

THE “ANY EXPOSURE” THEORY: AN UNSOUND BASIS FOR ASBESTOS CAUSATION AND EXPERT TESTIMONY

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Over the years asbestos litigation has morphed into a tort world all of its own.¹ Courts developed entire sets of rules in an attempt to manage efficiently their substantial asbestos dockets,² in the process dispensing with

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1. See Griffin B. Bell, *Asbestos Litigation and Judicial Leadership: The Courts’ Duty to Help Solve the Asbestos Litigation Crisis*, BRIEFLY, June 2002, at 1, 4; Mark A. Behrens, *Some Proposals for Courts Interested in Helping Sick Claimants and Solving Serious Problems in Asbestos Litigation*, 54 BAYLOR L. REV. 331, 336-42 (2002); Paul F. Rothstein, *What Courts Can Do in the Face of the Never-Ending Asbestos Crisis*, 71 MISS. L.J. 1, 4-9 (2001).

2. See *In re Combustion Eng’g, Inc.*, 391 F.3d 190, 200 (3d Cir. 2004) (“For decades, the state and federal judicial systems have struggled with an avalanche of asbestos lawsuits.”). The United States Supreme Court has described the litigation as a “crisis.” *Amchem Prods., Inc. v. Windsor*, 521 U.S. 591, 597 (1997). Through 2002, approximately 730,000 claims had been filed. STEPHEN J. CARROLL ET AL., ASBESTOS LITIGATION xxiv (RAND Inst. for Civil Justice 2005), available at http://www.rand.org/pubs/monographs/2005/RAND_MG162.pdf. “In August 2005, the Congressional Budget Office estimated that there were about 322,000 asbestos bodily injury cases pending in state and federal courts.” AM. ACAD. OF ACTUARIES’ MASS TORTS SUBCOMM., OVERVIEW OF ASBESTOS CLAIMS ISSUES AND TRENDS 5 (2007), available at http://www.actuary.org/pdf/casualty/asbestos_aug07.pdf.

many standard venue, discovery, and trial consolidation requirements.³ The changes almost universally favored plaintiffs and instead of affecting a reduction in congested dockets, the litigation became so malleable and lucrative that plaintiff attorneys have spent the last decade searching for the “next asbestos.” Practitioners in this field have come to know these asbestos rules well, whereas newcomers are often astounded to discover that their tort law frame of reference means little in the alternative universe of asbestos litigation.

One of the most substantial departures from black letter tort law is the *any exposure* theory of causation, sometimes referred to as the *any fiber* theory.⁴ In a nutshell, the *any exposure* theory contends that because asbestos disease is a cumulative, dose-response process, each and every exposure to asbestos during a person’s lifetime, no matter how small or trivial, substantially contributes to the ultimate disease (e.g., asbestosis, lung cancer, or mesothelioma).⁵ There is an important caveat, however, in that most proponents of this theory agree that *background* exposures to asbestos, even though they may contribute millions of fibers to an individual’s lungs over a lifetime, do *not* contribute to the development of disease.⁶ Only occupational or para-occupational (e.g., home remodeling or “shade tree” automotive brake repair) exposures count.⁷ The theory allows plaintiffs’ counsel to sue thousands of defendants every year whose “contribution” to disease is trivial and far below the type of doses actually known to cause disease, while at the same time excluding from causation another source of millions of fibers (i.e., background exposures).

In the last three years, more than a dozen courts in multiple jurisdictions have excluded or criticized *any exposure* causation testimony, either as unscientific under a *Daubert*⁸/*Frye*⁹ analysis or as insufficient to support causation.¹⁰ This pattern of decisions includes:

- the Texas Supreme Court in a mechanic/asbestosis case,

3. See Victor E. Schwartz & Rochelle M. Tedesco, *The Law of Unintended Consequences in Asbestos Litigation: How Efforts to Streamline the Litigation Have Fueled More Claims*, 71 MISS. L.J. 531, 542-47 (2001); Victor E. Schwartz & Leah Lorber, *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, 256-58 (2000).

4. See, e.g., *infra* notes 26, 30-31.

5. See, e.g., *infra* note 50.

6. See *Bartel v. John Crane, Inc.*, 316 F. Supp. 2d 603, 607-08 (N.D. Ohio 2004), *aff’d sub nom.* *Lindstrom v. A-C Prod. Liab. Trust*, 424 F.3d 488 (6th Cir. 2005).

7. See, e.g., *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 773 (Tex. 2007), *reh’g denied* (Oct. 12, 2007).

8. See *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579 (1993).

9. See *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923).

10. See, e.g., *infra* notes 11-19.

rejecting the testimony of Dr. Barry Castleman and another expert that mere proof of exposure is sufficient for causation;¹¹

- a Texas appellate court in a mesothelioma case, rejecting the testimony of Dr. Samuel Hammar that any dry wall exposures above 0.1 fibers/cc year would be a substantial contributing factor;¹²
- the Texas Multi-District Litigation (“MDL”) court, rejecting the testimony of Dr. Eugene Mark in a friction product case and other experts in an electrician/dry wall exposure case;¹³
- the Pennsylvania Supreme Court in a mesothelioma case against an auto parts company, rejecting the position espoused in affidavits by Drs. Richard Lemen, James Girard, and Arthur Frank;¹⁴
- an Ohio federal district court and the Sixth Circuit Court of Appeals in a gasket and packings case, rejecting the testimony of Drs. Arthur Frank and Yasunosuki Suzuki;¹⁵
- three Pennsylvania state trial courts, rejecting the *any exposure* testimony of Drs. John Maddox, Eugene Mark, William Longo, Jonathan Gelfand, and Arthur Frank in friction product cases and criticizing the theory’s application in a pleural disease case;¹⁶
- a federal bankruptcy court in litigation involving asbestos in vermiculite insulation, rejecting Dr. Henry Anderson’s *any exposure* approach;¹⁷
- a Mississippi appellate court, rejecting a medical monitoring class for persons allegedly exposed in a school building;¹⁸ and
- two Washington State trial court decisions by different judges, rejecting the opinions of Drs. Samuel Hammar and Carl

11. *See Flores*, 232 S.W.3d at 774.

12. *See Georgia-Pac. Corp. v. Stephens*, 239 S.W.3d 304, 320-21 (Tex. App. 2007), *reh’g overruled* (Oct. 13, 2007), *review denied* (Feb. 22, 2008).

13. *See Letter Ruling, In re Asbestos Litig.*, Cause No. 2004-03964 (Tex. Dist. Ct. Jan. 20, 2004); *Letter Ruling, In re Asbestos*, Cause No. 2004-3,964 (Tex. Dist. Ct. July 18, 2007).

14. *See Gregg v. V-J. Auto Parts, Inc.*, 943 A.2d 216, 218, 223, 226-27 (Pa. 2007).

15. *See Bartel*, 316 F. Supp. 2d at 611.

16. *See In re Toxic Substance Cases*, No. A.D. 03-319, 2006 WL 2404008 at *7-8 (Pa. Ct. Com. Pl. Aug. 17, 2006); *Basile v. Am. Honda Motor Co.*, No 11484 CD 2005 (Pa. Ct. Com. Pl. Feb. 22, 2007); *In re Asbestos Litig.*, Certain Asbestos Friction Cases Involving Chrysler LLC, No. 0001 Control #084682 (Pa. Ct. Com. Pl. Sept. 24, 2008); *Summers order v. Certainteed Corp.*, 886 A.2d 240, 244 (Pa. Super. Ct. 2005), *appeal granted*, 897 A.2d 460 (Pa. 2006).

17. *See In re W.R. Grace & Co.*, 355 B.R. 462, 474, 478 (Bankr. D. Del. 2006), *leave to appeal denied*, No. 07-MC-0005 RLB, 01-1139, 2007 WL 1074094 (D. Del. Mar. 26, 2007).

18. *See Brooks v. Stone Architecture, P.A.*, 934 So. 2d 350 (Miss. Ct. App. 2006).

Brodkin in heavy equipment mechanic cases.¹⁹

These are not insignificant courts—they include two state supreme courts, one federal appellate court, a federal bankruptcy court, and state appellate and trial courts in several jurisdictions.²⁰ In addition, the breadth of alleged exposures and diseases covered by these cases demonstrates that the *any exposure* theory is failing across the spectrum of asbestos cases, regardless of disease and type of exposure. Perhaps most remarkably, the experts whose testimony is being excluded are veterans in the litigation who have supported plaintiff cases for many years with little or no interference from the judiciary.²¹ The rejection of these experts' causation testimony, while a significant departure from past practice, reflects the sound application of standard causation rules to asbestos testimony²²—something that should have happened years ago and is finally gaining traction. These rulings also likely reflect a growing skepticism of many asbestos claims in the wake of findings of massive fraud in federal court silica litigation.²³

This Article discusses the underpinnings of the *any exposure* causation theory and why recent courts that have examined the theory more carefully

19. See *Anderson v. Asbestos Corp.*, No. 05-2-04551-5SEA, slip op. at 144-45 (Wash. King County Super. Ct. Oct. 31, 2006) (transcript of bench ruling) (Erllick, J.); *Free v. Ametek*, No. 07-2-04091-9-SEA (Wash. King County Super. Ct. Feb. 29, 2008) (Barnett, J.) (ruling on motion *in limine*).

20. See *supra* notes 11-19 and accompanying text.

21. See *infra* notes 50-53.

22. See, e.g., *Flores*, 232 S.W.3d at 770 (discussing the “substantial factor” test in causation); David E. Bernstein, *Getting to Causation in Toxic Tort Cases*, 74 BROOK. L. REV. 51, 59 (2008) (stating that “[t]he recent, increasingly strict exposure cases . . . reflect a welcome realization by state courts that holding defendants liable for causing asbestos-related disease when their products were responsible for only *de minimis* exposure to asbestos, and other parties were responsible for far greater exposure, is not just, equitable, or consistent with the substantial factor requirements of the *Restatement (Second)* and *Lohrmann [v. Pittsburgh Corning Corp.]*, 782 F.2d 1156 (4th Cir. 1986).”); cf. Lee S. Siegel, Note, *As the Asbestos Crumbles: A Look at New Evidentiary Issues in Asbestos-Related Property Damage Litigations*, 20 HOFSTRA L. REV. 1139, 1146 (1992) (“There is no merit to the one fiber theory, and the myth is slowly being dispelled.”).

23. See *In re Silica Prods. Liab. Litig.*, 398 F. Supp. 2d 563, 635 (S.D. Tex. 2005); Lester Brickman, *Disparities Between Asbestosis and Silicosis Claims Generated by Litigation Screenings and Clinical Studies*, 29 CARDOZO L. REV. 513 (2007); Lester Brickman, *On the Applicability of the Silica MDL Proceeding to Asbestos Litigation*, 12 CONN. INS. L.J. 289 (2006); see also Editorial, *Screening for Corruption*, WALL ST. J., Dec. 2, 2005, at A10, abstract available at 2005 WLNR 19447615; Editorial, *Silicosis, Inc.*, WALL ST. J., Oct. 27, 2005, at A20, abstract available at 2005 WLNR 17413061; Editorial, *The Silicosis Sheriff*, WALL ST. J., July 14, 2005, at A10, abstract available at 2005 WLNR 11084626; David Hechler, *Silica Plaintiffs Suffer Setbacks: Broad Effects Seen in Fraud Allegations*, NAT'L L.J., Feb. 28, 2005, at 1; Roger Parloff, *Diagnosis for Dollars: A Court Battle Over Silicosis Shines a Harsh Light on Mass Medical Screeners—The Same People Whose Diagnoses Have Cost Asbestos Defendants Billions*, FORTUNE, June 13, 2005, at 96, available at 2005 WLNR 8694138; Jonathan D. Glater, *Companies Get Weapon in Injury Suits Many Silica-Damage Plaintiffs Also Filed Claims Over Asbestos*, N.Y. TIMES, Feb. 2, 2005, at C1, available at 2005 WLNR 1415209.

have decided to reject it. These decisions reflect a proper assessment of the *dose requirement* of toxicology.²⁴ On the other hand, courts that continue to allow *any exposure* testimony to proceed unchallenged run the risk of encouraging a flood of speculative or trivial claims at a time when the litigation environment for asbestos claims appears to be regaining some semblance of control.²⁵ Such an outcome would reflect poor science and even poorer public policy.

I. THE TOXICOLOGICAL REQUIREMENT OF DOSE AND ITS APPLICATION IN THE TOXIC TORT CONTEXT

The *any exposure* theory can only be understood against the backdrop of widely accepted tort and medical causation principles because the theory departs so dramatically from those principles. Ordinarily, under long-standing rules of tort law, courts should require asbestos plaintiffs to demonstrate that each defendant’s product was either a “but-for” cause or a “substantial factor” in the cause of plaintiff’s disease.²⁶ 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.²⁷ The concept of a necessary dose goes back to the sixteenth century, when the “father of toxicology,” physician and philosopher Paracelsus, first articulated the principle that the dose makes the poison: “All substances are poisonous—there is none which is not; the dose differentiates a poison from a remedy.”²⁸ Examples are

24. See David E. Bernstein, *Keeping Junk Science Out of Asbestos Litigation*, 31 PEPP. L. REV. 11, 28 (2003) (“There is clearly some relationship between asbestos and diseases. The effects of exposure to asbestos on a particular individual, however, depend on the level of exposure and what type of asbestos one was exposed to and for how long.”).

25. See Mark A. Behrens & Phil Goldberg, *The Asbestos Litigation Crisis: The Tide Appears to Be Turning*, 12 CONN. INS. L.J. 477 (2006); James A. Henderson, Jr., *Asbestos Litigation Madness: Have the States Turned a Corner?*, MEALEY’S TORT REFORM UPDATE, vol. 3:6, Jan. 18, 2006, at 23; Patti Waldmeir, *The Americas: Asbestos Litigation Declines in Face of US Legal Reforms*, FIN. TIMES, July 24, 2006, at 2, available at 2006 WLNR 12719566; Martha Neil, *Backing Away from the Abyss: Courts May Be Starting to Get a Grip on Asbestos Litigation*, A.B.A. J., Sept. 2006, at 26.

26. See RESTATEMENT (SECOND) OF TORTS §§ 431, 433 (1965).

The word “substantial” is used to denote the fact that the defendant’s conduct has such an effect in producing the harm as to lead reasonable men to regard it as a cause . . . rather than in the so-called “philosophical sense,” which includes every one of the great number of events without which any happening would not have occurred.

Id. at § 431 cmt. a.

27. See *infra* notes 29-31 and accompanying text.

28. David L. Eaton, *Scientific Judgment and Toxic Torts—A Primer in Toxicology for Judges and Lawyers*, 12 J.L. & POL’Y 5, 11 (2003) (emphasis omitted) (internal quotation marks omitted).

commonplace—alcohol, aspirin, sunlight, even basic substances we eat in food and vitamins like zinc are not harmful at low levels, but can cause harm at higher doses.²⁹

This dose concept is widely recognized in both science and courts as the foundation of causation and the basis for many medical tort decisions.³⁰ Courts around the country, including at least five federal circuit courts, have recognized the necessity of proving an actual toxic dose in medical tort cases.³¹ As one leading researcher recently wrote: “Dose is the single most

(quoting CASARETT AND DOULL’S TOXICOLOGY: THE BASIC SCIENCE OF POISONS, Chs. 1, 4 (Curtis D. Klaassen ed., McGraw Hill 6th ed. 2001)).

29. A fundamental tenet of toxicology is that “the dose makes the poison.” Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology*, in FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 401, 403 (West Group 2d ed. 2000) (1994) (internal quotation marks omitted). Thus, courts routinely require plaintiffs to demonstrate not just some exposure, but “evidence from which the trier of fact could conclude that the plaintiff was exposed to levels of toxins sufficient to cause the harm complained of.” *Nelson v. Tenn. Gas Pipeline Co.*, No. 95-1112, 1998 WL 1297690, slip op. at *6 (W.D. Tenn. Aug. 31, 1998), *aff’d*, 243 F.3d 244 (6th Cir.), *cert. denied*, 534 U.S. 822 (2001) (citing *Wintz v. Northrop Corp.*, 110 F.3d 508, 513 (7th Cir. 1997) (internal citation omitted)); *see also Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1107 (8th Cir. 1996). This is as true for asbestos as for any other potentially toxic substance. *See Bartel*, 316 F. Supp. 2d at 611 (rejecting “one-fiber” asbestos theory as not supported by medical literature); *In re Toxic Substance Cases*, 2006 WL 2404008 at *7-8 (criticizing plaintiffs’ experts for failing to assess the dose for mechanic exposure).

30. *See, e.g., 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’”) (emphasis added) (quoting *Allen v. Pa. Eng’g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996)).

31. *See, e.g., id.* (explaining that plaintiffs must establish the level at which substance is harmful and that their exposures were of that level); *Nelson*, 1998 WL 1297690 at *6 (excluding opinion of expert who did not assess dose because “[a]n appropriate methodology requires evidence from which the trier of fact could conclude that the plaintiff was exposed to levels of toxin sufficient to cause the harm complained of.”); *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*, 91 F.3d at 1106); *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 RADS.”), *cert. denied*, 526 U.S. 1064 (1999); *Allen*, 102 F.3d at 199 (“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 to sustain the plaintiffs’ burden in a toxic tort case.”); *Cano v. Everest Minerals Corp.*, 362 F. Supp. 2d 814, 825 (W.D. Tex. 2005) (quoting *Merrell Dow Pharms., Inc. v. Havner*, 953 S.W.2d 706, 720 (Tex. 1997)) (“[A] claimant must not only introduce sufficient epidemiological evidence, he must also show that he is similar to those in the studies.”); *Nat’l Bank of Commerce v. Dow Chem. Co.*, 965 F. Supp. 1490, 1524 (E.D. Ark. 1996) (explaining plaintiff must provide evidence of level of exposure and show that the dose was likely to produce harm of the type experienced by plaintiff); *Louderback v. Orkin Exterminating Co., Inc.*, 26 F. Supp. 2d 1298, 1305 (D. Kan. 1998) (“[T]o recover in a toxic tort case, the plaintiff must prove the levels of exposure that are hazardous to human beings generally as well as the plaintiff’s actual level of

important factor to consider in evaluating whether an alleged exposure caused a specific adverse effect.”³²

Parker v. Mobil Oil Corp.,³³ a recent non-asbestos case involving benzene, illustrates the point and the reasoned approach of many courts. In *Parker*, a gas station attendant alleged that he developed acute myeloid leukemia (“AML”) from low level benzene exposures in gasoline.³⁴ Epidemiology studies have demonstrated that high exposures to pure benzene, typically in factory settings, can cause AML, but studies have not demonstrated the occurrence of disease from low-exposure gas station work where the exposures involved only a small amount (usually two to five percent) of benzene in gasoline.³⁵ Plaintiff’s experts, Drs. Phil Landrigan and Bernard Goldstein, extrapolated down from the high-dose, factory benzene exposure studies and cited to government regulations and mathematical modeling studies to opine that low level exposures would likewise cause the disease.³⁶ They did so, however, without any assessment of the actual dose from gas station work; they could not present any evidence that the plaintiff’s dose approached those shown to cause disease in the epidemiology studies of high-dose workers.³⁷ Instead, they expressed their opinions in subjective terms, referring to the plaintiff’s exposures as “substantial” or “significant” with no grounding in actual dose calculations or comparisons.³⁸

The New York Court of Appeals rejected this methodology as unreliable under New York’s general requirements for reliability and proper foundation to support an evidentiary submission.³⁹ The decision focused on the flawed approach to dose and unsupported assumptions that low doses produce the same effects as high doses:

The experts, although undoubtedly highly qualified in their respective fields, failed to demonstrate that exposure to benzene as a component of gasoline caused Parker’s AML. Dr. Goldstein’s general, subjective and

exposure to the toxic substance.”) (quoting *Wright*, 91 F.3d at 1106); *Mancuso v. Consol. Edison Co.*, 967 F. Supp. 1437, 1453 (S.D.N.Y. 1997) (explaining that expert’s testimony that plaintiffs’ ailments were caused by exposure to PCBs was inadmissible because, *inter alia*, expert “did not make sufficient determinations of environmental PCB levels, nor of the extent of the plaintiffs’ exposure thereto.”).

32. *Eaton*, *supra* note 28, at 11.

33. 857 N.E.2d 1114 (N.Y. 2006), *reargument denied*, 861 N.E.2d 104 (N.Y. 2007).

34. *Id.* at 1116.

35. *Id.* at 1117.

36. *Id.* at 1122.

37. *Id.*

38. *Id.* at 1121-22.

39. *Id.* at 1120-22.

conclusory assertion—based on Parker’s deposition testimony—that Parker had “far more exposure to benzene than did the refinery workers in the epidemiological studies” is plainly insufficient to establish causation. It neither states the level of the refinery workers’ exposure, nor specifies how Parker’s exposure exceeded it, thus lacking in epidemiologic evidence to support the claim.⁴⁰

The New York court thus rejected the notion that low level, unquantified exposures to a known harmful substance necessarily suffices as proof of causation of a disease the substance is known to produce at much higher exposure levels.⁴¹ This is classic toxicology, applied properly in the courtroom setting.

Parker has many antecedents similarly rejecting assumed causation at low levels, including, for instance, the United States Supreme Court’s *General Electric Co. v. Joiner* ruling,⁴² which rejected alleged PCB injury without a dose assessment,⁴³ and the Sixth Circuit’s *Nelson v. Tennessee Gas Pipeline Co.* decision,⁴⁴ which likewise rejected alleged environmental harm from PCB exposure without any assessment of the actual dose.⁴⁵ The concept of a sufficient dose to cause disease is fundamental to both science and tort law, and should not be jettisoned in favor of a mere “exposure only” approach.

II. THE ASBESTOS ANY EXPOSURE THEORY

In contrast to the traditional tort approach requiring some assessment of dose, some courts presiding over asbestos cases have permitted plaintiffs to demonstrate merely that they were exposed to a defendant’s product, rather than require proof that any particular exposure was high enough to cause a plaintiff’s disease.⁴⁶ The result is that the causation dose requirement—real exposure, at quantities known to cause disease—was reduced to an exposure test, and a minimal one at that. Some verdicts have stretched the

40. *Id.* at 1121-22.

41. *Id.* at 1122.

42. 522 U.S. 136 (1997).

43. *Id.* at 144-47.

44. 243 F.3d 244 (6th Cir.), cert. denied, 534 U.S. 822 (2001).

45. *Id.* at 252-54.

46. See, e.g., *Jones v. John Crane, Inc.*, 35 Cal. Rptr. 3d 144, 151-52 (Ct. App. 2005) (finding evidence of exposure to defendant’s asbestos products, regardless of level of exposure, was sufficient to establish causation); *Celotex Corp. v. Tate*, 797 S.W.2d 197, 203 (Tex. App. 1990), writ dismissed by agreement (Aug. 16, 1996); see generally Steven D. Wasserman et al., *Asbestos Litigation in California: Can it Change for the Better?*, 34 PEPP. L. REV. 883, 897-99 (2007) (discussing California cases involving *de minimis* exposures).

concept so far that virtually any exposure, regardless of degree or frequency, suffices.⁴⁷

The foundation for these opinions is the *any exposure* theory, sometimes called the *any fiber* theory.⁴⁸ Rather than assess dose, the experts who support this theory simply opine that any occupational or product-related exposure to asbestos fibers is sufficient—there is no minimum.⁴⁹ As a result, they regularly opine that every exposure a plaintiff received from any occupational or hobby-related work is a substantial factor in causing disease.⁵⁰ The opinions will encompass all such activities,

47. Some examples include a verdict upholding a \$4 million judgment against Union Carbide, based on the *any exposure* theory, when plaintiff could not even recall using defendant's product, see *California Court: Conflicting Evidence Could Have Resulted in Verdict for Asbestos-Exposed Man*, MEALEY'S LITIG. REP.: ASBESTOS, vol. 22:22, Dec. 12, 2007, at 4; a \$5 million verdict against John Crane based on any exposure to rope and gaskets without any assessment of the dose or fiber release from those products, see *Judge: Daughter's Showing That Father was Exposed, Product was Present Sufficient*, MEALEY'S LITIG. REP.: ASBESTOS, vol. 22:22, Dec. 12, 2007, at 5; a \$35 million verdict for “exposure” to Leslie Control's “small pump and valve components” in the Navy, ignoring large-scale exposure to Navy insulation, see *\$35.1M Awarded to Couple for Exposure to Asbestos in Navy*, MEALEY'S LITIG. REP.: ASBESTOS, vol. 22:19, Nov. 1, 2007, at 3; and a verdict of \$3.92 million against General Electric alleging exposure from brakes in cranes and a mill motor, apparently with no assessment of the minimal dose those exposures likely would produce, see *Maryland Asbestos Jury Awards \$3.92 Million to 3 Steelworkers' Families*, MEALEY'S ASBESTOS, BANKR. REP., vol. 7:1, Aug. 1, 2007, at 12. See also *Flores v. Borg-Warner Corp.*, 153 S.W.3d 209, 214 (Tex. App. 2004) (finding a product “emitting” dust or “working in the presence of” dust deemed sufficient for causation), *rev'd*, 232 S.W.3d 765 (Tex. 2007), *reh'g denied* (Oct. 12, 2007).

48. Some plaintiff experts have testified that breathing even a single fiber of asbestos could cause disease. When this approach began to be criticized, the theory became more commonly articulated as “every exposure,” “any exposure,” “every breath,” or similar phrases. Some plaintiffs' experts state simply that any exposure above background is sufficient, while others attach a number as a cutoff (e.g., Dr. Samuel Hammar's 0.1 fibers/cc year level, or Dr. John Maddox's 0.0003 fibers/cc single exposure cutoff), but the result is usually the same—most if not all occupational exposures are captured. See *infra* notes 50-51 and accompanying text.

49. See *Gregg*, 943 A.2d at 226 (“We recognize that it is common for plaintiffs to submit expert affidavits attesting that any exposure to asbestos, no matter how small, is a substantial contributing factor in asbestos disease.”); *Georgia-Pac. Corp.*, 239 S.W.3d at 308 (stating plaintiffs relied on “expert testimony that any exposure to asbestos contributes to cause mesothelioma”); *Lindstrom*, 424 F.3d at 498 (stating plaintiff experts contended that “[o]nce mesothelioma is diagnosed, it is impossible to rule out any of Mr. Lindstrom's exposures as being substantially contributory.”).

50. See *Georgia-Pac. Corp.*, 239 S.W.3d at 315 (stating opinion of plaintiffs' expert Jerry Lauderdale was “that every exposure does contribute to the development of potential to develop mesothelioma.”); *Summers*, 886 A.2d at 244 (quoting plaintiffs' expert Dr. Jonathan Gelfand stating, “Each and every exposure to asbestos has been a substantial contributing factor to the abnormalities noted.”); *Bartel*, 316 F. Supp. 2d at 611 (criticizing testimony of Drs. Arthur Frank and Yasunosuke Suzuki “that every exposure to asbestos [plaintiff] had during his working career, no matter how small, was a substantial factor in causing his peritoneal mesothelioma”); *In re Toxic Substance Cases*, 2006 WL 2404007 at *1 (rejecting testimony of plaintiffs' experts, Drs. Maddox and Laman, who opined that “every single exposure to every asbestos product is a

regardless of duration or dose—a single backyard brake job, one remodeling job using asbestos-containing joint compound, walking by a gasket repair job on an engine—all have been targeted by plaintiffs' experts as the cause of mesothelioma.⁵¹

The *any exposure* plaintiffs' experts typically make the following arguments to support their position:

(a) *A single fiber of asbestos can generate mesothelioma.* The exact mechanism by which asbestos causes cancer, including mesothelioma, is not known, but one theory is that the cancer is believed to be the result of inflammation or other factors that disrupt a cell's DNA and cause the cell to begin replicating out of control.⁵² The *any exposure* experts rely on this hypothesis to testify that exposure to a single fiber could, in theory, start the disease.⁵³ Once an individual has mesothelioma, these experts contend that we do not know and cannot determine which fiber (or more importantly, which *defendant's* fiber) caused the disease, and thus must assume that any and all exposures are the potential cause.⁵⁴ The experts exclude,

proximate cause of a subsequently diagnosed asbestos-related disease.”).

51. For instance, Dr. Arthur Frank, a proponent of the *any exposure* theory, has testified that a single brake job should be identified as a substantial factor in causing asbestos disease. See *Lulich v. Rapid Am. Corp.*, No. 2005 L004323 (Cir. Ct. Cook County, Ill.) (Deposition of Arthur Frank, Feb. 1, 2005, at 111 (“[S]omeone removing a set of brakes that contain brake dust where there is some percentage of untransformed chrysotile . . . I would say yes, it was a contributing factor to his mesothelioma.”)). The other examples are representative of allegations and expert testimony in numerous other cases. See, e.g., *Chavers v. General Motors Corp.*, 79 S.W.3d 361, 370 (Ark. 2002) (“The competent medical evidence presented in this case does not support the conclusion that a one-time exposure to asbestos-containing brakes was a substantial cause of Mr. Chaver’s mesothelioma.”); *Wilson v. A.P. Green Indus., Inc.*, 807 A.2d 922, 926 (Pa. Super. Ct. 2002) (affirming summary judgment for manufacturer where decedent was merely exposed to dust from defendant’s product “at one time or another.”).

52. See, e.g., *Hamilton v. Asbestos Corp.*, 998 P.2d 403, 407 (Cal. 2000) (describing mesothelioma disease process); Cheryl L. Fattman et al., *Experimental Models of Asbestos-Related Disease*, in *PATHOLOGY OF ASBESTOS-ASSOCIATED DISEASES* 256, 285 (Victor L. Roggli et al. eds., Springer Sci.+Bus. Media, Inc. 2d ed. 2004) (1992) (citing studies by Moalli).

53. See, e.g., *Bartel*, 316 F. Supp. 2d at 605 (Dr. Arthur Frank testified that a single fiber could cause disease); *Gregg*, 943 A.2d at 223 (stating plaintiffs’ expert Dr. Richard Lemen opined that there is no “safe” level of exposure to asbestos and that any level of exposure will place an individual at risk for developing asbestos-related conditions); *Bonnette v. Conoco, Inc.*, 837 So. 2d 1219, 1232 (La. 2003) (stating plaintiff’s expert Dr. Richard Lemen “testified that any level of exposure to asbestos will place an individual at risk for developing asbestos-related conditions.”); *Basile*, No. 11484 CD 2005, slip. op. at 9-12 (“The ‘single fiber’ theory [presented by plaintiff’s expert] holds that exposure to a single asbestos fiber can cause mesothelioma and other disease processes.”); *Georgia-Pac. Corp.*, 239 S.W.3d at 320 (“[T]he experts posited that all asbestos fibers cause mesothelioma because all asbestos fibers have the ability to cause cancer-inducing mutations in the cells and it is not possible to pinpoint which particular fibers actually caused the mutations.”); *In re Toxic Substance Cases*, 2006 WL 2404008 at *6 (stating plaintiff experts testified that a “single exposure” can cause disease).

54. See, e.g., *Georgia-Pac. Corp.*, 239 S.W.3d at 314-15, 320; *Gregg*, 943 A.2d at 223;

incongruously, background fibers as the potential initiating source, and they do not address or account for the body’s defensive mechanisms that actually protect against cancer caused by just one fiber or even many fibers entering the body.⁵⁵

(b) *Asbestos is a cumulative dose disease.* Asbestos disease is generally believed to result from the cumulative total dose of asbestos received over time rather than from an instantaneous exposure.⁵⁶ The *any exposure* proponents rely on the cumulative dose principle to conclude that every occupational exposure contributes to the disease, from the very smallest to the very highest, much like every drop of water contributes to filling a glass.⁵⁷ They do not factor in, however, the established differences in fiber potency,⁵⁸ any differences in duration of exposure across jobs or the

Bonnette, 837 So. 2d at 1232; *Basile*, No. 11484 CD 2005, slip. op. at 9-12; *In re Toxic Substance Cases*, 2006 WL 2404008 at *6.

55. See, e.g., *Flores*, 232 S.W.3d at 773 (stating expert acknowledged background fibers but did not suggest they were a cause of asbestosis); *Georgia-Pac. Corp.*, 239 S.W.3d at 315 (quoting Dr. Samuel Hammar’s testimony that the “level of exposure it takes to cause mesothelioma ‘could be any level above what is considered to be background’”); *In re Toxic Substance Cases*, 2006 WL 2404008 at *3 (“[B]ackground or ambient exposure is simply not sufficient to allow experts to causally attribute asbestos-related disease to it. Everyone, including the plaintiff’s experts, agrees that something greater is required.”). *Bartel*, 316 F. Supp. 2d at 607-08 (discussing background levels of asbestos).

56. National Cancer Institute, Fact Sheet, Asbestos Exposure: Questions and Answers 3 (Feb. 1, 2007), http://www.cancer.gov/images/Documents/5ac7d2fc-27df-4ecc-839f-dc5bc1909e01/fs3_21.pdf.

57. See, e.g., *Georgia-Pac. Corp.*, 239 S.W.3d at 320.

58. A great many studies and publications recognize that chrysotile is less potent in causing mesothelioma than the amphibole family of asbestos fibers, including amosite and crocidolite. See *Bartel*, 316 F. Supp. 2d at 606 (“[P]revailing scientific and medical view” supports lower chrysotile potency); *Becker v. Baron Bros., Coliseum Auto Parts, Inc.*, 649 A.2d 613, 620 (N.J. 1994) (holding that trial court erred in instructing jury that all asbestos-containing friction products without warnings are defective as a matter of law: “Our courts have acknowledged that asbestos-containing products are not uniformly dangerous and thus that courts should not treat them all alike.”); *Gideon v. Johns-Manville Sales Corp.*, 761 F.2d 1129, 1145 (5th Cir. 1985) (“[A]ll asbestos-containing products cannot be lumped together in determining their dangerousness.”); *Celotex Corp. v. Copeland*, 471 So. 2d 533, 538 (Fla. 1985) (“Asbestos products . . . have widely divergent toxicities, with some asbestos products presenting a much greater risk of harm than others.”); Charles M. Yarborough, *Chrysotile as a Cause of Mesothelioma: An Assessment Based on Epidemiology*, 36 CRITICAL REV. TOXICOLOGY 165, 165 (2006); U.S. ENVTL. PROT. AGENCY, REPORT ON THE PEER CONSULTATION WORKSHOP TO DISCUSS A PROPOSED PROTOCOL TO ASSESS ASBESTOS RELATED RISK viii (2003), http://www.epa.gov/oswer/riskassessment/asbestos/pdfs/asbestos_report.pdf; Andrew Churg, *Nonneoplastic Disease Caused by Asbestos*, in PATHOLOGY OF OCCUPATIONAL LUNG DISEASE 277, 314 (Andrew Churg & Francis H.Y. Green eds., 2d ed. 1998); B.T. Mossman et al., *Asbestos: Scientific Developments and Implications for Public Policy*, 247 SCIENCE 294, 296, 299 (1990), available at 1990 WLNR 2425147. The distinction is important for jobs such as automotive mechanics whose exposure is only to chrysotile fibers, because the difference in potency would indicate the need for a considerably higher dose to cause disease in that occupation.

dose of fiber received from any particular job, the removal of some fibers from the body,⁵⁹ or the frequency of exposure on any job. All asbestos types and all exposures are treated the same for purposes of their opinions.

(c) *The “no safe dose” or “no threshold” approach.* In keeping with the dose principle, virtually every toxin is believed to have a *threshold* level below which injury does not occur.⁶⁰ A dose of two aspirin, for instance, is below the threshold of injury for that drug.⁶¹ It is exceedingly difficult, however, to establish with certainty the level at which asbestos exposures do not cause mesothelioma.⁶² This is primarily because epidemiology studies—the “gold standard” for establishing causation—cannot easily identify differences in populations at low exposure levels approaching background. Because of the difficulty of proof that low exposures are safe, regulatory agencies such as OSHA have frequently stated that there is no *known* safe level of asbestos exposure and, therefore, set the regulatory limit at the lowest technologically feasible limit.⁶³

59. The body is capable of removing many inhaled fibers through defense mechanisms such as throat mucus, ciliary bodies, coughing and sneezing, the action of macrophage cells, and the lymph system. See generally Fattman, *supra* note 52, at 260-65. Chrysotile fibers, in particular, are removed fairly quickly, with a half life (the amount of time required to remove half the resident fibers from the body) of a few months for most fibers. The half life of amphibole fibers in contrast is measured in years or decades. See Churg, *supra* note 58, at 284-85; *Free*, No. 07-2-04091-9-SEA, slip op. at 2-3.

The notion that chrysotile fibers cause damage during their brief stay in the human body before their expulsion—known as the “hit and run” theory—is supported by plaintiff experts but rejected by many researchers. See, e.g., Richard A. Lemen, *Asbestos in Brakes: Exposure and Risk of Disease*, 45 AM. J. INDUS. MED. 229, 234 (2004) (stating plaintiff testifying expert Dr. Lemen argued that fast clearance of chrysotile does not eliminate possibility it caused disease before being eliminated); Kelly J. Butnor et al., *Exposure to Brake Dust and Malignant Mesothelioma: A Study of 10 Cases with Mineral Fiber Analyses*, 47 ANNALS OCCUPATIONAL HYGIENE 325, 239 (2000) (explaining why “hit and run” theory is “flimsy” and not plausible); Richard A. Lemen, *Reply to Victor L. Roggli and Arthur M. Langer*, 47 AM. J. INDUS. MED. 278, 278-79 (2005) (criticizing Roggli’s rejection of “hit and run” theory).

60. See Eaton, *supra* note 28, at 15.

61. Aspirin is a commonly-understood example. Others include alcohol, nitroglycerine, arsenic, and even water. See *In re Toxic Substance Cases*, 2006 WL 2404008 at *7.

62. *Id.* at *8-9; *Free*, No. 07-2-04091-9-SEA, slip op. at 4.

63. See, e.g., NIOSH-OSHA ASBESTOS WORK GROUP, WORKPLACE EXPOSURE TO ASBESTOS: REVIEW AND RECOMMENDATIONS DHHS (NIOSH) PUB. NO. 81-103 3 (1980), www.cdc.gov/niosh/topics/asbestos/pdfs/81103.pdf (“Evaluation of all available human data provides no evidence for a threshold or for a ‘safe’ level of asbestos exposure.”); 59 Fed. Reg. 40964-01, 40967 (Aug. 10, 1994) (stating OSHA believes that the regulatory limit of .1 fiber per cubic centimeter of air as an eight-hour time-weighted average is “the practical lower limit of feasibility for measuring asbestos levels reliably.”), available at 1994 WL 413576 (F.R.).

The basis for the 1975 proposal’s reduction in the permissible exposure limit to 0.5 f/cc was OSHA’s then-current policy for carcinogens that assumed that no safe threshold level was demonstrable and therefore that the Act required the Agency to set the PEL at a level as low as technologically and economically feasible.

51 Fed. Reg. 222612-01, 22614 (June 20, 1986), available at 1986 WL 103293 (F.R.).

The *any exposure* experts have converted this cautionary approach into an opinion that there *is* no safe dose of asbestos.⁶⁴ This conclusion, however, is clearly a non sequitur—the absence of conclusory proof as to *where* the threshold lies does not mean there is no threshold. These experts rely on, and often misstate, this concept to argue that since the safe level is unknown, then every exposure must be considered dangerous and contributory to disease.⁶⁵

(d) *The linear non-threshold theory and extrapolation down.* The *any exposure* theorists are often confronted with the lack of any epidemiology studies reasonably demonstrating that low levels of asbestos exposure produce any increased incidence of disease.⁶⁶ Because the plaintiffs’ experts have no such proof at the levels they claim are disease-inducing, they turn to an extrapolation methodology that relies on the assumption that high-dose studies can be used to estimate low-dose disease.⁶⁷ In the studies of high-incidence asbestos disease, typically in professions such as insulators, asbestos factory workers, miners, and textile workers, the disease follows a dose-response relationship that approaches, at least at the higher exposure levels experienced by those workers, a somewhat linear relationship between the lifetime fiber burden and the incidence of disease.⁶⁸

That data, however, *does not exist* at lower levels of exposure.⁶⁹ The two most likely explanations are: (1) the exposures do not cause disease at

64. See *In re Toxic Substance Cases*, 2006 WL 2404008 at *11.

While it may be a valid assertion that: if high dose asbestos exposure is bad for you, then low dose asbestos exposure may potentially be bad for you; it is not a valid assertion that because high dose exposure to asbestos is bad for you, then low dose exposure to asbestos is, in fact, bad for you, or that a specific plaintiff’s exposure at an unknown low dose exposure level, in fact, contributed to that plaintiff’s asbestos-related disease.

Id. (emphasis omitted).

65. See *In re Toxic Substance Cases*, 2006 WL 2404008 at *11 (“[Drs. John Maddox and David Laman] offer not a shred of independent corroboration of their opinion that each and every fiber causes or contributes to a Plaintiff’s disease process.”); *Brooks*, 934 So. 2d at 355 (stating plaintiffs’ expert Dr. Gaeton Lorino “was unable to cite a single study or publication to support his assertion” that mesothelioma is not a dose-related disease); *In re W.R. Grace*, 355 B.R. at 474-75 (discussing the fallacy of the “no safe dose” position).

66. See, e.g., B.T. Mossman et al., *supra* note 58, at 294 (“There are no available data showing health hazards due to low-level exposure . . .”).

67. The extrapolation-down approach of plaintiff experts was specifically addressed and rejected by the courts in *In re Toxic Substance Cases*, 2006 WL 2404008 at *7-8, and *Free*, No. 07-2-04091-9-SEA, slip op. at 3-4.

68. See John T. Hodgson & Andrew Darnton, *The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure*, 44 ANNALS OCCUPATIONAL HYGIENE 565, 578 fig. 6 (2000); *Free*, No. 07-2-04091-9-SEA, slip op. at 3 n.5 (discussing slope in Hodgson article).

69. See Hodgson & Darnton, *supra* note 68, at 578 fig. 6, 580 fig. 9 (identifying data points above 10 fibers/ml years).

lower levels and there is, quite plainly, nothing to find, or (2) the exposures may cause very low levels of disease, so low that their occurrence is not distinguishable from other causes of the disease. The *any exposure* experts, relying on a theoretical approach sometimes used by regulators, *assume* that the latter explanation is true.⁷⁰ They adopt a linear dose-response curve that extends in a straight line all the way to zero exposure.⁷¹ Most toxins do not follow such a line, but present a curvilinear relationship that drops to zero disease as the exposures approach the threshold (usually well above zero exposures).⁷² The assumed linear relationship at low levels produces a *theoretical* level of mesothelioma at extremely low levels of exposure, but these are theoretical and assumed cases only since no study has ever identified real disease at such low levels that is distinguishable from idiopathic or spontaneous mesothelioma.⁷³ The experts nevertheless testify, through this extrapolation down methodology, that disease must exist at low levels and that their calculated estimates prove that an individual plaintiff's low exposures contributed to his disease.⁷⁴

(e) *Reliance on case reports.* In some instances, lacking any supporting epidemiology, some *any exposure* experts resort to reliance on case reports of disease in persons exposed to low doses.⁷⁵ The most frequent application is in mechanic cases, where the epidemiology has consistently supported a *lack of disease* from chrysotile exposures, even among lifetime mechanics.⁷⁶ The experts reject the existing, contradictory epidemiology and rely on case reports instead.⁷⁷ “Case reports, by their

70. See *In re Toxic Substance Cases*, 2006 WL 2404008 at *6-7.

71. *Id.* at *7.

72. *Id.* (discussing threshold effect for common substances); Eaton, *supra* note 28, at 15-17.

73. See *In re Toxic Substance Cases*, 2006 WL 2404008 at *6; National Cancer Institute, *supra* note 56, at 3.

74. See *In re Toxic Substance Cases*, 2006 WL 2404008 at *7-8.

75. A case report is nothing more than an occurrence in which a person with a particular exposure also develops a particular disease. If epidemiology has established the link, a case report can potentially reflect a real causative source, for example, a heavy smoker who develops lung cancer. In most instances, however, case reports are at best suggestive of a possible link and frequently represent unrelated incidents. For example, case reports of coffee drinkers incurring pancreatic cancer a few years ago turned out to be false associations when epidemiology studies produced no evidence of a link. See American Cancer Society, *Pancreatic Cancer is Not Linked with Drinking Coffee or Alcohol* (July 17, 2001), http://www.cancer.org/docroot/NWS/content/NWS_1_1x_Pancreatic_Cancer_Is_Not_Linked_With_Drinking_Coffee_or_Alcohol.asp.

76. The studies are summarized and discussed in Francine Laden et al., *Lung Cancer and Mesothelioma among Male Automobile Mechanics: A Review*, 19 REV. ON ENVTL. HEALTH 39 (2004); Michael Goodman et al., *Mesothelioma and Lung Cancer among Motor Vehicle Mechanics: A Meta-analysis*, 48 ANNALS OCCUPATIONAL HYGIENE 309, 309 (2004); see also Yarbrough, *supra* note 58.

77. See, e.g., *In re Toxic Substance Cases*, 2006 WL 2404008 at *4-5.

very nature, can never prove causation.”⁷⁸ Consequently, some courts routinely reject case reports as proof of causation.⁷⁹ Nevertheless, some courts allow experts to rely on case reports as evidence in asbestos courtrooms where these experts are permitted to testify.

The proponents of the *any exposure* theory make little or no attempt to segregate real exposures from trivial or nonexistent exposures. The types of exposures sufficient to name a defendant can involve either a small number of exposure experiences, or a longer series of low dose exposures, such as mechanics doing brake jobs.⁸⁰ The lifetime dose from either type of exposure is minimal and far different from the world of known asbestos disease generated typically in dusty trades involving amphibole fibers.

Through this testimony, the *any exposure* experts are helping to extend the asbestos litigation to any entity that had any connection to asbestos.⁸¹

The “new” wave of asbestos cases, relying almost exclusively on the *any exposure* theory, typically involves a mesothelioma victim who, through attorney interviews, has identified any conceivable contact with asbestos in his or her lifetime.⁸² The contact can include household members who had direct contact and then allegedly brought fibers home, or “bystander” or “pass-by” exposures allegedly resulting from just being in the same building or vicinity as asbestos-related work.⁸³ In each case, the attorneys

78. Robert N. Jones, *Asbestos Medicine II*, SJ031 ALI-ABA 29, 29, 35 (Nov. 13-14, 2003).

79. See *Glastetter v. Novartis Pharms. Corp.*, 107 F. Supp. 2d 1015 (E.D. Mo. 2000), *aff’d*, 252 F.3d 986 (8th Cir. 2001); *Hollander v. Sandoz Pharms. Corp.*, 95 F. Supp. 2d 1230, 1235-38 (W.D. Okla. 2000), *aff’d in part*, 289 F.3d 1193 (10th Cir.), *cert. denied*, 537 U.S. 1088 (2002); *Brumbaugh v. Sandoz Pharm. Corp.*, 77 F. Supp. 2d 1153, 1157 (D. Mont. 1999); *Willert v. Ortho Pharm. Corp.*, 995 F. Supp. 979, 981 (D. Minn. 1998); *Boyles v. Am. Cyanamid Co.*, 796 F. Supp. 704, 708 (E.D.N.Y. 1992); *Casey v. Ohio Med. Prods.*, 877 F. Supp. 1380, 1385 (N.D. Cal. 1995); *In re Diet Drugs (Phentermine, Fenfluramine, Dexfenfluramine) Prods. Liab. Litig.*, MDL 1203, 2001 WL 454586 at *15 (E.D. Pa. Feb. 1, 2001) (memorandum and pretrial order No. 1685) (unreported).

80. See Dennis J. Paustenbach et al., *An Evaluation of the Historical Exposures of Mechanics to Asbestos in Brake Dust*, 18 APPLIED OCCUPATIONAL & ENVTL. HYGIENE 786, 786-804 (2003) (stating average lifetime mechanic exposures calculated at 0.04 f/cc or less, below OSHA standard of 0.1 f/cc); Brent L. Finley et al., *Cumulative Asbestos Exposure for US Automobile Mechanics Involved in Brake Repair (circa 1950s-2000)*, 17 J. EXPOSURE SCIENCE & ENVTL. EPIDEMIOLOGY (2007) 644, 644 (stating cumulative lifetime average exposures for automobile mechanics “are all substantially lower than the cumulative exposure of 4.5 f/cm³ year associated with occupational exposure to 0.1 f/cm³ of asbestos for 45 years that is currently permitted under the current occupational exposure limits in the US.”).

81. See Richard B. Schmitt, *Burning Issue: How Plaintiffs’ Lawyers Have Turned Asbestos into a Court Perennial*, WALL ST. J., Mar. 5, 2001, at A1, available at 2001 WLNR 2021814; Susan Warren, *Asbestos Quagmire: Plaintiffs Target Companies Whose Premises Contained Any Form of Deadly Material*, WALL ST. J., Jan. 27, 2003, at B1, available at 2003 WLNR 3099209.

82. See, e.g., *Chavers*, 79 S.W.3d at 370.

83. See, e.g., *Georgie-Pac. Corp.*, 239 S.W.3d at 315 (stating plaintiffs’ expert Dr. Samuel Hammer expressed opinion that “each and every exposure that an individual has in a bystander

then sue dozens of defendants associated with these contacts, many of whom have never made an asbestos product.⁸⁴ One-time contacts or events are treated equally as causes along with long-duration, high level exposures, such as Navy shipyard work.⁸⁵ Mesothelioma is particularly vulnerable, because it is readily associated with asbestos and, at least for amphiboles, requires a lower dose than other asbestos diseases.⁸⁶ Despite the wide agreement that a significant number (by some estimates, twenty to thirty percent) of mesotheliomas are not asbestos induced,⁸⁷ the *any exposure* theory is capable of converting every diagnosis of mesothelioma into an asbestos action. Countless individuals have had some contact with asbestos, either directly or through a family member, in their lifetime sufficient to satisfy the theory's minimal requirements. The *any exposure* cases are heavily weighted toward a handful of jurisdictions that continue to apply the "old" rules to all asbestos cases.⁸⁸

The massive expansion of the number of asbestos defendants brought about by this theory is highly problematic. When asbestos litigation focused on actual producers of asbestos and asbestos-containing products, defendants numbered in the hundreds (in 1982, about 300 such defendant

occupational setting causes their mesothelioma."); see also *Jackson v. Anchor Packing Co.*, 994 F.2d 1295 (8th Cir. 1993) (affirming dismissal of claimants who performed general "tireworker" duties and did not directly handle any of the defendant's asbestos products).

84. See, e.g., *Chavers v. Gatke Corp.*, 132 Cal. Rptr. 2d 198, 199 (Ct. App. 2003) (stating plaintiffs' complaint "joined as defendants scores of manufacturers, suppliers and distributors of friction brake products containing asbestos—59 named defendants and 800 'Doe' defendants . . ."); Susan Warren, *Asbestos Suits Target Makers of Wine, Cars, Soups, Soaps*, WALL ST. J., Apr. 12, 2000, at B1, available at 2000 WLNR 2042486.

85. See, e.g., *\$35.1M Awarded to Couple for Exposure to Asbestos in Navy*, MEALEY'S LITIG. REP.: ASBESTOS, vol. 22:19, Nov. 1, 2007, at 3 (stating \$35 million verdict for "exposure" to Leslie Control's "small pump and valve components" in the Navy, ignoring large-scale exposure to Navy insulation).

86. See 51 Fed. Reg. 22612-01, 22619 (June 20, 1986) (noting cases of mesothelioma, but not lung cancer, in low-exposed populations such as neighborhood and home exposures), available at 1986 WL 103293 (F.R.).

87. See Mayo Clinic Staff, *Mesothelioma* (Aug. 11, 2006), <http://www.mayoclinic.com/health/mesothelioma/DS00779/DSECTION=4> ("Asbestos exposure plays a role in 70 percent to 80 percent of mesothelioma cases, though the actual percentage could be higher."); Lawrence G. Cetrulo, *Asbestos Litigation & Tort Reform: Health Hazards/Diseases*, 3 TOXIC TORTS LITIG. GUIDE § 33:3 (updated Oct. 2007) ("Asbestos exposure is the dominant cause of mesothelioma, and accounts for 70 to 80 percent of all mesothelioma cases."); B.T. Mossman et al., *supra* note 58 ("[A]pproximately 20 to 30% of mesotheliomas occur in the general population in adults not exposed occupationally to asbestos."); Lester Brickman, *On the Theory Class's Theories of Asbestos Litigation: The Disconnect Between Scholarship and Reality*, 31 PEPP. L. REV. 33, 44 n.19 (2003) (stating that approximately twenty percent of malignant mesotheliomas have been attributed to causes other than exposure to asbestos).

88. See Wasserman et al., *supra* note 46, at 905-08 (discussing policy reasons why courts should reject *de minimis* and *any exposure* causation theories in asbestos litigation).

companies).⁸⁹ Now, over 8,500 defendants have been named,⁹⁰ as “the net has spread from the asbestos makers to companies far removed from the scene of any putative wrongdoing.”⁹¹ One well-known plaintiffs’ attorney has described the litigation as an “endless search for a solvent bystander.”⁹² Once a company is caught in this net, unless courts are willing to reject *any exposure* testimony prior to trial, it is nearly impossible for a defendant to escape without serious financial consequences.

III. COURT RULINGS REJECTING OR CRITICIZING THE *ANY EXPOSURE* APPROACH

In the last three years, the *any exposure* approach has been criticized or found inadmissible under both the *Frye* and *Daubert* tests, and under general requirements of reliability and foundation, before over a dozen courts in multiple jurisdictions.⁹³ These courts have recognized the extreme position the plaintiffs’ experts are taking, the lack of scientific proof supporting their theory, and the lack of logical or scientific support for their conclusions.⁹⁴

The *any exposure* theory was first criticized in a 2005 Ohio federal court case, *Bartel v. John Crane, Inc.*⁹⁵ In *Bartel*, plaintiff’s experts attempted to attribute plaintiff’s mesothelioma to exposure from handling the defendant’s gaskets and packing while in the Navy.⁹⁶ The plaintiff, like many Naval workers, had substantial exposure to large amounts of amphibole asbestos in ship insulation, but plaintiff either did not sue or had settled with the entities responsible for those extreme and clearly dangerous exposures.⁹⁷ Exposures from gaskets and packing, in contrast, are quite

89. See JAMES S. KAKALIK ET AL., VARIATION IN ASBESTOS LITIGATION COMPENSATION AND EXPENSES 5 (1984).

90. See Deborah R. Hensler, *California Asbestos Litigation—The Big Picture*, HARRISMARTIN COLUMNS: ASBESTOS, Aug. 2004, at 5.

91. Editorial, *Lawyers Torch the Economy*, WALL ST. J., Apr. 6, 2001, at A14, available at 2001 WLNR 1993314.

92. ‘*Medical Monitoring and Asbestos Litigation*’—A Discussion with Richard Scruggs and Victor Schwartz, MEALEY’S LITIG. REP.: ASBESTOS, vol. 17:3, Mar. 1, 2002, at 5 (quoting Mr. Scruggs); see also Steven B. Hantler et al., *Is the “Crisis” in the Civil Justice System Real or Imagined?*, 38 LOY. L.A. L. REV. 1121, 1151-52 (2005) (discussing spread of asbestos litigation to “peripheral defendants”). “Nontraditional [d]efendants [n]ow [a]ccount for [m]ore [t]han [h]alf of [a]sbestos [e]xpenditures.” CARROLL ET AL., *supra* note 2, at 94.

93. See *supra* notes 11-19.

94. See, e.g., *Flores*, 232 S.W.3d at 774.

95. 316 F. Supp. 2d at 611.

96. *Id.* at 604-05.

97. *Id.* at 604-06.

low—measured at 0.0062 fibers/cc in trial evidence.⁹⁸ Some of plaintiff's experts agreed that exposures approaching or below background, such as those from gasket work, would be insufficient for causation.⁹⁹ As to the remaining *any exposure* experts, who relied on this theory to point the finger at the minimal chrysotile exposures rather than plaintiff's insulation exposures, the court found their testimony unpersuasive:

The two experts who disagreed, Dr. Frank and Dr. Suzuki, testified that every exposure to asbestos Lindstrom had during his working career, no matter how small, was a substantial factor in causing his peritoneal mesothelioma If an opinion such as [this] . . . would be sufficient for plaintiff to meet his burden, the Sixth Circuit's "substantial factor" test would be meaningless

In addition, the opinion of Dr. Frank, that every breath Lindstrom took which contained asbestos could have been a substantial factor in causing his disease, is not supported by the medical literature.¹⁰⁰

This decision was upheld on appeal by the United States Court of Appeals for the Sixth Circuit: "[Plaintiff's expert argument] appears to be that a showing of any level of asbestos exposure attributable to John Crane's products was sufficient for the court to have entered a judgment in their favor. We reject plaintiffs-appellants' argument on this point."¹⁰¹

We believe the *Bartel* opinions were the first time that the *any exposure* theory was held insufficient to support causation. *Bartel* itself appears to have received little attention and did not quickly replicate itself in other courts. In the last two years, however, and largely independent of *Bartel*, the flawed *any exposure* approach has produced a raft of decisions that reject the theory as unscientific and/or exclude the expert testimony under *Daubert* or *Frye*.¹⁰²

The first and most influential of these subsequent decisions was that of Judge Colville in a Pennsylvania case, *Betz v. Pneumo-Abex*.¹⁰³ *Betz* initially involved a group of automotive mechanic cases in which plaintiffs'

98. *Id.* at 608. For comparison purposes, this figure is more than ten times lower than the current standard (0.1 fibers per cubic centimeter of air as an eight-hour time-weighted average) at which OSHA permits workers to be exposed every day for forty years without requiring any protection. See 29 C.F.R. § 1910.1001(c)(1) (2006).

99. *Bartel*, 316 F. Supp. 2d at 604-06.

100. *Id.* at 611.

101. *Lindstrom*, 424 F.3d at 498.

102. See *supra* notes 11-19.

103. The trial court opinion was entitled *In re Toxic Substances Cases*, 2006 WL 2404008 (Pa. Ct. Com. Pl. Aug. 17, 2006). The case has also been referred to as *Simikian* (another plaintiff), but on appeal the title changed to *Betz v. Pneumo Abex LLC*, No. 1058 WDA 2006 (Pa. Super. Ct. Jan. 16, 2007) (Betz is the administrator of the deceased plaintiff Vogelsberger's estate).

experts Drs. John Maddox and David Laman declared that the specific exposure facts of each mechanic were essentially irrelevant because any exposure was sufficient to support causation.¹⁰⁴ The experts thus rebuffed the need for any sort of dose assessment and opined that any level of mechanic work, regardless of duration, was sufficient to cause disease.¹⁰⁵ Judge Colville precluded this testimony, and in the process, addressed the key underpinnings of the theory and found each one illogical and unsupported.¹⁰⁶ We will cover Judge Colville’s reasoning in some detail, because it remains today the seminal and best evisceration of the grounds asserted by *any exposure* approach experts. Decisions that followed largely repeated and elaborated on Judge Colville’s arguments.

First, Judge Colville addressed the serious discrepancy between the claim that any exposure to an occupational fiber causes disease, and the experts’ candid, albeit incongruent, admission that a lifetime of background exposures to asbestos fibers does not cause disease.¹⁰⁷ In modern industrial society, urban and sometimes rural air has historically contained asbestos at low levels (some of this from natural asbestos outcrops), and thus most individuals over fifty will have millions of “background” fibers in their lungs even without any known occupational or other direct exposure to asbestos.¹⁰⁸ These levels have never been known to cause disease, primarily because the human body is capable of ejecting, absorbing, or otherwise dealing with these low exposures.¹⁰⁹ Plaintiff experts almost without exception readily admit this and exclude background exposures from their cumulative dose opinions.¹¹⁰ (This “admission” has the benefit for plaintiffs of preventing defendants from pointing to background exposures as contributory.) The fibers involved in these two types of exposures, however, are no different—only the dose distinguishes background from occupational exposures, and even then a low occupational exposure (such as the Crane gasket exposure of 0.0062 f/cc in the *Bartel* case above) can easily overlap or not exceed a higher background exposure.¹¹¹ Thus, there is no logic that permits these experts to categorically exclude background exposure, yet, at the same time,

104. *In re Toxic Substance Cases*, 2006 WL 2404008 at *1.

105. *Id.*

106. *Id.* at *11-12.

107. *Id.*

108. *Id.* at *3.

109. *Id.*

110. *Id.*

111. *Id.*

categorically include all occupational exposures as causative.¹¹²

Given the admission by plaintiff's experts that background exposures were not high enough to cause disease, Judge Colville recognized that it was incumbent on the experts to identify exactly *what dose would be sufficient* to cause disease:

For instance, experts suggest that the average ambient exposure in Pittsburgh is approximately .0001 fibers per milliliter of air No one, including the plaintiff's experts, proffers an opinion that this level of exposure creates an increased risk of the development of any asbestos-related disease The argument in this Frye challenge, in part, revolves around the question of how much greater quantity of exposure is necessary to permit the causal attribution of an asbestos-related disease to a particular asbestos-related exposure.¹¹³

Plaintiff's experts made no attempt to measure or quantify the mechanics' occupational doses or show how they were sufficient to cause disease when background exposures clearly are not.¹¹⁴ The court rejected the experts' complete failure to quantify or assess the mechanic's dose in any way because the lack of any measurement made it impossible for them to accurately distinguish low level occupational exposures from background exposures.¹¹⁵

Second, Judge Colville rejected the experts' attempt to "extrapolate down" from high-dose asbestos studies to prove that occupational exposures at low doses, above background or not, also must cause disease.¹¹⁶ The amphibole form of asbestos is widely recognized to cause disease at significant doses (e.g., in the shipyard, insulator, and asbestos factory professions), but there are no low-dose response curves for asbestos exposure and no studies demonstrating an increase in actual disease at very low doses, particularly for chrysotile.¹¹⁷ Drs. Maddox and Laman used the

112. *Id.* at *12.

113. *Id.* at *3 (emphasis added).

114. *Id.* at *6-7.

115. *Id.* at *6, 9, 11-13.

116. *Id.* at *6-7.

117. *Id.* at *6, 8. Whether chrysotile exposures cause mesothelioma at all is the subject of considerable debate currently in the scientific community. See *supra* notes 58-59; J. C. McDonald & A. D. McDonald, *Chrysotile, Tremolite and Carcinogenicity*, 41 ANNALS OCCUPATIONAL HYGIENE 699, 703 (1977); Thomas A. Sporn & Victor L. Roggli, *Mesothelioma*, in PATHOLOGY OF ASBESTOS-ASSOCIATED DISEASES 104, 108 (Victor L. Roggli et al. eds., Springer Sci.+Bus. Media, Inc. 2d ed. 2004) (1992) (stating the capacity of chrysotile to cause mesothelioma is "much debated"). Epidemiology studies have not demonstrated excess mesothelioma among populations exposed only to low levels of chrysotile. See, e.g., *supra* note 58 (vehicle mechanic studies show no increased mesothelioma); Jennifer Pierce et al., *An Evaluation of Reported No-Effect Chrysotile Asbestos Exposures for Lung Cancer and Mesothelioma*, 38 CRITICAL REV. IN

“extrapolate down” methodology to assume, based on high-dose studies, that low-dose studies would also cause disease in a linear fashion.¹¹⁸ Judge Colville rejected this approach:

The fallacy of the “extrapolation down” argument is plainly illustrated by common sense and common experience. Large amounts of alcohol can intoxicate, larger amounts can kill; a very small amount, however, can do neither. Large amounts of nitroglycerine or arsenic can injure, larger amounts can kill; small amounts, however, are medicinal. Great volumes of water may be harmful, . . . moderate amounts of water, however, are healthful. In short, the poison is in the dose.¹¹⁹

Judge Colville recognized that when experts attempt this kind of extrapolation downward, they are engaged in both a logical falsehood and scientific error:

[P]laintiffs have not proffered any generally accepted methodology to support the contention that a single exposure or an otherwise vanishingly small exposure has, in fact, in any case, ever caused or contributed to any specific individual’s disease, or even less so, that in this case such a small exposure did, in fact, contribute to this specific plaintiff’s disease.¹²⁰

Finally, Judge Colville rejected the experts’ reliance on the “no safe threshold” position.¹²¹ The court noted the very large difference between stating that the threshold is not known and claiming that *there is no threshold at all*.¹²² The court believed that such testimony improperly shifted the burden of proof to defendants when it is plaintiff’s burden to establish the known toxic level of a substance and that plaintiff experienced a dose consistent with that level.¹²³

Following Judge Colville’s ruling, a second Pennsylvania trial judge rejected the *any exposure* testimony of Dr. Maddox on similar reasoning in a case involving heavy equipment mechanic exposures.¹²⁴ Judge Colville’s decision is currently on appeal before Pennsylvania’s intermediate appellate court. The Pennsylvania Supreme Court, however, recently issued a decision in *Gregg v. V.J. Auto Parts, Inc.*¹²⁵ that clearly rejects the *any exposure* theory and may well offer a glimpse into how the court would ultimately deal with *Betz*. *Gregg* involved allegations that personal car

TOXICOLOGY 191 (2008) (identifying likely no-effect level in chrysotile studies).

118. *In re Toxic Substance Cases*, 2006 WL 2404008 at *7.

119. *Id.*

120. *Id.* at *8.

121. *Id.*

122. *Id.* at *8-9.

123. *Id.* at *8.

124. *See Basile*, No. 11484 CD 2005, slip op. at 5.

125. 943 A.2d at 226.

repair work on brakes and gaskets caused plaintiff's mesothelioma, resulting in a lawsuit against the auto parts store that sold Mr. Gregg the parts he used.¹²⁶ The primary holding in the case dealt with the application of the "frequency, proximity, and regularity" causation test, but in the course of the discussion the Court majority expressed a clear rejection of the *any exposure* approach:

We recognize that it is common for plaintiffs to submit expert affidavits attesting that any exposure to asbestos, no matter how minimal, is a substantial contributing factor in asbestos disease. However, we share Judge Klein's perspective, as expressed in the *Summers* [*v. Certaineed Corp.*, 886 A.2d 240 (Pa. Super. 2005), *appeal granted*, 897 A.2d 460 (Pa. 2006)] decision, that such generalized opinions do not suffice to create a jury question in a case where exposure to the defendant's product is *de minimis*, particularly in the absence of evidence excluding other possible sources of exposure (or in the face of evidence of substantial exposure from other sources). As Judge Klein explained, one of the difficulties courts face in the mass tort cases arises on account of a willingness on the part of some experts to offer opinions that are not fairly grounded in a reasonable belief concerning the underlying facts and/or opinions that are not couched within accepted scientific methodology.¹²⁷

While recognizing the occasional difficulty of proving which of plaintiff's exposures contributed to the disease, Pennsylvania's highest court nevertheless rejected the easy way out of simply stating that all exposures are responsible:

[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. The result, in our view, is to subject defendants to full joint-and-several liability for injuries and fatalities in the absence of any reasonably developed scientific reasoning that would support the conclusion that the product sold by the defendant was a substantial factor in causing the harm.¹²⁸

Thus, it now appears to be the law in Pennsylvania, as expressed by that state's highest court, that asbestos cases will have to follow the same dose and toxicity rules and proof as any other toxic tort case. A blanket assertion that each and every occupational exposure contributes to disease will no longer support an asbestos case in that state.

Pennsylvania's Supreme Court is not the only state supreme court to

126. *Id.* at 217-18.

127. *Id.* at 226 (citation omitted).

128. *Id.* at 226-27.

address this issue. Six months earlier, the Texas Supreme Court in *Borg-Warner Corp. v. Flores* became the first state court of last appeal to reject the *any exposure* theory.¹²⁹ The case involved a mechanic who worked most of his life doing brake and clutch jobs and claimed he developed asbestosis as a result of repeated, low-level exposures over a lifetime.¹³⁰ Following a line of Texas asbestos cases highly favorable to plaintiffs, the intermediate Corpus Christi appellate court had held that under Texas law it was sufficient for plaintiffs to show mere exposure to take a defendant to trial:

In 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

. . . .

[T]he plaintiffs offered evidence that the defendant’s products emitted dust containing respirable asbestos fibers, which one of the plaintiffs had inhaled. On appeal, this Court held that the evidence was sufficient to prove the defendant’s products injured both plaintiffs.

. . . .

“[W]ork[ing] in the presence of the asbestos-containing product” was “direct evidence” of causation and sufficient to uphold the jury’s finding [of liability].¹³¹

These statements reflect the older, shortcut approach to causation in asbestos cases designed to expedite cases to trial and alleviate plaintiffs of the burden of proving which exposures actually contributed to their disease.¹³² The Texas Supreme Court, however, rejected this approach as inconsistent with Texas tort and causation law:

While science has confirmed the threat posed by asbestos, we have not had the occasion to decide whether a person’s exposure to “some” respirable fibers is sufficient to show that a product containing asbestos was a substantial factor in causing asbestosis [W]e conclude that it is not¹³³

The court’s reasoning followed that of Judge Colville in recognizing the importance of dose, the need for a dose quantification, and the necessity of equating the plaintiff’s dose to those in the epidemiological literature

129. 232 S.W.3d at 774.

130. *Id.* at 766.

131. *Flores*, 153 S.W.3d at 213-14 (citations omitted).

132. See *supra* notes 1-3 and accompanying text.

133. *Flores*, 232 S.W.3d at 765-66.

documenting disease.¹³⁴ Since *Flores*, other Texas courts have rejected the *any exposure* approach, including in mesothelioma cases.¹³⁵

The federal bankruptcy court in Delaware (Judge Judith K. Fitzgerald) also rejected the *any exposure* theory in *In re W.R. Grace & Co.*¹³⁶ Plaintiff's experts contended that asbestos contamination in vermiculate attic insulation posed an unreasonable risk of harm to the homeowners because "any exposure to asbestos fibers is an unreasonable risk."¹³⁷ Their testimony was excluded under *Daubert*, however, because the experts failed to establish what level of exposure would actually cause disease and could not present any epidemiology studies demonstrating asbestos disease from exposure to vermiculite.¹³⁸ The court held, "[t]he use of the no safe level or linear 'no threshold' model for showing unreasonable risk 'flies in the face of the toxicological law of dose-response, that is, that 'the dose makes the poison'"¹³⁹

Other courts in Mississippi and Washington State have similarly rejected *any exposure* testimony.¹⁴⁰ The Mississippi decision came in the context of an allegation that exposure to asbestos in a school justified a medical monitoring award, but the court of appeals rejected that approach

134. *Id.* at 770-74.

135. See *Georgia-Pac. Corp.*, 239 S.W.3d at 321; Letter Ruling, *In re Asbestos*, No. 2004-3, 964 (Tex. Dist. Ct., July 18, 2007). The Texas MDL judge actually presaged the *Flores* ruling in rejecting similar *any exposure* testimony in 2004. See Letter Ruling, *In re Asbestos*, No. 2004-03964 (excluding plaintiff expert Eugene Mark on "any exposure" theory).

136. 355 B.R. at 474-78.

137. *Id.* at 474.

138. *Id.* at 468.

139. *Id.* at 476. A Delaware state judge in charge of asbestos litigation, Judge Joseph R. Sights III, has rejected a broad motion by automotive defendants to dismiss all mechanic cases, filed largely on the ground that the epidemiology did not support such cases. Even in rejecting this argument, however, Judge Sights expressed considerable skepticism that a no threshold, *any fiber* theory would be viable:

If, in a given case, a plaintiff must rely upon a no threshold theory to establish causation, the [c]ourt can determine the reliability of that testimony on a separate *in limine* motion. Suffice it to say, the testimony will be scrutinized carefully. See *Bartel v. John Crane, Inc.*, 316 F.[3] Supp.[2] 603, 611 (N.D. Ohio 2004) (finding Dr. Frank's single fiber theory to be inconsistent with prevailing scientific evidence, including the testimony of Drs. Lemen and Hammar).

In re Asbestos Litig., 911 A.2d 1176, 1209 n.202 (Del. Super. Ct.), cert. denied, 2006 WL 1579782 (Del. Super. Ct. June 7, 2006), appeal refused, 906 A.2d 806 (Del. 2006). The Michigan intermediate appellate court likewise failed to exclude the testimony of plaintiffs' expert Richard Lemen, but the decision turned on the vehicle mechanic epidemiology and not on a purported *any exposure* causation opinion. See *Chapin v. A & L Parts*, 732 N.W.2d 578, 587 (Mich. Ct. App.), appeal denied, 733 N.W.2d 23 (Mich. 2007), 733 N.W.2d 29 (Mich. 2007), and 733 N.W.2d 35 (Mich. 2007).

140. See *Brooks*, 934 So. 2d at 355-56; *Anderson*, No. 05-2-04551-5SEA, slip op. at 144-45 ("[T]his is not a theory which is generally accepted in the scientific community."); *Free*, No. 07-2-04091-9-SEA.

without some assessment that the dose was high enough to produce disease.¹⁴¹ In one the Washington decisions, a state trial judge held that Dr. Samuel Hammar’s testimony that any occupational exposure was sufficient to cause disease was “not a theory which is generally accepted in the scientific community” and thus prevented him from so testifying.¹⁴² This case illustrates the extremes of the theory, as the case went to trial against Caterpillar, not in regard to plaintiff’s extensive Navy exposures, but on the ground that plaintiff walked by Caterpillar engines while gaskets were being removed and thus must have breathed some asbestos fibers.¹⁴³ This ruling is believed to be the first substantive limitation on the testimony of Dr. Hammar, one of the most prominent of plaintiffs’ testifying experts. Since then, Dr. Hammar’s low dose testimony has been excluded by another Washington State trial judge (along with the testimony of another plaintiffs’ expert, Dr. Carl Brodtkin),¹⁴⁴ and in Texas in the *Georgia-Pacific Corp. v. Stephens*¹⁴⁵ mesothelioma case where Dr. Hammar testified that any exposure above 0.1 fibers/cc years would contribute to cause disease.¹⁴⁶

One of the opinions criticizing the *any exposure* approach, *Summers v. Certaineed Corp.*,¹⁴⁷ directly addresses the illogic of the cumulative dose approach many of these expert use to include every exposure in causation:

Dr. [Jonathan] Gelfand used the phrase, “Each and every exposure to asbestos has been a substantial contributing factor to the abnormalities noted.” However, suppose an expert said that if one took a bucket of water and dumped it in the ocean, that was a “substantial contributing factor” to the size of the ocean. Dr. Gelfand’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 thirty years at an asbestos factory making lagging, it can hardly be said that the one whiff of the asbestos from the brakes is a “substantial” factor in causing disease.¹⁴⁸

The *Summers* statement proved influential in convincing the Pennsylvania Supreme Court to reject the *any exposure* approach in the recent *Gregg* ruling.¹⁴⁹

141. See *Brooks*, 934 So. 2d at 355-56.

142. See *Anderson*, No. 05-2-04551-5SEA, slip op. at 145.

143. *Id.* at 95.

144. *Free*, No. 07-2-04091-9-SEA, slip. op. at 4-5 (ruling on motion *in limine*).

145. 239 S.W.3d at 304.

146. *Id.* at 316.

147. 886 A.2d 240 (Pa. Super. Ct. 2005), *appeal granted*, 897 A.2d 460 (Pa. 2006).

148. *Id.* at 244 (emphasis omitted).

149. See *Gregg*, 943 A.2d at 226.

Experts who continue to assert the *any exposure* basis for medical causation in asbestos cases are carrying a torch that is being extinguished repeatedly in asbestos cases around the country. As courts have held, “each and every exposure” testimony is, at best, an unproven hypothesis that ignores scientific principles and should not suffice for causation in an asbestos case.¹⁵⁰

IV. HOW THE *ANY EXPOSURE* THEORY FITS INTO THE SCIENTIFIC AND TORT LITIGATION WORLD

Plaintiffs’ experts who support the *any exposure* approach to asbestos litigation can speak at great length and cite to many materials to justify their approach. The discussion is a siren song of epidemiology, animal studies, the history of asbestos, fear of cancer, case reports of persons with mesothelioma and a certain exposure, and mathematical predictions of thousands of mesothelioma cases at even the lowest of doses.¹⁵¹ They can cite to a number of review articles and other published literature that support at least a portion of their approach, much of it written by other plaintiff testifying experts.¹⁵² Government publications also offer tacit support, since regulators take highly conservative approaches and rarely, if ever, declare any form of asbestos exposure to be “safe,” even when the literature supports an identifiable no-effect level.¹⁵³

150. See, e.g., *In re Toxic Substance Cases*, 2006 WL 2404008 at *6; *Bartel*, 316 F. Supp. 2d at 611; *Georgia-Pac. Corp.*, 239 S.W.3d at 320-21.

151. See, e.g., Lemen, *supra* note 59; David Egilman et al., *Exposing the “Myth” of ABC, “Anything But Chrysotile”: A Critique of the Canadian Asbestos Mining Industry and McGill University Chrysotile Studies*, 44 AM. J. INDUS. MED. 540 (2003); Laura S. Welch, *Asbestos Exposure Causes Mesothelioma, But Not This Exposure: An Amicus Brief to the Michigan Supreme Court*, 13 INTL. J. OCCUPATIONAL & ENVTL. HEALTH 318 (2007).

152. See, e.g., Lemen, *supra* note 59; see also Egilman et al., *supra* note 151; see also Welch, *supra* note 151; David S. Egilman, *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability*, 11 INT. J. OCCUPATIONAL & ENVTL. HEALTH 360 (2005); BARRY I. CASTLEMAN, *ASBESTOS: MEDICAL AND LEGAL ASPECTS* 539-80 (Aspen Publishers, Inc. 5th ed. 2005). All of these authors testify on behalf of plaintiffs in asbestos litigation.

153. See, e.g., Pierce et al., *supra* note 117, at 205 (calculating no-effect level for chrysotile exposures). For example, despite the extensive growing evidence that chrysotile is less potent than amphiboles and that short fibers do not cause disease, neither OSHA nor EPA has ever made any distinctions between the different exposures in their regulatory requirements. There are practical reasons for this (the difficulty of separating exposures in a workplace), but the justification is usually, “since we don’t know for sure we’ll just be cautious and regulate everything the same way.” While this approach may have some justification in the regulatory world, it should never serve as a basis for finding legal causation as to any exposure regardless of disease-inducing potency. EPA has announced the creation of a scientific advisory panel to assist

Nevertheless, despite these attempts to support it, the *any exposure* theory does not have any credible foundation in the scientific literature.¹⁵⁴ In fact, the *any exposure* theory is almost entirely a litigation construct and is not widely published or accepted in the peer-reviewed literature. Virtually nothing in the literature expressly states what these experts routinely say in court—that each and every occupational exposure, no matter how small, is a cause of disease.¹⁵⁵ There is great debate over related subjects such as whether chrysotile should be considered a cause of mesothelioma, whether short fibers contribute to disease, or whether occupations like vehicle mechanics are even subject to any asbestos disease at all, despite long-term, low-level exposures.¹⁵⁶ Even the most plaintiff-oriented of these articles, however, do not take the extreme position that there is no minimum. The litigation proponents of the theory themselves rarely, if ever, present the notion that *every occupational exposure is causative* to the general scientific community through publications or

the agency in deciding whether its asbestos risk assessment process should be modified to reflect the growing literature and findings.

154. See cases cited *supra* note 65.

155. Perhaps the closest enunciation is that of the “Helsinki Criteria,” a document generated by nineteen scientists in 1997 to develop their version of criteria for attributing lung cancer and mesothelioma to asbestos exposure. See *Asbestos, Asbestosis, and Cancer: The Helsinki Criteria for Diagnosis and Attribution*, 23 SCANDINAVIAN J. WORK ENV'T & HEALTH 311, 312-14 (1997). As to mesothelioma, the Helsinki Criteria states that a “significant occupational exposure” is adequate for attribution, but then later restates (confusingly) that “brief or low level exposures” are also sufficient. *Id.* at 313. There is no definition or even discussion of what would constitute a significant, brief, or low level exposure, and no justification provided for attributing disease to such exposures, particularly for chrysotile. Nor does the Helsinki Criteria document purport to provide any basis for determining which occupational exposures should be considered causative—it merely provides criteria for attributing a disease to occupational exposure generally. Even the Helsinki Criteria’s extreme approach to mesothelioma attribution does not state that every occupational exposure, or every exposure above background, should be considered disease-inducing. Instead, it clearly implies a universe of occupational exposures that are too low to be included and some judgment about dose and duration must be exercised before attributing a disease to an occupational exposure. See *id.*

156. Cf., e.g., Ronald F. Dodson et al., *Asbestos Fiber Length as Related to Potential Pathogenicity: A Critical Review*, 44 AM. J. INDUS. MED. 291 (2003); Phillip J. Landrigan et al., *The Hazards of Chrysotile Asbestos: A Critical Review*, 37 INDUS. HEALTH 271 (1999); William J. Nicholson, *The Carcinogenicity of Chrysotile Asbestos—A Review*, 39 INDUS. HEALTH 57 (2001); Gunnar Hillerdal, *Mesothelioma: Cases Associated with Non-Occupational and Low Dose Exposures*, 56 OCCUPATIONAL & ENVTL. MED. 505 (1999); with Hodgson & Darnton, *supra* note 68 (stating chrysotile far less potent than amphiboles); EASTERN RESEARCH GROUP, INC., REPORT ON THE EXPERT PANEL ON HEALTH EFFECTS OF ASBESTOS AND SYNTHETIC VITREOUS FIBERS: THE INFLUENCE OF FIBER LENGTH vi (2003), <http://www.atsdr.cdc.gov/HAC/asbestospanel/finalpart1.pdf>; EASTERN RESEARCH GROUP, INC., REPORT ON THE PEER CONSULTATION WORKSHOP TO DISCUSS A PROPOSED PROTOCOL TO ASSESS ASBESTOS RELATED RISK viii (2003), http://www.epa.gov/oswer/riskassessment/asbestos/pdfs/asbestos_report.pdf; Churg, *supra* note 58, at 314.

scientific conferences where it could be scrutinized and likely debunked. Thus, the theory completely fails the *Daubert* “peer review and publication” test and the “general acceptance” test under either *Daubert* or *Frye*.¹⁵⁷

The *any exposure* theory also does not fit well in the litigation world. It is largely an asbestos issue because in most toxic tort litigation the notion that any exposure to a toxin satisfies the substantial factor or but-for tests of causation would be considered so extreme that most experts would not have the temerity to present it. In the *Parker* case discussed previously, the plaintiff’s experts testified that plaintiff’s AML was caused by many years of gas station work exposures, not *any exposure*.¹⁵⁸ Imagine if they had been dealing with a long-time benzene factory worker—the exact population demonstrated by epidemiology to be at risk—but tried to blame the plaintiff’s disease on the few times he put gas in his own car and thus breathed miniscule amounts of benzene in gasoline. This is the equivalent of the *any exposure* theory carried over to non-asbestos litigation.

At best, the *any exposure* approach is only a theory, an unproven hypothetical concept. At worst and carried to extremes, it is almost certainly erroneous and misleading, and would never be permitted as a basis for testimony in most toxic tort causation contexts. Only in the world of special asbestos rules can such a theory gain a foothold.

V. ASBESTOS JURISPRUDENCE IN LIGHT OF THE DECISIONS REJECTING THE *ANY EXPOSURE* THEORY

The above decisions rejecting the *any exposure* theory have the potential to affect a sea of change in asbestos litigation causation testimony. The issues they raise will likely come to be litigated at some point in virtually every asbestos jurisdiction.

Many of the courts that will have to address *any exposure* motions have managed asbestos cases for years and have seen some of the plaintiffs’ experts testify perhaps hundreds of times without anyone challenging the scientific basis for their testimony. Some of these judges will be quite puzzled to understand why defendants are suddenly raising expert and

157. The *Daubert* test requires that the scientific methodology be reliable, one aspect of which is that the theory/methodology have been subjected to peer review and general acceptance in the scientific community. See *Daubert*, 509 U.S. at 593-94. The *Frye* test is more narrow, requiring simply that the theory be accepted in the scientific community before admission as evidence. See *id.* at 584-85. Both tests are designed to prevent theories from being presented as courtroom evidence before they have run the gauntlet of review and testing in the scientific community. See *id.* at 589-90.

158. See *supra* notes 33-41 and accompanying text.

causation issues not part of the previous asbestos landscape. For several reasons, courts should take a closer look at this causation testimony, even if they have not done so in the past.

A. Courts Should Begin to Apply Standard Tort Principles and Causation Rules to Asbestos Cases

For three decades, the special asbestos rules affecting causation created a unique opportunity for plaintiffs to prove their cases merely by identifying a defendant’s product and asserting, through plaintiff’s or co-workers’ testimony, that some fibers from that product were in the plaintiff’s breathing zone.¹⁵⁹ In the context of the “old” asbestos litigation, where, for example, an insulator might work with multiple insulation products but could not necessarily prove how much with each one, the rule served to overcome proof obstacles in occupations and industries clearly demonstrated through epidemiology to cause mesothelioma and other asbestos disease.¹⁶⁰

Whether the relaxation of causation rules was justified in this circumstance or not, the justification no longer exists where most of the defendants are not asbestos companies or insulation suppliers, but are companies that only sold or used products with limited asbestos in them. Often the asbestos in the products used or sold by these defendants was sealed in resins or binders and thus would not ordinarily produce much, if any, exposure. Mechanic exposures, for example, have historically been in the range of half the current OSHA standard of acceptable exposures in the workplace.¹⁶¹ Gaskets likewise produce very little exposure, as noted in the *Bartel* case.¹⁶² Nevertheless, in the face of allegations that the plaintiff worker or co-worker witnessed visible dust while working with asbestos parts, or disrupted these products through grinding, sanding, cutting, or drilling, it is nearly impossible for such a defendant to claim *zero* fiber exposure—the only defense that could avoid a trial under an *any exposure* attack.

These cases fall squarely into the world of the New York *Parker* case, where the causation theory is highly speculative and theoretical, and is not supported by epidemiology showing disease from these exposures or

159. See *supra* notes 1-3, 46-47 and accompanying text.

160. See *id.*

161. See Paustenbach et al., *supra* note 80.

162. See *Bartel*, 316 F. Supp. 2d at 608.

occupations.¹⁶³ If plaintiff law firms wish to develop a new wave of asbestos litigation against *de minimis* exposure defendants, based on a theory that is not generally accepted in the scientific community, they should not be permitted to rely on the old rules to do so. Instead, the principles of *Parker* and other traditional toxic tort cases should apply, and the testimony must be tested under *Daubert*, *Frye*, or other state evidentiary and expert requirements before trial. **Plaintiffs' experts should be required to assess the dose from an individual defendant's product or workplace and demonstrate that it is the kind of dose shown in established epidemiology studies to be capable of causing disease.**

B. The Expansion of Asbestos Litigation to a Wide Array of Minimal Exposure Defendants is Unjustified

The effect of the *any exposure* theory can be exceptionally unfair when applied to defendants connected with extremely small exposures, especially when plaintiff experts ignore far more significant exposures that almost certainly caused the disease. The unfairness is only multiplied in jurisdictions that will not permit the defendants remaining at trial to point to the plaintiff's real asbestos exposures from other sources.¹⁶⁴ The courts discussed above have sometimes reacted to the perverse effect of the *any exposure* theory as applied to asbestos cases. The Pennsylvania *Summers* court, for example, noted the incongruity of blaming a single brake job for plaintiff's mesothelioma when he was a lifelong insulator.¹⁶⁵ The *Gregg* court questioned why plaintiffs were trying to take a brake parts supplier to trial when the complaint alleged a forty-year history of occupational exposure.¹⁶⁶ The *Bartel/Lindstrom* cases noted that the claim against the provider of gaskets and packings seemed trivial in comparison to the worker's extensive insulation exposure.¹⁶⁷

These courts are recognizing that asbestos causation can get out of

163. See *Parker*, 857 N.E.2d at 1121-22.

164. In Illinois, for instance, the bizarre *Lipke v. Celotex Corp.*, 505 N.E.2d 1213 (Ill. App. Ct. 1987), rule, the only one of its kind in the country, prohibits a trial defendant from informing the jury about *any* of plaintiff's other asbestos exposures, even when those exposures were severe (e.g., in the Navy) and far more than sufficient to cause the disease. This rule, and others like it that prohibit references to bankrupt asbestos companies, leave the jury with few or no sources of asbestos to consider as the cause of the plaintiff's disease except for the minimal exposures of the remaining, low dose trial defendants. See generally Victor E. Schwartz et al., *Asbestos Litigation in Madison County, Illinois: The Challenge Ahead*, 16 WASH. U. J.L. & POL'Y 235 (2004).

165. See *Summers*, 886 A.2d at 244.

166. See *Gregg*, 943 A.2d at 226.

167. See *Bartel*, 316 F. Supp. 2d at 610-11.

control under the *any exposure* approach. Yet plaintiffs’ experts refuse to acknowledge that their theory attaches causation to trivial exposures, nor do they even attempt to separate work experiences that truly cause asbestos disease from those that contribute nothing but isolated or inconsequential exposures. Put bluntly, there is likely to be a credibility issue for courts that allow experts to testify that a single breath of exposure justifies taking that defendant to trial.¹⁶⁸

C. *The Science Requires More*

As documented in Judge Colville’s analysis¹⁶⁹ and others above, the *any exposure* experts do not have epidemiology or other scientific proof that these low exposures cause anything. They are testifying to a theory, a hypothesis, an estimate of disease derived from assumptions that may or may not be true. Courts cannot be in the business of allowing cases to go forward with only theory and speculation to prove causation. Under either *Frye* or *Daubert*, and for that matter under basic evidentiary and reliability principles, there must be more.¹⁷⁰ Traditional industrial hygiene and toxicology principles and methodologies exist under which non-litigation professionals can assess whether past exposures were sufficient to cause disease.¹⁷¹ The *Parker* opinion noted as much and found no merit to the contention that low dose cases cannot be proven unless plaintiffs are given the advantage of avoiding any dose assessment.¹⁷² The reality is that such cases can and should only be proven if the dose can be reasonably and scientifically assessed and there is credible science supporting causation at such doses. Otherwise, the experts should be excluded and the cases should not go to trial.

168. Virtually everyone who lives in an industrial society has been exposed to asbestos fibers and could be a potential plaintiff under the *any exposure* theory, even though there is no evidence low-level exposures consistent with background exposures actually cause disease.

[Because asbestos] is one of the most ubiquitous of the Earth’s minerals and in addition, millions of cars still spew thousands of asbestos fibers into the air each time a driver applies the brakes, many if not most adults in the general population have significant numbers of asbestos fibers in their lungs; however, despite breathing in millions of asbestos fibers annually, virtually none of the population thus exposed to ambient concentrations of asbestos fibers thereby suffer adverse effects on their health.

Brickman, *supra* note 87, at 49.

169. See *supra* notes 103-23 and accompanying text.

170. See *Frye*, 293 F. at 1013 (explaining admission of expert testimony depends on general acceptance within the scientific community); see also *Daubert*, 509 U.S. at 579 (explaining admission of expert testimony depends on reliability).

171. See, e.g., *Parker*, 857 N.E.2d at 1114.

172. See *supra* notes 33-41 and accompanying text.

D. It Is Not Sufficient to Let a Low-dose Case Simply Go to the Jury

Some courts are proponents of letting expert disputes, such as the level of exposure required, go to the jury in every instance. Similarly, some of the *any exposure* experts, when confronted with their failure to separate significant from insignificant exposures, respond that they are unwilling to deal with differences among defendants and simply expect the jury to figure it all out. This is not an acceptable approach, scientifically or legally. Without any expert testimony separating the exposures likely to have caused the disease from those unlikely to have done so, the jury has no basis to make the decision. The attribution of disease among different fiber types and exposure experiences is not within the province of a lay person but must be supported by expert testimony. Experts who abdicate that exercise should not be permitted to testify that all exposures “contribute” and then hand it over to the jury.

Likewise, the subject of “how much is enough” is without question one that is subject to a *Daubert/Frye* review of the experts’ methodology and testimony. Judges cannot abdicate their gatekeeping function on this critical expert issue,¹⁷³ but must determine whether the expert testimony and causation evidence pass scientific and legal muster. This is particularly true in complex science cases, in which juries of lay people are singularly ill-equipped to sort through the complex studies and scientific issues and instead often render decisions based on favorable reactions to witnesses, impressive testimony by the experts, and sympathy for a plaintiff who is likely to die soon (or already has) from a disease known to be caused by asbestos.

VI. CONCLUSION

Asbestos has for years held sway as perhaps the most feared of industrial exposures. At the same time, asbestos litigation has also earned a reputation as the most out-of-control of all tort litigation. The history of asbestos is indeed a terrible one, with great loss of life from exposures that predate the institution of OSHA workplace and other regulations. That history is not a basis for blaming every fiber and every breath for asbestos disease in today’s litigation environment. Courts must exercise control over the current state of litigation and the assertion of the *any exposure* theory. In light of the array of recent decisions rejecting that theory, courts that do so are clearly in good company and have substantial support from their colleagues in other jurisdictions.

173. See, e.g., *Daubert*, 509 U.S. at 597.