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

Mark A. Behrens*

William L. Anderson**†

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

* Mark A. Behrens is a partner in the Public Policy Group of Shook, Hardy & Bacon L.L.P. in Washington, D.C. He received his B.A. from the University of Wisconsin-Madison in 1987 and his J.D. from Vanderbilt University Law School in 1990, where he was a member of the *Vanderbilt Law Review*. Mark is a member of The American Law Institute and was named one of Washington, D.C.'s "top lawyers" by *Washingtonian* magazine in December 2007. Mr. Behrens may be contacted at mbehrens@shb.com.

** William Anderson is a partner with Crowell & Moring L.L.P. in Washington, D.C. He represents defendants in toxic tort and product liability litigation, including asbestos cases, and writes regarding the intersection of science and law. He received his B.A. from Hendrix College in 1975 and his J.D. from the University of Minnesota Law School in 1988, where he served as Editor-in-Chief of the *Minnesota Law Review*. Mr. Anderson may be contacted at wanderson@crowell.com.

† Research support for this Article was provided by funding from the Coalition for Litigation Justice, Inc. The views discussed herein are solely those of the authors.

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 testimony[22]—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) (Erlick, 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 § 431cmt. 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).