, but Not This Asbestos Exposure: An Amicus Brief to the Michigan Supreme Court, to support every exposure testimony. 13 INT’L J. OCCUPATION & ENV’T HEALTH 318 (2007). However, the Yates court and others have rejected this paper as a basis for expert testimony because it is a litigation brief and not a true scientific article. See Yates, 113 F. Supp. 3d at 846-47 (“The court has previously held that this document, which was initially prepared for purposes of litigation, is not one that ‘experts in the particular field would reasonably rely on’ for purposes of satisfying Federal Rule of Evidence 703.”) (citing Yates v. Ford Motor Co., No. 5:12-CV-752-FL, 2015 WL 3463559, at *9-11 (E.D.N.C. May 30, 2015)); accord Rockman, 266 F. Supp. 3d at 848; Vedros, 119 F. Supp. 3d at 564.

199 See Eaton, supra note 143, at 38-40; Green et al., supra note 39, at 552.
have ever published an article that articulates the scientific basis for their litigation opinion that any exposure is causative, nor have they submitted this form of opinion for peer review in recognized scientific journals. As the court observed in Daubert II, “[i]t’s as if there were a tacit understanding within the scientific community that what’s going on here is not science at all, but litigation.”

3. The Lack of Scientific Support and the *Ipse Dixit* Nature of the Testimony

The United States Supreme Court’s decision in *General Electric Co. v. Joiner*, which followed the Daubert opinion to provide a further explanation of the Daubert opinion’s impact, excluded testimony in part because it was founded on the expert’s own say-so, not in the actual science. Since then, many forms of what the court termed *ipse dixit* testimony—“it is because I say it is”—have been excluded in many contexts. The fundamental inquiry into expert causation testimony

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200 As one example, Dr. Arthur Frank utilizes a very long affidavit with citations to hundreds of studies, but none is his own published and peer-reviewed articulation of his courtroom testimony. See, e.g., Rockman, 266 F. Supp. 3d at 846; Suoja v. Owens-Ill., Inc., 211 F. Supp. 3d 1196, 1202-05 (W.D. Wis. 2016).


203 Joiner, 522 U.S. at 147.

needs to include an investigation into whether the expert is legitimately drawing from the cited studies or extending the testimony well beyond those studies to a personal opinion not in fact found in the literature.205

The every exposure experts have often run up against the ban on ipse dixit testimony.206 Virtually every court that has looked behind the curtain of claimed studies and government publications these experts raise to protect themselves has found that the experts’ statements are not supported by anything other than the experts’ own statements.207 The cited literature is typically irrelevant or inconsistent with the testimony or causation principle that the experts state in testimony.208 The following offer examples of key rulings on this front.

• The “no safe dose” argument. Plaintiff’s experts often support their testimony by relying on the notion that there is “no safe dose” of asbestos.209 The support for these statements is often taken from regulatory documents and other health assessments that do not assess

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205 Joiner, 522 U.S. at 146-47.


208 See, e.g., Krik, 870 F.3d at 675 (“[T]he experts . . . had provided only generalized citations to scientific literature with no indication that they were authorities upon which the experts would rely, did not identify any peer-reviewed scientific journal adopting this theory, did not cite any medical studies or discuss an error rate.” (citing Krik v. Crane Co., 76 F. Supp. 3d 747, 754 (N.D. Ill. 2014))); Rockman v. Union Carbide Corp., 266 F. Supp. 3d 839, 846 (D. Md. 2017) (“[B]oth experts have improperly drawn conclusions about this case, a case involving peritoneal mesothelioma and low-level bystander exposure to chrysotile asbestos, based on prior research studying pleural mesothelioma and primarily high-level exposures to amphibole asbestos.”); Comardelle, 76 F. Supp. 3d at 635 (“[P]laintiffs refer cursorily to a broad array of cases, studies, and regulatory materials. Suffice it to say, plaintiffs overstate or misstate the relevance of these sources.”); Vedros, 119 F. Supp. 3d at 564-65 “Plaintiffs refer cursorily to a broad array of cases, studies, and regulatory materials . . . [but] overstate or misstate the relevance of the sources cited.” (citations omitted)).

the degree of exposure needed to prove causation and are thus irrelevant to a causation determination.\textsuperscript{210}

As one New York court held, the assumption that there is “no safe dose” of a toxin is not a substitute for the required quantification of an individual plaintiff’s exposure, and “the reports and findings of governmental agencies [declaring there to be no safe dose of asbestos] are irrelevant as they constitute insufficient proof of causation.”\textsuperscript{211} As the court stated, these experts’ reliance on a “no safe dose” theory fundamentally exposes their inability to prove causation.\textsuperscript{212} This approach is not a proper basis for a causation opinion, as several courts have held, including, for example, a federal court in Louisiana: “Although there may be no known safe level of asbestos exposure, this does not support Dr. Hammar’s leap to the conclusion that therefore every exposure Comardelle had to asbestos must have been a substantial contributing cause of his mesothelioma.”\textsuperscript{213}

\textsuperscript{210} 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 v. Ford Motor Co., 113 F. Supp. 3d 841, 847 (E.D.N.C. 2015) (citing cases distinguishing regulatory pronouncements of “no safe dose” from causation standard).


\textsuperscript{212} Id. at 434.

\textsuperscript{213} Comardelle, 76 F. Supp. 3d at 634; see also Butler v. Union Carbide Corp., 712 S.E.2d 537, 551 n.37 (Ga. Ct. App. 2011) (citing Parker v. Mobil Oil Corp., 857 N.E.2d 1144, 1122 (N.Y. 2006) for its correct rejection of reliance on regulatory pronouncements); Bostic v. Georgia-Pacific Corp., 439 S.W.3d 332, 358 (Tex. 2014) (“[T]he failure of science to isolate a safe level of exposure does not prove specific causation.”). 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 also Comardelle, 76 F. Supp. 3d at 634 (finding the expert testimony unreliable and inadmissible because the expert did not rely on specific facts or circumstances of the plaintiff’s exposure, but the expert instead relied on general studies); Wannall v. Honeywell Int’l, Inc., 292 F.R.D. 26, 41-42 (D.D.C. 2013) (discussing the distinction between risk and cause when looking at levels of exposure); Bartel v. John Crane, Inc., 316 F. Supp. 2d 603, 611 (N.D. Ohio 2004) (disagreeing with experts that “every exposure to asbestos . . no matter how small, was a substantial factor in causing [Plaintiff’s] peritoneal mesothelioma”); In re W.R. Grace & Co., 355 B.R. 462, 493 (Bankr. D. Del. 2006) (holding “that [Zonolite Attic Insulation] does not pose an unreasonable risk of harm”); Crane Co. v. DeLisle, 206 So. 3d 94
“A single day of exposure can cause mesothelioma.” This is one of the every exposure experts’ most widely cited propositions, sometimes alternatively phrased as “only a short exposure” or “minimal exposure.” Yet the statement is not accurate as a scientific principle and, at best, conflates extremely heavy exposures to the most potent forms of asbestos with the categorically different kinds of exposures appearing in today’s litigation. The courts that have looked at this type of reasoning have concluded that the experts jump, without foundation, from the dramatic exposures in some studies to the minimal exposures at issue in the case. As the bankruptcy court found in In re Garlock Sealing Technologies, L.L.C., “[a] fundamental flaw in [the experts’] analyses is that the studies on which they rely all involve people in very high exposure settings—such as miners or manufacturing/textile workers.” One court referred to this exercise

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214 As only one example, Dr. Frank and other similar experts often claim that very short exposures to asbestos, even as small as one day, have been shown to cause mesothelioma. Rost v. Ford Motor Co., 151 A.3d 1032, 1039-40 (Pa. 2016).

215 See, e.g., Yates, 113 F. Supp. 3d at 853 (“The parties agree that amphibole asbestos is more potent than chrysotile asbestos, and that higher levels of exposure to chrysotile asbestos than amphibole asbestos are necessary to cause mesothelioma.”). Case reports are notoriously unreliable for deriving causation because they utilize no control group for comparison. See Betz v. Pneumo-Abex, L.L.C., 44 A.3d 27, 55-56 (Pa. 2012) (“Appellee’s efforts to invoke case reports, animal studies, and regulatory standards are also ineffectual in terms of substantial-factor causation, since the most these can do is suggest that there is underlying risk from the defendants’ products . . . .”).

216 See, e.g., Yates, 113 F. Supp. 3d at 852-53 (finding that an expert claiming “that visible dust rises to the level of being ‘hazardous to human beings generally’ for purposes of establishing causation” is not sufficient) (quoting Westberry v. Gislaved Gummi AB, 178 F.3d 257, 263 (4th Cir. 1999)); Betz, 44 A.3d at 55 (finding that a mere statement that there is an “underlying risk from the defendants’ products” is not sufficient); Gregg v. A-J Auto Parts, Co., 943 A.2d 216, 226 (Pa. 2007) (holding that “generalized [every exposure] opinions do not suffice to create a jury question in a case where exposure to the defendant’s product is de minimis”).

as trying to cross the Mississippi with “small bridges.” The authors of these studies do not themselves conclude that exposures as short as one day actually cause mesothelioma—that is an opinion incorrectly drawn from them by the testifying experts.

The cited studies also typically involve exposures to extreme amphiboles or mixed fiber exposures to support causation testimony in low-exposure chrysotile litigation. To illustrate, one of the favorite sources cited for this proposition is the International Agency for Research on Cancer’s (IARC) asbestos monograph.

This asbestos monograph cites to an article reporting on two mesotheliomas occurring in employees who worked with crocidolite (not chrysotile) in an enclosed hut from 1928-1929, with no ventilation and in conditions so dusty they could barely see across the room.

• The circular reasoning supporting every exposure testimony. Several courts have held that the every exposure experts’ opinions

218 Betz, 44 A.3d at 48.

219 See, e.g., Morris Greenberg & T.A. Lloyd Davies, Mesothelioma Register 1967-68, 31 BRIT. J. INDUS. MED. 91, 103 (1974) (“[T]he briefest occupational exposure to asbestos associated with a mesothelial tumour was three weeks, but if asbestos was a cause of mesothelioma it cannot be assumed that lesser exposures are safe.”). This statement reflects a hypothesis, with no reference to fiber type or degree of exposure, but experts like Dr. Frank rely on it as a finding supporting low dose chrysotile exposures.

220 See, e.g., Yates, 113 F. Supp. 3d at 853 (referring to the parties’ studies that “that amphibole asbestos is more potent than chrysotile asbestos, and that higher levels of exposure to chrysotile asbestos than amphibole asbestos are necessary to cause mesothelioma”).

221 The Occupational Safety and Health Administration (OSHA) published an informal web page in the summer of 2014 that cites four articles as support for the “few days” statement. None of the referenced articles make this statement. The closest relevant information—the referenced IARC publication (n.6)—refers to exposures of one year to crocidolite asbestos (the most potent form) by workers who toiled in a small “fiber hut” with no ventilation or personal protection. “The work there caused so much dust that the employees could barely see across the room.” Bjorn Hilt et al., Occurrence of Cancer in a Small Cohort of Asbestos-Exposed Workers, 7 SCAND. J. WORK & ENVTL. HEALTH 185, 185 (1981); see U.S. Dep’t Lab. Asbestos, OSHA, https://www.osha.gov/SLTC/asbestos (last visited Sept. 23, 2018).

engage in classic circular reasoning—if the plaintiff has mesothelioma, and mesothelioma is caused by asbestos exposure, then it must have been this plaintiff’s exposures, no matter how minimal, that caused his disease. 223 As one example, the experts often testify that “when you diagnose mesothelioma, it is considered caused by asbestos until proven otherwise.” 224 Several courts have criticized this reasoning as “entirely circular” and unsupported. 225

• The experts’ refusal to exclude non-asbestos, spontaneous causes. Mesothelioma is widely associated with certain types of asbestos exposures presumably because the research has focused on the occurrence of this disease in heavily-exposed asbestos workers. But like all cancers, mesothelioma has other known causes, 226 and is also likely produced by nothing more than the human body’s own DNA transcription errors that accumulate in certain cells over time. 227 In women in particular, some studies have estimated that as many as eight out of ten mesothelioma diagnoses occurring today are likely spontaneously induced and have nothing to do with asbestos. 228 Thus,

223 Yates, 113 F. Supp. 3d at 856-58.
225 See Yates, 113 F. Supp. 3d at 856 (holding that expert testimony about the each and every exposure theory was “entirely circular and conclusory” and unhelpful to the jury); Butler v. Union Carbide Corp., 712 S.E.2d 537, 550-51 (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.”) (quoting Mancuso v. Consol. Edison Co. of New York, Inc., 967 F. Supp. 1437, 1450 (S.D.N.Y. 1997)).
226 See Katherine D. Crew et al., Malignant Mesothelioma Following Radiation, in MALIGNANT MESOTHELIOMA ADVANCES IN PATHOGENESIS, DIAGNOSIS, AND TRANSLATIONAL THERAPIES 350 (Harvey I. Pass et al eds., 2005) (“Radiation, non-asbestos mineral fibers, organic chemicals, chronic inflammation, and simian virus 40 exposure have also been suggested as risk factors for mesothelioma in humans.”).
227 See Weinberg, supra note 33, at 89-90 (explaining the process of serial mutations in cells that can produce a cancer).
228 See, e.g., H. Weill et al., Changing Trends in US Mesothelioma Incidence, 61 OCCUPATION ENVTL. & MED. 438, 440 (2004) (“[O]nly about 20% of all mesotheliomas in women in the United States can be reasonably linked to past asbestos exposure.”). Several books and published articles support the human body’s DNA transcription process’s capacity to produce cancers without outside influence. See Cristian Tomasetti & Bert Vogelstein, Variation in Cancer Risk Among Tissues Can Be Explained by the Number of Stem Cell Divisions, 347 SCI. 78, 78 (2015) (discussing
mesothelioma in a female plaintiff with minimal exposure, which is typical in younger groupings of women alive today, is far more likely to be a case of a spontaneously-induced disease.\textsuperscript{229}

cancers that result from “random mutations arising during DNA replication in normal, noncancerous stem cells); Stanley Venitt, \textit{Mechanisms of Spontaneous Human Cancers}, 104 ENVTL. HEALTH PERSP. 633, 633-35 (1996) (discussing cancers that are unavoidable because they are spontaneous and “arise from endogenous processes”); WEINBERG, \textit{supra} note 33, at 59 (“Even the best-functioning cells will occasionally miscopy one in a million (or ten million) bases during each cycle of DNA replication.”).

The medical literature documents the existence of spontaneous cases of mesothelioma. British Thoracic Society, \textit{BTS Statement on Malignant Mesothelioma in the UK, 2007, 62 THORAX ii1, ii1 (2007)} (“[T]he rate of ‘spontaneous’ mesotheliomas each year . . . is around one per million . . . .”); J.E. Craighead, \textit{Epidemiology of Mesothelioma and Historical Background, in MALIGNANT MESOTHELIOMA 13, 13 (A. Tannapfel ed. 2011)} (“Many cases of mesothelioma are idiopathic, while some are caused by therapeutic irradiation or chronic inflammation in body cavities.”); Alastair J. Moore \textit{et al.}, \textit{Malignant Mesothelioma, 3 ORPHANET J. RARE DISEASES 34, 35 (2008)} (“Idiopathic or spontaneous mesothelioma can also occur in the absence of any exposure to asbestos in both animals and humans, and a recent review suggests a spontaneous mesothelioma rate in humans of around one per million.”); B.T. Mossman \textit{et al.}, \textit{Asbestos: Scientific Developments and Implications for Public Policy, 247 SCI. 294, 295 (1990)} (“Approximately 20 to 30% of mesotheliomas occur in the general population in adults not exposed occupationally to asbestos.”); Robert Spirtas \textit{et al.}, \textit{Malignant Mesothelioma: Attributable Risk of Asbestos Exposure, 51 OCCUPATION & ENVTL. MED. 804, 807 (1994)} (noting eleven percent of the study’s mesothelioma cases had no known source of asbestos exposure); Weill \textit{et al.}, \textit{supra}, at 440 (“Only about 20% of all mesotheliomas in women in the United States can be reasonably linked to past asbestos exposure.”); see also Butler, 712 S.E.2d at 542 (affirming the trial court’s exclusion of expert testimony that the decedent’s occupational exposure to asbestos was causative to decedent’s subsequently developing mesothelioma as based on unreliable methodology because scientific literature does not support the conclusion that one’s risk of developing mesothelioma is increased by exposure to chrysotile asbestos fibers).

In addition, there seems to be a current focus on the BAP1 gene as a possible precursor to mesothelioma, and the issue is in the beginning phases of being part of asbestos litigation. Given advances in genetic research, it is highly likely that additional gene mutations will be identified as associated with or in fact the cause of human mesothelioma. \textit{See, e.g.}, Michelle Carbone & Haining Yang, \textit{Mesothelioma: Recent Highlights, 5 ANNALS OF TRANSLATIONAL MED. 238, 240 (2017)} (“[W]e discovered that germline BAP1 truncating mutations caused a very high incidence of mesothelioma in some US families in the absence of occupational asbestos exposures.”).

\textsuperscript{229} Rake \textit{et al.}, \textit{Occupational, Domestic and Environmental Mesothelioma Risks in the British Population: A Case-Control Study, 100 BRITISH J. CANCER 1175, 1175 (2009)} (noting, of the study’s mesothelioma cases, “14% of male and 62% of female cases were not attributable to occupational or domestic asbestos exposure”). For a discussion of the different trend lines for mesothelioma, see Anderson, \textit{supra} note 81, at 113-15.
The *every exposure* experts refuse to acknowledge this reality and continue to speculate that the smallest of exposures—such as the “five to ten” brake jobs of the father in *Schwartz v. Honeywell International, Inc.*\(^{230}\)—are the causes of female disease, even from mere “take-home” exposures. A diagnosis that fails to rule out non-asbestos induced disease as a cause of plaintiff’s condition should not be considered a legitimate diagnosis.\(^{231}\)

• *Visible dust as a surrogate for a dose assessment.* The *every exposure* experts routinely rely on the asserted presence of “dust” in the plaintiffs’ or workers’ environment to claim that the exposures were significant, but again with no assessment of the actual dose involved in that dust exposure.\(^{232}\) In the *New York City Asbestos Litigation* case, for instance, the two experts relied on testimony that “dust” was created by the few brake jobs, and that any sign of visible dust meant that the exposures were far above OSHA standards.\(^{233}\)

A number of courts have heavily criticized the experts’ reliance on testimony of “visible dust” in lieu of a professional assessment of exposure and dose.\(^{234}\) It is, of course, extremely easy for a plaintiff to

\(^{230}\) 102 N.E.3d 477, 479 (Ohio 2018).

\(^{231}\) See *Union Carbide Corp. v. Synatzke*, 438 S.W.3d 39, 48 (Tex. 2014) (holding that a physician must rule out non-asbestos causes of injury when alleging asbestos exposure caused the plaintiff injury (citing TEX. CIV. PRAC. & REM. CODE ANN. § 90.010(f) (West 2015))).

\(^{232}\) See, e.g., *Yates v. Ford Motor Co.*, 113 F. Supp. 3d 841, 853 (E.D.N.C. 2015) (rejecting Dr. Mark’s reliance on “visible dust” as insufficient to prove a causative dose); *In re N.Y. City Asbestos Litig. (Juni I)*, 11 N.Y.S.3d 416 (N.Y. Sup. Ct. 2015) (noting neither expert witness could provide a scientific showing that the dust at issue contained amounts of asbestos sufficient to cause mesothelioma), appeal dismissed, *(Juni II)*, 148 A.D. 3d 233 (N.Y. App. Div. 2017) (Feinman, J., dissenting) (describing the defendant’s position that the “presence of dust” by itself does not prove “that a hazardous dosage of asbestos fibers was inhaled” and concluding that the verdict favoring the defendant was incorrect), *aff’d sub nom. (Juni III)*, 116 N.E.3d 75 (N.Y. 2018); *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 774 (Tex. 2007) (stating evidence of causation was legally insufficient because there was no evidence as to the content of the dust or the “approximate quantum of fibers” to which the plaintiff was exposed).

\(^{233}\) *In re N.Y. City Asbestos Litig. (Juni I)*, 11 N.Y.S.3d at 420-22.

\(^{234}\) *Id.* at 435; see also *Yates*, 113 F. Supp. 3d at 853 (rejecting the “dust” approach to causation in asbestos cases); *Flores*, 232 S.W.3d at 774 (rejecting reliance on testimony regarding clouds of visible dust as a substitute for the “approximate quantum” of actual exposure).
claim that “dust” was created by some activity—many activities create some amount of dust, including ordinary house cleaning. But dust from an asbestos-related product could well contain no or very little asbestos. 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.

4. The Failure of Every Exposure Testimony to Satisfy the Substantial Factor Causation Standard

Many courts have rejected every exposure testimony as a legal matter because that theory is in conflict with and cannot satisfy a substantial factor or similar causation test. Causation is “an essential element of

235 See, e.g., Yates, 113 F. Supp. 3d at 854-55 (discussing how “visible dust” testimony ignores differences in asbestos types and potency and how there is a limited amount of asbestos in brake dust); In re N.Y. City Asbestos Litig. (Juni I), 11 N.Y.S.3d at 435-36 (discussing expert testimony that brake “dust” contained “99 percent” non-toxic, non-asbestos material).

236 See Yates, 113 F. Supp. 3d at 855 (discussing plaintiff experts’ erroneous and misleading fiber calculations that were based on visible dust).

any tort claim.” The *Restatement (Second) of Torts* provides that a plaintiff must provide sufficient evidence to conclude that the actor’s conduct was a *substantial factor* in bringing about the resulting harm. The “substantial factor” causation standard has been adopted in some form in most jurisdictions, primarily to avoid attributing liability to minimal or “insubstantial” contributors. The substantial factor standard keeps the burden of proving causation where it has always belonged—on plaintiffs, who must demonstrate a causative dose. The numerous

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238 See *Lohrmann v. Pittsburgh Corning Corp.*, 782 F.2d 1156, 1162-63 (4th Cir. 1986) (holding that to show substantial factor causation, one must provide evidence of frequent, regular, and proximate contact with asbestos); *see also* *Jones v. Owens-Corning Fiberglass Corp.*, 69 F.3d 712, 716 (4th Cir. 1995) (applying the *Lohrmann* test and North Carolina law); *Jackson v. Anchor Packing Co.*, 994 F.2d 1295, 1303 (8th Cir. 1993) (applying the “frequency, regularity, and proximity” test); *Slaughter v. S. Tale Co.*, 949 F.2d 167, 171 (5th Cir. 1991) (adopting the *Lohrmann* test as a minimum showing of causation); *Sheffield v. Owens-Corning Fiberglass Corp.*, 595 So.2d 443, 451 (Ala. 1993) (holding that, in order to show causation, one must show that asbestos products manufactured by defendant were aboard the ship on which each plaintiff worked); *Thacker v. UNR Indus.*, 603 N.E.2d 449, 457 (Ill. 1992) (adopting *Lohrmann*’s “frequency, regularity, and proximity” test); *Eagle-Picher Indus. Inc. v. Balbos*, 604 A.2d 445, 460 (Md. 1992) (finding factors to be considered include “the nature of the product, the frequency of its use, the proximity, in distance and in time, of a plaintiff to the use of a product, and the regularity of the exposure of that plaintiff to the use of that product”); *Gorman-Rupp Co. v. Hall*, 908 So. 2d 749, 757 (Miss. 2005) (adopting the “frequency, regularity, and proximity test” for asbestos litigation); *James v. Bessemer Processing Co.*, 714 A.2d 898, 911 (N.J. 1998) (holding that plaintiff can establish causation with “factual proof of the plaintiff’s frequent, regular, and proximate exposure to a defendant’s products”); *Gregg v. V-J Auto Parts, Co.*, 943 A.2d 216, 227 (Pa. 2007) (holding it was appropriate for courts to assess “evidence concerning frequency, regularity, and proximity of [the] asserted exposure”); *Henderson v. Allied Signal, Inc.*, 644 S.E.2d 724, 727 (S.C. 2007) (adopting *Lohrmann*’s “frequency, regularity, and proximity test”); *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 770 (Tex. 2007) (holding that *Lohrmann*’s “frequency, regularity, and proximity” test is appropriate” and that it “requires that the exposure be a ‘substantial factor’ in causing the disease”); *Allen v. Asbestos Corp.*, 157 P.3d 406, 410-11 (Wash. Ct. App. 2007) (finding that “sales record evidence was not enough to support an inference” but should instead be evaluated “in light of the totality of the evidence”).
courts holding that every exposure testimony is insufficient to support a finding of substantial factor causation have recognized that plaintiffs in these cases fail to segregate meaningful or “substantial” exposures from those that are not. This testimony removes the “substantial” from substantial contributing factor and renders it meaningless. The United States Court of Appeals for the Sixth Circuit concluded the following:

While [the decedent’s] exposure to Garlock gaskets may have contributed to his mesothelioma, the record simply does not support an inference that it was a substantial cause of his mesothelioma. Given that the Plaintiff failed to quantify [the decedent’s] exposure to asbestos from Garlock gaskets and that the Plaintiff concedes that [the decedent] sustained massive exposure to asbestos from non-Garlock sources, there is simply insufficient evidence to infer that Garlock gaskets probably, as opposed to possibly, were a substantial cause of [the decedent’s] mesothelioma.

According to the Sixth Circuit, “saying that exposure to Garlock gaskets was a substantial cause of [the decedent’s] mesothelioma would be akin to saying that one who pours a bucket of water into the ocean has substantially contributed to the ocean’s volume.”

Likewise, the Ninth Circuit rejected every exposure causation testimony in a maritime case as “precisely the sort of unbounded liability that the substantial factor test was developed to limit.” The Seventh

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242 See sources cited supra note 237.
244 Moeller v. Garlock Sealing Techs., L.L.C., 660 F.3d 950, 955 (6th Cir. 2011); see also Pluck v. B.P. Oil Pipeline Co., 640 F.3d 671, 679 (6th Cir. 2011) (noting that the presence of a toxin is not enough to demonstrate causation); Martin v. Cincinnati Gas & Elec. Co., 561 F.3d 439, 443 (6th Cir. 2009) (stating that not every exposure to asbestos qualifies as a “substantial factor” otherwise the substantial factor test would become “meaningless”); Lindstrom, 424 F.3d at 493 (finding the plaintiff must demonstrate that the defendant’s product was a “substantial factor” in the injury).
245 Moeller, 660 F.3d at 955; see also Martin, 561 F.3d at 443 (noting the any exposure approach “would make every incidental exposure to asbestos a substantial factor”).
246 McIndoe v. Huntington Ingalls Inc., 817 F.3d 1170, 1177 (9th Cir. 2016).
Circuit has also held that “[r]equiring a defendant to exclude a potential cause of the illness, therefore, improperly shifts the burden to the defendants to disprove causation and nullifies the requirements of the ‘substantial factor’ test.”

State courts have also held that every exposure testimony does not support their versions of substantial factor causation. In the *Betz* opinion, the Supreme Court of Pennsylvania found that that theory was “in irreconcilable conflict with itself” because “one cannot simultaneously maintain that a single fiber among millions is substantially causative, while also conceding that a disease is dose responsive.” The court added: “[W]e do not believe that it is a viable solution to indulge in a fiction that each and every exposure to asbestos, no matter how minimal in relation to other exposures, implicates a fact issue concerning substantial-factor causation in every ‘direct-evidence’ case.”

Similarly, the Supreme Court of Texas in *Borg-Warner Corp. v. Flores*, an asbestos case brought by a retired brake mechanic, rejected the idea that mere proof of exposure is sufficient for causation. The Texas Supreme Court held that, to prove causation, a plaintiff must show “[d]efendant-specific evidence relating to the approximate dose to which the plaintiff was exposed, coupled with evidence that the dose was a substantial factor in causing the asbestos-related disease.” This reasoning was reaffirmed by the Supreme Court of Texas in *Bostic v. Georgia-

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247 Krik v. Exxon Mobil Corp., 870 F.3d 669, 677 (7th Cir. 2017); see also Haskins v. 3M Co., No. 2:15-cv-02086-DCN, 2017 WL 3118017, at *6 (D.S.C. July 21, 2017) (emphasizing the importance of plaintiff’s demonstration that the exposure was a “substantial factor” and that “minimal exposure” is not enough); Bell v. Foster Wheeler Energy Corp., No. 15-6394, 2017 WL 876983, at *2 (E.D. La. Mar. 6, 2017) (noting the difficulty in establishing when exposure qualifies as a “substantial factor”); Smith v. Ford Motor Co., No. 2:08-cv-630, 2013 WL 214378, at *3 (D. Utah Jan. 18, 2013) (finding a lack of evidence demonstrating that plaintiff’s exposure to asbestos was “sufficient” to cause his illness).


249 Id. at 56-57.

250 232 S.W.3d 765, 773 (Tex. 2007) (“[T]he court of appeals erred in holding that ‘[i]n the context of asbestos-related claims, if there is sufficient evidence that the defendant supplied any of the asbestos to which the plaintiff was exposed, then the plaintiff has met the burden of proof.’” (alteration in original) (emphasis omitted) (quoting *Borg-Warner Corp. v. Flores*, 153 S.W.3d 209, 213 (Tex. App. 2004))).

251 Id.
Pacific Corp., which held that “[p]roof of any exposure at all from a defendant should not end the inquiry and result in automatic liability.”

B. Why “Cumulative Exposure” Testimony Is No Different than Every Exposure Testimony

In recent years, the every exposure experts have begun to modify the terminology they use to describe their testimony. The change—as candidly admitted by one such expert—does not reflect any actual change in the science or methodology, but is instead intended to avoid exclusion in the many courts that have rejected every exposure experts. Rather than testify, as virtually all of these experts previously did, that every exposure above background is a cause of mesothelioma, many of these experts for the last several years have begun testifying that all of this plaintiff’s cumulative exposures together are the cause of her mesothelioma. Some of the experts now disavow the very every exposure opinion to which they previously testified, under oath to a reasonable degree of medical certainty, in dozens or hundreds of cases. Plaintiffs’ counsel, relying on the modification in the experts’ terminology, then argue to the court that a motion to exclude every exposure

252 439 S.W.3d 332, 341 (Tex. 2014).

253 See Rockman v. Union Carbide Corp., 266 F. Supp. 3d 839, 849 (D. Md. 2017) (“In fact, Dr. Frank has previously stated that he specifically avoids using the phrase ‘each and every’ in light of court rulings excluding testimony based on that theory.”).

254 Id.


testimony as to these experts is moot and misdirected because the experts are not utilizing the *every exposure* theory.\textsuperscript{257}

This not so subtle avoidance technique has surprisingly had some success.\textsuperscript{258} A few courts have failed to examine the two theories together and simply agreed on the face of the language used—the *cumulative exposure* experts assiduously avoid using the words “every exposure”—that the testimony seems different and will be allowed.\textsuperscript{259} Yet, as discussed below, multiple courts have grasped the nature of the artificial change in title and have held that the migration to the *cumulative exposure* theory is nothing more than semantic gymnastics.\textsuperscript{260} Those opinions now evidence a clear trend rejecting *cumulative exposure* testimony as well as *every exposure* testimony.\textsuperscript{261}

\textsuperscript{257} *Id.* at *3.


\textsuperscript{259} See, e.g., *Quirin*, 2014 WL 716162, at *6-7 (holding that Dr. Brodkin’s cumulative exposure testimony is not the same as the single fiber theory and was admissible).


\textsuperscript{261} See, e.g., *Krik*, 870 F.3d at 673 (affirming the trial court’s decision to exclude “cumulative exposure” testimony because it was “the same as ‘each and every exposure’”); *Haskins*, 2017 WL 3118017, at *6 (D.S.C. July 21, 2017) (finding *cumulative exposure* irrelevant and “any probative value . . . easily outweighed by [its]
In application, the cumulative exposure approach is no different than the every exposure theory, or the single fiber theory, that these same experts formerly articulated to the jury.\(^{262}\) As the Seventh Circuit Court of Appeals held, “the principle behind the ‘each and every exposure’ theory and the cumulative exposure theory is the same—that it is impossible to determine which particular exposure to carcinogens, if any, caused an illness.”\(^{263}\) Under the cumulative exposure theory, every instance of a workplace exposure for any particular plaintiff would be considered causative because all the fibers cumulatively contribute to the lung over a lifetime.\(^{264}\) This is almost exactly the same as the testimony under the every exposure theory—all of a plaintiff’s exposures beyond background exposures are part of the overall causative exposure.\(^{265}\) Based on this login, no exposure can escape being a cumulative exposure, no matter how trivial the exposure. Both the every exposure theory and cumulative formulation theory ignore the dose of a given exposure.\(^{266}\) In addition, both theories shift the burden of proof to the defendants.\(^{267}\) The experts even used the concept of “cumulative exposure” frequently when testifying based on the older every exposure theory to support the notion that every exposure is a cause—so even the new “cumulative” phraseology is actually not new at all.\(^{268}\)

The idea of cumulative exposure can create difficulties for courts. It is, in fact, partially true that asbestos exposures accumulate over a lifetime.\(^{269}\) And thus, there is some facial appeal to the idea that all of

\(^{262}\) Krik, 870 F.3d at 662,\(^{263}\) In re N.Y. City Asbestos Litig. (Juni I), 11 N.Y.S.3d 365, 370 (N.Y. App. Div. 2017).\(^{264}\) Id.\(^{265}\) Id.\(^{266}\) Id.\(^{267}\) Id.\(^{268}\) Id. at 676; see also McIndoe v. Huntington Ingalls Inc., 817 F.3d 1170, 1177 (9th Cir. 2016) (rejecting the argument that every exposure to asbestos above a threshold level is necessarily a substantial factor).\(^{269}\) Krik, 870 F.3d at 673 (citing Krik v. Owens-Ill., Inc., No. 10-cv-07435, 2015 WL 5050143, at *1 (N.D. Ill. Aug. 25, 2015)).
the fibers that get into the lung should count toward causation and this can likely even result in plaintiffs eliciting testimony from a defense expert agreeing to the principle that asbestos diseases result from cumulative exposure over time.

But there are two fundamental flaws with claiming that any amount of fibers contributed to the lungs also contributes to causation. First, not all such fibers remain in the lungs. All asbestos fiber types have a half-life reflecting the period of time needed for half the dose of fibers to escape the body. Chrysotile in particular has a very short half-life—a matter of weeks or months. Thus, considering all low-level exposures, particularly to chrysotile, as “cumulative” over a lifetime is misleading—many if not most of those fibers are not around a short time after the exposure. The actual dose incurred by a given exposure is again the most important element in proving causation.

The second flaw in considering cumulative exposure is that the body’s defenses can handle a certain amount of exposure to all kinds of potentially toxic substances, including asbestos. Unless the exposures exceed this “noise level” and an even higher causative threshold, they are not part of any cause—this is the fundamental lesson of Dr. Eaton’s article regarding dose.

For causation purposes, as discussed above in Section II, courts should consider only those exposures that reach a level tied to disease in the studies. Any other exposures are likely to be—until proven otherwise
by the science—trivial and medically meaningless, not “cumulative” in
the sense of contributing to the cause of the mesothelioma. As noted
above, some of these experts have used the analogy that a bucket of water
thrown into the ocean is part of the ocean’s cumulative mass. However,
some courts, such as the Sixth Circuit, have turned that analogy
around to point out how extreme and unscientific this testimony is.
The opinion in In re New York City Asbestos Litigation (Juni I),
which was recently affirmed by the New York Court of Appeals, was one
of the earliest to exclude specifically the cumulative exposure approach.
The Juni I trial court’s analysis and rejection of “cumulative” exposure
testimony is one of the most thorough and carefully reasoned such
opinions to date. The court’s rejection was based on the lack of logical
or scientific foundation for either and on the obvious reality that the
underpinnings of cumulative exposure testimony is nearly the same as
that for every exposure.

The Seventh Circuit has also issued an opinion dissecting the cumulative exposure theory. In Krik v. Exxon Mobil Corp., the plaintiff’s
expert Dr. Arthur Frank changed his testimony midstream from every
exposure to cumulative exposure after the initial trial judge rejected
every exposure testimony as inadmissible. The Seventh Circuit held that Dr.
Frank’s new-found cumulative exposure theory was “no different from

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review denied (Cal. June 14, 2017).
279 Moeller v. Garlock Sealing Techs., LLC, 660 F.3d 950, 955 (6th Cir. 2011)
280 See id. (noting that the substantial factor test would be rendered meaningless if
the court were to accept augments that any level of exposure, as opposed to “a high
enough level of exposure,” was indeed a substantial cause of the disease).
282 See In re N.Y. City Asbestos Litig. (Juni 1), 11 N.Y.S.3d at 436 (stating plaintiffs
failed to offer sufficient evidence to support their cumulative exposure theory).
283 See id. at 437 (“Many of those courts [addressing sufficiency of the expert
evidence] require specific proof of exposure and have rejected the so-called cumulative
exposure theory and its variant, the ‘each and every’ exposure theory.”).
284 870 F.3d 669 (7th Cir. 2017).
285 Krik, 870 F.3d at 672-73.
Federal district courts in South Carolina, Maryland, Wisconsin, and North Carolina have also rejected cumulative exposure testimony on the same grounds as every exposure testimony.\textsuperscript{287}

The most recent appellate ruling on this issue comes from the Supreme Court of Ohio in \textit{Schwartz v. Honeywell International, Inc.}\textsuperscript{288} In \textit{Schwartz}, Dr. Carlos Bedrossian testified that the total cumulative dose of asbestos exposure causes disease and the plaintiff’s bystander exposure to a small number of brake jobs would have been a substantial factor in causing disease because it contributed to her cumulative dose.\textsuperscript{289} Dr. Bedrossian’s testimony, seemingly consistent with both every exposure and cumulative exposure theorists, “does not rely upon any particular dose or exposure to asbestos, but rather [opines that] all exposures contribute to a cumulative dose.”\textsuperscript{290} The court held that the use of cumulative exposure testimony cannot support a verdict based on substantial factor causation.\textsuperscript{291}

\textbf{C. The Gatekeeping Error—
The Failure of Courts Accepting \textit{Every Exposure} Testimony to Engage in Sufficient Gatekeeping}

Despite the many flaws associated with every exposure testimony, a number of courts have permitted plaintiff experts to present to the jury some version of every exposure testimony or held that such testimony

\textsuperscript{286} \textit{Id.} at 675.


\textsuperscript{288} \textit{102} N.E.3d 477 (Ohio 2018).

\textsuperscript{289} \textit{Schwartz}, 102 N.E.2d at 177.

\textsuperscript{290} \textit{Id.} at 181 (quoting Krik v. Exxon Mobil Corp., 870 F.3d 669, 677 (7th Cir. 2017)).

\textsuperscript{291} \textit{Id.} at *5.
is sufficient proof of causation to go to the jury. The discussion below highlights the flaws in the reasoning of these courts, all of which arise from the one unifying factor—a complete failure to investigate the bases for the experts’ own *ipse dixit*, self-supporting statements.

More than anything else, engaging in meaningful gatekeeping is the most critical step in any review of the admissibility or sufficiency of expert testimony. Judges have the authority—and the obligation—to ask hard questions of experts who appear in their courts. Those questions go beyond “what is your opinion?” and “what are you relying on for that opinion?” The inquiry requires engaging in the underlying studies and science, understanding the principles at issue, and examining whether the expert’s statements are backed up by acceptable logic and actual evidence. As the Ninth Circuit Court of Appeals held, a “strong divide among both scientists and courts on whether [every exposure testimony] is relevant in asbestos-related cases” is not sufficient to pass


294 See FED. R. EVID. 702 advisory committee’s note to 2000 amendment (explaining and defining gatekeeping as established under *Daubert*).

295 See, e.g., Estate of Barabin v. AstenJohnson, Inc., 740 F.3d 457, 464 (9th Cir. 2014) (finding the court failed to act as the gatekeeper in part by not examining “every exposure” theory).

296 See, e.g., Moeller v. Garlock Sealing Techs., LLC, 660 F.3d 950, 954 (6th Cir. 2011) (“After conducting our own careful review of the record, including the testimony of each expert, we must conclude that the Plaintiff failed to prove that Garlock’s product was a substantial factor in bringing about the harm.”).
the issue to the jury—the court must still examine whether the testimony meets the Rule 702 threshold for admissibility.297

Judicial review of low exposure cases, of course, can be problematic—judges are loath to dismiss a potentially deserving plaintiff for lack of concrete scientific evidence, and determining exactly where the line is between proof of causation and speculation is often difficult.298 The science itself is in part to blame—as noted in Section I, subsection A, these cases almost always involve diseases occurring many years after the claimed exposure, with no clear temporal link to which litigants can point.299 The disease could have been caused by many events in between, and proof of a causative link is potentially challenging given the long passage of time. Epidemiology—the “gold standard” for proving causation for latent disease300—is an indirect science at best that is relatively easy to discount, misinterpret, or distort. The science often changes constantly as new studies appear, and new studies could even occur during the course of the litigation itself.301 As one dramatic example, the

297 Barabin, 740 F.3d at 464 (internal quotation marks omitted).
299 See supra subsection I(A); see generally, e.g., Rutherford, 941 P.2d 1203, 1208 (presenting evidence that the plaintiff contacted lung cancer because of asbestos exposure at his long-time job even though his particular work duties did not include installing asbestos insulation).
300 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 Agent Orange and Bendectin Litigation, 86 NW. U. L. REV. 643, 646-47 (1992); see also id. at 647 (“[E]pidemiology assess the likelihood that the agent caused a specific individual disease.”); Mary Andrues, Proof of Cancer Causation in Toxic Waste Litigation: The Case of Determinancy Versus Indeterminancy, 61 S. CAL. L. REV. 2075, 2088 (1988) (“The only valid way to identify human carcinogens and establish medical causation is to observe differences in the incidence of cancer between humans exposed to toxic wastes and those who are not.”); Bert Black & David Lilienfeld, Epidemiologic Proof in Toxic Tort Litigation, 52 FORDHAM L. REV. 732, 736 (1984) (“Epidemiology is the only generally accepted scientific discipline . . . to identify and establish the causes of human diseases.”).
breast implant litigation induced extensive settlement payments until epidemiology studies caught up with the litigation and demonstrated no link between the implants and the claimed diseases.\textsuperscript{302} Judges who engage in a serious review of the expert testimony are truly entering into the “brave new world” predicted by the Ninth Circuit \textit{Daubert} court.\textsuperscript{303}

Notwithstanding these difficulties, courts have an obligation to ensure that experts are standing on solid footing before allowing them to testify to the jury about the most speculative of causation inquiries—the long ago exposure that supposedly produced current disease.\textsuperscript{304} Testimony about such circumstances by experts can be very influential and thus needs vigorous testing before allowing experts to confuse or mislead the jury.\textsuperscript{305} Thus, most, if not all, jurisdictions offer the opportunity for some form of pre-testimony gatekeeping review.\textsuperscript{306} And presumably any jurisdiction would at least consider motions for judgment based on insufficiency of the expert evidence.\textsuperscript{307} In addition, judges often have to engage in difficult reviews in an array of cases, including patent litigation, antitrust litigation, medical malpractice, and even complex commercial cases.\textsuperscript{308} There is nothing so unique about toxic tort or

\textsuperscript{302} See Kristin E. Schleiter, \textit{Silicone Breast Implant Litigation}, 12 J. ETHICS AMA 389, 389 (2010) (describing how the developing science undercut the litigation, leading to judicial decisions excluding the expert testimony); \textit{see also} MARCIA ANGEL, SCIENCE ON TRIAL 128-29 (1996) (describing the lack of epidemiology that ultimately led to the Supreme Court’s and Ninth Circuit’s \textit{Daubert} decision).

\textsuperscript{303} \textit{Daubert} v. Merrell Dow Pharms., Inc., 43 F.3d 1311, 1315 (9th Cir. 1995).

\textsuperscript{304} \textit{See} Krik v. Exxon Mobil Corp., 870 F.3d 669, 674 (7th Cir. 2017) (stating courts can exclude evidence if the evidence being introduced does not assist the jury with their determination of fact).

\textsuperscript{305} \textit{See}, e.g., \textit{Daubert} v. Merrell Dow Pharms., Inc., 509 U.S. 579, 595 (1993) (citing Jack B. Weinstein, \textit{Rule 702 of the Federal Rules of Evidence Is Sound; It Should Not Be Amended}, 138 F.R.D. 631, 632 (1991)) (“Judge Weinstein has explained: ‘Expert evidence can be both powerful and quite misleading because of the difficulty in evaluating it. Because of this risk, the judge in weighing possible prejudice against probative force under Rule 403 of the present rules exercises more control over experts than over lay witnesses.’”).

\textsuperscript{306} \textit{See} David Bernstein & Jeffrey Jackson, \textit{The Daubert Trilogy in the States}, 44 JURIMETRICS J. 351, 355-56 (2004) (indicating that, as of 2004, a majority of states have adopted \textit{Daubert} or interpreted their law consistently with \textit{Daubert}).

\textsuperscript{307} \textit{See}, e.g., cases cited supra notes 235-47.

\textsuperscript{308} \textit{See generally} Michael Baye & Joshua Wright, \textit{Is Antitrust too Complicated for Generalist Judges? The Impact of Economic Complexity and Judicial Training on
product litigation that would justify a lesser review for experts supporting such claims.  

The distinction between gatekeeping and simply waving a hand at the expert as he or she passes by becomes apparent by contrasting several of the every exposure opinions. In 2011, the Georgia intermediate appellate court allowed Dr. Jerold Abraham to testify that “several” or “multiple” exposures in a paper felt manufacturing plant were the cause of plaintiff’s mesothelioma. But, neither the experts nor the court identified any form of dose quantification or even an estimate. The defendant, Scapa Dryer Fabrics, challenged the evidence under Georgia’s Daubert standard. The court’s analysis is a classic example of a failed gatekeeping—the court relied heavily on several statements made by Dr. Abraham, without once examining the logical or scientific basis for those statements. The court merely repeated, without examination, many of the expert’s statements, and ultimately agreed with the expert without ever critiquing or examining the basis of the claim that identified exposures “above background” were not “trivial” and were therefore causative.

But this is not a de minimis exposure case. Scapa was responsible for considerably more than de minimis exposure. As the testimony of Dr. Abraham established, the exposures for which Scapa is responsible were “substantial causes” of Knight’s mesothelioma, and the jury indeed found Scapa substantially liable in the amount of 40 percent.

Dr. Abraham’s only basis for “establishing” the substantiality of the exposures was that he himself declared them to be substantial—with no

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Appeals, 54 J. L. & ECON. 1, 2 (2011) (discussing the complexity of antitrust law for generalist judges).

309 See Kathleen Michon, Toxic Torts Overview, Nolo, https://www.nolo.com/legal-encyclopedia/toxic-torts-overview-32204.html (last visited Mar. 3, 2019) (“Although each toxic tort case is unique . . . there are some common issues that crop up in many toxic tort cases.”).


311 Id. at 347-48.

312 Id. at 339.

313 Id. at 340-42.

314 Id. at 339-40 (emphasis added).
hint of a dose assessment.\textsuperscript{315} The court repeated the same “analysis” later, again falling into the trap of simply repeating the expert’s testimony: “Dr. Abraham testified, ‘those exposures [for which Scapa was responsible] would have been substantial causes for [Knight’s] mesothelioma.’”\textsuperscript{316} Nor did the court note, or even challenge, why Dr. Abraham had failed to produce any epidemiology studies documenting that a few such exposures would be sufficient.\textsuperscript{317} The intermediate court’s opinion and inadequate analysis were later reversed by the Georgia Supreme Court, finding that Dr. Abraham’s “cumulative exposure” testimony failed to consider or account for the dose involved.\textsuperscript{318}

The Scapa intermediate court is not alone in relying exclusively on the experts’ statements rather than examining their underlying trustworthiness and value. The Ninth Circuit Court of Appeals reversed a federal judge because the judge “failed to act as a gatekeeper,” in part by affirming admission of “every exposure” testimony based on the mere fact that there was a dispute among the experts over the issue.\textsuperscript{319} Likewise, in \textit{In re Asbestos Products Liability Products Litig.}, the federal MDL (multi-district litigation) judge overseeing a large docket of asbestos cases, despite performing an enormous benefit by dismissing many cases and clearing out that docket, allowed \textit{every exposure} experts to testify repeatedly.\textsuperscript{320} The court’s various statements, however, are replete with mere references to the experts’ testimony—“Dr. Hammar has testified . . .,” “Dr. Hammar reviewed. . .,” “Dr. Hammar notes . . .”—with no investigation whatsoever into the validity of those

\textsuperscript{315} Id. at 340.
\textsuperscript{316} Id. at 341 (emphasis added).
\textsuperscript{318} Id. at 425. In a somewhat unusual approach, the Georgia Supreme Court found that Dr. Abrahams’s testimony did not “fit” the facts of the case, thus relying on \textit{Daubert}’s second prong of inquiry rather than the reliability prong. \textit{Id.} at 426. The reasoning—accurate although not often followed by these courts—is that the jury needs to hear more than “any exposure will do” in order to determine the significant of the exposures. \textit{Id.} Thus, the opinion was unhelpful to the jury, did not fit the inquiry, and was inadmissible under \textit{Daubert}. \textit{Id.}
\textsuperscript{319} Estate of Barabin v. AstenJohnson, Inc., 740 F.3d 457, 464 (9th Cir. 2014).
statements. After remand of one of these cases to its home court in Utah, the Utah federal judge excluded the same experts, finding in part that the expert’s statements were not supported by the cited studies.

The intermediate Ohio appellate court decision in Schwartz also illustrates the same gatekeeping error—the panel repeatedly referred to statements made by the experts themselves as support for the reliability of their own testimony under Rule 702. Over forty times in the Schwartz intermediate court opinion, the panel simply restated the expert’s testimony by noting that the expert “testified,” “opined,” “found,” “discussed,” “considered,” or “stated” certain opinions. Yet, not a single time did the court actually examine the basis for those statements or decide whether they were credible and derived from a scientific methodology.

Contrast this laissez-faire approach with that of the very same court under the same legal standard—the Ohio intermediate court in Watkins v. Affinia Group applying Ohio’s Daubert approach. After taking a much closer look at the testimony, that court held: “The trial court did not properly execute its duty as gatekeeper because, without a hearing, the court could not independently examine and evaluate the reliability of Drs. Frank’s and Strauchen’s expert testimony. Therefore, their testimony was admitted in error.”

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. If the court declines to pull back

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321 See, e.g., id. at *1-7 (reserving challenges to reliability for cross-examination).
322 Anderson v. Ford Motor Co., 950 F. Supp. 2d 1217, 1223 (“Plaintiff’s experts are unable to point to any studies showing that ‘any exposure’ to asbestos above the background level of asbestos in the ambient air is causal of mesothelioma.”).
324 Id.
327 Watkins, 54 N.E.3d at 182. Both of these opinions were issued by the Eighth District Ohio Court of Appeals. The contrast between them illustrates how the driving factor is the court’s failure to act as gatekeeper and not the standard applied.
328 See, e.g., Waite v. All Acquisition Corp., 194 F. Supp. 3d 1298, 1314-17 (S.D. Fla. 2016) (containing repeated references to Dr. Frank’s testimony); Neureuther v. Atlas Copco Compressors, LLC, No. 13-cv-1327-SMY-SCW, 2015 WL 4978448, at
the curtain, the charade goes unchecked. As one of the earliest opinions on every exposure testimony held, judges are not required to sign off on a “hired expert” every exposure opinion:

Just because a hired expert makes a legal conclusion does not mean that a trial judge has to adopt it if it is not supported by the record and is devoid of common sense. . . . [The expert’s] statement saying every breath is a “substantial contributing factor” is not accurate. If someone walks past a mechanic changing brakes, he or she is exposed to asbestos. If that person worked for a factory making lagging, it can hardly be said that one whiff of the asbestos from the brakes is a “substantial factor” in causing disease.

The underpinnings of every exposure testimony fall apart under any level of reasonable examination. The many opinions rejecting this form of testimony are replete with clear analyses of the experts’ arguments and supposed support, and documenting their inadequacy or irrelevance. The discussion above in Section III, for instance, includes court rejection of expert reliance on the “no safe dose” approach as, at most, a hypothesis that is unproven and unprovable. Other courts have rejected reliance on government pronouncements about asbestos as irrelevant to court causation standards. Several courts have rejected the experts’ reliance

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331 See, e.g., Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 855-63 (E.D.N.C. 2015) (surveying literature and other bases for “special exposure” opinion and finding that under close consideration none of the cited studies or arguments supported the actual opinion).

332 See supra Section III.

on irrelevant epidemiology studies, especially the attempt to use mixed exposure or amphibole studies to prove a chrysotile case—and they identified the flaw because these courts chose to examine the studies and understanding that the experts were misleading the jury as to their meaning. Other courts have rejected the unscientific reliance of the experts on mere dust to prove causation. Some courts have reviewed the studies cited by these experts in detail and found that not a single one contained the expert’s every exposure proposition or supported the idea that no dose assessment is necessary. As one such example, the most frequently cited source as supporting every exposure testimony—the so-called Helsinki Criteria—never states that every identifiable workplace exposure is a cause of mesothelioma but instead requires a “significant” exposure.

(“The court finds no probative value to the statements of safety and regulatory agencies or to the warnings contained in Garlock’s own Materials Safety Data Sheets. Such statements simply involve something quite different than the issues involved here. Many, if not all, safety and regulatory bodies have issued statements, policies or regulations regarding asbestos exposure. But, these cannot be probative on the issue of causation because of the differences in the way courts and regulatory authorities assess risk.”); In re W.R. Grace & Co., 355 B.R. 462, 468-69 (Bankr. D. Del. 2006), appeal denied, 2007 WL 1074094 (D. Del. Mar. 26, 2007) (discussing EPA studies showing the connection between Zonolite Attic Insulation and health risks).


See supra cases accompanying notes 234-36.


See A. Tossavainen, Asbestos, Asbestosis, and Cancer: The Helsinki Criteria for Diagnosis and Attribution, 23 SCAND. J. WORK ENVIRON. HEALTH 4, 311, 313 (1997) (“In the absence of such markers, a history of significant occupational, domestic, or environmental exposures to asbestos will suffice for attribution.”) (emphasis added). The Helsinki committee did not consider specific fiber types and toxicities, or establish that every single exposure no matter how small is causative. It instead required “significant” workplace exposures before attributing mesothelioma causation. See Butler v. Union Caribe Corp., 712 S.E.2d 537, 552 (2011). Several courts have rejected plaintiff expert reliance on the Helsinki Criteria as a basis for testifying without providing a causative dose quantification. Yates, 113 F. Supp. 3d at 861-62; Rockman, 266 F. Supp. 3d at 847 (rejecting Helsinki Criteria as a basis for failure to demonstrate a significant exposure); Betz, 44 A.3d at 55-576 n.35 (Helsinki Criteria “do not embody the any-exposure theory.”). In 2014 the Finnish Institute of Occupational Health updated the criteria, and the update again repeated the need for a “significant” exposure.
The flaws in the \textit{every exposure} theory are not difficult to discern, unless the court decides to gloss over the expert testimony and let it go to the jury without examination.\footnote{See, e.g., Estate of Barabin v. AstenJohnson, Inc., 740 F.3d 457, 464 (9th Cir. 2014) (finding that the district court failed in its gatekeeping role by allowing the jury to decide whether the expert testimony was relevant and reliable because the court should have determined that first); Scapa Dryer Fabrics, Inc. v. Knight, 788 S.E.2d 421, 427-28 (Ga. 2016) (finding that the appellate court failed in its role as gatekeeper by affirming the trial court’s allowance of letting the jury hear the expert’s testimony about causation because the testimony did not meet Georgia law requirements for causation).} Fortunately, most of the courts reviewing \textit{every exposure} testimony have done more than this, and as a result the vast majority of opinions have found that testimony inadmissible or insufficient.\footnote{See, e.g., Krik v. Exxon Mobil Corp., 870 F.3d 669, 672 (7th Cir. 2017) (rejecting the plaintiff’s expert’s testimony about the \textit{every exposure} theory finding the theory insufficient); Vedros v. Northrop Grumman Shipbuilding, Inc., 119 F. Supp. 3d 556, 562-63 (E.D. La. 2015) (noting that courts generally exclude expert testimony of the \textit{every exposure} theory because it is insufficient).} The \textit{every exposure} approach cannot survive a legitimate gatekeeping inquiry.\footnote{See Crane Co. v. DeLisle, 206 So. 3d 94 (Fla. App. 2016) (stating “gatekeeping . . . requires more than simply taking the expert’s word for it” when it comes to scientific knowledge) (citing United States v. Frazier, 387 F.3d 1244, 1265 (11th Cir. 2004)), \textit{decision quashed by} DeLisle v. Crane Co., 258 So. 3d 1219 (Fla. 2018).}

IV. The Judicial Roadmap: An Analytical Framework for Assessing the Admissibility of Low-Exposure Causation Testimony

The problem of variation in judicial opinion in low-dose cases could be rectified if all of these courts followed a scientifically-based approach to review expert testimony in these cases. The judicial review effort often varies so widely across cases that the selection of jurisdiction itself can be dispositive. In Louisiana, for instance, state courts have not yet rejected \textit{every exposure} testimony outright and, according to some for mesothelioma attribution. H. Wolff, reporter, et al., \textit{Asbestos, Asbestosis, and Cancer, the Helsinki Criteria for Diagnosis and Attribution 2014: Recommendations}, 10 \textit{Scand J. Work Environ Health Online} at 2 (“In the absence of such markers, a history of significant occupational, domestic or environmental exposure will suffice for attribution.”).\footnote{See, e.g., H. Wolff, reporter, et al., \textit{Asbestos, Asbestosis, and Cancer, the Helsinki Criteria for Diagnosis and Attribution 2014: Recommendations}, 10 \textit{Scand J. Work Environ Health Online} at 2 (“In the absence of such markers, a history of significant occupational, domestic or environmental exposure will suffice for attribution.”).}
opinions, may have accepted it. Some five consecutive federal courts have held that any exposure testimony is inadmissible. Some degree of variation is expected, but for obvious reasons, it would be beneficial for the overall tort system, not to mention the future of asbestos litigation, if courts applied a reasonably uniform approach.

Rational judicial gatekeeping decisions should at least keep the litigation consistent with the declining exposure scenarios in the population at large. To assist judges in this process, after addressing the relevance of the review standard to the proposed analysis, we provide a three-step approach derived from the case law for courts to follow in addressing low toxic exposure matters, including asbestos.

A. Daubert or Frye—Does It Matter?

As an initial issue, the question might be posed whether the jurisdiction’s adherence to Daubert versus Frye (or some other review standard) matters. The short answer to that question is “not much.” Courts have rejected every exposure testimony many times under both Daubert and Frye and under several variations of the two, and a few

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343 See infra Section IV(B).

344 For examples of courts rejecting every exposure testimony under state or federal versions of Daubert, see Haskins v. 3M Co., 103 Fed. R. Evid. Serv. 1286, 6-8 (D.S.C. 2017); Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 846 (E.D.N.C. 2015); Butler v. Union Carbide Corp., 712 S.E.2d 537, 541-42 (Ga. Ct. App. 2011); Watkins v. Affinia Grp., 54 N.E.3d 174, 181 (Ohio Ct. App. 2016). For examples of courts that have rejected every exposure testimony under Frye-type standards, see In re N.Y. City...
courts have accepted some version of the testimony under both standards.\footnote{345}

As a brief primer, both federal and state courts largely utilized the original \textit{Frye} standard or some version of it since the seminal decision by the District of Columbia Court of Appeals in 1923.\footnote{346} The \textit{Frye} opinion established the “general acceptance” test in which scientific testimony would be kept away from the jury if the relevant scientific community had not generally accepted the principles on which it was founded.\footnote{347} The United States Supreme Court’s \textit{Daubert} opinion, issued seventy years later in 1993, created a different approach intended to give judges more flexibility in the analysis, including the potential to accept sufficiently reliable but somewhat new scientific testimony.\footnote{348} The \textit{Daubert} court focused on the methodology of the expert and held that testimony should not be admitted unless the methodology was reliable and the testimony fit the facts of the case.\footnote{349} The Court utilized four, non-exclusive factors for its review: (1) whether the methodology was

\footnote{345}{For examples of courts that have permitted \textit{every exposure} testimony under \textit{Daubert}, see King v. Burlington Northern Santa Fe Ry. Co., 762 N.W.2d 24, 48 (Neb. 2009); Estate of Barabin v. AstenJohnson, Inc., 740 F.3d 457, 461 (9th Cir. 2014) (refusing to “decide whether an expert may offer testimony that each and every exposure to asbestos is a significant cause of mesothelioma’ and allowing testimony that “each ‘identified exposure’ was a substantial cause of injury”). For examples of similar decisions under \textit{Frye}-based standards, see Northrop Grumman Sys. Corp. v. Britt, 241 So. 3d 208, 214-15 (Fla. Dist. Ct. App. 2017); and Jones v. John Crane, Inc., 35 Cal. Rptr. 3d 144, 151 (Ct. App. 2005).}

\footnote{346}{Daubert v. Merrell Dow Pharms., Inc., 509 U.S. 579, 585 (1993) (stating \textit{Frye} has been the “dominant” standard since its formulation and has been followed by “a majority of courts”).}

\footnote{347}{\textit{Id.} (discussing \textit{Frye}’s “general acceptance” test).}

\footnote{348}{\textit{Id.} at 587-97 (discussing the replacement of \textit{Frye} with the new criteria under \textit{Daubert}).}

\footnote{349}{\textit{Id.} at 592-93; see also Kumho Tire Co. v. Carmichael, 526 U.S. 137, 141 (1999) (emphasizing \textit{Daubert}’s test for reliability is “flexible”); United States v. Crisp, 324 F.3d 261, 265-66 (4th Cir. 2003) (reviewing the factors \textit{Daubert} set forth to test reliability and acknowledging that the test “must be a flexible one”).}
testable, (2) its rate of error, (3) whether it had been published and peer-reviewed, and (4) whether it was generally accepted, incorporating Frye.\textsuperscript{350}

The Daubert standard has since been codified in the Federal Rules of Evidence under Rule 702.\textsuperscript{351} Many states have also adopted Daubert or some version of it, while other states remain adherents of the Frye approach or utilize their own version of review standard.\textsuperscript{352} Daubert is now often, but not universally, recognized as a more potent form of court review that often results in defense wins.\textsuperscript{353} But, as discussed below, the outcome for the every exposure theory is not as dependent on the review standard because the theory is (1) inadmissible under both, but (2) still susceptible to admission under both for courts who wish to avoid a rigorous review of the theory.\textsuperscript{354}

Under a Frye analysis, applied faithfully, it is unlikely that every exposure testimony would be admissible.\textsuperscript{355} No scientific or medical body has adopted the “principle” that the smallest exposure to a carcinogen is a known cause of cancer.\textsuperscript{356} Nor have the experts who expound this theory in court ever tried to publish their litigation testimony in a

\textsuperscript{350} Daubert, 509 U.S. at 593-94 (reviewing the four, nonexclusive factors).

\textsuperscript{351} See Fed. R. Evid. 702 advisory committee’s note to 2000 Amendments (“Rule 702 has been amended in response to Daubert . . . and to the many cases applying Daubert . . . .”).

\textsuperscript{352} Edward K. Cheng & Albert H. Yoon, Does Frye or Daubert Matter? A Study of Scientific Admissibility Standards, 91 Va. L. Rev. 471, 472-73 (2005) (reviewing state and federal court use of Daubert versus Frye and the effect on judicial decisions). This article supports generally the proposition that the review standard is not terribly relevant to the outcome, and the decisions instead turn on judicial preference for controlling evidence versus letting the jury deal with expert disputes.

\textsuperscript{353} Id. (stating the Daubert “legacy” is relatively clear as a “potent weapon” of tort reform and that the effects have been “pro-defendant”).

\textsuperscript{354} See generally Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 846 (E.D.N.C. 2015) (stating the “each and every exposure theory” should be excluded under Daubert); Betz v. Pneumo Abex, LLC, 44 A.3d 27, 56 (Pa. 2012) (rejecting the every exposure theory under Frye-like standards).

\textsuperscript{355} See generally Blum ex rel. Blum v. Merrell Dow Pharm., Inc., 564 Pa. 3, 4-5 (2000) (concluding that expert testimony would not be admissible under Frye because Frye “bars novel scientific evidence until it has achieved ‘general acceptance’ in the relevant scientific community”).

\textsuperscript{356} See generally Yates, 113 F. Supp. 3d at 847 (explaining that the “each and every exposure theory has no sufficient supporting facts or data”).
peer-reviewed scientific journal to obtain “general acceptance” of the principle.\textsuperscript{357} To achieve acceptance under \textit{Frye}, courts would have to revert to a review only of the sources of information on which the experts rely.\textsuperscript{358} In such a case, the experts would claim to rely on epidemiology, animal studies, government publications and the like—all of which are in some sense “generally accepted.”\textsuperscript{359} In 2018 Florida affirmed its commitment to this approach in \textit{DeLisle v. Crane Co}.\textsuperscript{360} Under this most generous analysis, it does not matter whether an expert uses a hammer to saw a board or a screwdriver to pound a nail, as long as those tools are in the toolbox.

A more straightforward and realistic approach to gatekeeping under \textit{Frye} would focus on the scientific principle involved, hinging on the universal failure of the scientific community to adopt a completely dose-less form of causation analysis. Causation based merely on limited, above-background exposures, “dust,” is universally rejected in the scientific universe (which relies instead on dose assessments and competent epidemiology reflecting comparable exposure scenarios).\textsuperscript{361}

Under \textit{Daubert}, the \textit{every exposure} theory should not fare any better—a methodology that ignores dose is by definition unreliable. Applying the four \textit{Daubert} factors leads to the same conclusion. The theory cannot be tested because it presumes the outcome—an exposure to asbestos is the cause regardless of testing or evidence.\textsuperscript{362} This testimony has an enormous rate of error because it points to asbestos as the cause of many spontaneous and other mesotheliomas; however, no

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\item \textsuperscript{357} See generally Cavallo v. Star Enter., 892 F. Supp. 756, 772 (E.D. Va. 1995) (noting the expert “could cite no studies or published literature to support adverse effects from [the] level of exposure to [the specific toxic agent]”).
\item \textsuperscript{358} See Berry v. CSX Transp., Inc., 709 So. 2d 552, 565 (Fla. App. 1988); Crane Co. v. DeLisle, 206 So. 3d 94, 100 n.7 (Fla. Dist. Ct. App. 2016), decision quashed by DeLisle v. Crane Co., 258 So. 3d 1219 (Fla. 2018).
\item \textsuperscript{359} Id.
\item \textsuperscript{360} 258 So. 3d 1219, 1229-30 (Fla. 2018); see also Castillo v. E.I. Du Pont de Nemours & Co., 854 So. 2d 1264, 1273 (Fla. 2003) (permitting expert testimony relying on \textit{in vitro} and \textit{in vivo} studies without testing the expert’s approach to drawing conclusions from them as to human causation).
\item \textsuperscript{361} See supra Section III.
\end{enumerate}
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competent evidence demonstrates that asbestos induces instances of such disease.\textsuperscript{363} The experts have not managed to obtain peer-reviewed publication, nor acceptance, of their litigation testimony.\textsuperscript{364} And the theory, as noted under the \textit{Frye} analysis above, is not generally accepted in the scientific community as a basis for attributing causation.

The reality is that the admissibility of the every exposure theory seems to have little to do with the analytical approach adopted in the relevant jurisdiction and much more to do with the court’s willingness to permit an asbestos case to proceed to the jury. Both standards of review provide sufficient gatekeeping authority to trial judges to exclude every exposure testimony.\textsuperscript{365} Whether judges will apply that authority to asbestos litigation is the driving factor. Thus, the analytical framework below is available and applicable regardless of the review standard used in any given jurisdiction. Every exposure theory is so far off the beaten path of scientific methodology and thinking that it should be relatively easy for judges to exclude compared to some of the more difficult causation inquiries courts often face.\textsuperscript{366}

\section*{B. Three Key Steps to Low Dose Litigation}

\subsection*{1. Define the Exposure and Causation Issue in a Given Case}

Asbestos causation experts who correctly apply scientific principles should address the actual product and exposure at issue and not be permitted to obscure the inquiry by pointing to entirely different sets of studies and exposures.\textsuperscript{367} Thus, it is vital for the reviewing court to

\textsuperscript{363} Id.; see text accompanying supra note 228.

\textsuperscript{364} Parker v. Mobil Oil Corp., 857 N.E.2d 1114, 1121-22 (N.Y. 2006).

\textsuperscript{365} See Julia Luyster, \textit{Frye} and \textit{Daubert} Challenges: Unreliable Options vs. Unreliable Science, 26 No. 2 TRIAL ADVOC. Q. 29, 29 (2007) (“The \textit{Frye} and \textit{Daubert} tests are somewhat different evidentiary standards utilized, in state and federal court respectively, to accomplish the same defensive maneuver: to prevent scientifically unreliable testimony from reaching the trier of fact.”).

\textsuperscript{366} Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 846 (E.D.N.C. 2015) (noting “numerous courts” have excluded the every exposure expert testimony and the theory has not be “been published in peer-reviewed works”).

understand the nature of the claimed exposure and ensure that the experts are addressing it properly and accurately. An overly precise definition of the issue is not required, but it is surely an appropriate step to measure the experts’ testimony against the science involving the same chemical or material and under similar exposure circumstances. In *Parker v. Mobil Oil Corp.*, for example, plaintiffs tried to demonstrate that benzene caused plaintiff’s acute myeloid leukemia (AML). But, the case did not involve exposure to pure benzene, such as that experienced by the factory workers handling large amounts of benzene, as to whom several studies identified an increased risk of AML. Instead, the court correctly identified the product at issue as gasoline, which contains only about two to five percent benzene. All studies of workers exposed to gasoline, rather than benzene itself, found no link between those exposures and AML.

Similarly, in asbestos cases the plaintiffs’ experts seem to frequently merge the product at issue with statements about very different asbestos exposures and studies. As one example, in cases involving motor vehicle brakes, which contained chrysotile asbestos, the experts have frequently resorted to expansive statements such as “all fiber types are known to cause mesothelioma” and “studies show that only a few days of exposure can produce mesotheliomas.” What they fail to disclose is that the

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368 *Id.* at 762, 769-70 (quoting *In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 745 (3d Cir. 1994) (“[A]ny step that renders the analysis unreliable under the *Daubert* factors renders the expert’s testimony inadmissible. This is true whether the step completely changes a reliable methodology or merely misapplies that methodology.”)).

369 *Id.* at 769-70 (“[The expert witness] is unable to provide any scientifically valid basis to support the leap from those studies to his opinion in this case. . . . Thus, while the agreed-upon methodology appears to be scientifically valid, it does not appear to have been faithfully applied. . . . As a result, Dr. Monroe’s opinion is not ‘scientific knowledge’ and must be excluded.”) (citations omitted).

370 857 N.E.2d 1114, 1116 (N.Y. 2006).

371 *Parker*, 857 N.E.2d at 1118.

372 *Id.* at 1116-17.

373 *Id.* at 1119, 1122.

studies they rely on for these propositions involve either extremely high chrysotile exposures (typically from the old textile factory work or chrysotile mining) or heavy amphibole exposure.\textsuperscript{375} The Maryland case of \textit{Rockman v. Union Carbide Corp.} illustrates this point. That federal court criticized the experts for “conflat[ing] data on \textit{pleural} mesothelioma and \textit{amphibole} asbestos with a case involving \textit{peritoneal} mesothelioma and \textit{chrysotile} asbestos” to justify their opinion that mere residence in a home undergoing remodeling resulted in enough bystander exposure to chrysotile to cause mesothelioma.\textsuperscript{376} By mis-defining the exposure at issue, the experts tried to testify to causation even though they could not “identify any study associating \textit{peritoneal} mesothelioma with \textit{chrysotile} bystander exposures,” the actual exposure at issue.\textsuperscript{377} Thus, as \textit{Rockman} correctly concluded, the issue was not whether asbestos causes mesothelioma, but whether remote bystander exposure to chrysotile causes peritoneal mesothelioma, and the experts could cite to no studies demonstrated causation in that circumstance.\textsuperscript{378}

Similarly, as the New York \textit{Parker} court found that gasoline studies showed no increased AML from the small amount of benzene in that

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\textsuperscript{375} Many occupations involving chrysotile exposure have not produced excess mesothelioma, including from occupations such as mining where the exposures would have been far more significant than in today’s litigation. \textit{See}, \textit{e.g.}, \textit{In re Garlock Sealing Techs.}, LLC, 504 B.R. 71, 75, 78 (W.D.N.C. 2014) (“[I]t is clear under any scenario that chrysotile is far less toxic than other forms of asbestos,” and “[t]he most reliable and probative [peer-reviewed scientific] reports confirm[] that exposure to asbestos from end users of encapsulated asbestos products is minimal.”); \textit{In re Asbestos Litig.}, 911 A.2d 1176, 1181 (Del. Super. Ct. 2006) (“[I]t is generally accepted in the scientific community and among government regulators that amphibole fibers are more carcinogenic than serpentine (chrysotile) fibers.”); Bartel \textit{v. John Crane, Inc.}, 316 F. Supp. 2d 603, 605, 610 (N.D. Ohio 2004), \textit{aff’d sub nom.} Lindstrom \textit{v. A-C Prod. Liab. Trust}, 424 F.3d 488 (6th Cir. 2005) (“[I]t is generally accepted that it takes a far greater exposure to chrysotile fibers than to amphibole fibers to cause mesothelioma.”); David Rees \textit{et al.}, \textit{Case-Control Study of Mesothelioma in South Africa}, 35 AM. J. INDUS. MED. 213, 220-21 (1999) (stating no mesothelioma was identified in a cohort of thousands of long-time South African chrysotile miners).


\textsuperscript{377} \textit{Id.} at 848.

\textsuperscript{378} \textit{Id.} at 854.
\end{footnotesize}
product,\textsuperscript{379} at least twenty-one out of twenty-two epidemiology studies have never found that brake work produces any excess mesothelioma in automotive mechanics.\textsuperscript{380} As the largest study of mesothelioma in Britain recently noted: “We found no evidence of increased risk associated with non-industrial workplaces or those that were classified as ‘low risk’, including motor mechanics and workers handling gaskets and mats that may have contained asbestos.”\textsuperscript{381} Two New York Supreme Court opinions since then have noted that the issue for examination in a similar case was brake exposure and epidemiology, and not other, irrelevant exposure circumstances.\textsuperscript{382}

Expert testimony that misleads a jury by avoiding the product or exposure at issue should not be countenanced. The court as gatekeeper needs to define the relevant exposure accurately and then examine the relevance of the experts’ claimed studies to that exposure.\textsuperscript{383}

2. Require a Dose Assessment and Evaluation

As set forth in Section II above, once the relevant exposure is identified, the court should next require the testifying experts to develop a competent dose assessment for that product or exposure. This is the fundamental lesson of the many decisions rejecting every exposure testimony and far more decisions requiring a dose calculation or quantification in many other contexts.\textsuperscript{384} The sources of scientific

\textsuperscript{379} 857 N.E.2d at 449-50.


\textsuperscript{381} Julian Peto et al., Occupational, Domestic and Environmental Mesothelioma Risks in Britain, 100 BRITISH J. CANCER 1175, 1175 (2009); Rake et al., supra note 229, at 1182.


\textsuperscript{384} See text accompanying supra note 186.
evidence to develop such an estimate could include any air monitoring of the involved activity by the company, if it exists. But, if specific air monitoring does not exist, which is not uncommon, the inquiry does not end, because it is equally valid to rely on published or unpublished studies assessing similar exposures from other, comparable work activities.\textsuperscript{385} The expert can also conduct or consider simulation studies that accurately predict exposures from that work activity, but those studies should accurately reproduce the exposure circumstances and bear at least a modest consistency with published data.\textsuperscript{386} Finally, the expert can utilize published survey articles that examine the scope of exposures found in multiple studies and then determine the likely overall dose for that particular work activity, such as brake work.\textsuperscript{387}

In addition, a dose assessment involves more than merely identifying the snapshot exposure involved in one-time work activity measurement. Plaintiffs’ industrial hygienists, if they identify an exposure level at all, often focus on a single moment of work activity—for example, such an expert might state that “plaintiff was exposed to in excess of 2.0 fibers/cc each time he removed a brake.” These snapshot figures are meaningless from a health perspective because they do not take into account the eight-

\textsuperscript{385} See supra Section II.

\textsuperscript{386} For examples of review articles assessing the dose expected for vehicle mechanics, whether or not an individual mechanic’s actual exposures were measured at the time of his alleged work activity, see Anderson et al., Round II, supra note 7, at 1 (describing the “average lifetime mechanic exposures calculated at 0.04 f/cc or less, below OSHA standard of 0.1 f/cc”) (quoting Dennis J. Paustenbach et al., An Evaluation of the Historical Exposures of Mechanics to Asbestos in Brake Dust, 18 APPLIED OCCUPATIONAL & ENVT'L HYGIENE 786, 786-804 (2003)); Brent Finley et al., Cumulative Asbestos Exposure for U.S. Automobile Mechanics Involved in Brake Repair (circa 1950s-2000), 17 J. EXPOSURE SCIENE & ENVT'L EPIDEMIOLOGY 641, 641 (2007) (Cumulative lifetime average exposures for automobile mechanics “are all substantially lower than the cumulative exposure of 4.5 f/cm(3) year associated with occupational exposure to 0.1 f/cm(3) of asbestos for 45 years that is currently permitted under the current occupational exposure limits in the US.”).

For examples of simulation studies published in peer-reviewed journals and sometimes utilized for dose testimony in asbestos litigation, see Fred W. Boelter et al., Heavy Equipment Maintenance Exposure Assessment: Using a Time-Activity Model to Estimate Surrogate Values for Replacement of Missing Data, 4 J. OCCUPATIONAL ENVTL. HYGIENE 525, 525-37 (2007); A.K. Madl et al., Airborne Asbestos Concentrations Associated with Heavy Equipment Brake Removal, 53 ANNALS OCCUPATIONAL HYG. 8 (2009); Jennifer Sahmel et al., Evaluation of Take-Home Exposure and Risk Associated with the Handling of Clothing Contaminated with Chrysotile Asbestos, 34 RISK ANALYSIS 1148, 1448-68 (2014).

\textsuperscript{387} See Finley et al., supra note 386 (discussing dose reconstruction).
hour time-weighted average degree of exposure (OSHA-measured intensity), how long the activity lasts (duration), how many times the work activity is performed in a given month or year (frequency), and how many years the activity continues (overall dose). For this reason among others, OSHA requires an eight-hour average over a given day and then permits that level of exposure for a working lifetime of forty-five years. If plaintiffs’ causation experts cannot identify and are not relying on a competent assessment of the overall lifetime dose contributed by a particular activity, they are not engaging in either a scientific or a logical exercise. In many of today’s cases, if the experts followed the science, the dose assessments would fall well below even today’s OSHA standard and often less than a lifetime of mere background exposures.

Mere “exposure” to a product or substance should never be sufficient proof. If plaintiffs’ experts cannot produce or are not relying on a competent dose assessment, then summary judgment should be entered for defendant. Either the expert testimony is inadmissible under either Daubert or Frye, or the evidence (sans dose) is not sufficient to support a verdict.

3. Require a Demonstration of Actual Disease in Populations with Exposures Similar to Plaintiff

If the testifying experts manage to produce and competently rely on a dose assessment, the gatekeeping inquiry is not over. The court should

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388 OSHA established its limit of 0.1 fibers per cc on an 8-hour time-weighted average basis in 1996. Under the regulations, employers are in compliance with that standard, and the exposure is considered “acceptable,” as long as the daily exposure is under the standard each day based on the daily average. See Finley et al., supra note 386, at 645, 653-54 (describing the calculation of an OSHA lifetime level).


390 See, e.g., Finley et al., supra note 386, at 651 (discussing brake work exposures over a lifetime during 1950 to 2000 were well below today’s OSHA lifetime exposure standard of 4.54 E/cm3).

391 See Lindstrom v. A-C Prod. Liab. Trust, 424 F.3d 488, 596 (6th Cir. 2005) (affirming motions for summary judgement in favor of the defendants, because the plaintiff failed to show causation between his mesothelioma and the defendant companies).
then ensure that the experts are properly comparing that dose to similar populations and relying on studies that consistently demonstrate excess disease from those exposures. Typically, this inquiry would require comparison to studies of the same product (or one with a similar exposure profile), from activities that would produce reasonably similar exposure levels, and that show statistically significant excess disease across a range of such studies to avoid false positives from a single study or two.  

If the experts and court are following the science, the resort to epidemiology in a latent disease case is virtually mandatory. Because of the lack of temporal connection (i.e., many years pass between exposure and disease), and the likelihood of intervening events such as other exposures or spontaneous cancers, epidemiology is likely the only means of confirming that the alleged exposure in fact produces human disease and at what dose. The intricacies of epidemiological research are beyond the scope of this article, but many resources are available to help courts interpret and understand them. The key point is that the gatekeeper should not permit the experts to rely on irrelevant studies of other products/exposures, to “cherry-pick” one study out of many that favors the expert’s testimony, or to dismiss a large set of contrary epidemiology as “inconclusive.”

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393 See 2 DAVID OWEN & MARY DAVIS, OWEN & DAVIS ON PROD. LIAB. § 11:18, Westlaw (database updated May 2018).
394 See Eaton, supra note 143, at 39-40; Green et al., supra note 39, at 608-18.
395 See, e.g., Yates v. Ford Motor Co., 113 F. Supp. 3d 841, 858-59 (E.D.N.C. 2015) (quoting Eghnayem v. Boston Sci. Corp., 57 F. Supp. 3d 658, 676 (S.D.W. Va. 2014) (“An expert’s opinion may be unreliable if he fails to account for contrary scientific literature and instead ‘selectively chooses his support from the scientific landscape.’”); see also Norris v. Baxter Healthcare Corp., 397 F.3d 878, 886 (10th Cir. 2005) (affirming exclusion of expert testimony that failed to account for epidemiological evidence); In re Zoloft (Sertraline Hydrochloride) Prods. Liab. Litig., 26 F. Supp. 3d 449, 460-61 (E.D. Pa. 2014) (“The Court finds that the expert report prepared by Dr. Bérard does selectively discuss studies most supportive of her conclusions, as Dr. Bérard admitted in her deposition, and fails to account adequately for contrary evidence, and that this methodology is not reliable or scientifically sound.”); Pooshs v. Phillip Morris USA, Inc., 287 F.R.D. 543, 546 (N.D. Cal. 2012) (“A methodology may not be reliable if an expert fails to address and exclude alternative explanations for the data on which he bases his findings or rejects studies reporting contrary empirical findings.”) (internal quotation marks omitted)); In re Rezulin Prod. Liab. Litig., 369 F. Supp. 2d 398, 425 (S.D.N.Y. 2005) (“[I]f the relevant scientific literature contains
Of the many examples in the case law, the following should be sufficient to illustrate the analysis. Using the New York example of *Parker v. Mobil Oil Corp.*, that court correctly looked to the gasoline epidemiology literature, which found no link with AML, and rejected the experts’ inappropriate reliance on pure benzene studies.\(^{397}\) In the *Daubert* series of cases, the testifying experts “reinterpret” a series of studies involving the morning sickness drug Bendectin, none of which found any link with birth defects.\(^{398}\) The experts’ refusal to countenance such a strong, contrary body of literature doomed their testimony.\(^{399}\) In the breast implant litigation, large settlements were paid out before the epidemiology caught up with the litigation and found no demonstrable link between the implants and the alleged connective tissue disease.\(^{400}\) Finally, in the asbestos world, several courts have noted that virtually every study of vehicle mechanics has produced no excess mesothelioma.\(^{401}\) Specifically, courts have prevented the experts from ignoring that literature (by claiming it is “biased” or “inconclusive”), to selectively rely on a single study that found a modest increased risk, or resort to studies of irrelevant amphibole and high-level exposures in a different context.\(^{402}\)

Epidemiology can be difficult to understand and interpret. But, in the world of low-dose exposures, only rarely do epidemiology studies demonstrate any link with disease.\(^{403}\) Instead, these low-dose cases are evidence tending to refute the expert’s theory and the expert does not acknowledge or account for that evidence, the expert’s opinion is unreliable.”).\(^{397}\) 857 N.E.2d 1114, 1119 (N.Y. 2006).

\(^{398}\) MARCIA ANGELL, SCIENCE ON TRIAL 128-29 (1996) (describing the lack of epidemiology that ultimately led to the Supreme Court’s and Ninth Circuit’s *Daubert* decision).

\(^{399}\) Id.

\(^{400}\) See Kristin Schleiter, *Silicone Breast Implant Litigation*, 12 J. ETHICS AMA 389 (May 2010) (describing how the developing science undercut the litigation, leading to judicial decisions excluding the expert testimony).

\(^{401}\) See, e.g., *Yates*, 113 F. Supp. 3d at 858-59 (criticizing an expert’s refusal to acknowledge studies contradicting his opinion); *In re N.Y. City Asbestos Litig. (Juni II)*, 148 A.D.3d 233, 247 (N.Y. App. Div. 2017) (stating twenty-one out of twenty-two studies showed no link), aff’d sub nom. (*Juni III*), 116 N.E.3d 75 (N.Y. 2018).

\(^{402}\) See, e.g., *Yates*, 113 F. Supp. 3d at 858-59; *In re N.Y. City Asbestos Litig. (Juni II)*, 148 A.D.3d at 247.

\(^{403}\) See, e.g., *In re N.Y. City Asbestos Litig. (Juni II)*, 148 A.D.3d at 247, 251 (noting the single study finding an increased risk to contract mesothelioma differed from the general type of study).
often supported by expert distortions of the literature or reliance on irrelevant studies. Thus, courts functioning as gatekeepers in a low-exposure case should if anything be even more diligent to ensure the epidemiology provides logical and scientific support for the claim before letting it go to the jury.

C. Why the “Split-the-Baby” Decisions Are Insufficient

Some state courts have opted for a third path that falls somewhere in between rejecting or allowing every exposure testimony. These courts seem to resort to a split the baby approach to avoid a supposedly harsh outcome for plaintiffs, while recognizing that every exposure testimony is too inclusive of minimal exposures. These decisions are not well-taken because they ultimately force the trial judges to play the role of experts and decide how much exposure is enough.

Most of these courts resort to an old causation standard in asbestos litigation widely known as the approach in Lohrmann v. Pittsburgh Corning Corp. The test of asbestos testimony in Lohrmann derives

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404 See, e.g., id. (stating the expert discussed 21 studies concerning mesothelioma but also explained that the studies were not relevant to the plaintiff’s scenario).


406 Sweredoski, WL 3010419, at *2; Flores, 232 S.W.3d at 770-72.

407 Sweredoski, WL 3010419, at *2; Flores, 232 S.W.3d at 770-72.

408 782 F.2d 1156, 1162 (4th Cir. 1986) (citing Robin Express Transfer, Inc. v. Canton R.R., 338 A.2d 335, 343 (Md. Ct. Spec. App. 1975) (“To establish proximate causation . . . the plaintiff must introduce evidence . . . [showing] . . . it is more likely than not that the conduct of the defendant was a substantial factor in bringing about the result.”); see also Sweredoski, 2013 WL 3010419, at *2 (quoting Almonte, 46 A.3d at 18) (“With regard to causation, ‘[a] plaintiff must not only prove that a defendant is the cause-in-fact of an injury, but also must prove that a defendant proximately caused the injury.’”); Flores, 232 S.W.3d at 770-72 (stating that “proof of mere frequency, regularity, and proximity is necessary but not sufficient, as it provides none of the quantitative information necessary to support causation under Texas law”).
from a Maryland Court of Appeals case requiring frequent, regular, and proximate exposure as a sort of substitute dose assessment. At the time, the adoption of this ruling appeared favorable to defendants because it prevented plaintiffs from arguing that limited exposure from “fiber drift” or bystander work would suffice in the context of insulators or asbestos factory workers, even though the plant or job produced mesotheliomas in other, more heavily-exposed workers.

The Lohrmann standard did in fact help rein in some excesses in the litigation, but it is a limited and flawed form of substitute for an actual and competent dose assessment. The “frequent, regular, and proximate” approach does not solve the problem—it only begs the question of how frequent, how regular, and how proximate the exposure should be to suffice.

A Maryland every exposure case illustrates the difficulty. In Dixon v. Ford Motor Co., the plaintiff was the wife of a man who worked as a mechanic performing two brake jobs a week for about thirteen years. The court rejected every exposure testimony but resorted to the Lohrmann standard and determined that 1,000 brake jobs over thirteen years was easily sufficient frequency and regularity. Not only that, but the court then extended its scientific opinion to conclude that the wife’s contact with the husband’s clothing (not the brake work itself) was also sufficient proximity and degree of exposure, without taking into account the diminishing degree of take-home exposures. Nowhere does the court

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409 Lohrmann, 782 F.2d at 1162 (citing Robin Express Transfer, Inc., 338 A.2d. at 343).
410 For a discussion of the “fiber drift” theory, see Sanders, supra note 1, at 1163 nn.74-75.
412 See, e.g., Flores, 232 S.W.3d at 770-72 (adding a dose assessment to the established Lohrmann requirement).
413 See, e.g., Moeller v. Garlock Sealing Techs., LLC, 660 F.3d 950, 955 (6th Cir. 2011) (“Plaintiff failed to establish how many Garlock gaskets he removed, or how frequently he removed—as opposed to installed—they.”).
414 70 A.3d 328 (Md. 2013).
415 Dixon, 70 A.3d at 330-33.
416 Id. at 334-36.
417 Id. at 334, 336.
analyze the dose from this exposure or even require any kind of dose estimate, nor did the court indicate why 1,000 low-level exposures from brake work are enough to produce disease, much less the even lower take-home exposures of the wife. 418 The court also failed to acknowledge the importance of the many epidemiological studies failing to find mesothelioma among full-time and lifelong brake workers, some of whom surely performed many thousands of brake jobs, much less the complete lack of any epidemiological study finding excess mesothelioma among mechanics’ spouses. 419 The gist of the opinion is that court made its own determination as to “how much is enough” based on its assumption that 1,000 brake jobs should be enough. 420 The result is an opinion based not on science but on some “gut” feeling of the court.

Earlier, this same court examined a different exposure scenario and reached a similar result, apparently based largely on a “we’ll know it when we see it” analysis. In Scapa Dryer Fabrics, Inc. v. Saville, the court deemed only one year of “regularly” handling a non-friable asbestos product on a daily basis sufficient to go to the jury. 421 Again, the decision did not include any dose assessment or proof that handling such products had produced mesothelioma in the past. 422 The court resorted to its own instincts as to how much is enough. In 2012, the Nevada Supreme Court in Holcomb v. Georgia Pacific, LLC 423 declined to adopt the near every exposure test that the plaintiff asserted applied under the Rutherford v. Owens-Illinois Inc. 424 case in California, and the court also declined to adopt the more rigorous (but scientifically accurate) Texas’s

418 Id. at 337.
419 Id. at 334-36 (finding that the plaintiff expert’s characterization of epidemiological studies, in particular the case-control studies, which “have shown a connection between working on brakes and mesothelioma” were not subject to Frye analysis because the court did not find them to be a “novel scientific method”); In re N.Y. City Asbestos Litig. (Juni II), 148 A.D.3d 233, 237 (N.Y. App. Div. 2017) (finding the expert acknowledged twenty-one out of twenty-two brake worker studies show no association with mesothelioma), aff’d sub nom. (Juni III), 116 N.E.3d 75 (N.Y. 2018).
420 Dixon, 70 A.3d at 336.
421 16 A.3d 159, 164 (Md. 2011).
422 Scapa, 16 A.3d at 168.
Borg-Warner Corp. v. Flores test. The court instead adopted what it called “a balanced approach” that relied on the Lohrmann standard. The exposure testimony in this case was extremely vague, boiling down to plaintiff testifying that he used defendants’ products “numerous times.” Thus, the “balance” this court attempted to achieve resulted in the case going to the jury without any scientific analysis.

Courts adopting this compromise approach seem to believe they are achieving a goal for both sides. But, in today’s low-dose asbestos litigation, it is not difficult for plaintiffs to alleging a number of exposure incidents that will satisfy the Lohrmann frequent, regular, and proximate standard in nearly every case. This flaw is especially applicable if courts believe that five or six times is “frequent” and “regular” enough and being inside a factory building with asbestos in it is “proximate” enough. However, the low number of exposures in no way approach the kinds of doses demonstrated in epidemiology studies that are required to cause asbestos-related disease.

These courts call their approach a “balanced” one, but in reality, they have adopted a test with one thumb on the scale; it will be very difficult for a defendant to obtain dismissal if plaintiffs simply couch their testimony with phrases like “numerous times” and “nearby.” The result is a set of determinations with no scientific foundation, and these determinations will extend unwarranted asbestos cases into the future indefinitely.

More critically for a gatekeeping analysis, these courts are asking their state’s trial judges to make the critical and difficult expert determinations as to how much exposure to asbestos is enough to sufficiently link

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232 S.W.3d 765 (Tex. 2007).
Holcomb, 289 P.3d at 193-95.
Id. at 195-96 (citing Lohrmann v. Pittsburgh Corning Corp., 782 F.2d 1156, 1162-63 (4th Cir. 1986)).
Id. at 198.
See id. at 190 (stating that taking a balancing approach “is not overly rigorous or too relaxed in order to ensure protection for both manufacturers and consumers”).
Anderson & Tuckley, supra note 7, at 281-83.
Id. at 281-83.
Id. at 281, 283.
causation to a disease such as mesothelioma. These determinations by courts are made with no guidance based on medical science (at least from the plaintiffs’ experts) to sort frequent from infrequent or regular from insufficient exposures. The trial judges are essentially forced into this position by plaintiffs’ experts, who have seemingly abandoned their role to help the jury (and the judge) sort causative exposures from those that are not. The trial judge’s role is to be the gatekeeper of expert testimony, not to sit in the expert’s seat by rendering an opinion that a certain number or type of exposures is sufficient for a jury’s causation determination. Decisions on the degree and type of exposure necessary to cause mesothelioma are the subject of hundreds of scientific articles and intense medical debate in current literature. Professionals in several fields—epidemiology, toxicology, occupational medicine, oncology, industrial hygiene, and others—regularly apply their expertise and extensive knowledge to assess, for instance, whether a long-term exposure to chrysotile could ever be considered a cause of mesothelioma. Courts adopting the compromise approach, however, simply declare a certain number of exposures to be sufficient. Doing so is quite a leap for a judge, given the degree of scientific knowledge required to make such a determination.

434 Id. at 282.
435 Id. at 283.
436 Id.
437 Anderson & Tuckley, supra note 7, at 284-85.
438 See, e.g., John T. Hodgson & Andrew Darnton, The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure, 44 ANNALS OCCUPATIONAL HYGIENE 565, 565 (2000) (providing extensive analysis of the exposure levels and types of fibers causing asbestos disease); Rake et al., supra note 229, at 1175 (discussing a population-based study of the “risks and numbers of cases caused by specific occupational and environmental exposures” from asbestos).
439 See, e.g., Holcomb v. Georgia Pacific, LLC, 289 P.3d 188, 195 (Nev. 2012) (citing Lohrmann v. Pittsburgh Corning Corp., 782 F.2d 1156, 1162 (4th Cir. 1986)) (adopting the Lohrmann “frequency, regularity, proximity” test which looked at whether a specific defendant’s product caused the plaintiff’s disease and rejecting the Flores test which required a plaintiff to prove the total asbestos dosage to which the plaintiff was exposed).
440 The Maryland Court of Appeals in Dixon v. Ford Motor Co. (cited by the plaintiffs) fell into this trap—the court decided one year of brake work was enough, under that state’s “frequency, regulatory, and proximity” test for asbestos causation,
Trial judges operating in these states face an enormous difficulty: Is six months of contact with asbestos products enough? Three? What if the exposures cover ten years but occur only once a month? What if the exposure is merely handling a brake pad with no disruption of the resin bound material, or merely a resident in the worker’s home who never touches the product at all? The answers to these and an infinite number of similar questions lie in the science of dose, exposure, and epidemiology.441 If the experts offer scientific testimony on how much exposure in fact causes disease, a trial court can perform the required gatekeeping function and decide whether the expert’s analysis of the data is based on a reliable methodology.442 However, where the expert simply refuses to perform this analysis at all, resorting instead to “no safe dose” formulations, there is nothing with which the court can work.443 Courts adopting the compromise position should not have tried to fill in the gap created by this testimony and instead should have dismissed the cases for lack of adequate expert causation testimony.

Very few courts to date have taken the supposedly middle road.444 In reality, when courts adopt this “middle” approach, the result is nearly identical to courts that allow every exposure testimony—the supposedly balanced approach leads to speculative expert testimony going to the jury. The effect then is not meaningfully different from admission of every exposure testimony.445 Moreover, states like Maryland and Illinois that

441 See Rake et al., supra note 229, at 1175 (examining dosage exposure to asbestos in an epidemiological case study).

442 See United States v. Frazier, 387 F.3d 1244, 1260 (11th Cir. 2004) (“As the Supreme Court made abundantly clear in Dabuert, Rule 702 compels the district courts to perform the critical “gatekeeping” function concerning the admissibility of expert scientific evidence.”).

443 See Pluck v. BP Oil Pipeline Co., 640 F.3d 671, 675 (6th Cir. 2011) (excluding “no safe does” testimony because it was discredited by other courts).

444 See generally Holcomb, 289 P.3d at 195 (citing Lohrmann, 782, F.2d 1156) (adopting “a balanced approach” that relied on the Lohrmann standard).

445 See supra Part III, “Court Rulings Rejecting Every Exposure Testimony and the Erroneous Basis for Recent Contradictory Opinions,” and accompanying footnotes.
still rely on Lohrmann need to understand that the Lohrmann test has become wholly insufficient to keep unwarranted cases away from a jury. Courts faced with low exposure testimony should not hesitate to test the science and apply the rigor of the above three-part analysis. Even the compromise approach is just another form of speculative causation case, this time with speculation sponsored by the trial judge.\footnote{See Danielle Conway-Jones, \textit{Factual Causation in Toxic Tort Litigation: A Philosophical View of Proof and Certainty in Uncertain Disciplines}, 35 U. RICH. L. REV. 875, 920-22 (2002) (noting how judges “retain significant power to either admit or exclude, consistent with their respective philosophical approaches, expert testimony on factual causation in toxic tort litigation” and how vague standards make it challenging for litigants and courts to know admissibility standards).}

**Conclusion**

Most toxic tort litigation has long since passed the era when workers and others were exposed to large amounts of materials in doses believed to be safe at the time that turned out not to be. We are left today with much lower contact with substances that can be hazardous, but only at higher doses. Courts are thus going to be increasingly faced with the need to sort legitimate cases from those that are speculative. Compounding the problem, with the development of amazingly sensitive testing techniques, experts can now detect chemicals and substances at incredibly low levels—parts per trillion and even lower. The use of scanning electron microscopy in asbestos analysis performs much the same miracle of identification. But in no way should the mere detection of these materials suffice for causation in a court of law. Courts must sharpen their gatekeeping focus to deal with these cases and be prepared to exclude experts who cannot provide real proof of causation. The result otherwise will be a flood of cases with no scientific support—a reality that is unfortunately well underway in the asbestos world.