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Getting to Causation in Toxic Tort Cases

David E. Bernstein[†]

INTRODUCTION

Since the issue first arose in earnest in the 1970s, courts have struggled to create rules for causation in toxic tort cases¹ that are both consistent with longstanding tort principles and fair to all parties. Faced with conflicting and often novel expert testimony, scientific uncertainty, the gap between legal and scientific culture, and unprecedented claims for massive damages, common-law courts needed time to adjust and accommodate themselves to the brave new world of toxic tort litigation. Eventually, however, courts around the country reached a broad consensus on what is required for a toxic tort plaintiff to meet his or her burden of proof.

While there is a voluminous scholarly literature on various aspects of toxic tort litigation, this Article's unique contribution is to articulate the new consensus on causation standards, document and criticize the various ways plaintiffs attempt to evade these standards, and defend the courts' adherence to traditional notions of causation against their critics.

Part I of this Article explains that to prove causation in a toxic tort case, a plaintiff must show that the substance in question is capable, in general, of causing the injury alleged, and also that exposure to the substance more likely than not caused *his* injury. When a plaintiff was exposed to a single toxin from multiple sources, to prove causation by a

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¹ While "there is no single universally accepted definition of what constitutes a toxic tort," Kyle D. Logue, *Reparations as Redistribution*, 84 B.U. L. REV. 1319, 1334 (2004), for purposes of this Article toxic tort cases involve plaintiffs who have been exposed to allegedly toxic substances, such as chemicals, asbestos fibers, or a pharmaceutical product, and allege that this exposure has caused their cancer, birth defect, or other injury.

specific defendant the plaintiff must show that the actions of that defendant were a “substantial factor” in causing the alleged harm.²

Part II discusses plaintiffs’ attempts to evade these standards by hiring experts to present various types of unreliable causation evidence. Examples of such evidence include testimony based on high-dose animal studies, anecdotal case reports, analogizing from the known effects of “similar” chemicals, preliminary epidemiological studies that have not been peer-reviewed, and differential etiologies used to “rule in” an otherwise unknown causal relationship. Additionally, when multiple defendants have contributed to the plaintiffs’ exposure to a potentially toxic substance, plaintiffs often present experts who claim, with no reliable scientific grounding, that the level of exposure (“dose”) is irrelevant to causation.

Part III of this Article argues that courts should be steadfast in requiring toxic tort plaintiffs to meet their burden of proof. Traditional tort principles require that plaintiffs bear the burden of proving actual causation by a preponderance of the evidence, not merely that they were exposed to a risk. To hold otherwise and essentially shift the burden to defendants to disprove causation would open the floodgates to all manner of speculative claims, with potentially devastating consequences for Americans’ well-being. Similarly, with regard to cases in which a plaintiff alleges injury after exposure to a toxin from multiple sources, a given defendant may only be held liable if the plaintiff proves by a preponderance of the evidence that exposure to *that* defendant’s products was a “substantial factor” in causing that injury. To hold otherwise would amount to an implicit adoption of a system of broad, collective liability that courts have rejected when the issue has been raised explicitly. This section concludes by discussing the negative consequences that arise from speculative toxic tort litigation unsupported by reliable scientific evidence.

I. CAUSATION IN TOXIC TORT LITIGATION

As discussed in detail below, American courts have reached a broad consensus on what a plaintiff must show to prove causation in a toxic tort case. First, a plaintiff must show that the substance in question is capable of causing the injury in question. This is known as “general causation.”³ Second, a plaintiff must show that this substance caused *his* injury.⁴ This is known as “specific causation.”⁵ Because proof of general

² The same test may apply when a plaintiff was exposed to multiple toxins by several different defendants, but here the law is murky.

³ See, e.g., *Jaros v. E.I. Dupont (In re Hanford Nuclear Reservation Litig.)*, 292 F.3d 1124, 1133 (9th Cir. 2002) (General or generic causation involves “whether the substance at issue had the capacity to cause the harm alleged . . .”).

⁴ See *Kelley v. Am. Heyer-Schulte Corp.*, 957 F. Supp. 873, 882 (W.D. Tex. 1997) (excluding specific causation testimony “[i]n the absence of any evidence regarding general

causation cannot satisfy a plaintiff's burden without proof of specific causation, and proof of specific causation implicitly requires proof of general causation, the focus of inquiry in toxic tort cases typically is on the existence of specific causation.

Proof of specific causation has two elements. The plaintiff must initially show that the *level of the toxin* he was exposed to *can* cause the illness he contracted.⁶ Here, epidemiology (the study of the cause and distribution of illness in human populations) becomes vitally important, as many courts have emphasized.⁷ A noted legal scholar explains that “[t]here plainly is a hierarchy to these different indirect forms of toxic effect evidence. Epidemiology is at the top, and structural similarity, in vitro testing, and case reports are at the bottom.”⁸

causation”); *Hall v. Baxter Healthcare Corp.*, 947 F. Supp. 1387, 1413 (D. Or. 1996) (explaining that specific causation proof “assumes that general causation has been proven”) (emphasis omitted).

⁵ See, e.g., *In re Bextra & Celebrex Mktg. Sales Practices & Prod. Liab. Litig.*, 524 F. Supp. 2d 1166, 1172 (N.D. Cal. 2007) (“Specific causation refers to whether a particular individual suffers from a particular ailment as a result of exposure to a substance.”).

⁶ 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 plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs’ burden”) (citing *Allen v. Pa. Eng’g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996)); *Mitchell v. Gencorp, Inc.*, 165 F.3d 778, 781 (10th Cir. 1999) (“It is well established that a plaintiff in a toxic tort case must prove that he or she was exposed to and injured by a harmful substance manufactured by the defendant. . . . In order to carry this burden, a plaintiff must demonstrate ‘the levels of exposure that are hazardous to human beings generally as well as the plaintiff’s actual level of exposure to the defendant’s toxic substance before he or she may recover.’” (quoting *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1106 (8th Cir. 1996))); *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1106 (8th Cir. 1996) (“[A] plaintiff in a toxic tort case must prove 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.”); *Allen v. Pa. Eng’g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996) (“[S]cientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiff’s burden in a toxic tort case.”); *Parker v. Mobil Oil Corp.*, 857 N.E.2d 1114, 1120-21 (N.Y. 2006) (“It is well-established that an opinion on causation should set forth a plaintiff’s exposure to a toxin, that the toxin is capable of causing the particular illness (general causation) and that plaintiff was exposed to sufficient levels of the toxin to cause the illness (specific causation).”).

⁷ See *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 882 (10th Cir. 2005) (“We agree with the district court that epidemiology is the best evidence of general causation in a toxic tort case.”); *Allison v. McGhan Med. Corp.*, 184 F.3d 1300, 1316 (11th Cir. 1999) (“[I]n the face of controlled, population-based epidemiological studies which find otherwise, these case studies [of alleged breast implant injury] pale in comparison.”); *Allen*, 102 F.3d at 199 (“[T]he most useful and conclusive type of evidence in a case such as this [ethylene oxide toxic tort claim] is epidemiological studies.”); *Chambers v. Exxon Corp.*, 81 F. Supp. 2d 661, 664 (M.D. La. 2000) (“Without a controlled [epidemiological] study, there is no way to determine if CML is more common in people who are exposed to benzene than those who are not. . . . [I]n a case such as this [benzene toxic tort claim], the most conclusive type of evidence of causation is epidemiological evidence.”), *aff’d*, 247 F.3d 240 (5th Cir. 2001)); *In re Breast Implant Litig.*, 11 F. Supp. 2d 1217, 1224 (D. Colo. 1998) (“The most important evidence relied upon by scientists to determine whether an agent (such as breast implants) cause [sic] disease is controlled epidemiologic studies.”); *Hall*, 947 F. Supp. at 1403 (“[T]he existence or nonexistence of relevant epidemiology can be a significant factor in proving general causation in toxic tort cases.”); *Conde v. Velsicol Chem. Corp.*, 804 F. Supp. 972, 1025-26 (S.D. Ohio 1992) (“Epidemiologic studies are the primary generally accepted methodology for demonstrating a causal relation between a chemical compound and a set of symptoms or a disease.”).

⁸ Michael D. Green, *Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of Agent Orange and Bendectin Litigation*, 86 Nw. U. L. REV. 643, 658 (1992).

For an epidemiological study to be used to properly prove specific causation, moreover, the plaintiff must show that the exposure at issue did not simply slightly raise the hypothetical risk of injury, but in fact more than *doubled the risk* of the harm.⁹ Courts, borrowing scientific terminology, often refer to the doubling of the risk as a “relative risk” of greater than two.¹⁰ In legal terms, this equates to ensuring that a preponderance of the evidence shows that the relevant exposure was the cause of his injury.

For example, consider the following hypothetical: If a study found that 20 out of 1000 women exposed to a toxin were diagnosed with breast cancer and 5 out of 1000 unexposed women (the “control” group) with otherwise matching characteristics were diagnosed with breast cancer, the relative risk of exposure to the toxin is 4.0, or four times as great as the risk of breast cancer without exposure. This is so because the proportion of women in the exposed group with breast cancer is 0.02 (20/1000) and the proportion of women in the control group with breast cancer is 0.005 (5/1000), and 0.02 divided by 0.005 is 4.0. In that hypothetical, assuming the reliability and validity of the underlying study, the crude mathematics of the relative risk suggest that exposure to the toxin caused the breast cancer of three of every four women who were both exposed to the toxin and were diagnosed with breast cancer. A “three in four chance,” in turn, satisfies the preponderance of the evidence standard.

Of course, epidemiology is not an exact science, and epidemiologists often prefer to present data as a range of possibilities, rather than as a single, potentially misleading figure.¹¹ But while mathematical precision is not required, a plaintiff has the burden to present evidence, epidemiological or otherwise, “from which a

⁹ See *Merrell Dow Pharm., Inc. v. Havner*, 953 S.W.2d 706, 716 (Tex. 1997) (stating that many courts have “found that the requirement of a more than 50% probability means that epidemiological evidence must show that the risk of an injury or condition in the exposed population was more than double the risk in the unexposed or control population,” and agreeing with those courts).

¹⁰ See, e.g., *Cagle v. The Cooper Companies (In re Silicone Gel Breast Implants Prod. Liab. Litig.)*, 318 F. Supp. 2d 879, 892 (C.D. Cal. 2004) (“The relative risk is obtained by dividing the proportion of individuals in the exposed group who contract the disease by the proportion of individuals who contract the disease in the non-exposed group.”); see also *Daubert v. Merrell Dow Pharm., Inc.*, 43 F.3d 1311, 1321 (9th Cir. 1995) (for epidemiological testimony to be admissible to prove specific causation, there must have been a relative risk for the plaintiff of greater than two); *In re Bextra*, 524 F. Supp. 2d at 1172 (epidemiological studies “can also be probative of specific causation, but only if the relative risk is greater than 2.0, that is, the product more than doubles the risk of getting the disease”).

¹¹ Moreover, individual studies generally cannot be deemed reliable in the absence of independent, confirmatory data, epidemiological or otherwise. See generally Gary Taubes, *Epidemiology Faces its Limits*, 269 SCIENCE 164 (1995) (noting that epidemiology is subject to systematic errors, biases, and confounders). Note that the lack of available evidence on either side does not permit the plaintiff to satisfy his burden of proof by presenting speculation and conjecture that is not scientifically valid. See, e.g., *In re Bextra*, 524 F. Supp. 2d at 1181 (“Plaintiffs cite no case, however, that suggests that they can satisfy their burden of proof based on a lack of evidence . . .”).

reasonable person could conclude that a defendant's emission has *probably* caused [the plaintiff] the kind of harm of which he or she complains."¹²

Beyond general and specific causation, an additional causation issue arises when multiple defendants are responsible for exposing the plaintiff to a harmful substance. The most common example is a plaintiff who contracts an asbestos-related disease, such as lung cancer or asbestosis, and was exposed to asbestos from multiple sources. Assuming the plaintiff is able to show that his disease was more probably than not caused by asbestos exposure, he still has to prove that a particular defendant's asbestos-containing product was a "proximate cause" of that injury to recover damages from that defendant.

Courts, building on the *Restatement (Second) of Torts*, have concluded that plaintiffs must provide sufficient evidence for a jury to conclude that exposure to the defendant's asbestos or asbestos-containing product was a "substantial factor" in promoting the disease.¹³ As the comments to the *Restatement (Second)* note, if other actors' conduct is the predominant factor in bringing the harm at issue, then a defendant's action is not a "substantial factor" in causing the harm, and thus it is not the legal cause of the harm.¹⁴

Asbestos plaintiffs have faced the problem that in most cases they were exposed to asbestos many years earlier and are unable to prove with any precision how much exposure they received from any particular defendant's products.¹⁵ Given that this could prove an insuperable barrier to many deserving plaintiffs, courts have overwhelmingly held that proximate cause in the asbestos context should be considered in light of the "frequency, regularity, proximity test" pioneered by the Fourth Circuit Court of Appeals in *Lohrmann v. Pittsburgh Corning Corp.*¹⁶

¹² *Parker v. Mobil Oil Corp.*, 857 N.E.2d 1114, 1121 (N.Y. 2006) (quoting *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1107 (8th Cir. 1996)) (emphasis added); *see also Daubert*, 43 F.3d at 1321 n.16 (Because individual plaintiffs may have a different background risk than the average subject of an epidemiological study, it is possible that "[a] statistical study showing a relative risk of less than two could be combined with other evidence to show it is more likely than not that the accused cause is responsible for a particular plaintiff's injury."). An expert, however, cannot simply assert without evidence that a plaintiff's circumstances differ in a relevant way from the circumstances of the subjects of a scientific study. *See Parker*, 857 N.E.2d at 1121-22 ("Dr. Goldstein's general, subjective and 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." (quoting Dr. Goldstein)).

¹³ This test is based on the *Restatement (Second) of Torts*. RESTATEMENT (SECOND) OF TORTS §§ 431, 433 (1965).

¹⁴ *Id.* § 433 cmt. d.

¹⁵ *See Thacker v. UNR Indus.*, 603 N.E.2d 449, 456 (Ill. 1992) ("[A] plaintiff injured by asbestos fibers often does not know exactly when or where he was injured and therefore is unable to describe the details of how such injury occurred.").

¹⁶ 782 F.2d 1156, 1162-63 (4th Cir. 1986); *see also Slaughter v. S. Talc Co.*, 949 F.2d 167, 171 (5th Cir. 1991) ("The most frequently used test for causation in asbestos cases is the 'frequency-regularity-proximity test' announced in *Lohrmann*."). For examples of courts that have

This test attempts to reduce the evidentiary burden on plaintiffs while still absolving defendants who were not responsible for plaintiffs' injuries.¹⁷

The *Lohrmann* court held that when a plaintiff alleges multiple sources of exposure to asbestos, the plaintiff must present evidence: (1) of exposure to a "specific product" attributable to the defendant, (2) "on a regular basis over some extended period of time," (3) "in proximity to where the plaintiff actually worked," [or where he otherwise claims to have been exposed to asbestos], (4) such that it is probable that the exposure to the defendant's product caused plaintiff's injuries.¹⁸ The court found it insufficient to raise an inference of causation that the plaintiff claimed exposure to a specific asbestos-containing product ten to fifteen times for a duration of one to eight hours over a thirty-nine year period.¹⁹

Some courts, while purporting to apply the *Lohrmann* test, ignored both that case's specific holding and the teachings of the *Restatement (Second)* regarding substantial factor causation. These courts effectively held that any exposure to asbestos is sufficient to make such exposure a "substantial factor" in disease causation, even when the

adopted this test, see, e.g., *Chism v. W.R. Grace & Co.*, 158 F.3d 988, 992 (8th Cir. 1998) (applying Missouri law); *Jones v. Owens-Corning Fiberglas Corp.*, 69 F.3d 712, 716 (4th Cir. 1995) (applying North Carolina law); *Jackson v. Anchor Packing Co.*, 994 F.2d 1295, 1303 (8th Cir. 1993) (applying Arkansas law); *Dillon v. Fibreboard Corp.*, 919 F.2d 1488, 1491 (10th Cir. 1990) (applying Oklahoma law); *Robertson v. Allied Signal, Inc.*, 914 F.2d 360, 380 (3d Cir. 1990) (applying Pennsylvania law); *Lyons v. Garlock, Inc.*, 12 F. Supp. 2d 1226, 1229 (D. Kan. 1998) (applying Kansas law); *Kraus v. Celotex Corp.*, 925 F. Supp. 646, 652 (E.D. Mo. 1996) (applying Missouri law); *Chavers v. General Motors Corp.*, 79 S.W.3d 361, 369 (Ark. 2002); *Thacker v. UNR Indus., Inc.*, 603 N.E.2d 449, 457 (Ill. 1992); *Spaur v. Owens-Corning Fiberglas Corp.*, 510 N.W.2d 854, 859 (Iowa 1994); *Eagle-Picher Indus., Inc. v. Balbos*, 604 A.2d 445, 460 (Md. 1992); *Monsanto Co. v. Hall*, 912 So. 2d 134, 137 (Miss. 2005); *Gorman-Rupp Co. v. Hall*, 908 So. 2d 749, 757 (Miss. 2005); *James v. Bessemer Processing Co., Inc.*, 714 A.2d 898, 911 (N.J. 1998); *Sholtis v. American Cyanamid Co.*, 568 A.2d 1196, 1207 (N.J. Super. Ct. App. Div. 1989); *Gregg v. V-J Auto Parts, Co.*, 943 A.2d 216, 227 (Pa. 2007); *Eckenrod v. GAF Corp.*, 544 A.2d 50, 53 (Pa. Super. Ct. 1988); *Henderson v. Allied Signal, Inc.*, 644 S.E.2d 724, 727 (S.C. 2007); *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 770 (Tex. 2007); *Vaughn v. Ford Motor Co.*, 91 S.W.3d 387, 393 (Tex. App. 2002) (applying Illinois law). *But see* *Ingram v. ACandS, Inc.*, 977 F.2d 1332, 1343-44 (9th Cir. 1992) (applying Oregon law); *Blackston v. Shook & Fletcher Insulation Co.*, 764 F.2d 1480, 1486 (11th Cir. 1985) (applying Georgia law); *Bailey v. N. Am. Refractories Co.*, 95 S.W.3d 868, 872 (Ky. Ct. App. 2001); *Purcell v. Asbestos Corp., Ltd.*, 959 P.2d 89, 94 (Or. App. 1998), *modified on reconsideration*, 963 P.2d 729 (Or. App. 1998) (applying Oregon law). The *Lohrmann* causation standard has been adopted by statute in a number of states. See FLA. STAT. ANN. § 774.203(30)-204 (2008) (applying to certain claims); GA. CODE ANN. § 51-14-3(23) (2008); OHIO REV. CODE ANN. § 2307.96(B) (2008).

¹⁷ For a discussion, see *Tragarz v. Keene Corp.*, 980 F.2d 411, 419-21 (7th Cir. 1992); see also Victor E. Schwartz et al., *A Letter to the Nation's Trial Judges: Serious Asbestos Cases—How to Protect Cancer Claimants and Wisely Manage Assets*, 30 AM. J. TRIAL ADVOC. 295, 316 (2006) ("The frequency, regularity, and proximity test offers a rational method for eliminating inconsequential exposure cases consisting of one-time or infrequent exposures.") (emphasis omitted).

¹⁸ *Lohrmann*, 782 F.2d at 1162-63.

¹⁹ *Id.* at 1163.

plaintiff was exposed to much more asbestos from other sources.²⁰ Plaintiffs in turn bolstered their cases by hiring experts to present highly dubious testimony, not supported by reliable scientific studies,²¹ that exposure to even one fiber of asbestos can cause disease.²² The result was a litigation explosion,²³ in which hundreds of thousands of claims were filed by claimants with little or no physical impairment,²⁴ and thousands of defendants were named.²⁵ The claims, concentrated in jurisdictions

²⁰ See, e.g., *Thacker*, 603 N.E.2d at 457 (The substantial factor test was satisfied even though the court acknowledged that “significantly less” than 3% of plaintiff’s total workplace exposure to asbestos came from defendant’s product.); *Spain v. Owens Corning Fiberglass Corp.*, 710 N.E.2d 528, 535 (Ill. App. Ct. 1999) (referring to Illinois’s test as a “*de minimis*” test and noting that Illinois courts ban the admission of evidence that the plaintiff was exposed to asbestos from sources other than the defendant); *Hoerner v. ANCO Insulations, Inc.*, 812 So. 2d 45, 56 (La. Ct. App. 2002) (holding that a defendant’s asbestos could satisfy the substantial factor test even if it was not a proximate cause of the plaintiff’s injury).

²¹ See *Bartel v. John Crane, Inc.*, 316 F. Supp. 2d 603, 611 (N.D. Ohio 2004), *aff’d sub nom.*, *Lindstrom v. A-C Prod. Liab. Trust*, 424 F.3d 488 (6th Cir. 2005) (rejecting the one-fiber theory as not scientifically valid). In *In re Toxic Substances Cases*, the court stated:

Generally accepted scientific methodology may well establish that certain “high dose” asbestos exposure causes, or contributes to, a specific hypothetical plaintiff’s disease, but the plaintiffs 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.

In re Toxic Substance Cases, No. A.D. 03-319, 2006 WL 2404008, at *8 (Pa. Ct. Com. Pl. 2006).

²² See *Gregg v. V-J Auto Parts, Co.*, 943 A.2d 216, 226-27 (Pa. 2007) (“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.”). For a general discussion of the misuse of expert testimony in asbestos litigation, see David E. Bernstein, *Keeping Junk Science Out of the Asbestos Litigation*, 31 PEPP. L. REV. 11 (2003) [hereinafter Bernstein, *Junk Science*].

²³ During the 1990s, the number of asbestos cases pending nationwide doubled from 100,000 to more than 200,000. See *The Fairness in Asbestos Compensation Act of 1999: Hearing on H.R. 1283, Before the H. Comm. on the Judiciary*, 106th Cong. 62 (1999) (statement of Christopher Edley, Jr., Professor, Harvard Law School). By 2002, approximately 730,000 claims had been filed, more than 100,000 additional claims filed in 2003 alone—“the most in a single year.” STEPHEN J. CARROLL ET AL., RAND INST. FOR CIVIL JUSTICE, ASBESTOS LITIGATION xxiv (2005) [hereinafter RAND REP.]; Editorial, *The Asbestos Blob, Cont.*, WALL ST. J., Apr. 6, 2004, at A16. In August 2005, the Congressional Budget Office estimated that approximately 322,000 asbestos-related claims were pending in state and federal courts. See AMERICAN ACADEMY OF ACTUARIES’ MASS TORTS SUBCOMMITTEE, OVERVIEW OF ASBESTOS CLAIMS AND TRENDS 5 (2007), available at http://www.actuary.org/pdf/casualty/asbestos_aug07.pdf [hereinafter Am. Acad. Actuaries].

²⁴ James A. Henderson, Jr. & Aaron D. Twerski, *Asbestos Litigation Gone Mad: Exposure-Based Recovery for Increased Risk, Mental Distress, and Medical Monitoring*, 53 S.C. L. REV. 815, 823 (2002) (“By all accounts, the overwhelming majority of claims filed in recent years have been on behalf of plaintiffs who . . . are completely asymptomatic.”).

²⁵ See JAMES S. KAKALIK ET AL., RAND INST. FOR CIVIL JUSTICE, COSTS OF ASBESTOS LITIGATION 12 (1983). At least one company in nearly every U.S. industry is involved in the litigation. See Am. Acad. Actuaries, *supra* note 23, at 5. One plaintiffs’ attorney has described the litigation as an “endless search for a solvent bystander.” Commentary, *‘Medical Monitoring and Asbestos Litigation’—A Discussion with Richard Scruggs and Victor Schwartz*, MEALEY’S LITIG. REP.: ASBESTOS, Mar. 1, 2002, at 33, 37 (quoting Richard Scruggs).

that had the most lenient causation and jurisdictional requirements,²⁶ sought to hold marginal asbestos defendants fully responsible for hundreds of billions of dollars of damages.²⁷ By 1997, the Supreme Court observed that the United States was in the throes of an “asbestos-litigation crisis.”²⁸

In a series of recent decisions, however, courts relying on *Lohrmann* have applied the substantial factor test far more literally and sensibly. The Pennsylvania Supreme Court recently endorsed a lower court opinion that rejected testimony by a “hired expert” that “[e]ach and every exposure to asbestos has been a *substantial* contributing factor to the abnormalities noted.”²⁹ The lower court noted that such a proposition was as nonsensical as saying “that if one took a bucket of water and dumped it into the ocean, that was a substantial contributing factor to the size of the ocean.”³⁰ Several other courts have recently rejected the “any exposure” approach to asbestos causation.³¹

²⁶ See James L. Stengel, *The Asbestos End-Game*, 62 N.Y.U. ANN. SURV. AM. L. 223, 236 (2006) (noting the dramatic increase in asbestos claims because of “the erosion or elimination of standards of recovery, particularly causation and product identification”); see generally Michelle J. White, *Asbestos Litigation: Procedural Innovations and Forum Shopping*, 35 J. LEGAL STUD. 365 (2006) (discussing “forum-shopping” by asbestos plaintiffs). For example, Madison County, Illinois, has been a popular destination for asbestos claim filings. 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) (discussing Madison County’s tendency to attract asbestos litigation). California courts also have attracted considerable attention. See generally Steven D. Wasserman et al., *Asbestos Litigation in California: Can it Change for the Better?*, 34 PEPP. L. REV. 883 (2007) (critiquing the state of asbestos litigation in California); Dominica C. Anderson & Kathryn L. Martin, *The Asbestos Litigation System in the San Francisco Bay Area: A Paradigm of the National Asbestos Litigation Crisis*, 45 SANTA CLARA L. REV. 1 (2004) (discussing the management of asbestos litigation in the Bay Area). As one set of commentators recently explained, “plaintiffs’ firms are steering cases to California, partly to the San Francisco-Oakland area, which is traditionally a tough venue for defendants, but also Los Angeles, which was an important asbestos venue in the 1980s but is only recently seeing an upsurge in asbestos cases.” Patrick M. Hanlon & Anne Smetak, *Asbestos Changes*, 62 N.Y.U. ANN. SURV. AM. L. 525, 599 (2007).

²⁷ See Griffin B. Bell, *Asbestos Litigation and Judicial Leadership: The Courts’ Duty to Help Solve the Asbestos Litigation Crisis*, 6:6 BRIEFLY 1, 29 (June 2002); 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).

²⁸ *Amchem Prods., Inc. v. Windsor*, 521 U.S. 591, 597 (1997); see also *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.”).

²⁹ *Gregg v. V-J Auto Parts, Co.*, 943 A.2d 216, 223 (Pa. 2007) (quoting *Summers v. Certaineed Corp.*, 886 A.2d 240, 244 (Pa. Super. 2005)).

³⁰ *Id.* (internal quotation marks omitted). Consider, analogously, a situation in which three nurses are sued for malpractice for providing an overdose of a drug to a patient via three simultaneous IVs. The doctor asked each nurse to provide five ccs of medication to the patient, for a total of fifteen ccs. Up to seventy-five ccs is considered safe, while anything more than that triples the risk of a stroke. The patient inadvertently receives 223 ccs, suffers a stroke, and dies. Under one scenario, Nurse A inadvertently provided six ccs of medication to the patient, Nurse B inadvertently provided seven ccs of medication to the patient, and nurse C inadvertently provided 210 ccs to the patient. Although Nurse A and Nurse B’s action contributed in some metaphysical sense to the patient’s death, only Nurse C’s actions were a “substantial factor” in that death. See RESTATEMENT (SECOND) OF TORTS § 431 cmt. a (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 ‘philosophic sense,’ which includes every one of the great

The Texas Supreme Court in 2007 issued perhaps the strictest recent opinion on exposure. The court noted that a plaintiff must show that his exposure to a defendant's asbestos-containing products "was a substantial factor in contributing to the aggregate *dose* of asbestos the plaintiff . . . inhaled or ingested, and hence to the *risk* of developing asbestos-related cancer."³² The plaintiff in the Texas case was a long-time brake mechanic, whose routine, low-level exposures to fibers in brakes and gaskets arguably satisfied the frequency, regularity, and proximity test articulated in *Lohrmann*. The court noted, however, that the plaintiff must also prove that the cumulative dose from an extended, low-level exposure rose to a level sufficient to cause disease. *Lohrmann* itself had held that the exposure level must make it probable that the exposure to the defendant's product caused plaintiff's injuries,³³ but many courts had given this aspect of *Lohrmann* short shrift. The recent, increasingly strict exposure cases described above 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*. 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.³⁴

number of events without which any happening would not have occurred."). On the other hand, if Nurse A had provided sixty ccs, Nurse B ninety ccs, and Nurse C seventy-three ccs, it seems fair to say that each nurse's negligence was a "substantial factor" in the patient's death. Or, to take a slightly different scenario, let us assume that the patient should not have been provided with *any* of the medication, and that each cc created a small risk of stroke, but it is not clear exactly at what point the stroke occurred. (This is analogous to arguing that a single fiber caused an asbestos-related disease, as opposed to the cumulative effects of multiple exposures.) Under the first scenario described above, it would make little sense to claim that Nurse A or B "caused" the stroke for legal purposes, when the likelihood that they caused the injury is well under 5% for each, while there is a greater than 90% chance that Nurse C was responsible.

³¹ See *Bartel v. John Crane, Inc.*, 316 F. Supp. 2d 603, 610-11 (N.D. Ohio 2004), *aff'd sub nom. Lindstrom v. A-C Prod. Liab. Trust*, 424 F.3d 488 (6th Cir. 2005); *In re W.R. Grace & Co.*, 355 B.R. 462, 474-78 (Bankr. D. Del. 2006); *Brooks v. Stone Architecture, P.A.*, 934 So. 2d 350, 352-54 (Miss. Ct. App. 2006); *Georgia-Pac. Corp. v. Stephens*, 239 S.W.3d 304, 320-21 (Tex. App. 2007).

³² *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 773 (Tex. 2007) (quoting *Rutherford v. Owens-Illinois, Inc.*, 941 P.2d 1203, 1219 (Cal. 1997)); see also Andrew K. Meade, *Asbestos Litigation Update: Texas Abandons the 'One Fiber' Causation Theory and Imposes a High Burden of Proof for Plaintiffs in Asbestos Cases*, THE HOUSTON LAWYER, Nov./Dec. 2007, at 16.

³³ *Lohrmann v. Pittsburgh Corning Corp.*, 782 F.2d 1156, 1162-63 (4th Cir. 1986).

³⁴ See generally *In re Silica Prods. Liab. Litig.*, 398 F. Supp. 2d 563 (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; Roger Parloff, *Diagnosing 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. As *The New York Times* reported,

Finally, the cases reflect judicial recognition that courts have a gate keeping obligation to keep “junk science” out of the courtroom.³⁵ There are sound public policy reasons for requiring trial courts to carefully evaluate expert testimony to ensure both its relevance and reliability. Expert testimony, whether presented by plaintiffs or defendants, can strongly influence juries. As the United States Supreme Court recognized, “[e]xpert evidence can be both powerful and quite misleading because of the difficulty in evaluating it.”³⁶ Expert witnesses are given extraordinary privileges in court. Unlike lay witnesses, “an expert is permitted wide latitude to offer opinions, including those that are not based on firsthand knowledge or observation.”³⁷ In addition, courts may permit expert witnesses to testify on the ultimate issue to be decided by the jury.³⁸ And perhaps most significant, unlike with regard to lay witnesses, parties presenting experts can often choose from an almost unlimited supply of potential experts, and present only the testimony of those who agree with the proffering party’s perspective, or are willing to say they do in exchange for an hourly fee.³⁹

Even beyond the asbestos context, the battle over causation standards in toxic tort litigation has come to focus on the admissibility and sufficiency of expert testimony. Plaintiffs contend that juries should be permitted to rule in their favor based on testimony from experts who are willing to speculate regarding causation based on what objectively appears to be meager, tangentially relevant data. Defendants, by contrast, insist that plaintiffs are obligated to present reliable evidence of specific causation. As discussed in Part II, below, defendants are gradually prevailing in this dispute, as courts become increasingly skeptical of various categories of “proof” of causation presented by plaintiffs.

[United States District Judge Janis Graham Jack in Corpus Christi, Texas] not only called for sanctions on one of the plaintiffs’-side law firms, but she also slammed the whole process that led to the claims landing in court in the first place. The medical findings underlying the claims, based on X-ray screenings paid for by lawyers looking for potential clients, were worthless, she wrote.

“It is apparent that truth and justice had very little to do with these diagnoses—otherwise more effort would have been devoted to ensuring they were accurate,” Judge Jack wrote. “These diagnoses were driven by neither health nor justice: they were manufactured for money. The record does not reveal who originally devised this scheme, but it is clear that the lawyers, doctors and screening companies were all willing participants.”

Jonathan D. Glater, *The Tort Wars, at a Turning Point*, N.Y. TIMES, Oct. 9, 2005.

³⁵ See generally Bernstein, *Junk Science*, *supra* note 22 (discussing the requirements of Federal Rule of Evidence 702 and state equivalents and how they affect the admissibility of dubious scientific evidence in the asbestos litigation).

³⁶ *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 595 (1993) (quoting Jack B. Weinstein, *Rule 702 of the Federal Rules of Evidence Is Sound; It Should Not Be Amended*, 138 F.R.D. 631, 632 (1991)).

³⁷ *Id.* at 592.

³⁸ FED. R. EVID. 704.

³⁹ See David E. Bernstein, *Expert Witnesses, Adversarial Bias, and the (Partial) Failure of the Daubert Revolution*, 93 IOWA L. REV. 451, 455 (2008) [hereinafter Bernstein, *Expert Witnesses*].

II. SPECULATIVE CAUSATION CASES

Despite the black letter requirement that plaintiffs provide sufficient evidence to prove general and specific causation by a preponderance of the evidence, plaintiffs have often brought toxic tort claims where the evidence of causation ranges from extremely speculative to entirely fanciful. While plaintiffs must present expert testimony for their lawsuits to be viable, “there is no shortage of credentialed scientists in the world who will confuse hypothesis with confirmed fact, and testify (sincerely), to the actual existence of causal relations or substantially enhanced risks on weak or no evidence.”⁴⁰

Among the dubious categories of evidence frequently relied on by expert witnesses in toxic tort cases are high-dose animal studies, anecdotal case reports, analogizing from known effects of “similar” chemicals, preliminary epidemiological studies that have not been peer-reviewed, and differential etiologies used to “rule in” an otherwise unknown causal relationship. Each of these categories of evidence will be discussed below in turn.

Anecdotal case reports depict a temporal relationship between exposure to a substance and the manifestation of disease. For example, a physician may report that a pregnant patient to whom he prescribed a new drug gave birth to a child with birth defects. With rare exceptions, case reports at best suggest a hypothesis that needs further testing.⁴¹ As one medical expert has explained, “[c]ase reports, by their very nature, can never prove causation.”⁴² Not surprisingly, courts routinely reject case reports as proof of causation.⁴³ A few courts, however, allow experts

⁴⁰ D. Michael Risinger, *Preliminary Thoughts on a Functional Taxonomy of Expertise for the Post-Kumho World*, in MODERN SCIENTIFIC EVIDENCE: THE LAW AND SCIENCE OF EXPERT TESTIMONY § 2:15, 144 (David L. Faigman et al. eds., 2d ed. 2005).

⁴¹ See David H. Kaye & David A. Freedman, *Reference Guide on Statistics*, in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 91-92 (2d ed. 2000) (discussing the general unreliability of anecdotal evidence of causation); see *In re Breast Implant Litig.*, 11 F. Supp. 2d 1217, 1228 (D. Colo. 1998) (“To the extent there are case or anecdotal reports noting various symptoms or signs in breast implanted women, without controls, these suggest only a potential, untested hypothesis that breast implants may be their cause.”); *In re Toxic Substances Cases*, No. A.D. 03-319, 2006 WL 2404008, at *4-5 (Pa. Ct. Com. Pl. Aug. 17, 2006) (case reports may suggest associations and may be used to generate hypotheses to be tested, but they do not show causal connections). Available anecdotal evidence can occasionally be so strong as to create a reasonable, but tentative, inference of causation. See KATHLEEN R. STRATTON ET AL., ADVERSE EVENTS ASSOCIATED WITH CHILDHOOD VACCINES: EVIDENCE BEARING ON CAUSALITY 22 (1994) (case reports can sometimes be persuasive evidence of causation of injury from a vaccine); Michael D. Green, *Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of Agent Orange and Bendectin Litigation*, 86 NW. U. L. REV. 643, 658 (1992) (“Occasionally, when the effect of the agent is powerful enough, scientists will tentatively accept case reports as sufficient to establish a causal relation.”). Even then, such inferences cannot stand in the face of stronger contrary evidence.

⁴² Robert N. Jones, M.D., *Asbestos Medicine II*, SJ031 ALI-ABA *29, *35 (Nov. 13-14, 2003).

⁴³ See *Glastetter v. Novartis Pharm. Corp.*, 107 F. Supp. 2d 1015, 1028, 1030-31 (E.D. Mo. 2000), *aff'd*, 252 F.3d 986 (8th Cir. 2001); *Hollander v. Sandoz Pharm. Corp.*, 95 F. Supp. 2d 1230, 1236-38 (W.D. Okla. 2000), *aff'd in part*, 289 F.3d 1193 (10th Cir. 2002); *Brumbaugh v.*

to speculate wildly based on case reports. One court, for example, argued that “[i]n science, as in life, where there is smoke, fire can be inferred, subject to debate and further testing.”⁴⁴ Such reasoning reflects the *post hoc ergo propter hoc* (after which therefore because of which) logical fallacy.⁴⁵ Although medical journals do publish case reports to alert readers to potentially significant clinical evidence, no respectable journal will permit an author to make an unsubstantiated inference of causation from such a report, because such inferences are simply not scientifically valid.⁴⁶

To understand why an isolated case report is not reliable evidence of causation, consider the following example. Hundreds of thousands of infants may receive measles vaccines every year, and a few of them, by chance, will develop brain tumors. A physician who happens to see one of these unfortunate children and is at a loss to explain the tumor may be tempted to attribute the tumor to the vaccine. She may even publish a case report in a medical journal. However, having seen only a skewed data sample including one of the few infants to develop a brain tumor after a measles vaccine, but not including many thousands who did not, the physician is simply not in a position to infer that the measles vaccine causes brain tumors.

Other experts rely on extrapolation from animal studies to prove human causation. This practice is very problematic because not only are animal studies typically conducted at much higher levels than relevant human exposure,⁴⁷ but a substance that is carcinogenic or teratogenic (causing birth defects) in one species (e.g., rabbits) is not necessarily carcinogenic or teratogenic in another (e.g., humans). A recent study shows that a substance that is toxic to both rodents and non-rodents predicts toxicity in humans 71% of the time, toxicity in nonrodent

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).

⁴⁴ Brasher v. Sandoz Pharm. Corp., 160 F. Supp. 2d 1291, 1296 (N.D. Ala. 2001).

⁴⁵ Richard H. Underwood, *Logic and the Common Law Trial*, 18 AM. J. TRIAL ADVOC. 151, 175-78 (1994) (explaining the *post hoc ergo propter hoc* logical fallacy).

⁴⁶ See, e.g., Hall v. Baxter Healthcare Corp., 947 F. Supp. 1387, 1411 (D. Or. 1996) (“[C]ase reports and case studies are universally regarded as an insufficient scientific basis for a conclusion regarding causation because case reports lack controls.”); Muzzey v. Kerr-McGee Chem. Corp., 921 F. Supp. 511, 519 (N.D. Ill. 1996) (case reports “are not reliable bases to form a scientific opinion about a causal link”); *In re TMI Litig. Cases Consol. II*, 911 F. Supp. 775, 801 (M.D. Pa. 1996) (excluding testimony relying on a case report and not meeting “the most basic standards of scientific validity”), *aff’d*, 193 F.3d 613 (3d Cir. 1999); Cavallo v. Star Enter., 892 F. Supp. 756, 765-66 n.18 (E.D. Va. 1995) (“[C]ase reports are not reliable scientific evidence of causation, because they simply describe[] reported phenomena without comparison to the rate at which the phenomena occur in the general population or in a defined control group; do not isolate and exclude potentially alternative causes; and do not investigate or explain the mechanism of causation.”) (quoting *Casey v. Ohio Med. Prods.*, 877 F. Supp. 1380, 1385 (N.D. Cal. 1995)), *aff’d in pertinent part*, 100 F.3d 1150 (4th Cir. 1996).

⁴⁷ Beyond the well-known principle, “the dose makes the poison,” the chronic irritation that many chemicals produce at high exposure levels is itself a cause of cancer. Bruce N. Ames & Lois S. Gold, *Too Many Rodent Carcinogens: Mitogenesis Increases Mutagenesis*, 249 SCI. 970, 970 (1990).

species predicts toxicity in humans 63% of the time, and toxicity in rodents predicts toxicity in humans 43% of the time.⁴⁸ As one court has concluded, “[b]ecause of the difference in animal species, the methods and routes of administration of the suspect chemical agent, maternal metabolisms and other factors, animal studies, taken alone, are unreliable predictors of causation in humans.”⁴⁹

The United States Supreme Court addressed many of the typical problems attendant to relying on animal studies to prove causation in toxic tort cases in *General Electric Co. v. Joiner*.⁵⁰ The plaintiff’s experts sought to rely on mice studies to prove he contracted cancer based on his exposure to PCBs. The Court noted:

Joiner [the plaintiff] was an adult human being whose alleged exposure to PCBs was far less than the exposure in the animal studies. The PCBs were injected into the mice in a highly concentrated form. The fluid with which Joiner had come into contact generally had a much smaller PCB concentration of between 0–500 parts per million. The cancer that these mice developed was alveogenic adenomas; Joiner had developed small-cell carcinomas. No study demonstrated that adult mice developed cancer after being exposed to PCBs. One of the experts admitted that no study had demonstrated that PCBs lead to cancer in any other species.⁵¹

Because the animal studies presented by the plaintiff were not reliable evidence of causation, the Court upheld the district court’s grant of summary judgment to General Electric. More generally, as a recent law review article explained,

[i]f a laboratory test on a rat shows a chemical to be carcinogenic to the rat at a high exposure level, the test will not prove carcinogenicity in humans if the expert cannot explain how rats and animals have a similar physiological makeup and rate of chemical absorption, or if the expert cannot provide an

⁴⁸ Harry Olson et al., *Concordance of the Toxicity of Pharmaceuticals in Humans and in Animals*, 32 REG. TOXICOLOGY & PHARMACOLOGY 56, 59-60 (2000).

⁴⁹ Nat’l Bank of Commerce v. Dow Chem. Co., 965 F. Supp. 1490, 1527 (E.D. Ark. 1996), *aff’d*, 133 F.3d 1132 (8th Cir. 1998). Regulatory agencies such as the EPA nevertheless rely on animal studies in formulating proactive regulations. They do so not because such studies are reliable, but because in the absence of human data, such studies are often the best (and perhaps the only) evidence of human toxicity the agencies have. See Robert M. Sussman, *Science and EPA Decision-Making*, 12 J.L. & POL’Y 573, 584 (2004). It is one thing, however, to say that an animal study is suggestive of a possible risk to humans and therefore it is prudent to take precautions through the regulatory process, and quite another to say that an animal study proves that human exposure to the particular substance at a much lower dose more likely than not caused a specific individual’s injury. For cases rejecting reliance on government regulatory action as evidence of causation, see *infra* note 75.

⁵⁰ 522 U.S. 136 (1997). Though *Joiner* involved cancer, similar issues arise when animal studies are used to prove the causation of birth defects. For discussions of the difficulty of extrapolating teratogenicity to humans from animal studies, see, e.g., Gideon Koren et al., *Drugs in Pregnancy*, 338 NEW ENG. J. MED. 1128, 1131 (1998); Louis Lasagna, *Predicting Human Drug Safety from Animal Studies*, 12 J. TOXICOLOGICAL SCI. 439, 442-43 (1987).

⁵¹ *Joiner*, 522 U.S. at 143.

accurate minimal dose-response relationship in humans based on the animal results.⁵²

Another gambit used by experts to find causation is to claim that because a similar chemical is known to cause harm, the chemical at issue in the case must also cause harm. As several courts have recognized, such reasoning is fallacious.⁵³ In *DeLuca v. Merrell Dow Pharmaceuticals*,⁵⁴ for example, the court concluded that the fact that other drugs with similar chemical structures are associated with birth defects is not competent evidence that Bendectin is associated with birth defects. Similarly, in *Lofgren v. Motorola, Inc.*,⁵⁵ the court determined that it is not generally accepted among scientists to rely on data regarding one chemical to determine the carcinogenicity of another, and that such testimony therefore must be excluded under the *Frye* general acceptance test.⁵⁶

Experts often try to bolster their causation theories by claiming that they have undertaken a “differential diagnosis” (really, differential etiology)⁵⁷ in which they have considered and eliminated other plausible causes of the disease.⁵⁸ In fact, a differential etiology cannot possibly determine that substance A caused disease B in the absence of prior, reliable independent evidence that substance A can cause disease B. Without such evidence, the substance of these purported differential etiologies usually amounts to an expert asserting that the product or substance at issue in this case must have caused the plaintiff’s injury,

⁵² See Craig T. Smith, *Peering into the Microscope: The Rise of Judicial Gatekeeping After Daubert and its Effect on Federal Toxic Tort Litigation*, 13 B.U. J. SCI. & TECH. L. 218, 233 (2007).

⁵³ David L. Eaton, *Scientific Judgment and Toxic Torts—A Primer in Toxicology for Judges and Lawyers*, 12 J.L. & POL’Y 5, 10 (2003) (“[E]ven though different chemicals of the same general type (e.g., solvents) may have some common effects, they may also differ dramatically in other effects. For example, . . . benzene is toxic to the bone marrow and can increase the risk of leukemia in workers, whereas these serious toxic effects have not been found for toluene [a similar solvent].”).

⁵⁴ *DeLuca v. Merrell Dow Pharm., Inc.*, 791 F. Supp. 1042, 1054 (D.N.J. 1992), *aff’d*, 6 F.3d 778 (3d Cir. 1993).

⁵⁵ *Lofgren v. Motorola Inc.*, No. CV 93-05521, 1998 WL 299925 (Ariz. Super. Ct. June 1, 1998).

⁵⁶ *Id.* at *15. The test articulated in *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923), and still applied in several states, requires that expert scientific testimony is admissible only when the theory or technique underlying the testimony is “sufficiently established to have gained general acceptance in the particular field in which it belongs.” *Id.* at 1014.

⁵⁷ See David L. Faigman, *The Limits of Science in the Courtroom*, in BEYOND COMMON SENSE 303, 307 (Eugene Borgida & Susan T. Fiske eds., 2008) (“Properly understood, differential diagnosis refers to the identification of the illness or behavioral condition that a person is experiencing. Differential etiology refers to the cause or causes of that condition.”) (emphasis omitted); Joseph Sanders & Julie Machal-Fulks, *The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law*, L. & CONTEMP. PROBS., Autumn 2001, at 107, 108 (“differential etiology” is a more appropriate moniker for this sort of analysis).

⁵⁸ Sanders & Machal-Fulks, *supra* note 57, at 111 (“Because courts have generally refused to relieve the plaintiff from proving specific causation, differential diagnosis evidence is often a crucial component of the plaintiff’s case.”).

merely because he cannot establish any other causal mechanism, even though there is no sound evidence linking the product or substance to the relevant injury.⁵⁹ A Florida Supreme Court justice has provided an illustrative, albeit extreme, example:

[A] patient suffering from depression sees a doctor because her arm hurts. She does not know why her arm hurts. The doctor diagnoses a broken arm. The patient cannot tell the doctor how she broke her arm. The doctor may, through performing tests and interviewing the patient, conclude that it could not have been a car accident (the patient was not involved in an accident) and it could not have been playing sports (the patient does not play sports), but the doctor cannot then conclude that it must have been the depression that caused the broken arm—unless, of course, the doctor can show that the theory that depression can cause a broken arm is generally accepted in the scientific community.⁶⁰

Most courts exclude differential etiology evidence used to support a previously unsupported causal relationship as unreliable,⁶¹ though some jurisdictions hold that how much to credit such evidence is an issue of weight, not admissibility.⁶² Even assuming, however, that flaws in the use of differential etiology go to weight and not admissibility, a plaintiff cannot properly use differential etiology to “rule in” a cause and avoid summary judgment, much less satisfy his burden of proof.⁶³

Another tactic used by experts to pursue speculative causation theories is to rely on preliminary, unpublished epidemiological studies that have not been scrutinized by peers in the scientific community. For example, the plaintiffs in the litigation alleging that the morning sickness drug Bendectin caused birth defects sought, ultimately unsuccessfully, to rely on an unpublished (and, as it turns out, never published) reanalysis

⁵⁹ Cf. Edward J. Inwinkelried, *The Admissibility and Legal Sufficiency of Testimony About Differential Diagnosis (Etiology): Of Under- and Over-Estimations*, 56 BAYLOR L. REV. 391, 406 (2004) (An opinion based on differential etiology “seems to be at most an educated guess” with regard to general causation.).

⁶⁰ *Marsh v. Valyou*, 977 So.2d 543, 565 (Fla. 2007) (Cantero, J., dissenting).

⁶¹ See *Smith*, *supra* note 52, at 225 (“The majority of courts hold that an expert must ‘rule in’ an agent as a potential cause of the plaintiff’s injury, then ‘rule out’ other possible causes until only the most likely cause remains.”).

⁶² See, e.g., *Zuchowicz v. United States*, 140 F.3d 381, 387 (2d Cir. 1998) (“[D]isputes as to . . . faults in [the] use of differential etiology as a methodology, or lack of textual authority for [an] opinion, go to the weight, not the admissibility of [the] testimony.”) (quoting *McCullock v. H.B. Fuller Co.*, 61 F.3d 1038, 1044 (2d Cir. 1995)).

⁶³ See, e.g., *In re Rezulin Prods. Liab. Litig.*, No. MDL 1348, 00 Civ. 2843(LAK), 2004 WL 2884327, at *3 (S.D.N.Y. Dec. 10, 2004) (differential etiology may not be used to prove general causation); *Cavallo v. Star Enter.*, 892 F. Supp. 756, 771 (E.D. Va. 1995) (it is not enough for an expert to rule out other possible causes if he has no sound evidence that allows him to “rule in” the purported cause), (*aff’d on this issue, rev’d in part on other grounds*, 100 F.3d 1150 (4th Cir. 1996)). See generally *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 596 (1993) (If a court holds that marginal evidence on causation is admissible, but the evidence is not sufficient to meet the plaintiff’s burden, the court should grant summary judgment to the defendant.).

of earlier epidemiological studies.⁶⁴ Likewise, one of the most important early breast implant cases involved an expert who purportedly relied on “a preliminary epidemiological study involving over 200 women.”⁶⁵ This study, too, was never published.

Epidemiology, even when peer-reviewed and published, is subject to systematic errors, biases, and confounders.⁶⁶ Retrospective studies are especially problematic because they do not have the same controls as prospective, double-blind studies. With few exceptions, a retrospective study purported to show a causal association between a substance and disease is presumptively unreliable until confirmed by additional studies.⁶⁷ Relying on a single study that has not even received the basic scrutiny that attends publication in a peer-reviewed journal is that much more dubious. Even if a particular study has been peer-reviewed, if conflicting studies exist, it is improper for an expert to rely on that study without a valid scientific explanation as to why he discounts the conflicting studies.⁶⁸

Other plaintiffs claim, in defiance of both science and common sense, that trivial exposure to a substance was the proximate cause of their injuries. Sometimes plaintiffs’ experts explicitly argue that any amount of exposure, no matter how minimal, was a “cause” of the plaintiff’s injury.⁶⁹ Other times, this argument is implicit, as when plaintiffs’ experts opine on causation despite their total failure to investigate the dose to which the plaintiff was exposed.⁷⁰

⁶⁴ See *Daubert v. Merrell Dow Pharm., Inc.*, 951 F.2d 1128, 1131 (9th Cir. 1991), *vacated*, 509 U.S. 579 (1993).

⁶⁵ *Hopkins v. Dow Corning Corp.*, 33 F.3d 1116, 1125 (9th Cir. 1994). On the importance of *Hopkins* in unleashing a flood of breast implant litigation, see David E. Bernstein, *The Breast Implant Fiasco*, 87 CAL. L. REV. 457, 472, 484-85 (1999).

⁶⁶ See Taubes, *supra* note 11, at 167.

⁶⁷ *Id.* at 168.

⁶⁸ *Lofgren v. Motorola Inc.*, No. CV 93-05521, 1998 WL 299925, at *16-17 (Ariz. Super. Ct. June 1, 1998).

⁶⁹ See, e.g., *Lindstrom v. A-C Prod. Liab. Trust*, 424 F.3d 488, 493 (6th Cir. 2005) (“Dr. Corson does not specifically reference the product of any particular Defendant. Rather, he opines that there is no safe level of asbestos exposure, and that every exposure to asbestos, however slight, was a substantial factor in causing Lindstrom’s disease.”); *In re Toxic Substances Cases*, No. A.D. 03-319, 2006 WL 2404008, at *2 (Pa. Ct. Com. Pl. Aug. 17, 2006) (reporting that the experts argued that each and every exposure to asbestos was a proximate cause of plaintiffs’ injury).

⁷⁰ See *Nelson v. Tenn. Gas Pipeline Co.*, 243 F.3d 244, 252-53 (6th Cir. 2001) (“[P]laintiffs cannot dispute that [their expert] made no attempt to determine what amount of PCB exposure the [plaintiffs] had received and simply assumed that it was sufficient to make them ill.”); *Wintz v. Northrop Corp.*, 110 F.3d 508, 513-14 (7th Cir. 1997) (rejecting an expert’s causation opinion because he did not “know how frequently, in what quantity, or in what form” the plaintiff had been exposed to the chemical at issue); *Adams v. Cooper Indus., Inc.*, No. 03-476-JBC, 2007 WL 2219212, at *5 (E.D. Ky. July 30, 2007) (the plaintiffs’ experts “made no inquiry into the amounts of the chemicals to which the plaintiffs were exposed”); *Chikovsky v. Ortho Pharm. Corp.*, 832 F. Supp. 341, 345 (S.D. Fla. 1993) (the plaintiff’s expert “[did] not know how much Retin-A [the plaintiff] might have absorbed through her skin during her pregnancy”); *O’Conner v. Commonwealth Edison Co.*, 807 F. Supp. 1376, 1396 (C.D. Ill. 1992) (expert failed to consider whether plaintiff was exposed to sufficient dosage of radiation to cause cataracts); *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 772 (Tex. 2007) (the plaintiff presented no evidence regarding how

To the extent that experts try to explain why they ignore one of what the Federal Judicial Center's *Reference Manual on Scientific Evidence* calls the "central tenets" of toxicology—"the dose makes the poison"⁷¹—they rely on a variety of invalid theories. Some experts, especially in asbestos cases, argue that exposure is cumulative and contributes to disease.⁷² As noted previously, however, to argue that *any* exposure to asbestos, no matter how minimal, is a "substantial factor" in the development of asbestos-related disease regardless of much more extensive exposures is the equivalent of arguing that dumping a bucket of water in the ocean is a "substantial factor" in the size of the ocean.⁷³

Other experts argue that the safety of the substance at issue has a non-linear threshold or no threshold. These experts typically have no sound reason to believe that the particular substance at issue fits this theory, and courts consistently reject such evidence of proof of causation.⁷⁴ Experts sometimes note that the exposure at issue exceeded stringent regulatory standards, but, as the New York Court of Appeals recently concluded, ultra-conservative "standards promulgated by regulatory agencies as protective measures are inadequate to demonstrate legal causation."⁷⁵ The reason is that the task of an agency like OSHA, NIOSH, or EPA is to define and/or enforce exposure limitations for hazardous substances that the agency believes will adequately protect the public from harm. To do this, the agencies often have to work without adequate evidence, and they will take the conservative approach of issuing warnings and setting standards to avoid the *potential* of harm well below any known exposure level associated with actual injury. They typically do not set regulatory limits at the dose of *known causation*, and

much asbestos plaintiff inhaled or "what percentage of that indeterminate amount may have originated in Borg-Warner products").

⁷¹ See Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology, in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE* 401, 403 (2d ed. 2000) ("There are three central tenets of toxicology. First, 'the dose makes the poison'; this implies that all chemical agents are intrinsically hazardous—whether they cause harm is only a question of dose. Even water, if consumed in large quantities, can be toxic."); Eaton, *supra* note 53, at 11 (in toxicology "[d]ose is the single most important factor to consider in evaluating whether an alleged exposure caused a specific adverse effect"); Faigman, *supra* note 57, at 309 ("The first principle of toxicology is that the dose is the poison . . .") (citation and internal quotation marks omitted).

⁷² See *Borg-Warner*, 232 S.W.3d at 771-73 (Tex. 2007); see also Meade, *supra* note 32 (analyzing the impact of *Borg-Warner*).

⁷³ See *supra* note 30 and accompanying text.

⁷⁴ See, e.g., *Cano v. Everest Mineral Corp.*, 362 F. Supp. 2d 814, 849 (W.D. Tex. 2005) ("Several courts have considered and rejected the use of the linear no-threshold model in the litigation context."); *Sutera v. Perrier Group of Am., Inc.*, 986 F. Supp. 655, 666 (D. Mass. 1997) ("[T]here is no scientific evidence that the linear no-safe threshold analysis is an acceptable scientific technique used by experts in determining causation in an individual instance.").

⁷⁵ *Parker v. Mobil Oil Corp.*, 857 N.E.2d 1114, 1122 (N.Y. 2006); see also *Gehl v. Soo Line R.R.*, 967 F.2d 1204, 1208 (8th Cir. 1992); *Sprinkle v. Bower Ammonia & Chem. Co.*, 824 F.2d 409, 416-17 (5th Cir. 1987); *Siharath v. Sandoz Pharm., Corp.*, 131 F. Supp. 2d 1347, 1366 (N.D. Ga. 2001), *aff'd*, 295 F.3d 1194 (11th Cir. 2002); *Glastetter v. Novartis Pharm. Corp.*, 107 F. Supp. 2d 1015, 1036 (E.D. Mo. 2000), *aff'd*, 252 F.3d 986 (8th Cir. 2001); *Hollander v. Sandoz Pharm. Corp.*, 95 F. Supp. 2d 1230, 1235-38 (W.D. Okla. 2000), *aff'd in part*, 289 F.3d 1193 (10th Cir. 2002).

they do not rely on proof of causation to set these levels. As explained by the Tenth Circuit Court of Appeals:

The methodology employed by a government agency “results from the preventive perspective that the agencies adopt in order to reduce public exposure to harmful substances. The agencies’ threshold of proof is reasonably lower than that appropriate in tort law, which traditionally makes more particularized inquiries into cause and effect and requires a plaintiff to prove it is more likely than not that another individual has caused him or her harm.”⁷⁶

Still other experts decline to estimate the plaintiff’s exposure. They rely instead on terms such as “substantial,” “significant,” or “high”—terms that have no objective scientific meaning in the absence of a defined baseline.⁷⁷ Some experts simply ignore mainstream, reliable toxicology and make a “simple logical error”⁷⁸ by “extrapolating down,” arguing that if exposure to large amounts of a substance can cause disease, exposure to a small amount of the same substance can also cause disease.⁷⁹ As a Pennsylvania court recently noted:

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, greater volumes or an extended absence of water can be lethal; moderate amounts of water, however, are healthful. In short, the poison is in the dose.⁸⁰

Finally, some experts reason backwards, arguing that because the plaintiff acquired a disease that can be caused by intense exposure to a

⁷⁶ Mitchell v. Gencorp Inc., 165 F.3d 778, 783 n.3 (10th Cir. 1999) (quoting Allen v. Pa. Eng’g Corp., 102 F.3d 194, 198 (5th Cir. 1996)).

⁷⁷ Parker, 857 N.E.2d at 1122 (noting that the plaintiff’s expert argued that the plaintiff was “frequently” exposed to “excessive” amounts of gasoline and had “extensive exposures . . . in both liquid and vapor form,” and concluding that “even given that an expert is not required to pinpoint exposure with complete precision” this expert’s statements could not “be characterized as a scientific expression of Parker’s exposure level”).

⁷⁸ *In re Toxic Substances Cases*, No. A.D. 03-319, 2006 WL 2404008, at *7 (Pa. Ct. Com. Pl. Aug. 17, 2006).

⁷⁹ For example, in *In re Bextra*, the court stated:

Although Dr. Doherty acknowledges that dose matters with Celebrex, he simply takes the relative risk point estimate of APC for 400 mg/d and cuts it in half (ignoring the confidence interval) to support his opinion that Celebrex at 200 mg/d can cause a heart attack. When the Court asked Dr. Doherty if there is anything in the scientific literature to support such primitive extrapolation, he failed to identify any scientific support for his method other than his own judgment. . . . Such an unscientific, untested methodology cannot support the proffered opinion of causation

In re Bextra & Celebrex Mktg. Sales Practices & Prod. Liab. Litig., 524 F. Supp. 2d 1166, 1180 (N.D. Cal. 2007) (internal citations omitted); see also *In re Toxic Substances Cases*, 2006 WL 2404008, at *7 (“Drs. Maddox and Laman attempt to ‘extrapolate down’ reasoning that if high dose exposure is bad for you, then surely low dose exposure (indeed, no matter how low) must still be bad for you.”).

⁸⁰ *In re Toxic Substances Cases*, 2006 WL 2404008, at *7.

particular substance, the plaintiff must have been intensely exposed to that substance.⁸¹ Unless the disease at issue is a rare “signature disease” always or almost always caused by exposure to a specific substance, this reasoning neglects the possibility that the disease at issue was caused by some other factor. Plaintiffs have been able to get away with such speculations because until recently many judges were reluctant to require plaintiffs’ experts to quantify, or even estimate, plaintiffs’ exposures to the alleged toxin at issue.

Thus, as we have seen, courts, bolstered by modern strict rules for the admissibility of expert testimony, are increasingly rejecting certain categories of expert evidence presented by plaintiffs—including testimony based on animal studies, case reports, analogies to “similar” chemicals, unpublished epidemiological studies, and differential etiologies—when such testimony is misused to assert causal inferences that the underlying studies do not support. But courts, supported by some legal commentators, are often sorely tempted to loosen causation standards to ease plaintiffs’ path to recovery. Part III of this Article discusses why courts should resist this temptation and require plaintiffs to meet their burden of proof by presenting reliable evidence of causation.

III. COURTS SHOULD REQUIRE PLAINTIFFS TO MEET THEIR BURDEN OF PROOF

Some readers will undoubtedly question whether courts are enforcing causation requirements that are too strict when they refuse to allow plaintiffs to rely on speculative or unreliable evidence of causation. After all, in some cases defendants did in fact negligently expose plaintiffs to at least the *risk* of harm. Even if causation cannot be proven, perhaps holding defendants liable for their negligence by shifting the burden of proof to them to *disprove* causation is appropriate.⁸²

There are several difficulties with this approach. First, adopting such a system would lead virtually any company that manufactured a

⁸¹ See, e.g., *Wills v. Amareda Hess Corp.*, No. 98-CV-7126, 2002 WL 140542, at *10 (S.D.N.Y. Jan. 31, 2002) (The plaintiff’s expert did not attempt to quantify the decedent’s exposure, but was nevertheless “ready to form a conclusion first, without any basis, and then try to justify it” by claiming that the fact that decedent had contracted an aggressive form of cancer was itself evidence that proved intensive exposure to carcinogenic hydrocarbons.). Relatedly, one expert recently attempted to argue that concentrations of toxins in plaintiffs’ blood level were “abnormal” because they exceeded the median in the population as a whole. The court rejected this testimony, noting that scientists consider “normal” to include concentrations present in the 95th percentile of the population. *Adams v. Cooper Indust., Inc.*, No. 03-476-JBC, 2007 WL 1805586, at *5 (E.D. Ky. June 21, 2007).

⁸² Early in the toxic tort era, one court explicitly shifted the burden of proof to defendants. See *Allen v. United States*, 588 F. Supp. 247, 415 (D. Utah 1984), *rev’d*, 816 F.2d 1417 (10th Cir. 1987) (holding that the jury in a toxic tort case may find for the plaintiff “absent persuasive proof to the contrary offered by the defendant”); see generally Thomas W. Henderson, *Toxic Tort Litigation: Medical and Scientific Principles in Causation*, 132 AM. J. EPIDEMIOLOGY S69 (Supp. 1990).

potentially hazardous product to be dragged into litigation based on a combination of alleged misbehavior and speculative expert testimony. The experts engaging in such guesswork will be adversarial, partisan experts chosen because the plaintiffs' attorney knows that they are willing to testify that they agree with his theory of the case.⁸³ Some such experts are "hired guns," while others are sincere "outliers" with views well outside the scientific mainstream. Between hired guns and outliers, plaintiffs' attorneys have had no difficulty finding qualified experts willing to testify to causal relationships lacking sound scientific support, even when, as was the case with the Bendectin litigation, a solid line of epidemiological studies contradicted their views.⁸⁴

Placing the burden of proof on the defendants is especially problematic because it requires them to prove a negative, that their substances did not cause harm. Yet "[s]cience can never demonstrate the absence of hazard," but "can only place an upper limit on risk."⁸⁵ Allowing cases to go to the jury based on a combination of extremely speculative evidence and perceived bad behavior by the defendants runs a significant risk that juries will focus on punishing unpopular defendants, rather than on determining whether the evidence presented supports causation.⁸⁶

⁸³ For more on the problem of "adversarial bias," see generally Bernstein, *Expert Witnesses*, *supra* note 39.

⁸⁴ See David E. Bernstein, *Learning the Wrong Lessons from "An American Tragedy": A Critique of the Berger-Twerski Informed Choice Proposal*, 104 MICH. L. REV. 1961, 1964-65 (2006) [hereinafter Bernstein, *Wrong Lessons*].

⁸⁵ See PHANTOM RISK: SCIENTIFIC INFERENCE AND THE LAW 435 (Kenneth R. Foster et al. eds., 1993). One cannot, as a general matter, prove a negative, and certainly not with epidemiological studies or other tools currently at scientists' disposal. See Margaret A. Berger, *Converting Unknown Risk into Phantom Risk*, in *Books-on-Law: Book Reviews* (Sept. 1999), <http://jurist.law.pitt.edu/lawbooks/revsep99.htm#Berger> (reviewing *Phantom Risk*) ("Epidemiological studies are incapable of proving that something has no effects . . .").

⁸⁶ See Bernstein, *The Breast Implant Fiasco*, *supra* note 65, at 473, 478, 486 (explaining how attorneys in the breast implant litigation emphasized defendants' misconduct to distract the jury from the fact that their causation evidence was extremely weak); Edwin J. Jacob, *Of Causation in Science and Law: Consequences of the Erosion of Safeguards*, 40 BUS. LAW. 1229, 1238-40 (1985). This risk is compounded by the fact that laypeople find it extremely difficult to comprehend probabilistic evidence, and therefore are tempted to focus on more readily accessible issues. See, e.g., Isaac M. Lipkus et al., *General Performance on a Numeracy Scale Among Highly Educated Samples*, 21 MED. DECISION MAKING 37, 37 (2001) ("[E]ven highly educated participants have difficulty with relatively simple numeracy questions . . ."). Moreover, juries are apt to give too much weight to evidence that speaks to their emotions. See Kari Edwards & Tamara S. Bryan, *Judgmental Biases Produced by Instructions to Disregard: The (Paradoxical) Case of Emotional Information*, 23 PERSONALITY & SOC. PSYCHOL. BULL. 849, 856 (1997) (concluding that information that elicits emotions is especially difficult to ignore).

Things get even dicier when not one but several allegedly harmful products or substances are at issue. For example, there are different asbestos fiber types, some of which are more harmful than others, and there are different asbestos-containing products, some with high friable fiber potential, some with very low friable fiber potential. Yet in cases where a claimant alleges exposure to multiple defendants' asbestos products that are materially different from one another, courts often shift the burden to defendants to disprove causation by allowing plaintiff experts to infer that the cumulative effect of the exposure proves causation, without individualized evidence about the dangers of specific products.

Moreover, allowing plaintiffs to place the burden of proof on defendants to disprove causation means that allegations presented in toxic tort claims will be limited only by the imaginations of plaintiffs' attorneys. Consider that one plaintiffs' expert made the wildly implausible allegation that silicone breast implants caused illnesses and symptoms ranging from auto-immune disease to "suicidal depression" to hair loss.⁸⁷ No rational legal system would ask a defendant to disprove that each of these symptoms was caused by its product simply because a paid expert for the other side has made such claims.

Even if plaintiffs provide reliable evidence that a defendant's actions have raised the risk of disease, but plaintiffs cannot prove specific causation by a preponderance of the evidence, sound, fundamental tort principles prohibit plaintiffs from recovering. Under standard tort principles, "cause" and "risk" are distinct, with only the former creating a viable tort action.⁸⁸ Consider a hypothetical truck driver who drives through a school zone at 2:35 p.m., just after school lets out, at ninety miles per hour. He is very drunk, on tranquilizers, is legally blind, and is knowingly driving a truck with shoddy brakes. Miraculously, he does not hit any children, and makes it safely to his next stop. This driver has engaged in extremely *risky* behavior, but he did not commit a tort because he did not cause an injury. The driver, of course, can and should get a ticket, and even go to jail, for reckless driving and driving while intoxicated, just as a company that is responsible for chemical exposures that caused no harm cognizable by the tort system is still subject to civil and criminal penalties. But punishing risky behavior that did not lead to proven harm is the responsibility of the criminal justice system and regulatory agencies, not common law courts acting through the tort system.

Courts that relax the "substantial factor" test in asbestos cases, meanwhile, so that any slight exposure to asbestos fibers satisfies the

⁸⁷ The symptoms and illnesses identified by the expert in question included suicidal depression, mental lapses, pain in gallbladder, loss of sex drive, chronic exhaustion, night sweats, insomnia, flu-like symptoms, mouth ulcers, poor concentration, memory failure, abdominal pain, pain in groin, fluid retention, asthma-like wheezing, frequent urination, unexplained rashes, arthritis, difficulty swallowing, swollen lymph nodes, dry eyes/mouth, shortness of breath, difficulty breathing, crushing chest pain, muscle weakness, gallbladder pain, gallbladder polyps, scleroderma, rheumatic disease, human adjuvant disease, auto-immune disease, connective-tissue disease, emotional breakdown, appetite loss, heart attack symptoms, depression, hypertension, tremors, weight loss, weight gain, joint pain, dizzy spells, hair loss, numbness in limbs and head, burning, tingling, hardening of breast, gastrointestinal problems, urinary tract problems, irritable bowel, sleep disturbances, redness of the palms (palmar erythema), blurred vision, neck pain, fibromyalgia, rheumatoid arthritis, low grade fevers, nausea, tender "points" on body, kidney failure, facial pain, double vision, vertigo, pleurisy, lung pain, migraine headaches, cold sensitivity, multiple sclerosis, small areas of muscle that quiver, twitches, back pain, neck pain, chronic cough, multiple environmental allergies, chronic bronchitis, lupus, Sjogren's disease, heart palpitations, joint inflammation, clumsiness, and morning stiffness. FRANK B. VASEY & JOSH FELDSTEIN, *THE SILICONE BREAST IMPLANT CONTROVERSY: WHAT WOMEN NEED TO KNOW passim* (1993).

⁸⁸ See, e.g., RESTATEMENT (THIRD) OF TORTS § 29 (Proposed Final Draft No. 1, 2005) ("An actor's liability is limited to those physical harms that *result* from the risks that made the actor's conduct tortious.") (emphasis added).

test, also ignore fundamental tort principles. In jurisdictions that allow plaintiffs to pursue actions based on the “any exposure” theory, plaintiffs sue every entity that may have been responsible for any of their asbestos exposure, no matter how minimal the alleged relative or absolute contribution, and then coerce settlements from them all on pains of potentially being held responsible for damages attributable primarily to other entities responsible for much greater asbestos exposure.⁸⁹

By relaxing proximate cause standards to the vanishing point, such courts are essentially, and likely inadvertently, creating broad collective liability for asbestos defendants without due consideration of the consequences. When courts *have* explicitly considered the issue, and carefully weighed the costs and benefits, they have overwhelmingly rejected imposing enterprise, alternative, or market share liability on asbestos defendants.⁹⁰

Putting the special liability issues relating to asbestos to one side, the damage that has been caused by toxic litigation unsupported by reliable scientific evidence is considerable. Consider the case of the morning sickness drug Bendectin, which was (and remains) the only FDA-approved drug to treat nausea and vomiting in pregnancy (NVP). Faced with the expense of thousands of lawsuits alleging that the drug caused birth defects, Bendectin’s manufacturer pulled the drug from the United States.⁹¹ Hospitalization rates for NVP doubled when Bendectin was removed from the market following the litigation scare of the early 1980s.⁹²

⁸⁹ See, e.g., *Chavers v. Gatke Corp.*, 107 Cal. App. 4th 606, 610 (2003) (plaintiffs’ complaint “joined as defendants scores of manufacturers, suppliers and distributors of friction brake products containing asbestos—59 named defendants and 800 ‘Doe’ defendants”); *\$35.1M Awarded to Couple for Exposure to Asbestos in Navy*, MEALEY’S LITIG. REP.: ASBESTOS, Nov. 1, 2007, at 3 (\$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 also Richard B. Schmitt, *Burning Issue: How Plaintiffs’ Lawyers Have Turned Asbestos into a Court Perennial*, WALL ST. J., Mar. 5, 2001, at A1; Susan Warren, *Asbestos Suits Target Makers of Wine, Cars, Soups, Soaps*, WALL ST. J., Apr. 12, 2000, at B1; Susan Warren, *Plaintiffs Target Companies Whose Premises Contained Any Form of Deadly Material*, WALL ST. J., Jan. 27, 2003, at B1.

⁹⁰ See, e.g., *Jackson v. Anchor Packing Co.*, 994 F.2d 1295, 1303 (8th Cir. 1993); *Souder v. Owens-Corning Fiberglas Corp.*, 939 F.2d 647, 650 (8th Cir. 1991); *Robertson v. Allied Signal, Inc.*, 914 F.2d 360, 179-80 (3d Cir. 1990); *White v. Celotex Corp.*, 907 F.2d 104, 106 (9th Cir. 1990); *Blackston v. Shook & Fletcher Insulation Co.*, 764 F.2d 1480, 1483 (11th Cir. 1985); *Perrin v. Owens-Corning Fiberglas Corp.*, 871 F. Supp. 1092, 1096 (N.D. Iowa 1994), *aff’d sub nom.* *Perrin v. Acands*, 68 F.3d 1122 (8th Cir. 1995); *In re Asbestos Litig.*, 509 A.2d 1116, 1118 (Del. Super. Ct. 1986), *aff’d sub nom.* *Nicolet, Inc. v. Nutt*, 525 A.2d 146 (Del. 1987); *Celotex Corp. v. Copeland*, 471 So. 2d 533, 534, 538-39 (Fla. 1985); *Leng v. Celotex Corp.*, 554 N.E.2d 468, 470-71 (Ill. App. Ct. 1990); *Sholtis v. American Cyanamid Co.*, 568 A.2d 1196, 1204 (N.J. Super. Ct. App. Div. 1989); *Black v. Abex Corp.*, 603 N.W.2d 182, 191-92 (N.D. 1999); *Horton v. Harwick Chem. Corp.*, 653 N.E.2d 1196, 1203 (Ohio 1995); *Goldman v. Johns-Manville Sales Corp.*, 514 N.E.2d 691, 696-97, 699-701 (Ohio 1987); *Case v. Fibreboard Corp.*, 743 P.2d 1062, 1067 (Okla. 1987); *Gaulding v. Celotex Corp.*, 772 S.W.2d 66, 69, 70-71 (Tex. 1989).

⁹¹ Bernstein, *Wrong Lessons*, *supra* note 84, at 1965-66, 1968.

⁹² See Melanie Ornstein et al., Editorial, *Bendectin/Diclectin for Morning Sickness: A Canadian Follow-up of an American Tragedy*, 9 REPROD. TOXICOLOGY 1, 2-3 (1995).

Even though there was overwhelming evidence by the late 1980s that Bendectin was safe, and even though its manufacturer eventually won every lawsuit filed against it (at a cost of over \$100 million in direct litigation expenses),⁹³ Bendectin remains off the market in the United States. As a result, “American patients tended to lose, on average, more weight during their NVP, were hospitalized more often than their Canadian counterparts [who can get prescriptions for the generic equivalent of Bendectin] despite similar distribution of the severity of symptoms, and lost more time from paid work.”⁹⁴

Publicity over the litigation claiming that Bendectin caused birth defects led at least seven women to abort their unborn children because they were afraid that their ingestion of Bendectin would lead to birth defects.⁹⁵ Other women needlessly suffered guilt,⁹⁶ falsely believing that their attempt to relieve their morning sickness had caused their children’s birth defects.

Similarly, many women unnecessarily had their breast implants explanted after claims that implants are associated with immune system disease or cancer were circulated in the media by litigants and activist groups.⁹⁷ Others underwent costly, unnecessary, and risky treatments to combat nonexistent implant-related ailments.⁹⁸ As social scientists have explained, warnings of purported risks “may create stress whether the warnings are realistic or not.”⁹⁹

“Phantom risk” litigation over products such as Bendectin and breast implants also inhibits innovation. Unjustified litigation claiming that products such as Bendectin, spermicides, and birth control pills caused birth defects spurred a decline in contraceptive research.¹⁰⁰ Likewise, unjustified lawsuits against vaccines led to a decline in vaccine research.¹⁰¹

⁹³ Bernstein, *The Breast Implant Fiasco*, *supra* note 65, at 461.

⁹⁴ P. Mazzotta et al., *Attitudes, Management and Consequences of Nausea and Vomiting of Pregnancy in the United States and Canada*, 70 INT’L J. GYNECOLOGY & OBSTETRICS 359, 364 (2000).

⁹⁵ Thomas H. Strong, Jr., *Alternative Therapies of Morning Sickness*, 44 CLINICAL OBSTETRICS & GYNECOLOGY 653, 656 (2001).

⁹⁶ See Margaret A. Berger & Aaron D. Twerski, *Uncertainty and Informed Choice: Unmasking Daubert*, 104 MICH. L. REV. 257, 259, 280-81, 289 (2005) (discussing the guilt and emotional distress felt by mothers who had ingested Bendectin and subsequently gave birth to children with birth defects).

⁹⁷ See, e.g., *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 880 (10th Cir. 2005) (plaintiff had her implants removed because she feared that they were causing “silicone-induced lupus”).

⁹⁸ See MARCIA ANGELL, *SCIENCE ON TRIAL: THE CLASH OF MEDICAL EVIDENCE AND THE LAW IN THE BREAST IMPLANT CASE* 147-51 (1996).

⁹⁹ Paul R. Lees-Haley & Richard S. Brown, *Biases in Perception and Reporting Following a Perceived Toxic Exposure*, 75 PERCEPTUAL & MOTOR SKILLS 531, 532 (1992).

¹⁰⁰ Elizabeth B. Connell, *The Cost of Frivolous Lawsuits*, FAMILY PRAC. NEWS, Jan. 15, 2004, at 14, available at <http://www.familypracticenews.com/article/PIIS0300707304712220/fulltext>.

¹⁰¹ See, e.g., Henry I. Miller, Guest Commentary, *Vaccine Shortages*, NAPLES DAILY NEWS, Jan. 9, 2005, available at http://www.naplesnews.com/news/2005/jan/09/ndn_guest_

The economic costs to the companies involved in toxic tort litigation can also be enormous. Dow Corning, a leading breast implant manufacturer and Fortune 500 company, was forced into bankruptcy.¹⁰² Dozens of asbestos defendants have been forced into bankruptcy,¹⁰³ and as their remaining assets have been dissipated, plaintiffs have gone after thousands of solvent defendants with ever-more tenuous or marginal ties to asbestos, resulting in insurance chaos, financial uncertainty, and the loss of jobs.¹⁰⁴

In sum, as tempting as it may be for courts to relieve plaintiffs claiming grievous injuries of their burden of proof against often unsympathetic corporate defendants, it is bad policy as well as bad law. Shifting or lowering the burden of proof, or failing to enforce the rules of evidence, opens up the floodgates to speculative claims invented by plaintiffs' attorneys and their hired experts, unsupported by objective data. The result is a grave risk of driving safe, useful products off the market, stifling innovation, sowing fear and confusion among consumers, and creating massive economic burdens for innocent companies.

CONCLUSION

Settled principles of tort law require that plaintiffs who allege an injury must prove that a defendant caused that injury by a preponderance of the evidence. In the context of toxic tort cases, plaintiffs must prove both general and specific causation. With regard to cases in which plaintiffs can trace their disease to exposure to a particular toxic substance, but were exposed to that substance from many sources, plaintiffs must prove that the actions of one or more individual defendants were a substantial factor in causing the injury.

Unfortunately, plaintiffs have no trouble finding experts who are either professional outliers or hired guns who will draw inferences of causation from the shakiest of evidence. As a result, American courts have been flooded with toxic tort claims that rely on extremely dubious causation theories. Judges have the duty, under both the rules of evidence and tort principles, to police plaintiffs' causation theories to ensure that plaintiffs are satisfying their burden of production.

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¹⁰² Bernstein, *The Breast Implant Fiasco*, *supra* note 65, at 480.

¹⁰³ See RAND Rep., *supra* note 23, at xxvii.

¹⁰⁴ See DEBORAH R. HENSLER ET AL., RAND INST. FOR CIVIL JUSTICE, ASBESTOS LITIGATION IN THE U.S.: A NEW LOOK AT AN OLD ISSUE 8 (2001); Am. Acad. Actuaries, *supra* note 23, at 7-8; Raji Bhagavatula et al., *Asbestos: A Moving Target*, BEST'S REVIEW, Sept. 1, 2001, at 85.