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PROPOSAL FOR LINKING CULPABILITY AND CAUSATION TO ENSURE CORPORATE ACCOUNTABILITY FOR TOXIC RISKS

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Less than a week before this Symposium convened, *The Washington Post* reported that a prominent international drug company had arranged during the late 1990s to test an exceedingly profitable drug in human beings for effectiveness in treating Hepatitis B.¹ Although the initial results were promising, the principle investigator, Hong Kong virologist Dr. Nancy Leung, discovered that patients taking the drug for more than one year became infected by a highly pathogenic mutant virus that appeared to cause liver failure. She reported that the treatment may have resulted in the death of one subject. The company, however, belittled her concerns and continued to publish over her name an “upbeat” scientific abstract that did not mention her concerns.² The company also provided Dr. Leung with slides for a presentation at an international scientific meeting that portrayed her research in a misleading fashion. The Food and Drug Administration (“FDA”) approved the drug in 1998 based upon one-year’s worth of results from research conducted by Leung and others. When an FDA investigator visited Dr. Leung’s laboratory during a pre-approval investigation, the company provided a report that contained no reference to the dead patient that Dr. Leung had mentioned in her original report, an omission that was later discovered only because Dr. Leung’s hospital provided the investigator with an unaltered version of her full report.³

Although it is too early to gauge the FDA’s reaction to this very recent revelation, it is entirely possible, indeed probable, that the drug will remain on the market (perhaps with an additional side-effect warning) and that many cases of fatal liver failure will result from the mutant viruses.

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¹ Deborah Nelson, *Drug Giant’s Spin May Obscure Risk*, WASH. POST, Mar. 18, 2001, at A14.

² See *id.*

³ See *id.*

Should the heirs of one of the victims sue the company responsible for marketing the drug, their attorneys will face an uphill battle in proving that the victim's liver failure was caused by a mutant virus that resulted from the plaintiff's treatment with the defendant's drug. After all, persons suffering from Hepatitis B frequently suffer liver failure, and no one knows for sure whether the mutant virus actually causes liver failure. The most that some future epidemiological study can say with confidence is that it appears that more patients with Hepatitis B who take the drug suffer from liver failure than similarly situated patients who do not take the drug. Depending upon the power of the statistical analysis suggesting an association between the drug and the increased incidence of liver failure, it may be difficult for a plaintiff to find an attorney willing to invest the considerable time and resources required to bring such a lawsuit against a drug company anxious to protect its hundred-million-dollar-a-year product. If attempts to sue the manufacturer therefore fail and if, as is likely, the FDA does not take any punitive action against the company, then the company will have avoided all responsibility for reprehensible conduct that at the very least put patients at risk. The company might be held accountable in corporate heaven, but not down here on earth.

Let me begin with a controversial assessment and a debatable prediction, neither of which will I attempt to support empirically in this article.⁴ My assessment of the past twenty years of developing toxic tort law is that any realistic threat of a "liability crisis"⁵ ended years ago with the widespread adoption of tort reform legislation in state legislatures, careful screening by White House and Justice Department officials of prospective federal judges for their views on "judicial activism" with respect to judicially imposed constraints on business enterprises,⁶ and the pervasive airing of tort liability "horror stories" in the media. Highly publicized, but largely unwarranted claims that the common law courts and federal regulatory agencies (the two primary governmental institutions for ensuring corporate accountability for health and environmental risks)

⁴ The task of providing an empirical basis for the assumptions made in this article is the task of a larger work in progress to which future events will provide additional support.

⁵ For assertions that a "liability crisis" was overwhelming corporate America and the insurance industry, see MICHAEL J. MOORE & W. KIP VISCUSI, *COMPENSATION MECHANISMS FOR JOB RISKS* 136 (Princeton Univ. Press 1990) (alluding to the "emerging product liability crisis"); see also George L. Priest, *The Current Insurance Crisis and Modern Tort Law*, 96 YALE L.J. 1521 (1987).

⁶ See SHARON BUCCINO ET AL., *HOSTILE ENVIRONMENT: HOW ACTIVIST JUDGES THREATEN OUR AIR, WATER, AND LAND* (2001).

were relying too heavily upon “junk science” brought about changes in both of those institutions that have caused them to be far more cautious about regulating and imposing liability upon business entities. This reticence on the part of existing constraining institutions came at a time of booming economic expansion and rapid technological developments (especially in the areas of pharmaceutical and agricultural technologies) that had a significant potential to do great harm to human health and the environment. My prediction is that these developments will precipitate an “accountability crisis” that will begin to intrude upon the public consciousness in the coming years as stories of unpunished corporate malfeasance, like the one above, appear in the media on a regular basis.⁷

If my assessment and prediction are wrong, then the law of toxic torts as administered by the federal courts and most state courts is moving in roughly the right direction. Stiff limitations on the admissibility of scientific evidence in toxic torts lawsuits were badly needed to keep the liability crisis from overwhelming vital American producers and to keep American companies competitive in a robust global economy. The courts must continue to demand that scientific expert testimony meets clear threshold demonstrations of scientific validity and proper litigative fit to put the world on notice that judges will not tolerate attempts by uninjured plaintiffs and greedy trial lawyers to use the tort regime to punish corporations for conduct that federal and state regulatory agencies are fully capable of addressing.

In short, the reader who is convinced that the *Daubert* line of cases in the Supreme Court brought much needed balance to the law of toxic torts, whether legitimately or not, may stop reading here. This article does not challenge *Daubert*'s legitimacy. Others have persuasively argued that the Supreme Court, in the *Daubert* line of cases, displayed a woeful misunderstanding of science and scientific values, subtly usurped substantive state tort law in derogation of the *Erie* doctrine, and snatched away the jury's fact-finding power in full view of the Seventh Amendment to the United States Constitution.⁸ My criticism of the *Daubert* line of

⁷ Others have concluded that the existing tort and regulatory regimes are failing. See, e.g., Margaret A. Berger, *Eliminating General Causation: Notes Towards a New Theory of Justice and Toxic Torts*, 97 COLUM. L. REV. 2117 (1997) (“Under our current system, the process of guarding against risk and unraveling uncertainty is impeded because corporations often disregard the development and dissemination of critical information despite the supposed inducements provided by both regulation and toxic tort litigation.”).

⁸ See generally *id.* (arguing that the substantive law governing toxic torts should be recast); Erica Beecher-Monas, *A Ray of Light for Judges Blinded by Science: Triers of*

cases is that its comforting message to corporate America that companies will not be held liable in tort for damages they did not clearly cause will be heard by at least some companies as an invitation to press the limits of corporate responsibility. The recently exposed tobacco documents reveal with startling clarity how potential toxic tort defendants can "bend science" to meet their litigative and public relations needs. That capacity and the general inability of resource-strained regulatory agencies to uncover and punish illegitimate attempts to manipulate the regulatory process will combine to produce an accountability crisis that will ultimately precipitate strong political demands to change the system.

This article offers, perhaps somewhat prematurely, a partial solution to the impending accountability crisis that combines the institutional strengths of courts and regulatory agencies while at the same time sending a message to American companies that irresponsible manipulation of scientific information in the regulatory process will not go unnoticed and unpunished. This article proposes a combined administrative/litigative approach that links causation to culpability by applying various culpability-based presumptions to the conclusions of a federal agency charged with assessing scientific evidence that is reasonably reliable in the agency's policy-informed exercise of scientific judgment.

The political momentum appears at the moment to be moving rather powerfully in the opposite direction. But the momentum will shift in the not-too-distant future as we realize that a global economy with few effective health and environmental protections is a frightening place. If I am correct in predicting that an impending accountability crisis will provide a political setting in which far-reaching changes in the existing tort regime are legitimately on the table, the relatively modest changes suggested here may forestall more aggressive changes aimed at shifting the burden of proof altogether,⁹ or replacing the common law with a full-fledged administrative reparations regime.¹⁰

Science and Intellectual Due Process, 33 GA. L. REV. 1047 (1999) (focusing on the troubling scientific issues and discussing the failures of *Daubert*); Lucinda M. Finley, *Guarding the Gate to the Courthouse: How Trial Judges are Using Their Evidentiary Screening Role to Remake Tort Causation Rules*, 49 DEPAUL L. REV. 335 (1999) (exploring substantive law implications in the wake of *Daubert*); Michael A. Gottesman, *From Barefoot to Daubert to Joiner: Triple Play or Double Error*, 40 ARIZ. L. REV. 753 (1998) (tracing the Supreme Court's "erratic course" in addressing whether federal judges may bar scientific testimony).

⁹ See Berger, *supra* note 7.

¹⁰ See Richard A. Nagareda, *Future Mass Tort Claims and the Rule-Making/Adjudication Distinction*, 74 TUL. L. REV. 1781 (2000) (alluding to "an emerging consensus, as

I. CAUSATION AS A HURDLE IN TOXIC TORT LITIGATION

Fifteen years ago, as the phrase "toxic tort" was first entering the trial lawyers' lexicon, they were optimistic that the common law would prove an effective vehicle for compensating persons injured by exposure to toxic substances, for exacting retribution against those who callously exposed innocent people to toxic risks, and for sending a message to companies that pollution had a steep price that could be avoided only by limiting polluting activities.¹¹ One of the earliest practitioners of the newly emerging discipline predicted that "[c]hanges in laws to lessen the victim's causation burden are making it easier for plaintiffs to recover in toxic-tort cases."¹² Another pioneer of toxic tort litigation argued that judicial recognition of claims based upon the new science of "clinical

manifested by the actual behavior of attorneys on the front lines of mass tort litigation, as to the desirability of developing private administrative systems for the handling of future mass tort claims").

¹¹ I mean to incorporate within the rubric of "toxic torts" claims for compensation from persons who allege that they have been injured by virtue of an exposure to a substance or product for which the defendant is responsible. I acknowledge that this includes a large number of claims that may have little in common beyond the critical element of exposure to an allegedly toxic substance. See E. Donald Elliott, *Causation and Financial Compensation: Goal Analysis Versus Institutional Analysis of Toxic Compensation Systems*, 73 GEO. L.J. 1357, 1369 (1985) (noting that the "toxics problem" is not a single problem, but many problems that may have little in common). The institutional solution should be broadly applicable to a wide range of toxic torts involving exposures to products, drugs, and environmental contaminants. Nonetheless, it will probably not be necessary to address acute toxicity easily attributable to a single source (e.g., Bhopal), and it will not be useful in some contexts, such as the environmental contaminant that originates from thousands of sources. See *id.* at 1370-71. The discussion here is aimed primarily at the middle range of toxic exposures in which the primary evidence of cause-in-fact comes from epidemiological studies and animal studies.

¹² Paul D. Rheingold, *New Frontiers in Causation and Damages*, TRIAL, Oct. 1986, at 42-44. Rheingold mentioned recent holdings allowing claims for enhanced risk, mental distress resulting from fear of contracting disease, bodily changes, medical monitoring, and property damage as examples of courts reducing causation barriers. *Id.* In subsequent years, the courts have not been at all receptive to claims for increased risk or for mental distress unaccompanied by any physical injury caused by the toxic insult. Plaintiffs have successfully pursued medical monitoring claims in a few cases, and claims for property damage directly caused by polluting activities have also met with some success. Overall, however, the anticipated changes have not come about.

ecology" would "substantially increase opportunities for victims to recover for their injuries."¹³

Time has not borne out those rosy projections. The courts have repeatedly rejected attempts to sidestep the traditional "but for" cause-in-fact showing in toxic tort litigation. In retrospect, causation has proven a very effective stumbling block that has not only precluded compensation for all but the most clearly understood environmentally caused diseases, but has also stood in the way of ambitious attempts to protect the public health generally through toxic tort litigation. Jonathan Haar's widely read account of the *Woburn* litigation painted a stark, but accurate picture of the enormous difficulties that a plaintiff's attorney faces in attempting to recover damages for physical harm caused by exposure to environmental contaminants.¹⁴

As plaintiff's lawyers moved beyond mesothelioma and other "signature diseases" for which exposure to a single substance was clearly the dominant among possible causes, potential defendants and their trade associations began to worry in a serious way about the potential that the common law of torts had to effect profound changes in the way they did business.¹⁵ Business groups and conservative foundations created "scores of coalitions and task forces,"¹⁶ including the American Tort Reform Association, and funded academic research aimed at limiting "lawsuit abuse."¹⁷ Manhattan Institute Fellow Peter Huber wrote a well-publicized polemic entitled "Galileo's Revenge," in which he argued that plaintiffs' claims of causal association between exposures to toxic substances and

¹³ Anthony Z. Roisman, *Proving Cause in Toxic-Tort Litigation*, TRIAL, Oct. 1986, at 59-60.

¹⁴ See JONATHAN HAAR, *A CIVIL ACTION* (First Vintage Books 1996); see also Joseph Sanders, *The Bendectin Litigation: A Case Study in the Life Cycle of Mass Torts*, 43 HASTINGS L.J. 301 (1992).

¹⁵ See Heidi Li Feldman, *Science and Uncertainty in Mass Exposure Litigation*, 74 TEX. L. REV. 1 (1995).

¹⁶ Maxwell Glen, *Congress Joins the Hue and Cry Over Liability Insurance Crisis*, 18 NAT'L J. 380 (1986).

¹⁷ See Burt Solomon, *Finger-Pointing Distinguishes Attempts To Fix Blame for Liability Crisis*, 18 NAT'L J. 378 (1986) (identifying the American Tort Reform Association as "a coalition of policyholders assembled last month to lobby for changes in tort law"). The American Tort Reform Association gradually evolved into a fully funded American Tort Reform Foundation. See Letter from Sherman Joyce, American Tort Reform Foundation, to "Professor" (Dec. 11, 1998) (on file with author).

disease were frequently based upon “junk science.”¹⁸ Although the empirical basis for these criticisms was never convincingly established,¹⁹ Vice President Dan Quayle’s Council on Competitiveness drafted fifty proposals for reforming the civil justice system, six of which related to tightening judicial scrutiny of scientific expert testimony.²⁰ The overtly political purpose of the proposed reforms was to enhance the

¹⁸ PETER HUBER, *GALILEO’S REVENGE: JUNK SCIENCE IN THE COURTROOM* (1991); see W. John Moore, *Free-Lance Critic Hits Shackles of Regulation*, NAT’L J. 2797 (1986) (counsel for Products Liability Alliance refers to Huber’s work as “the intellectual underpinning” of the tort reform effort of the mid-1980s).

¹⁹ Professor Huber’s mostly anecdotal accounts were heavily relied upon by the tort reformers, but were harshly reviewed by academics and practitioners. See Robert F. Blomquist, *Science, Toxic Tort Law, and Expert Evidence: A Reaction to Peter Huber*, 44 ARK. L. REV. 629 (1991); Kenneth J. Chesebro, *Peter Huber’s Junk Scholarship*, 42 AM. U. L. REV. 1637 (1993); John F. Duffy, *The FCC and the Patent System: Progressive Ideals, Jacksonian Realism, and the Technology of Regulation*, 71 U. COLO. L. REV. 1071, 1076-77 (2000) (contrasting Huber’s praise of the common law with Huber’s criticism of the common law); Jeff L. Lewin, *Calabresi’s Revenge? Junk Science in the Work of Peter Huber*, 21 HOFSTRA L. REV. 183, 186 (1993) (book review).

Reacting to the widespread criticism of the way courts and juries were treating scientific evidence, the Carnegie Commission on Science, Technology and Government conducted a multi-year examination of that topic through a task force that included prominent members of the scientific community, as well as the bench and bar. CARNEGIE COMMISSION ON SCIENCE, TECHNOLOGY, AND GOVERNMENT, *SCIENCE AND TECHNOLOGY IN JUDICIAL DECISIONMAKING: CREATING OPPORTUNITIES AND MEETING CHALLENGES* (1993) [hereinafter CARNEGIE COMMISSION REPORT ON JUDICIAL DECISIONMAKING]. The task force’s report, published just before the Supreme Court issued its *Daubert* opinion, took a much less pessimistic view of the ability of courts and juries to cope with the complex scientific issues that arise in toxic tort litigation. It pointed out that “many of the criticisms directed at the operation of our court system arise—quite understandably—from misperceptions about the differing methodologies and goals of science and law, and from the consequent failure to comprehend the diverse roles and expertise of ‘judge,’ ‘juror,’ and ‘scientist.’” *Id.* at 12. While the Carnegie Commission may have been correct in concluding that the existing court system was not broken, this Article takes the position that the federal judiciary’s rather extreme response to the criticism, signaled by *Daubert/Joiner*, has contributed to a crisis of accountability that does call for institutional change. See generally *id.*

²⁰ PRESIDENT’S COUNCIL ON COMPETITIVENESS, *AGENDA FOR CIVIL JUSTICE REFORM IN AMERICA* 11, 21-22 (1991), reprinted in Dan Quayle, *Agenda for Civil Justice Reform in America*, 60 U. CIN. L. REV. 979 (1992). The Vice President’s tort reform initiative relied heavily upon Huber’s analysis of the failings of the tort regime. See Chesebro, *supra* note 19, at 1645.

competitiveness of U.S. companies in global markets by relieving them from the threat of expensive and occasionally ruinous civil liability.²¹

The *Daubert* case gave the Supreme Court an opportunity to examine the “junk science” issue in a critical legal context—determining the admissibility of expert testimony.²² Because nearly all causation claims in toxic tort litigation necessarily rest on expert testimony,²³ one way to ensure the validity of such claims and, incidentally, to reduce the incidence of successful lawsuits is to raise the bar on admissibility of expert testimony. Unlike the Quayle Commission’s tort reform initiative, which precipitated a loud political debate and ultimately failed,²⁴ the Supreme Court could stem the daunting flow of resource-intensive toxic tort lawsuits through a politically invisible interpretation of the words “scientific” and “knowledge” in the obscure Federal Rules of Evidence. Many observers believe that this is exactly what the Court had in mind in the *Daubert* line of cases.²⁵

Prior to *Daubert*, defendants had in several cases successfully persuaded courts to raise the bar of admissibility to the *Frye* “general acceptance in the scientific community” standard.²⁶ The Supreme Court in *Daubert* declined to employ that test, but it nevertheless raised the bar by interpreting Rule 104(a) of the Federal Rules of Evidence to require the district judge to perform a “gatekeeper” role in determining the admissibility of “scientific, technical, or other specialized knowledge”

²¹ See Dan Quayle, *Protecting America's Greatness*, WASH. POST WKLY. REP., Dec. 16-22, 1991, at 29. See also MICHAEL D. GREEN, BENEDICTIN AND BIRTH DEFECTS 19-22 (1996).

²² *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579 (1993). See Joseph Sanders, *Scientific Validity, Admissibility, and Mass Torts After Daubert*, 78 MINN. L. REV. 1387, 1389 (1994) (suggesting that the “emerging belief that an increase in ‘junk science’ in the courtroom requires greater judicial vigilance in admitting expert opinion” fueled a sense of emergency that persuaded the Supreme Court to examine the role of expert testimony in toxic torts cases twenty years after the Federal Rules of Evidence had been adopted).

²³ See Beecher-Monas, *supra* note 8, at 1063.

²⁴ See, e.g., Marc Galanter, *Bumblng on Billions*, AM. LAW., Apr. 1992, at 86; see also Quayle's *Crusade*, 17 NAT'L J. 344 (1992); Owen Ullmann, *President Quayle*, WASHINGTONIAN, Sept. 1992, at 68.

²⁵ See Finley, *supra* note 8, at 335, pt. III; see also Gottesman, *supra* note 8, at 756-59.

²⁶ *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923). See Peter A. Bell, *Strict Scrutiny of Scientific Evidence: A Bad Idea Whose Time Has Come*, BNA SAFETY AND LIAB. REP. 47 (1992). See, e.g., *Christophersen v. Allied Signal Corp.*, 939 F.2d 1106 (5th Cir. 1991) (noting that the court affirmed grant of defendant's motion of summary judgment on ground that plaintiff's causation expert not admissible).

under Rule 702.²⁷ Henceforth, the trial judge would have to determine whether offered expert testimony was “relevant and reliable” before allowing the jury to consider that testimony, and the reliability of scientific proof was to be determined by reference to its “scientific validity” when measured against the methods and procedures of science.²⁸ Although the Court gave the district judges some guidance,²⁹ the ultimate test remained quite subjective.

By rejecting the *Frye* rule in civil cases, the *Daubert* opinion could have reversed a growing trend in the lower courts toward strict judicial scrutiny of expert testimony in toxic tort cases.³⁰ Many early observers believed, however, that *Daubert* invited the lower courts to play an even more aggressive role in evaluating the “scientific validity” of expert testimony in cases involving toxic causation.³¹ The terse and ambiguous guidance that the Court provided could have allowed the law to evolve in either direction.³² But the Court’s elaboration on the *Daubert* criteria in *General Electric Co. v. Joiner*,³³ where it clarified the trial judge’s role of

²⁷ *Daubert*, 509 U.S. 579.

²⁸ *Id.* at 590; see also sources cited *supra* note 8.

²⁹ The Court suggested several factors that should bear on the trial judge’s inquiry. First, the judge should ask whether the scientific proposition can be tested through scientific methods. Second, the judge should consider whether the evidence has been subject to peer review. Third, the judge should take into account the “known or potential rate of error, and the existence and maintenance of standards controlling the technique’s operation.” Finally, the court should consider the extent to which the information is accepted in the scientific community. *Daubert*, 509 U.S. at 593-95.

³⁰ Some early commentators were convinced that *Daubert* would result in greater opportunities for juries to evaluate scientific evidence in toxic tort cases. See, e.g., Anthony Z. Roisman, *Conflict Resolution in the Courts: The Role of Science*, 15 CARDOZO L. REV. 1945 (1994); see also Kenneth Chesebro, *Taking Daubert’s “Focus” Seriously: The Methodology/Conclusion Distinction*, 15 CARDOZO L. REV. 1745 (1994).

³¹ See, e.g., David E. Bernstein, *The Admissibility of Scientific Evidence after Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 15 CARDOZO L. REV. 2139 (1994); see also Robert F. Blomquist, *The Dangers of “General Observations” on Expert Scientific Testimony: A Comment on Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 82 KY. L.J. 703, 719 (1993-94).

³² David L. Faigman, *The Law’s Scientific Revolution: Reflections and Ruminations on the Law’s Use of Experts in Year Seven of the Revolution*, 57 WASH. & LEE L. REV. 661, 663-64 (2000) (noting that “*Daubert* contained considerable language to support the view that more, rather than less, expert testimony might be the result of the opinion”). For critiques of the Court’s guidance in *Daubert* and *Joiner*, see Beecher-Monas, *supra* note 8, at 1051-62; and Sanders, *supra* note 22, at 1391-92.

³³ 522 U.S. 136 (1997).

ensuring that the scientific testimony "fit" the judge's view of the relevant issues of the case, had the foreseeable affect of nudging the lower courts toward the strict scrutiny end of the spectrum.³⁴ Dicta in the *Joiner* opinion suggesting that the trial court was obliged to evaluate the "scientific validity" of an expert's *conclusions*, as well as the data and methodology that the expert employed, accelerated an already existing trend in the lower courts toward aggressive judicial scrutiny of plaintiffs' expert testimony.³⁵ Finally, Rule 702 has recently been amended to incorporate the *Daubert/Joiner* tests.³⁶

It is now clear after almost a decade's experience with *Daubert* that the lower courts have applied it quite vigorously to exclude expert testimony.³⁷ Because the plaintiff ordinarily has the burden of proof in tort litigation, this aggressive invocation of the judge's new role as guardian of the purity of scientific evidence has had a disproportionate impact on plaintiffs. With remarkable speed, judges have gone far beyond throwing the clinical ecologists out of the courtroom.³⁸ They are now excluding the

³⁴ The actual holding of *Joiner* was that the courts of appeals should not review district court applications of *Daubert* to exclude scientific testimony with any greater scrutiny than other evidentiary rulings.

³⁵ *Joiner*, 522 U.S. at 146. See also D. Alan Rudlin, *The Judge as Gatekeeper: What Has Daubert-Joiner-Kumho Wrought?*, 29 BNA PROD. SAFETY & LIAB. REP. 329 (2001) (arguing that as a result of *Daubert/Joiner/Kumho* federal trial judges now "play an active role in deciding what expert testimony goes to the jury"). Professor Finley reads *Joiner* to express "a normative judgment that judges are to be trusted more than juries (and sometimes more than scientists) in areas where law intersects with science." Finley, *supra* note 8, at 345.

³⁶ FED. R. EVID. 702. The Advisory Committee Notes state that Rule 702 was amended "in response to" *Daubert* and "to the many cases applying *Daubert*." *Id.* advisory committee's note.

³⁷ Jeffrey D. Cutler, *Implications of Strict Scrutiny of Scientific Evidence: Does Daubert Deal a Death Blow to Toxic Tort Plaintiffs?* 10 J. ENVTL. L. & LITIG. 189, 214 (1995) ("[I]t doesn't take a rocket scientist to figure out that a four or five part test including 'general acceptance' as one factor will be more difficult to meet than a test based on 'general acceptance' alone"); see also Faigman, *supra* note 32, at 664 (concluding that "[e]xperience soon showed, however, that *Daubert* had the effect of excluding a lot of evidence that had been admitted previously"); Finley, *supra* note 8, at 341-42; Sanders, *supra* note 14, at 391.

³⁸ In the mid-1980s, the "science" of "clinical ecology" appeared to be the answer to the causation conundrum for plaintiffs' attorneys. Professor Elliott described the phenomenon as follows:

For a price, some clinical ecologists will testify that exposure to even very small amounts of a wide range of chemicals suppresses the immune system, thereby weakening the body's ability to ward off

testimony of well-regarded experts based upon smoke screens thrown up by artful defense counsel.³⁹ A plaintiff's attorney must come to court prepared not only to establish the expert's qualifications, but also to demonstrate to a skeptical trial judge that the testimony forms scientifically reliable conclusions based upon reliable data and that those conclusions "fit" the legal requirements for establishing cause-in-fact.⁴⁰ If the plaintiff's attorney fails, everyone goes home and no one knows whether the plaintiff was a victim of cruel fate or the defendant's possibly unconscionable conduct. If the plaintiff's attorney succeeds, the judge and a jury must sit through days of confusing and conflicting expert testimony at the end of which the jury may still decide that the plaintiff's attorney did not sustain the plaintiff's burden of proof.

The analysis that follows of the burdens plaintiffs' attorneys face in establishing cause-in-fact in all but the very rare cases of "signature diseases" should demonstrate that the common law, as currently implemented by trial judges in a post-*Daubert/Joiner* world, is incapable of providing adequate compensation to those injured by toxic products and environmental contaminants. Consequently, the tort reparations regime is incapable of punishing culpable behavior or of creating adequate incentives to protect potential victims of toxic risks in the future. The causation hurdle as presently administered allows far too many "blameworthy, but fortunate" defendants, in Richard Nagareda's memorable phrase,⁴¹ to avoid both punishment and their social obligation to compensate the victims of their culpable conduct.

disease. This weakening, in turn, allegedly makes the plaintiff vulnerable to virtually all diseases known to humankind, including 'nervousness,' 'malaise,' and other conditions that present only subjective symptoms.

E. Donald Elliott, *Toward Incentive-Based Procedure: Three Approaches for Regulating Scientific Evidence*, 69 B.U. L. REV. 487 (1989). See also Peter Huber, Comment, *A Comment On Toward Incentive-Based Procedure: Three Approaches For Regulating Scientific Evidence By E. Donald Elliott*, 69 B.U. L. REV. 513, 515 (1989).

³⁹ For an excellent example, see Professor Beecher-Monas' thorough and devastating critique of the Eighth Circuit's post-*Daubert* opinion in *Wright v. Willamette Industries, Inc.*, 91 F.3d 1105 (8th Cir. 1996). Erica Beecher-Monas, *The Heuristics of Intellectual Due Process: A Primer for Triers of Science*, 75 N.Y.U. L. REV. 1563, 1637 (2000).

⁴⁰ See Harvey Brown, *Eight Gates For Expert Witnesses*, 36 HOUS. L. REV. 743 (1999) (detailing eight "gates" through which the proponent of expert testimony must navigate in order to demonstrate that the testimony is admissible).

⁴¹ Richard A. Nagareda, *Outrageous Fortune and the Criminalization of Mass Torts*, 96 MICH. L. REV. 1121 (1998).

A. *Proof of Causation in Toxic Torts Cases After Daubert/Joiner*

The plaintiff in a common law toxic tort action must plead and prove by a preponderance of the evidence that the defendant was responsible for his or her exposure to a toxic substance and that the exposure was a cause-in-fact of damage to the plaintiff.⁴² Although plaintiffs have attempted to finesse the causation issue by attempting to recover for risk alone or by alleging unique forms of damage, like mental distress, increased need for medical monitoring, and immune system disruption, the courts have generally been hesitant to accept such novel claims.⁴³ The essential toxic tort claim remains an action for physical disease or injury caused by the

⁴² W. PAGE KEETON ET AL., PROSSER AND KEETON ON THE LAW OF TORTS § 41, at 263 (5th ed. 1984); see also Shelly Brinker, Comment, *Opening the Door to the Indeterminate Plaintiff: An Analysis of the Causation Barriers Facing Environmental Toxic Tort Plaintiffs*, 46 UCLA L. REV. 1289, 1302 (1999). Two possible exceptions to the requirement that a plaintiff establish a cause-in-fact relationship between exposure to a defendant's toxic substance and a particular damage are toxic batteries and toxic trespasses. For both of these claims, damage is generally not a necessary element. Most courts do not recognize a toxic trespass claim for pollution, but instead require plaintiffs to bring their claims in nuisance. In *Bradley v. American Smelting and Refining Co.*, 709 P.2d 782 (Wash. 1985), the court recognized a toxic trespass claim, but required the plaintiffs to show damage.

⁴³ See *Metro-North Commuter Railroad v. Buckley*, 521 U.S. 424, 432 (1997) (observing that "[w]ith only a few exceptions, common-law courts have denied recovery to those who . . . are disease and symptom free"); see also Troyen A. Brennan, *Environmental Torts*, 46 VAND. L. REV. 1, 64 (1993) (courts slow to adopt recovery for risk); Andrew R. Klein, *A Model for Enhanced Risk Recovery in Tort*, 56 WASH. & LEE L. REV. 1173 (1999) (Courts "have not favored enhanced risk claims, and have placed significant barriers in front of those who have filed such actions."); Nagareda, *supra* note 10, at 1797 (noting that "[m]ost [jurisdictions] . . . do not permit exposed-but-as-yet-unimpaired persons to sue").

The one innovation that appears to have met with some success is recovery of medical monitoring expenses. See, e.g., *Merry v. Westinghouse Elec. Corp.*, 684 F. Supp. 847 (M.D. Pa. 1988); *Ayers v. Township of Jackson*, 525 A.2d 287 (N.J. 1987). See generally Brennan, *supra*, at 68 (collecting cases).

A few courts have allowed recovery when the plaintiff can prove to a "reasonable medical certainty" that future harm will result from a past exposure, especially when causation has been established for other physical injuries. See, e.g., *Sterling v. Velsicol Chem. Corp.*, 855 F.2d 1188 (6th Cir. 1988); *Jackson v. Johns-Manville Sales Corp.*, 781 F.2d 394 (5th Cir.), *cert. denied*, 478 U.S. 1022 (1986); see also *Gideon v. Johns-Manville Sales Corp.*, 761 F.2d 1129 (5th Cir. 1985); *Eagle-Picher Indus., Inc. v. Cox*, 481 So. 2d 517, 519-20 (Fla. Dist. Ct. App. 1985). *Daubert* has unquestionably had an impact on this theory, however, as it becomes more difficult for the plaintiffs to qualify an expert who is prepared to testify "to a reasonable medical" certainty that the plaintiff will suffer a disease in the future.

plaintiff's exposure to a substance for which the defendant was legally responsible.

The plaintiff must establish both "general causation" and "specific causation."⁴⁴ To establish general causation, the plaintiff must prove that human exposure to the toxic agent at issue is capable of causing or exacerbating an identifiable disease from which the plaintiff suffers. To establish specific causation, the plaintiff must prove that his or her exposure to the toxic material caused the plaintiff's particular affliction.⁴⁵ The first showing generally requires scientific data in the nature of epidemiological studies demonstrating a statistical association between exposure to the substance and an increase in the incidence of the plaintiff's disease.⁴⁶ The second showing typically requires direct or expert testimony regarding the extent to which the plaintiff was exposed to the toxic agent at issue, and some courts require additional expert testimony to the effect that the particular plaintiff's particular affliction was more likely than not caused by the plaintiff's exposure to the substance at issue.⁴⁷

1. A Brief Primer on Epidemiology

When regulatory agencies are deciding how stringently they will regulate human exposure to toxic agents, they first attempt to assess the risks posed by the agent. Risk assessment is an analytical process that "uses available scientific information on the properties of an agent and its effects in biological systems to provide an evaluation of the potential for harm as a consequence of environmental exposure to the agent."⁴⁸ Risk assessment must be distinguished from "risk management," which consists of the actions that individuals and regulatory agencies take to reduce or

⁴⁴ See *In re "Agent Orange" Prod. Liab. Litig.*, 597 F. Supp. 740, 835 (E.D.N.Y. 1984); see also Klein, *supra* note 43, at 1196-97; Nagareda, *supra* note 41, at 1130; Richard A. Nagareda, *Turning from Tort to Administration*, 94 MICH. L. REV. 899, 910 (1996).

⁴⁵ See Nagareda, *supra* note 10, at 1787 (observing that "the defense may properly contest the existence of specific causation (whether the particular plaintiff's disease was caused by the defendant's product), even when general causation (the proposition that the product is capable of causing the disease in humans) has been established").

⁴⁶ See Cutler, *supra* note 37, at 200-01.

⁴⁷ See *In re "Agent Orange" Prod. Liab. Litig.*, 597 F. Supp. 740 (alluding to the "strong" and "weak" versions of the preponderance of the evidence rule).

⁴⁸ Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17,960, 17,963 (Apr. 23, 1996).

eliminate the risks that human beings encounter.⁴⁹ As currently conceived, risk assessment itself consists of four more or less discrete exercises: hazard assessment; dose response assessment; exposure assessment; and risk characterization.⁵⁰ Of these functions, hazard identification is the most relevant to toxic tort litigation.

Hazard assessment is "the process of determining whether exposure to an agent can cause an increase in the incidence of a health condition."⁵¹ Obviously, this function is quite similar, if not identical, to the general causation determination in tort litigation. Human health hazard assessment typically relies upon one or more of four different kinds of information: (1) controlled human experiments; (2) epidemiological studies; (3) animal testing studies; and (4) various studies of chemical structure, reactivity, mutagenicity, and DNA damage and repair.⁵²

Ordinarily, controlled human tests are the best evidence of toxicity to humans.⁵³ In the case of carcinogenicity, which may take decades to manifest itself and is often fatal, ethical considerations preclude direct human testing.⁵⁴ In the case of reproductive and developmental toxicity, human testing is inappropriate because of serious ethical questions

⁴⁹ See COMMITTEE ON THE INSTITUTIONAL MEANS FOR ASSESSMENT OF RISKS TO PUBLIC HEALTH, NATIONAL ACADEMY OF SCIENCES, *RISK ASSESSMENT IN THE FEDERAL GOVERNMENT: MANAGING THE PROCESS* 18-19 (1983) [hereinafter *NAS RED BOOK*] (stating that:

[r]isk management, which is carried out by regulatory agencies under various legislative mandates, is an agency decision-making process that entails consideration of political, social, economic, and engineering information with risk-related information to develop, analyze, and compare regulatory options and to select the appropriate regulatory response to a potential chronic health hazard).

⁵⁰ *Id.* at 3.

⁵¹ See *id.* at 19.

⁵² See *id.* at 20 ("Four general classes of information may be used in this step: epidemiologic data, animal-bioassay data, data on in vitro effects, and comparisons of molecular structure."); see also Scientific Bases for Identification of Potential Carcinogens and Estimation of Risks: Report of the Work Group on Risk Assessment, 44 Fed. Reg. 39,858 (June 26, 1979).

⁵³ Guidelines for Reproductive Toxicity Risk Assessment, 61 Fed. Reg. 56,274, 56,309 (Oct. 31, 1996); Guidelines for Developmental Toxicity Risk Assessment, 56 Fed. Reg. 63,798, 63,809 (Dec. 5, 1991).

⁵⁴ *NAS RED BOOK*, *supra* note 49, at 12 (stating "[e]thical considerations prevent deliberate human experimentation with potentially dangerous chemicals").

concerning the ability of the subject to consent to the testing.⁵⁵ Thus, as a practical matter, cost and ethical considerations severely limit the extent to which controlled human studies are available for determining the toxicity of potentially toxic agents.⁵⁶

The next best evidence for use in hazard assessment comes from epidemiological studies of human exposures that occur outside the context of a controlled experiment.⁵⁷ Epidemiology is a relatively new science, and epidemiologists have only within the last quarter-century developed consistent definitions and prescribed evaluative criteria for epidemiological studies.⁵⁸ An epidemiological study consists of the statistical comparison of human beings who have received a higher than normal exposure to a particular agent with humans who have received little or no exposure.⁵⁹ The two broad types of epidemiological studies are "cohort" studies and "case-control" studies. In cohort studies, groups of individuals who have received high exposures to the substance being studied are identified and compared to groups of similarly situated individuals with low exposures to determine differences, if any, in the occurrence of particular diseases.⁶⁰ Cohort studies can be conducted prospectively by identifying the two cohorts in advance of the exposure and following the groups through time or retrospectively through the use of historical records.⁶¹ In case-control studies, groups of similarly situated individuals with and without a particular disease are compared to determine the extent of any differences in exposure to the substance being

⁵⁵ See Lainie Friedman Ross, *Children as Research Subjects: A Proposal to Revise the Current Federal Regulations Using a Moral Framework*, 8 STAN. L. & POL'Y REV. 159 (1997); see also Michael J. Loscialpo, Note, *Nontherapeutic Human Research Experiments on Institutionalized Mentally Retarded Children: Civil Rights and Remedies*, 23 NEW ENG. J. ON CRIM. & CIV. CONFINEMENT 139 (1997).

⁵⁶ KENNETH J. ROTHMAN & SANDER GREENLAND, *MODERN EPIDEMIOLOGY* 72 (2d ed. 1998).

⁵⁷ Guidelines for Reproductive Toxicity Risk Assessment, 61 Fed. Reg. at 56,297.

⁵⁸ ROTHMAN & GREENLAND, *supra* note 56, at 4.

⁵⁹ Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17,960, 17,972 (Apr. 23, 1996) ("The goals of cancer epidemiology are to identify differences in cancer risk between different groups in a population or between different populations, and then to determine the extent to which these differences in risk can be attributed causally to specific exposures to exogenous or endogenous factors.").

⁶⁰ ROTHMAN & GREENLAND, *supra* note 56, at ch. 6; see also Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17,973.

⁶¹ *Id.* at 17,973.

studied.⁶² If the group with the disease has a higher degree of exposure, a statistical association between the exposure and the disease may exist.

The statistical power of an epidemiological study to support conclusions about cause-effect relationships depends upon four factors: (1) the size of the study group; (2) the level and duration of exposure; (3) the frequency of the relevant disease outcome in the general population; and (4) the level of excess risk to be identified.⁶³ The confidence with which one may draw conclusions from studies in which no adverse effects are detected depends entirely on the power of the study to detect such effects.⁶⁴ Unfortunately, cost considerations generally yield epidemiological studies that are not especially powerful. Therefore, a negative epidemiological study rarely warrants a strong conclusion that there is no cause-effect relationship between human exposure to a substance and disease.⁶⁵

In recent years, scientists have learned how to enhance the power of individual epidemiological studies through "meta-analysis" of the data from several such studies. Meta-analysis is the process of statistically combining the results of many studies dealing with similar diseases and risk factors to yield additional information that can enhance the epidemiologist's understanding of associations between potentially toxic agents and their effects.⁶⁶ Many epidemiological studies (especially case-control studies) observe only a very small number of individuals and therefore lack sufficient power to detect modest increases in relative risk that could be quite significant in the case of an agent to which there is widespread human exposure. Meta-analysis allows epidemiologists to

⁶² ROTHMAN & GREENLAND, *supra* note 56, at ch. 7; *see also* Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17,973.

⁶³ Guidelines for Reproductive Toxicity Risk Assessment, 61 Fed. Reg. 56,274, 56,299 (Oct. 31, 1996); Guidelines for Developmental Toxicity Risk Assessment, 56 Fed. Reg. 63,798, 63,809 (Dec. 5, 1991).

⁶⁴ Guidelines for Reproductive Toxicity Risk Assessment, 61 Fed. Reg. at 56,299.

⁶⁵ Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17,967 ("[W]hen cancer effects are not found in an exposed human population, this information by itself is not generally sufficient to conclude that the agent poses no carcinogenic hazard to this or other populations of potentially exposed humans . . . because epidemiologic studies usually have low power to detect and attribute responses.").

⁶⁶ ROTHMAN & GREENLAND, *supra* note 56, at ch. 32; *see also* Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17,974 ("When utilized appropriately, meta-analysis can enhance understanding of associations between sources and their effects that may not be apparent from examination of epidemiologic studies individually.").

increase the observational power of the studies by combining their results.⁶⁷

Epidemiologists must carefully avoid bias in obtaining and analyzing epidemiological data.⁶⁸ Unfortunately, limitations in the sources of data ordinarily available to epidemiologists provide ample opportunities for unintended bias.⁶⁹ According to the Environmental Protection Agency's ("EPA") proposed carcinogen assessment guideline revisions:

[b]ias can arise from several sources, including noncomparability between populations of factors such as general health, diet, lifestyle, or geographic location; differences in the way case and control individuals recall past events; differences in data collection that result in unequal ascertainment of health effects in the populations; and unequal follow-up of individuals.⁷⁰

One especially prevalent form of bias in epidemiology is the "confounding factor," which is a risk factor for the disease at issue that is associated with the exposure in the population under study that is not otherwise affected by the exposure or the disease.⁷¹ If confounding factors unrelated to the exposure of the substance at issue can account for the observed differences in disease rates, then it may be inappropriate to conclude that the exposure

⁶⁷ In the words of the National Academy of Sciences Committee on the Epidemiology of Air Pollution: "In essence, meta-analysis assumes that the results of studies can themselves be treated as random variables with predictable distributions." COMM. ON THE EPIDEMIOLOGY OF AIR POLLUTION, NAT'L ACAD. OF SCIENCES, EPIDEMIOLOGY AND AIR POLLUTION (Nat'l Academy Press 1985). Meta-analysis is not appropriate, however, when the studies being combined are not comparable or contain substantial confounding or other biases for which the statistical analysis does not adjust. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17,974 (Meta-analysis may not be useful "when there are substantial confounding or other biases that cannot be adjusted for in the analysis.").

⁶⁸ See generally GREEN, *supra* note 21, at 30-34.

⁶⁹ See ROTHMAN & GREENLAND, *supra* note 56, at 199 (citing one study that "listed dozens of possible biases that can distort the estimation of an epidemiologic measure"). See also Sanders, *supra* note 22, at 1420-21 (explaining recall bias in cases involving pharmaceuticals).

⁷⁰ Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17,973. See also ROTHMAN & GREENLAND, *supra* note 56, at 119-23.

⁷¹ ROTHMAN & GREENLAND, *supra* note 56, at 123-25; see also Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17,974.

to the substance is responsible for those differences.⁷² Epidemiologists must therefore adjust the study design or the statistical analyses to reduce or eliminate potential “confounders” as the explanation for the observed differences.⁷³ When complete adjustment for confounding factors is not possible, however, it may still be possible to draw conclusions about the ability of an agent to cause disease from the fact that multiple studies with different potential confounders yield similar results.⁷⁴

2. General Causation

As previously noted, a plaintiff in a toxic tort action has the burden of proving that the substance for which the defendant is responsible is capable of causing the disease from which the plaintiff suffers in a population of human beings that includes the plaintiff. Because the general causation issue does not involve factual evidence about the individual plaintiff, defendants frequently raise the general causation issue early in the development of a trial by way of motions for summary judgment.⁷⁵ The basis for the motion, which has by now become almost boiler-plate, is that the plaintiff will present no admissible evidence on the issue of general causation and therefore cannot sustain the burden of proof on that issue. The motion thus requires the court to conduct a *Daubert* assessment of the plaintiff's expert witnesses on general causation based upon affidavits and deposition testimony from the experts themselves and from the defendant's experts. In theory, the trial judge does not resolve the conflict in testimony at this stage, but rather determines whether the plaintiff's experts on general causation meet the *Daubert/Joiner* tests for relevance and scientific reliability.

a. The Corpuscular Approach to Expert Testimony

⁷² ROTHMAN & GREENLAND, *supra* note 56, at 59.

⁷³ Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17,974.

⁷⁴ *Id.* (“If consistent increases in cancer risk are observed across a collection of studies with different confounding factors, the inference that the agent under investigation was the etiologic factor is strengthened, even though complete adjustment for confounding factors cannot be made and no single study supports a strong inference.”).

⁷⁵ See Beecher-Monas, *supra* note 8, at 1074 (observing that “the causation issue frequently surfaces in the context of a dual motion challenging the admissibility of expert testimony and moving for summary judgment”).

In the wake of *Daubert* and *Joiner*, most courts have adopted a “corpuscular” approach to determining the admissibility of expert testimony in toxic tort cases. Under this approach, the party offering scientific expert testimony must establish the relevance and reliability under the *Daubert/Joiner* criteria of each individual study upon which the expert relies as well as the relevance and reliability of the expert’s overall conclusions.⁷⁶ If the plaintiff fails to establish the relevance and scientific reliability of a sufficient number of the individual studies, the trial judge will exclude the expert’s testimony and (in the absence of other relevant and reliable expert testimony on causation) grant the defendant’s motion for summary judgment before the jury ever enters the picture. This approach in practice places a great burden on the plaintiff to “validate” each of the studies relied upon by the plaintiff’s experts as well as to establish the scientific reliability of their overall conclusions. Furthermore, this approach invites defendants to focus upon flaws in the corpuscles of data underlying the testimony, rather than upon the scientific reliability of the expert’s overall conclusions.

Epidemiological studies are exceedingly difficult to conduct in a world in which health and mortality records are notoriously bad. Data must frequently be drawn from human recollections and it is impossible to control against every possible confounding factor or source of bias. As a consequence, the conclusions of individual epidemiological studies cannot be stated with a high degree of certainty. Indeed, the one thing that can be said with a great deal of confidence about epidemiological studies is that they are likely to contain flaws and potential biases. The corpuscular approach invites parties seeking to exclude expert testimony to search every detail of each epidemiological study for possible flaws in the statistical analysis and to speculate at great length about potential confounding factors and other possible sources of bias. Given the practical impossibility of conducting a perfect epidemiological study, the search is nearly always fruitful.

Perhaps the best example of the corpuscular approach in action is the majority opinion in *Joiner* itself.⁷⁷ In that case a plaintiff with well-documented exposure to PCBs offered the testimony of two experts who concluded that the PCB exposure had probably caused the plaintiff’s small cell lung cancer. In the process of holding that the appropriate standard of

⁷⁶ See *id.* at 1057, 1067 (noting that the courts have frequently read *Daubert* to require them to evaluate each study underlying an expert’s conclusion sequentially to determine admissibility).

⁷⁷ *Gen. Elec. Co. v. Joiner*, 522 U.S. 136 (1997).

review for a trial court's admissibility determinations was the "abuse of discretion" test, the Court addressed the trial judge's application of the *Daubert* criteria to the plaintiff's expert testimony. According to the Court, the testimony of both experts was based primarily upon on a laboratory animal study and four epidemiological studies, none of which could validly support a reliable scientific conclusion that PCBs were capable of causing lung cancer in humans.

The animal study could not validly support that conclusion because the animals were young (not middle-aged like the plaintiff), the route of administration was different (direct injection of single doses into the stomach as opposed to continuous dermal and inhalation exposure), the doses the animals received were much larger than the plaintiff's exposure, and the mice developed a different form of cancer. Although the Court was willing to entertain the possibility that a trial court could appropriately admit relevant expert testimony based upon valid animal studies that were relevant to a plaintiff's exposure, the studies in this case "were so dissimilar to the facts presented in this litigation that it was not an abuse of discretion for the District Court to have rejected the experts' reliance on them."⁷⁸ In other words, the district court was appropriately performing its *Daubert*-assigned gatekeeper role when it concluded, apparently as a matter of "sound science," that the plaintiff's experts could not validly rely upon the animal study.

The four epidemiological studies upon which the plaintiff's experts relied were likewise scientifically invalid for the purpose of demonstrating that PCB exposure could cause lung cancer in humans. The first study of workers exposed to PCBs in an Italian capacitor factory could not validly support the plaintiff's experts' conclusion because although the rate of lung cancer deaths was "higher than might have been expected" in the exposed cohort, the authors of the study concluded "there were apparently no grounds for associating lung cancer deaths (although increased above expectations) and exposure in the plant."⁷⁹ Since the authors of the study were unwilling to conclude that the study demonstrated a causal relationship between PCB exposure and lung cancer, the plaintiff's experts could not validly rely upon that study, no matter what additional supporting scientific information might be available.

⁷⁸ *Id.* at 144-45.

⁷⁹ *Id.* at 145 (quoting Bertazzi et al., *Cancer Mortality of Capacitor Manufacturing Workers*, 11 AM. J. INDUS. MED. 165, 172 (1987)). Neither the trial court nor the Supreme Court inquired into what might have motivated the authors to conclude that the PCB exposures were not associated with the lung cancers.

The second study of workers exposed to PCBs at the Monsanto Corporation's Sauget, Illinois PCB manufacturing facility also found the incidence of lung cancer among PCB-exposed workers to be "somewhat higher" than would ordinarily be expected, but its authors concluded that since the increase was not "statistically significant," the data did not suggest a link between PCB exposure and lung cancer.⁸⁰ Neither the trial court nor the Supreme Court elaborated on the degree of statistical significance that would be required to support a valid conclusion that an elevated incidence in lung cancer in PCB-exposed workers was linked to the exposure. They were apparently content to rely upon the level of significance demanded by the scientists who conducted the study. The study could therefore not validly be relied upon by the plaintiff's experts to support an overall conclusion that PCBs cause lung cancer in humans.

Although the third study of Norwegian employees of a cable manufacturing plant did report a statistically significant increase in lung cancer, it could not validly be relied upon by the plaintiff's experts because it made no mention of PCBs and was limited to the particular mineral oil to which the workers were exposed.⁸¹ The Court did not address the fact, obviously relied upon by the plaintiff's experts, that many kinds of mineral oil contain PCBs. The plaintiff's experts apparently had reason to believe that the mineral oil to which the Norwegian employees had been exposed contained PCBs, but because the published study made no mention of PCBs, it was, in the Court's view, irrelevant.

The fourth study appeared at first glance to be the holy grail for which the trial judge was apparently searching. That study detected a statistically significant increase in lung cancer deaths in a PCB-exposed human cohort in Japan. Alas, that study, too, had to be rejected because the "subjects of this study . . . had been exposed to numerous potential carcinogens, including toxic rice oil that they had ingested."⁸² Without relying upon any expert statistical analysis, the trial judge apparently concluded as a scientific matter that this confounding factor, which is likely to be present in any Japanese cohort, had not been adequately accounted for. The study was therefore invalid and could not support an overall conclusion that PCBs cause lung cancer in humans.

⁸⁰ *Id.* Again, neither the trial court nor the Supreme Court inquired into what might have motivated the authors (who no doubt undertook the study at the behest of the Monsanto Corporation) to conclude that the increase in lung cancer was not "statistically significant." *Id.*

⁸¹ *Id.* at 145-46.

⁸² *Id.* at 146.

As the *Joiner* case very clearly reveals, the corpuscular approach effectively prevents the expert in toxic torts cases from applying the "weight-of-the-evidence" approach that regulatory agencies universally employ in assessing the risks that toxic substances pose to human beings.⁸³ Under the weight-of-the-evidence approach, the agency considers all of the proffered studies and determines the weight to be afforded each study on the basis of the identified strengths and weaknesses of that study. Some studies are so poorly conducted that they are entitled to no weight at all, but many studies that are otherwise flawed in one or more regards may be appropriately considered to the extent that they add to or detract from conclusions based upon studies in which the agency is inclined to place more confidence.⁸⁴ Animal studies are properly considered under the weight-of-the-evidence approach, as are meta-analyses of epidemiological studies that may be flawed to some extent.⁸⁵ The weight-of-the-evidence

⁸³ See Brown, *supra* note 40, at 845 (arguing that although not specifically addressed in *Joiner*, the court "implicitly rejected" the weight-of-the-evidence approach); see also Gottesman, *supra* note 8, at 769-70; Beecher-Monas, *supra* note 8, at 1068 (noting that "[a]lthough the relevance of a single study may be a fairly straightforward determination, relevance becomes more complicated when a number of studies are involved, each of which is only marginally relevant, but which together purport to form the basis of an expert's conclusions"); Finley, *supra* note 8, at 336-37 (observing that "[j]udges have applied *Daubert* to subject each item of expert proof proffered by plaintiffs to substantive causation law scrutiny, to see if it, standing alone, would prove both general and specific causation"); Sanders, *supra* note 22, at 1416-17 (criticizing courts that do not preclude an expert from relying on all of the available evidence). At least one post-*Daubert* court has explicitly rejected an expert's conclusions based on a weight-of-the-evidence approach. See *Allen v. Pennsylvania Eng'g Corp.*, 102 F.3d 194, 196-98 (5th Cir. 1996).

⁸⁴ See Sanders, *supra* note 22, at 1392 (noting that "scientific validity has multiple meanings and is always a matter of degree").

⁸⁵ EPA explained the weight-of-the-evidence approach in the context of carcinogen risk assessment as follows:

Judgment about the weight of evidence involves considerations of the quality and adequacy of data and consistency of responses induced by the agent in question. The weight of evidence judgment requires combined input of relevant disciplines. Initial views of one kind of evidence may change significantly when other information is brought to the interpretation. For example, a positive animal carcinogenicity finding may be diminished by other key data; a weak association in epidemiologic studies may be bolstered by consideration of other key data and animal findings. Factors typically considered are illustrated in figures below. Generally, no single weighing factor on either side determines the overall weight. The factors are not scored mechanically by adding pluses and minuses; they are judged in combination.

approach, in fact, closely resembles the fact finding function of the jury in civil trials in which testimony of varying degrees of quality and credibility is offered from percipient witnesses.

The weight-of-the-evidence approach focuses upon the totality of the scientific information and asks in a holistic way whether a cause-effect conclusion seems warranted. Given the inevitability of flaws in individual studies and the fact that some of the studies were not undertaken with the litigative or regulatory process in mind, this necessarily involves the exercise of scientific judgment grounded in scientific expertise. The corpuscular approach focuses upon the inevitable flaws in individual studies and asks whether a sufficient number of unflawed studies that are sufficiently relevant to the causation issue remain to support a conclusion that is in itself relevant and reliable. Under the corpuscular approach, a study is either valid or invalid, and it is either relevant or irrelevant. A conclusion based upon invalid or irrelevant studies cannot be relevant and reliable and must therefore be rejected. Thus, the courts applying the corpuscular approach to determining the admissibility of expert testimony have adopted a remarkably different, and arguably much less scientific,⁸⁶ approach to causation than the regulatory agencies charged with the responsibility of protecting citizens from toxic risks.

b. Preponderance of the Evidence and Statistical Proof Under the Corpuscular Approach

In the *Daubert* remand, the court of appeals concluded that California tort law, like the law of most states, required the plaintiff to establish that the exposure to the defendant's toxic agent "more likely than not" caused the plaintiff's disease.⁸⁷ Writing for the majority, Judge Kozinski observed that in statistical terms this required the plaintiff to demonstrate that the substance "more than doubled" the likelihood that the plaintiff's disease resulted from exposure to the defendant's toxic agent. Because the "background" incidence of birth defects of the sort that the plaintiffs suffered was one per thousand births, the plaintiffs would have to introduce epidemiological evidence showing that "among children of mothers who took Bendectin the incidence of such defects was more than

Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17,960, 17,963, 17,981 (Apr. 23, 1996).

⁸⁶ See *id.* (criticizing *Daubert/Joiner* as being inconsistent with the scientific method).

⁸⁷ *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 43 F.3d 1311 (9th Cir. 1995).

two per thousand.”⁸⁸ This suggests that a relative risk of greater than 2.0 in a well-conducted epidemiological study is sufficient to establish general causation.⁸⁹

The *Daubert* remand opinion is less clear as to whether a well-conducted epidemiological study demonstrating a doubling of the relative risk is *necessary* to establish general causation in toxic tort cases. Several courts have read the *Daubert* remand opinion to conclude that an epidemiological study demonstrating a relative risk of greater than 2.0 is in fact the minimum showing necessary to prove general causation.⁹⁰ This position is by no means compelled by the Supreme Court's *Daubert* opinion, which does not even preclude a jury's finding of causation based exclusively upon animal studies.⁹¹ Other courts have sensibly refused to make such a showing a threshold requirement.⁹²

⁸⁸ *Id.* at 1320.

⁸⁹ The term “relative risk” in epidemiology is defined as the ratio $R1/R2$, where $R1$ is the risk of contracting the relevant disease as measured in the exposed population and $R2$ is the risk of contracting the disease in an equivalent unexposed population. *See* GERALD W. BOSTON & M. STUART MADDEN, *LAW OF ENVIRONMENTAL AND TOXIC TORTS* 3, 352, 354 (1994); *see also* GREEN, *supra* note 21, at 28; Beecher-Monas, *supra* note 39, at 1600-01.

⁹⁰ *See In re Breast Implant Litigation*, 11 F. Supp. 2d 1217 (D. Colo. 1998); *see also* Kelley v. American Heyer-Schulte Corp., 957 F. Supp. 873 (W.D. Tex. 1997); Hall v. Baxter Healthcare Corp., 947 F. Supp. 1387, 1403 (D. Or. 1996); Merrell Dow Pharmaceuticals v. Havner, 953 S.W.2d 706, 716 (Tex. 1997); Finley, *supra* note 8, at 339, 350 (concluding that appellate courts in the pre-*Daubert* and post-*Daubert* Bendectin cases through various devices gradually adopted a legal rule that “unless plaintiffs could produce a consistent body of statistically significant epidemiological studies that showed that Bendectin at least doubled the risk of birth defects, plaintiffs did not have sufficient evidence of causation to support a verdict”); Mark Geistfeld, *Scientific Uncertainty and Causation in Tort Law*, 54 VAND. L. REV. 1011, 1012-13 (2001).

⁹¹ Professor Finley suggests that federal judges have employed their *Daubert*-inspired screening role to change the substantive law of toxic torts by demanding plaintiffs put on proof of causation that is not only scientifically reliable, but also meets a substantive criterion imposed by the judges. Finley, *supra* note 8, at 335.

⁹² *See* Schudel v. Gen. Elec. Co., 120 F.3d 991 (9th Cir. 1997); Pick v. Am. Med. Sys., Inc., 958 F. Supp. 1151, 1160 (E.D. La. 1997); Landrigan v. Celotex Corp., 605 A.2d 1079 (N.J. Sup. Ct. 1992) (observing that “a relative risk of 2.0 is not so much a password to a finding of causation as one piece of evidence, among others, for the court to consider in determining whether the expert has employed a sound methodology in reaching his or her conclusion”).

Professor Klein has offered an interesting permutation in which tort law would “permit enhanced risk recovery on a proportional basis [prior to contracting the disease], but only when a plaintiff can prove that the toxic exposure has more than doubled her risk of contracting disease in the future.” Klein, *supra* note 43, at 117. Under the proposal

There is nothing magical about a relative risk of 2.0.⁹³ A relative risk exceeding 1.0 indicates that there is an association between the exposure and the disease, and a relative risk of 2.0 by no means guarantees that any particular incidence of disease is attributable to the exposure.⁹⁴ The EPA, for example, concluded that nonsmoker exposure to environmental tobacco smoke caused lung cancer on the basis of a "meta-analysis" combining many studies that indicated a relative risk of 1.42.⁹⁵ The level of relative risk sufficient to support governmental action (in this instance, shifting wealth from the manufacturer of a product to persons who have been exposed to the product and suffer from a disease associated with the product) is ultimately a policy decision, not a matter of science. The courts that insist upon a relative risk of 2.0 in all cases have employed a very conservative policy of requiring manufacturers to pay money to claimants only when the decisionmaker is very confident that the

anyone who could demonstrate that exposure to a toxic substance increased the relative risk of contracting a disease by 2.0 or greater could recover immediately for the risk of contracting the disease, whether or not the plaintiff had actually contracted the disease. *See id.* at 119. One wonders how frequently plaintiffs would recover under this proposed regime. It takes an especially rare cancer or an especially powerful chemical to produce a relative risk of 2.0. The proposal also has no role for animal studies to play in establishing causation. *Id.*

⁹³ One perceptive state court noted that such a threshold requirement would mean that no plaintiff could recover for exposure to a substance that presented a relative risk of 1.99 whereas every plaintiff could recover when the relative risk was 2.01. *Grassis v. Johns-Manville Corp.*, 591 A.2d 671, 676 (N.J. Super. Ct. App. Div. 1991).

⁹⁴ *See Beecher-Monas, supra* note 39, at 1601. Scientists are generally more confident that the apparent association is real if the "confidence interval" (the statistical range through which the statistician is ninety-five percent confident the real result lies) does not go below 1.0. *See Brown, supra* note 40, at 843 (explaining why most courts require a "p" value of 0.05 or less). The number 95, like the number 2.0, has no magical significance. The level of statistical significance that a scientist demands is merely a numerical representation of the willingness of the scientist to draw an erroneous conclusion. *See Beecher-Monas, supra* note 8, at 1095. Most epidemiologists are skeptical of binary approaches to epidemiological data that reject altogether studies that do not achieve a particular level of statistical significance. *See ROTHMAN & GREENLAND, supra* note 56, at 6 (complaining of "the practice of declaring associations in data as 'statistically significant' or 'nonsignificant,' using arbitrary criteria that became conventional"). By filtering out all statistical studies that reject the null hypothesis with a degree of confidence less than ninety-five percent, the courts are applying a "mighty damned confident" standard to statistical evidence that may in many cases be the only available evidence on causation.

⁹⁵ U.S. ENVTL. PROT. AGENCY, RESPIRATORY HEALTH EFFECTS OF PASSIVE SMOKING: LUNG CANCER AND OTHER DISORDERS 5-2 (EPA/600/6-90/006F 1992) [hereinafter FINAL EPA ETS REPORT].

claimants are in fact victims.⁹⁶ One could just as easily view this as a policy of shielding manufacturers of dangerous products from accountability through tort law.

c. Laboratory Animal Testing

From a corpuscular perspective, laboratory animal studies are generally superior to epidemiological studies, because they can be designed in advance to meet stringent scientific criteria.⁹⁷ Although flaws inevitably arise in the execution of animal studies, they are generally more difficult to challenge on scientific grounds. Consequently, defendants go to great lengths to persuade trial judges to exclude animal studies on relevance grounds. This tactic has proved highly successful in cases in which reliable epidemiological studies are not available.⁹⁸ If anything, the post-*Daubert* courts have shown more reluctance than pre-*Daubert* courts to admit expert testimony based solely upon animal testing data,⁹⁹ evidence that administrative agencies like EPA and the Occupational Health and Safety Administration ("OSHA") have for three decades found

⁹⁶ See Finley, *supra* note 8, at 363 (concluding that a court that demands epidemiological studies demonstrating a relative risk greater than 2.0 as proof of causation in toxic tort cases exalts epidemiology above other scientific disciplines, and makes the same highly conservative value choice on causation that epidemiology itself makes: it is worse to conclude that there is a causal relationship when in fact there turns out not to be one (a Type I false positive error), than it is to conclude that there is no causal relationship when in fact there is such a relationship (a Type II false negative error)). See also Brennan, *supra* note 43, at 62.

⁹⁷ See Beecher-Monas, *supra* note 8, at 1064-65. The usefulness of laboratory animal data can therefore be enhanced considerably by pharmacokinetic studies on the animals and on human beings. Guidelines for Reproductive Toxicity Risk Assessment, 61 Fed. Reg. 56,274, 56,302 (Oct. 31, 1996); Guidelines for Developmental Toxicity Risk Assessment, 56 Fed. Reg. 63,798, 63,813 (Dec. 5, 1991).

⁹⁸ See Beecher-Monas, *supra* note 39, at 1609.

⁹⁹ See *Wade-Greaux v. Whitehall Labs., Inc.*, 874 F. Supp. 1441, 1480 (D.V.I. 1994) (finding that the "notion that one can accurately extrapolate from animal data to humans to prove causation without supportive positive epidemiologic studies is scientifically invalid"), *aff'd*, 46 F.3d 1120 (3d Cir. 1994); *Merrell Dow Pharms., Inc. v. Havner*, 953 S.W.2d 706, 729-30 (Tex. 1997) (rejecting animal studies as unreliable under Texas equivalent of *Daubert*); Beecher-Monas, *supra* note 8, at 1064-65 (collecting cases); Cutler, *supra* note 37, at 216 (observing that "the pre-*Daubert* trend in excluding opinions based on animal studies is continuing"). But see *In re Paoli Ry. Yard PCB Litig.*, 35 F. 3d 717, 743 (3d Cir. 1997) (expressing willingness to find animal testing admissible when the expert demonstrates good grounds for the toxicological conclusions drawn by the studies and good grounds for extrapolating from animal studies to human exposures).

fully relevant to assessing and managing the risks posed by toxic substances.¹⁰⁰ Scientists and federal regulators are well aware of the limitations of animal studies, but they know that it is unscientific to disregard that important source of information altogether, and they are frequently willing to draw conclusions about human health risks without confirming epidemiological studies.¹⁰¹

¹⁰⁰ The pesticides aldrin/dieldrin and heptachlor/chlordane were canceled on the basis of animal studies, even though the registrants were able to offer limited epidemiological research that showed no increase in relative risk. *Environmental Defense Fund, Inc. v. EPA*, 548 F.2d 998 (D.C. Cir. 1976), *cert. denied*, 431 U.S. 925 (1977) (heptachlor/chlordane); *Environmental Defense Fund, Inc. v. EPA*, 510 F.2d 1242 (D.C. Cir. 1975) (aldrin/dieldrin). *See also* *Synthetic Organic Mfrs. Ass'n v. Brennan*, 506 F.2d 385, 387 (1974) (ruling that OSHA may rely upon animal studies in setting occupational health standards); *see also* *Synthetic Organic Mfrs. Ass'n v. Brennan*, 503 F.2d 1155, 1159 (3d Cir. 1974), *cert. denied*, 420 U.S. 973 (1975) (same); *see also* *Cutler*, *supra* note 37, at 202.

¹⁰¹ *See* NAT'L RES. COUNCIL, *RISK ASSESSMENT IN THE FEDERAL GOVERNMENT: MANAGING THE PROCESS* 22 (1983); *see also* Troyen A. Brennan & Robert F. Carter, *Legal and Scientific Probability of Causation of Cancer and Other Environmental Disease in Individuals*, 10 J. HEALTH POL'Y & L. 33, 34 (1985); *see also* Beecher-Monas, *supra* note 8, at 1064 (suggesting that the reluctance of some courts to admit expert testimony based upon animal studies "reflects the courts' ignorance of basic scientific precepts").

In recent years, scientists have developed less expensive tests for carcinogenicity that probe, *inter alia*, the mutagenic potential of an agent in *in vitro* conditions. *See* NAS RED BOOK, *supra* note 49, at 22 ("Considerable experimental evidence supports the proposition that most chemical carcinogens are mutagens and that many mutagens are carcinogens."). Although such tests can provide "supportive evidence" of carcinogenicity, they are not sufficiently accurate to support such a finding standing alone. *Id.* at 23 ("Such data, in the absence of a positive animal bioassay, are rarely, if ever, sufficient to support a conclusion that an agent is carcinogenic."). Risk assessors can also consider the chemical structure of an agent as compared with the chemical structure of a substance with a known toxicity to assess the hazard that it might pose to humans. *Id.* ("Comparison of an agent's chemical or physical properties with those of known carcinogens provides some evidence of potential carcinogenicity."). Since it is well known that two chemicals that have almost identical chemical structures can produce very different toxicity end points, however, chemical structure is even weaker support for a hazard assessment than *in vitro* tests. U.S. ENVTL. PROT. AGENCY, *GUIDANCE FOR IDENTIFYING PESTICIDE CHEMICALS AND OTHER SUBSTANCES THAT HAVE A COMMON MECHANISM OF TOXICITY* 4, 8 (Jan. 29, 1999); *see also* NAS RED BOOK, *supra* note 49, at 23 ("[S]uch studies are best used to identify potential carcinogens for further investigation and may be useful in priority-setting for carcinogenicity testing."). No court has allowed a plaintiff to establish cause-in-fact in a toxic tort case based solely upon either *in vitro* studies or structure-activity relationships or both, and no court is likely to in the near future. *See* Sanders, *supra* note 22, at 1412-15.

3. Specific Causation

If the toxic tort plaintiff is able to produce scientifically reliable expert testimony to establish that the substance at issue is capable of causing the plaintiff's disease, the plaintiff must still produce relevant and reliable evidence that the plaintiff's own exposure to the substance caused the plaintiff's disease. Specific causation poses difficult problems of proof for toxic torts plaintiffs. First, the plaintiff must document exposure to the substance or substances at issue.¹⁰² The plaintiff's lawyer can accomplish this through direct testimony from the plaintiff or others when the substance is well known to the plaintiff and the exposure is easily detectable through sight or smell.¹⁰³ Even then, however, the quantitative dose that the plaintiff received can rarely be established by direct lay testimony.¹⁰⁴ Experts must extrapolate from the testimony to an overall dose estimation through various modeling exercises. The growing insistence of district courts upon fairly precise dose estimates means that at the very least plaintiffs must establish the relevance and reliability of expert testimony on that topic, and this frequently means that the plaintiff is out of court.¹⁰⁵

Second, specific causation has frequently proven to be an insuperable barrier in toxic torts cases because of the long latency periods that often exist between the exposure to a toxic substance and the onset of

¹⁰² See Brown, *supra* note 40, at 843-44.

¹⁰³ For example, an employee of a contractor who was told to remove liquid from a tank marked "benzene" can probably establish dermal exposure to benzene by testifying as to the content of the label and the fact that the benzene splashed on his hands and arms. Inhalation exposure can be documented by direct testimony concerning fumes and foul odors, though even this sort of testimony is unavailable in the case of odorless gasses. Similarly ingestion of chemicals in drinking water can sometimes be documented by testimony about the taste of the water.

¹⁰⁴ Dose is fairly easily documented in cases involving prescription drugs, because the dose is specified by a doctor. The problem of identifying which of many companies is responsible for the particular drug that damaged the plaintiff has troubled the courts, but has not proven an insurmountable obstacle to recovery. See, e.g., *Hymowitz v. Eli Lilly and Co.*, 539 N.E.2d 1069 (N.Y. 1989); see also *Collins v. Eli Lilly and Co.*, 342 N.W.2d 37 (Wis. 1984); *Sindell v. Abbott Labs.*, 607 P.2d 924 (Cal. 1980).

¹⁰⁵ See Robert F. Blomquist, *Emerging Themes and Dilemmas in American Toxic Tort Law, 1988-1991: A Legal-Historical and Philosophical Exegesis*, 18 S. ILL. U. L.J. 1, 42 (1993); see also Brinker, *supra* note 42, at 1300.

disease.¹⁰⁶ Given all of the other exposures that the plaintiff will have encountered during that time period, defendants have ample opportunities to suggest alternative explanations for all but the clearest of signature diseases, thereby casting doubt on the plaintiff's expert's testimony on specific causation.¹⁰⁷ Indeed, a defendant can usually find something in the plaintiff's own lifestyle or genetic makeup that could also have caused the plaintiff's disease,¹⁰⁸ and this invites probing and sometimes embarrassing inquiries into the plaintiff's personal life.

4. Finding Flaws with Scientific Studies under the Corpuscular Approach

The reluctance of the lower courts to accept testimony based exclusively upon animal studies is profoundly conservative in that it forces plaintiffs in toxic tort cases to rely almost exclusively upon epidemiological studies. In addition to being very expensive and time consuming to perform¹⁰⁹ epidemiological studies are by their very nature laden with practical methodological difficulties that make them easy prey for trial lawyers bent on casting doubt upon their "scientific validity."¹¹⁰

¹⁰⁶ Cutler, *supra* note 37, at 199.

¹⁰⁷ See Berger, *supra* note 7, at 2121-22; see also Brennan, *supra* note 43, at 47.

¹⁰⁸ See Brinker, *supra* note 42, at 1299-1300.

¹⁰⁹ See GREEN, *supra* note 21, at 29 (noting that a large cohort study can cost as much as \$100 million); see also Geistfeld, *supra*, note 90, at 1012.

¹¹⁰ See Berger, *supra* note 7, at 2129 (noting that "epidemiological studies . . . are prone to design errors and interpretative disputes"); Cutler, *supra* note 37, at 200-01; Sanders, *supra* note 22, at 1417 (noting that epidemiology "inevitably suffers from some internal validity problems"). Professor Feldman provides a brief synopsis of the problems that plaintiffs may encounter in attempting to establish causation on the basis of epidemiological studies:

First, epidemiological studies are generally unable to detect small increases in the risk of harmful effects from a substance, especially if the background risks of the same harmful effects are extremely low. Second, epidemiological studies typically lack follow-up times long enough to ensure that diseases with long latency periods are discovered. Third, epidemiological studies may suffer from a number of biases such as selection bias, which occurs when the exposed group is selected in a way that makes it more or less susceptible to disease for reasons independent of exposure; diagnostic bias, which occurs when the disease in question is not accurately determined; exposure bias, which is the danger of selecting a study population especially likely or unlikely to have been exposed to the disease; and recall bias, which is

Professor Sanders has perceptively noted that “[m]uch of what goes on at trial in America is a process of deconstructing science.”¹¹¹ The remarkable array of internal documents produced during the recent tobacco litigation, makes it clear that the process of deconstruction starts long before the trial. The planned obfuscation begins when the first scientific studies and reports begin to appear, and it continues well beyond the point at which most reasonable members of the scientific community have drawn scientific conclusions based upon the weight of the available evidence.¹¹²

As the evidence mounted in the mid-1950s that cigarette smoking caused lung cancer in smokers, the industry’s public relations firm, Hill & Knowlton, developed a four-part proactive strategy for dealing with the emerging scientific studies that included:

(a) smearing and belittling them; (b) trying to overwhelm them with mass publication of the opposed viewpoints of other specialties; (c) debating them in the public arena; or (d) we can determine to raise the issue far above them, so they are hardly even mentioned¹¹³

The industry created the Tobacco Industry Research Committee (“TIRC”), later renamed the Council for Tobacco Research (“CTR”), to fund projects aimed at accomplishing these goals.¹¹⁴ Under the broad oversight of a

the tendency of those who are in a study to recall incorrectly whether they were exposed to the agent being studied. Fourth, epidemiological studies may be flawed because of the presence of unaccounted-for confounders—undiscovered factors that independently affect disease rates in the studies population.

Feldman, *supra* note 15, at 25.

¹¹¹ Sanders, *supra* note 22, at 1437.

¹¹² The following discussion draws upon a much larger work in progress on how the tobacco companies and others have attempted to “bend science” to shield themselves from regulation and liability.

¹¹³ Memorandum from Hill & Knowlton, to Members of the Planning Committee, *quoted in* DAVID KESSLER, *A QUESTION OF INTENT: A GREAT AMERICAN BATTLE WITH A DEADLY INDUSTRY* 200 (2001).

¹¹⁴ KESSLER, *supra* note 113, at 200-01; *see also* RICHARD KLUGER, *ASHES TO ASHES* 205-09 (1996). An internal memo, written at a time of transition, explains the Council’s functions as follows:

- (1) The existence of The Council demonstrates that the industry is acting in good faith in supporting a serious scientific effort to determine the effects of smoking on human health.

prestigious Scientific Advisory Board,¹¹⁵ the institution spent millions of dollars funding research that was only very remotely connected to smoking and health, but which allowed the industry to claim that it was searching for the answers.¹¹⁶ The research projects were frequently aimed at drawing attention away from tobacco as a cause of disease.

With the publication of the Report of the Surgeon General's Advisory Committee on Smoking and Health in 1964, the industry recognized that it needed additional support from scientists who were not so clearly associated with the tobacco business. It therefore created a "Special Projects" fund to support a network of consultants who could be depended upon to take the industry position in scientific publications, congressional and administrative presentations, and in expert testimony in court.¹¹⁷ The Special Projects were administered over the years by the Committee of Counsel without any oversight by the CTR Scientific Advisory Board.¹¹⁸ As its name suggested, the Committee of Counsel was composed of the general counsels of all of the major tobacco companies and attorneys from four major industry firms.¹¹⁹ Its mission was "to seed

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- (2) The Council provides the industry with a direct avenue of contact and intelligence in the field of medical research into tobacco use and health.
 - (3) The Council provides the industry with its own scientific experts who may also serve as scientific spokesmen.
 - (4) Research supported by The Council may either disprove the allegations that smoking is a primary etiological factor in some diseases or it may suggest ways of adapting or modifying cigarettes.

Memorandum from Simon O'Shea, to W. T. Hoyt (Aug. 18, 1965) (on file with author).

¹¹⁵ See Letter from Robert C. Hockett, to Theodor D. Sterling (Feb. 13, 1963) (on file with author) (explaining the role of the TIRC Scientific Advisory Board).

¹¹⁶ See KLUGER, *supra* note 114, at 206, 208.

¹¹⁷ See KESSLER, *supra* note 113, at 203. Former FDA Commissioner David Kessler noted that "[s]pecial [p]rojects had no official address, no incorporation papers, no board of directors, no by-laws, and no accountability." *Id.* at 204.

¹¹⁸ See Letter from Robert C. Hockett, to Theodor D. Sterling (June 15, 1964) (on file with author) (explaining that the persons approving special projects were "not bound in any way by [the Scientific Advisory Board's] judgments or advice but are free to consider any suggestions we can get from them on their merits").

¹¹⁹ KESSLER, *supra* note 113, at 204.

the universities with research money and grow an unassailable crop of advocates."¹²⁰

The tobacco industry is by no means the only industry to attempt to "deconstruct science" by financing scientific research aimed specifically at discrediting published studies and government reports.¹²¹ After a cellular telephone company was sued in 1993 by the husband of a frequent cell phone user who had died of brain cancer, the cellular telephone industry's trade association followed the tobacco industry model in virtually every particular.¹²² As news of the lawsuit caused cell phone company stock prices to drop precipitously, the Cellular Telecommunications Industry Association ("CTIA") mounted a well-financed public relations campaign to put the industry's spin on the issue.¹²³ The industry recognized, however, that it would have to launch a scientific offensive as well as a PR offensive. With much fanfare, the CTIA announced that the industry would sponsor a \$25 million Wireless Technology Research ("WTR") program to get to the bottom of the issue.¹²⁴ The trade association's press statement announced that it was "time for truth and good science to replace emotional videotape and unsupported allegations."¹²⁵

The CTIA hired epidemiologist George Carlo in 1993 to direct the WTR, the overt goal of which was to "re-validate" the studies showing that cell phones were safe.¹²⁶ A covert purpose of the project was to find flaws with studies that were beginning to indicate that cell phones did pose health risks.¹²⁷ Like the TIRC/CTR, Carlo's project created a Scientific Advisory Board to oversee particular projects.¹²⁸ He also commissioned

¹²⁰ DAN ZEGART, *CIVIL WARRIORS* 290 (Delacorte Press 2000). The Zegart book relates the fascinating story of Gary Huber, a prominent beneficiary of grants from the CTR and attorneys for the tobacco industry. *Id.* at 287-303.

¹²¹ See generally SHELDON RAMPTON & JOHN STAUBER, *TRUST US, WE'RE EXPERTS* 200 (2001) (noting that "[t]he tobacco industry is hardly alone in attempting to influence the scientific publishing process").

¹²² GEORGE CARLO & MARTIN SCHRAM, *CELL PHONES: INVISIBLE HAZARDS IN THE WIRELESS AGE* (Carroll & Graf 2001) (relating the experience of a scientist who was hired by the cell phone industry to study the health risks of cell phones, but lost his funding and was sharply criticized by other scientists hired by the cell phone industry after he concluded that cell phones posed some significant risks).

¹²³ *Id.* at 9.

¹²⁴ *Id.*

¹²⁵ *Id.*

¹²⁶ *Id.* at 9-10.

¹²⁷ *Id.* at 6.

¹²⁸ CARLO & SCHRAM, *supra* note 122, at 11-13.

the Harvard Center for Risk Analysis to assemble a Peer Review Board to review the project's outputs.¹²⁹ Like the TIRC/CTR, the WTR sponsored meetings of top scientists in an attempt to build a scientific consensus that cell phone radiation was not dangerous.¹³⁰ These measures were well received in the scientific community and at the FDA, which had regulatory authority over the health and safety aspects of cell phones.¹³¹

With an expensive Cray computer that the WTR made available to him, one of the top radiation scientists in the country discovered that the radiation patterns in petri dishes and test tubes were such that the places in each of those vessels containing most of the cells being tested were "cold spots" that did not in fact receive very much of the radiation that the cell phone emitted. This rendered suspect all of the previously conducted *in vitro* experiments in which scientists had concluded that cell phone radiation had no impact upon human tissue.¹³² This did not mean that the previous studies had reached erroneous conclusions, but it did suggest that the cells used in those studies had not received as high a dose of radio frequency radiation as the scientists had thought. Consequently, the conclusions of previous studies that cell phones were safe were suspect.

At the same time, the WTR found flaws with all of the studies that began to appear in the scientific literature in the early and mid-1990s indicating that cell phones did present a cancer risk.¹³³ When an Australian study showed that whole-body exposure to radio frequency waves caused a statistically significant increase in lymphomas in female transgenic mice, the WTR scientific advisory board found numerous flaws in the study and concluded that it did not require a public warning for cell phones.¹³⁴ A subsequent industry-sponsored scientific workshop concluded that more research was needed before studies with transgenic animals could support conclusions about human health hazards.¹³⁵ WTR scientists were careful to acknowledge that the findings of the studies were not necessarily wrong, but they did question the validity of each of the studies as they appeared.¹³⁶

¹²⁹ *Id.* at 13.

¹³⁰ *Id.* at 23.

¹³¹ *Id.* at 13.

¹³² *Id.* at 25-26.

¹³³ *Id.* at 107.

¹³⁴ CARLO & SCHRAM, *supra* note 122, at 115-18.

¹³⁵ *Id.* at 118.

¹³⁶ *Id.* at 107.

When the WTR appeared to be acting too independently of the industry trade association's lobbyists, the industry became hostile.¹³⁷ Three years after creating the WTR, the CTIR stopped funding it.¹³⁸ It refused to fund epidemiological studies attempting to follow up on complaints by cell phone users. One executive for a cell phone manufacturer, when asked how much money his company was willing to devote to follow up surveillance studies replied, "Zero. Zero surveillance. We're going to do enough research so that we can prove safety—and then we can stop doing research."¹³⁹ With this attitude prevalent in the industry, it should be a very long time before reliable epidemiological studies are available on the health effects of cell phones. It is entirely possible (in my view, probable) that experience will ultimately demonstrate that cell phones are reasonably safe for the human population in general. Yet, it is also possible that cell phones will cause some harm to a small class of especially vulnerable cell phone users. No plaintiff has recovered damages from a cellular telephone manufacturer for damages caused by such telephones. Given the difficulty of assembling scientific data capable of surviving *Daubert/Joiner* scrutiny, it is unlikely that any plaintiff in the future will receive compensation, even if subsequent federal agency risk assessments conclude that exposure to radio frequency radiation from cell phones does increase the risk of contracting brain cancer or some other disease.

The preceding examples suggest that entities who know that they are potential defendants in toxic tort litigation can, in a post-*Daubert/Joiner* regime, insulate themselves from liability (or at least delay liability for a very long time) by paying scientists to analyze and critique scientific studies as they are published in the scientific literature (or even before they are published). Lawyers for defendants in toxic tort litigation have staked out the position that courts should "exclude experts' opinions not only in the absence of reliable support from hard data and accepted methodologies, but also where the subject of the opinion is unsettled and a matter of substantial scientific debate."¹⁴⁰ Under this approach, a defendant may secure the exclusion of scientific expert testimony by creating the appearance of a substantial scientific debate. As the examples above have demonstrated, generating the appearance of a substantial

¹³⁷ *Id.* at 92.

¹³⁸ *Id.* at 97, 126-27.

¹³⁹ *Id.* at 141.

¹⁴⁰ Rudlin, *supra* note 35, at 336.

scientific debate is a fairly inexpensive proposition for most companies. It is not at all clear that lay judges have the wherewithal to distinguish unreliable expert testimony from reliable testimony based upon scientific studies that have been "deconstructed" by paid industry consultants.

II. THE WEAK DETERRENT EFFECT OF TOXIC TORT LITIGATION

One of the primary goals of tort law is to deter conduct that poses unreasonable risks to others.¹⁴¹ Like the prospect of punishment through criminal sanctions and administrative penalties, the threat of tort liability should encourage potential wrongdoers to behave properly. Yet, if plaintiffs rarely recover in toxic tort litigation because of difficulties in proving causation under the corpuscular approach, the tort system will send very weak signals to potential defendants to adjust their conduct so as to prevent future harm.¹⁴²

Little empirical evidence exists to support the proposition that the threat of being sued for a toxic tort has much of a deterrent effect on those who expose humans to toxic risks.¹⁴³ The absence of evidence is not at all surprising because a company has very little to gain and nothing to lose from keeping itself and the world ignorant of the risks that its products pose to others.¹⁴⁴ There is, however, much anecdotal evidence that the net result of setting a high threshold for admissibility of expert testimony in toxic substances is that potential toxic tort defendants have little to fear in cases not involving signature diseases.

When EPA issued a comprehensive risk assessment (after four years of deliberation and two reviews by EPA's Science Advisory Board) that concluded that Environmental Tobacco Smoke ("ETS") was a "Group A"

¹⁴¹ See Brennan, *supra* note 43, at 5; see also Nagareda, *supra* note 41, at 1128.

¹⁴² See Feldman, *supra* note 15, at 41 (placing the burden of proving cause-in-fact on the plaintiff in mass toxic tort cases "will not reliably promote optimal levels of safety: it will underdeter excessively risky behavior"); see also Clayton P. Gillette & James E. Krier, *Risks, Courts, Agencies*, 138 U. PA. L. REV. 1027, 1046 (1990) (alluding to "access bias," resulting from the decision by potential plaintiffs (and their potential attorneys) that a lawsuit, however justified in theory, will not be worthwhile because environmental risks are "diffuse in their impacts, and of low probability"); Klein, *supra* note 43, at 1176.

¹⁴³ See Brennan, *supra* note 43, at 6 (concluding that "[e]mpirical evidence suggests that environmental torts suits currently send a weak deterrent signal"); see also Beecher-Monas, *supra* note 8, at 1092.

¹⁴⁴ Wendy E. Wagner, *Choosing Ignorance in the Manufacture of Toxic Products*, 82 CORNELL L. REV. 773, 774, 794-95 (1997).

human carcinogen (the most potent category in the agency's lexicon), a prominent law firm wrote a memorandum for the Tobacco Institute to circulate to employers, the hospitality industry, and others who might anticipate liability actions due to cancers arising from ETS exposures. After attacking the scientific validity of the EPA report, the memo noted that:

[e]ven if EPA's report was viewed in court as establishing that ETS can cause lung cancer, that still would leave unsatisfied the other prerequisites to a successful claim. In any particular case, a nonsmoking employee who has contracted lung cancer would have to prove both that his or her lung cancer was caused by exposure to ETS (not something else) and that his or her exposure to ETS in the place under the employer's control (not other places) was the proximate cause of the illness . . . Even after the EPA's report, the plaintiff bears the burden of proof on both points, and it is a very heavy burden to satisfy. Proving causation from occasional exposure in a restaurant to other public settings would be even more difficult. Not many plaintiff's lawyers are likely to find the prospect of pursuing cases of this nature attractive.¹⁴⁵

Company documents produced during toxic tort litigation involving the W.R. Grace insulating product Monokote revealed that the company knew in 1977 that the product contained low levels of a relatively rare form of asbestos called tremolite, but elected not to label the product or otherwise inform consumers of that fact out of fear that labeling would severely dampen sales of the product. The risk of liability to workers and others of contracting cancer from exposure to Monokote was, in the view of the company's lawyers, only "moderate."¹⁴⁶ A 1977 strategy memorandum concluded that "it seems unlikely that bona fide

¹⁴⁵ Memorandum, Legal Significance of EPA's Classification of ETS as a "Group A" Carcinogen (Jan. 18, 1993), PM Bates No. 2024103846. A memorandum of the same date from the Tobacco Institute to various recipients indicates that this memorandum was prepared by the law firm of Covington & Burling. Memorandum from Patrick B. Donoho & Ronald C. Morris, to TI Legislative Consultants (Jan. 18, 1993), PM Bates No. 2047045168.

¹⁴⁶ Michael Moss & Adrienne Appel, *Company's Silence Countered Safety Fears About Asbestos*, N.Y. TIMES, July 9, 2001, at A1.

cases of personal harm could be well documented considering the pattern of use and exposure levels of our customers.”¹⁴⁷ Thus, the threat of potential liability had little impact on the corporate decision to continue to market the product without warnings. Although W.R. Grace recently declared bankruptcy as a result of liabilities incurred from litigation involving earlier products that contained much higher levels of a more potent form of asbestos, it may never be held accountable for its carefully calculated decision to preserve its markets at the expense of putting its customers at risk.¹⁴⁸

An absolute requirement that the toxic tort plaintiff present expert testimony based upon a virtually flawless epidemiological study demonstrating a relative risk of 2.0 or greater will effectively eviscerate tort law as a deterrent to corporate misbehavior. Although most new chemicals receive some sort of *in vitro* testing prior to marketing, there is no general requirement that any testing be undertaken.¹⁴⁹ Most epidemiological studies of chemicals are undertaken long after the chemical has entered commerce and humans have been exposed for a substantial period of time. Because epidemiological studies are very expensive, they exist for only a very few chemicals.¹⁵⁰ Once evidence that a chemical may be toxic begins to accumulate, the manufacture is likely to take measures to limit exposure to it, thereby reducing the number of exposed individuals available for inclusion in epidemiological investigations.¹⁵¹ No sane attorney is likely to take a case that has such a small prospect of success and such a large potential for bankrupting plaintiff's counsel.

¹⁴⁷ *Id.*

¹⁴⁸ See also Wagner, *supra* note 144, at 823-24 (citing evidence of “[c]orporate concealment of adverse testing results occurring in the marketing of asbestos, the Dalkon Shield, and breast implants” as “dramatic evidence that at least some corporations perceive liability for certain types of latent harms as either unlikely or a risk that should be significantly discounted as long as their own in-house research is concealed”).

¹⁴⁹ The Toxic Substances Control Act only requires that a manufacturer of a new chemical provide EPA with advance notice of its intent to market the product. Toxic Substance Control Act, 15 U.S.C. § 2604 (1998). EPA may require testing only upon making one of several findings related to the potential for the substance to cause adverse environmental effects or to result in large exposure to human beings. See *id.* § 2603. I am aware of no instance in which EPA has required a company to conduct an epidemiological study.

¹⁵⁰ *Ambrosini v. LaBarraque*, 101 F.3d 129 (D.C. Cir. 1996), *cert. dismissed*, 520 U.S. 1205 (1997); see Geistfeld, *supra* note 90, at 1013.

¹⁵¹ See Gottesman, *supra* note 8, at 767.

A legal system that demands a well-conducted epidemiological study demonstrating a relative risk of greater than 2.0 as a necessary condition to a plaintiff's toxic tort recovery is a legal system that is willing "to tolerate or even encourage a high degree of uncertainty about the dangers of some products, to reward manufacturers for ignoring risk, and to require consumers to bear more of the burden of scientific uncertainty than manufacturers."¹⁵² It is, in short, a system that is unwilling to assign to tort law the function of holding companies accountable for the consequences of their misbehavior, however culpable. It is my contention that a legal regime that leaves corporate accountability entirely up to the regulatory agencies and district attorneys enforcing the criminal law will not easily withstand public scrutiny in the upcoming accountability crisis.

III. LINKING CULPABILITY TO CAUSATION IN A MIXED REGIME

The essence of the impending accountability crisis is the growing public realization that major participants in the global economic marketplace who have engaged in culpable, even outrageous conduct frequently escape both punishment and their obligation to make their victims whole. Although outrageous conduct is no less outrageous merely because no one has been demonstrably injured,¹⁵³ the tort reparations regime has traditionally demanded that a plaintiff demonstrate that the defendant's act or failure to act caused some damage to the plaintiff as a precondition to asking the law to shift wealth from a culpable defendant to that plaintiff's pocket. The substantive law of torts, however, need not require courts to adopt a corpuscular approach toward admitting scientific evidence on causation. In my view, judicial adherence to the corpuscular approach is not only unscientific, in that it is not how scientists go about drawing scientific conclusions,¹⁵⁴ it is also contrary to the letter and spirit

¹⁵² Finley, *supra* note 8, at 368.

¹⁵³ See Nagareda, *supra* note 10, at 1790 ("[O]utrage may arise—for instance, over defendants' suppression of information about product risk—wholly apart from whether the product is ultimately proven as a scientific matter to cause the disease from which plaintiffs suffer.").

¹⁵⁴ See Gottesman, *supra* note 8, at 772 (arguing that "[i]t is not intrinsically 'unscientific' for experienced professionals to arrive at a conclusion by weighing all available scientific evidence—this is not the sort of junk science with which *Daubert* was concerned"); Sanders, *supra* note 22, at 1416-17 (arguing that "from a scientific validity perspective it would be preferable to form one's opinion about [toxic substances] based

of the original Rule 702. That rule, in the Court's words, was intended to "allow district courts to admit a somewhat broader range of scientific testimony than would have been admissible under *Frye*." The courts have clearly steered things in the opposite direction.

To some extent this fairly strong judicial tendency to exclude controversial testimony may be attributable to an understandable, if inappropriate simplicity that has pervaded the post-*Daubert/Joiner* judging process. Guided by the Federal Judicial Center's *Reference Manual on Scientific Evidence*, courts may be demanding a degree of perfection in the design and execution of the scientific studies underlying scientific expert testimony that has seldom characterized the scientific process and has never characterized the process of practical decisionmaking in other contexts involving scientific uncertainty.¹⁵⁵ Judges are not scientists and they have many other things to do with their limited time and resources. The suggestion, inherent in the corpuscular approach, that careful attention to the briefs and a quick read through the *Reference Manual* can provide sufficient understanding of subtle scientific concepts to warrant wholesale exclusion of scientific information from the scientific assessment process betrays an unwarranted degree of confidence in the scientific sophistication of the federal judiciary.¹⁵⁶ To allow legally trained judges, however intelligent, to serve as arbiters of "sound science" is to invite injustice and, in the final analysis, ridicule.¹⁵⁷ The end result will be, and, frankly, should be, a decline in the respect that other institutions and the public afford the judiciary.

on all of the available evidence: epidemiological, animal studies, in vitro and perhaps even structure-activity").

¹⁵⁵ See Beecher-Monas, *supra* note 8, at 1076-77.

¹⁵⁶ One solution to the problem of judicial ignorance is better guidance to the judges on the nature of scientific proof and techniques for evaluating scientific information, and legal scholars have offered many suggestions for filling that need. One of the most thoughtful attempts to provide an heuristic for determining the admissibility of scientific evidence after *Daubert* is Professor Beecher-Monas' "primer." Beecher-Monas, *supra* note 39. See also Bert Black et al., *Science and the Law in the Wake of Daubert: A New Search for Scientific Knowledge*, 72 TEX. L. REV. 715, 782-86 (1994). I have concluded, however, that the problem runs deeper than judicial ignorance.

¹⁵⁷ Courts can acquire subtler input by assembling a panel of scientists with relevant expertise to aid the judge in evaluating subtle scientific arguments. In addition to being resource-intensive and time consuming, this vehicle for educating the judge still raises questions about the ability of consensus-oriented groups to provide an adequate translation of the nature of the scientific disagreements. See Laurens Walker & John Monahan, *Scientific Authority: The Breast Implant Litigation and Beyond*, 86 VA. L. REV. 801, 813-17 (2000).

The post-*Daubert/Joiner* judicial eagerness to exclude expert testimony may also reflect a growing distrust of juries on the part of the current personnel on the bench.¹⁵⁸ Judges may not believe that lay jurors can assign the proper weight to the scientific studies underlying expert testimony, even with the aid of vigorous cross-examination. The corpuscular approach, with its binary view of the validity of scientific studies, "stands in stark contrast to traditional and proper practice, which sees the admissibility of evidence as a question quite distinct from the sufficiency of evidence to meet a plaintiff's burden of proof."¹⁵⁹ A similar judicial distrust of the capacity of jurors to give proper weight to scientific evidence may motivate the courts to exclude expert testimony based upon animal studies. There is, of course, a certain arrogance in the implicit assumption that lay judges can do a significantly better job than lay jurors of determining the validity of scientific studies and the reliability of expert testimony based upon scientific studies.

The judges may, however, be influenced by more than skepticism about the intellectual capacity of jurors. Careful observers have noted that jurors in toxic tort cases "might overlook weak evidence of causation when confronted with strong evidence of misconduct on the part of the defendant."¹⁶⁰ It is, in my view, highly likely that the judiciary's eager acceptance of the *Daubert/Joiner* corpuscular approach is motivated by a reluctance to allow juries to commingle separate legal questions in this fashion. Yet the decision to prevent such commingling is in fact a normative policy judgment. There is nothing *per se* irrational about a decisionmaker wanting to know the whole story when evaluating the consequences of his or her decision. The constraints that the law places on juries knowing the full story are generally motivated by "profoundly normative" policy concerns "about the social allocation of risk and who

¹⁵⁸ See Cutler, *supra* note 37, at 212-13.

¹⁵⁹ Finley, *supra* note 8, at 337.

¹⁶⁰ Nagareda, *supra* note 41, at 1168. See Elliott, *supra* note 38, at 788 ("[T]oday's toxic tort cases are really white-collar crime cases; they have a lot more to do with the morality of the defendants' conduct than they do with estimating dose-response relationships."); Joseph Sanders, *From Science to Evidence: The Testimony on Causation in the Bendectin Cases*, 46 STAN. L. REV. 1 (1993) (noting that in the Bendectin litigation "plaintiffs often attempted to commingle elements, thereby bolstering weak evidence on causation with stronger proof of breach of duty and damages"); Wagner, *supra* note 144, at 828-29 (finding it "more than coincidental that in those cases in which juries have awarded damages in spite of weak causation evidence, the defendant manufacturers' negligence in testing often rose to the level of gross negligence or recklessness sufficient to support the simultaneous award of punitive damages").

should bear the burden of scientific uncertainty.”¹⁶¹ The current judicial tendency to exclude scientific expert testimony out of fear of jury commingling reflects a policy of shielding culpable defendants from liability for harm they did not clearly cause. Whether it is a good policy or a bad one is a matter of legitimate debate.

It appears that a substantial number of sitting judges, persuaded by a decade’s worth of well-publicized “junk science” claims by industry-sponsored tort reformers, are raising the bar to the admissibility of scientific testimony in toxic torts cases out of a fear that the threat of jury awards will impede economic development.¹⁶² Witness Justice Breyer’s invocation, in his *Joiner* concurring opinion, of the trial judge’s obligation to “assure that the powerful engine of tort liability, which can generate strong financial incentives to reduce, or to eliminate, production, points towards the right substances and does not destroy the wrong ones.”¹⁶³ The recent judicial embrace of the corpuscular approach and the manifest judicial hostility to expert testimony based on animal studies represents a policy judgment not to subject manufacturers of valuable products to the “powerful engine of tort liability” for substances that have not clearly been shown to cause toxic effects in humans. This judicially adopted policy of erring on the side of protecting the assets of risk-producing enterprises varies dramatically from the policy of erring on the side of safety employed by federal regulatory agencies operating under federal health and safety statutes.¹⁶⁴

The quiet judicial tort reform achieved by *Daubert/Joiner* predates the extraordinary revelations during the recent tobacco litigation of the extent to which some products liability defendants are prepared to go to manipulate scientific data to avoid accountability. As time passes and more apparently deserving plaintiffs are deprived of their day in court by aggressive admissibility rulings, the role that *Daubert/Joiner* has played in the resulting accountability crisis will become apparent. The calls that are already being heard to repair a broken system of business-oriented judicial lawmaking will intensify as more stories of unpunished corporate malfeasance impinge upon the public consciousness.¹⁶⁵

¹⁶¹ Finley, *supra* note 8, at 335-36.

¹⁶² See Gottesman, *supra* note 8, at 759.

¹⁶³ Gen. Elec. Co. v. Joiner, 522 U.S. 136, 148-49 (1997). See Finley, *supra* note 8, at 344.

¹⁶⁴ The policy judgment also varies from the policies underlying the substantive law of torts in most states. See Gottesman, *supra* note 8, at 762.

¹⁶⁵ See BUCCINO ET AL., *supra* note 6.

One possible "quick fix" to forestall the upcoming accountability crisis would be for Congress to amend the Federal Rules of Evidence to remove (or greatly reduce) the trial judge's screening role. Unfortunately, it is probably too late in the day to attempt to induce the courts to abandon the corpuscular approach and restore the power of juries to assess the weight of the evidence through a more liberal interpretation of Rule 702. With surprising speed, the lower courts wove *Daubert/Joiner* into the fabric of tort law, and it will be difficult to undo those changes with language that will itself be open to judicial interpretation. Indeed, Rule 702 has recently been amended to incorporate the *Daubert/Joiner* tests.¹⁶⁶ In any event, subtle changes in the text of obscure rules will do little to alleviate public pressure for substantive reform. More radical institutional surgery will probably be required.

In the remainder of this article, I propose an institutional reform that will draw upon the ability of federal agencies to employ a weight-of-the-evidence approach toward scientific proof and that will acknowledge the tendency of the jury to commingle evidence of causation with evidence of culpability in basing verdicts on their conception of the "whole story." The regime described below retains the basic structure of the tort regime, but incorporates an administrative agency's assessment of the scientific evidence and employs rebuttable presumptions that vary with the degree of the defendant's culpability.

A. *The Possibility of Presumptions in Tort Litigation*

A mandatory legal presumption is a proven technique for arresting the tendency of judges to take decisionmaking power away from juries. If judicial adoption of a corpuscular approach to admissibility in combination with the common law rule that the plaintiff has the burden of proof means as a practical matter that plaintiffs can never win,¹⁶⁷ then one solution may be to lighten the burden of proof with a presumption.¹⁶⁸

¹⁶⁶ FED. R. EVID. 702. The Advisory Committee Notes state that Rule 702 was amended "in response to" *Daubert* and "to the many cases applying *Daubert*." *Id.* advisory committee's note.

¹⁶⁷ See Feldman, *supra* note 15, at 44 (suggesting that this is the case).

¹⁶⁸ See *Allen v. United States*, 588 F. Supp. 247, 438, 439 n.197 (D. Utah 1984) (employing a presumption in favor of causation in the context of egregious conduct on the part of the United States government in exposing plaintiffs to heavy doses of radiation).

Courts frequently create presumptions to correct imbalances caused by one party's greater access to reliable evidence.¹⁶⁹ Some presumptions are simply created by courts to "favor certain parties or claims for social and economic reasons."¹⁷⁰ Administrative compensation regimes, for example, frequently erect rebuttable presumptions to advance particular social goals.¹⁷¹ Courts also erect presumptions on particular contested issues as a way of sanctioning improper conduct that occurs during

without their knowledge). See generally Brinker, *supra* note 42, at 1321-23; Gottesman, *supra* note 8, at 779-80.

Professor Geistfeld argues that burden shifting is not necessary to alleviate unfairness in products liability litigation regarding toxic products. He believes that the plaintiff can ensure that her case comes before the jury by claiming that the manufacturer failed adequately to warn. Once that claim is proved, the plaintiff can produce evidence of increased risk of contracting a disease as part of the proof of damages. Geistfeld, *supra* note 90, at 1014. While Professor Geistfeld persuasively argues that the courts should not require epidemiological proof of a relative risk of 2.0 at the damages phase of a failure to warn products liability case, his solution leaves out a vast number of toxic tort plaintiffs. Plaintiffs who allege damage due to involuntary exposure environmental contaminants, for example, do not have an obvious failure to warn claim. In these cases, Professor Geistfeld suggests that it is not especially unfair to place the burden of proof on the plaintiff, because to shift the burden would be equally unfair to the innocent defendant. *Id.* at 1023-32. This may be true, but it does not address intermediate solutions short of placing a burden of demonstrating a relative risk of greater than 2.0 by epidemiological evidence. Courts could, for example, allow the jury to find causation based upon animal studies or epidemiological studies demonstrating a relative risk of less than 2.0, a possibility that Professor Geistfeld recognizes. *Id.* at 1032-36. Or courts could, as suggested here, demand that the plaintiff establish a high degree of culpability on the part of the defendant as a precondition to burden shifting, thus alleviating any unfairness to innocent defendants.

¹⁶⁹ See, e.g., KEETON ET AL., *supra* note 42, § 39, at 242 (explaining the doctrine of *res ipsa loquitur*).

¹⁷⁰ SUPERFUND § 301(E) STUDY GROUP, INJURIES AND DAMAGES FROM HAZARDOUS WASTES—ANALYSIS AND IMPROVEMENT OF LEGAL REMEDIES (July 1982) reprinted in *Hearings on Compensation for Exposure to Hazardous Substances Before the Subcomm. on Investigations and Oversight of the House Comm. on Sci. and Tech.*, 97th Cong. 154, 182 (1982) [hereinafter CERCLA SUPERFUND § 301(E) STUDY GROUP REPORT].

¹⁷¹ For example, the Federal Coal Mine Health and Safety Act of 1969, as amended, erects a rebuttable presumption that any miner suffering from black lung disease (pneumoconiosis) who can demonstrate that he or she has been exposed to coal mines for more than ten years contracted the disease as a result of that exposure. 30 U.S.C. §§ 801-962, 921(c) (1977). See CERCLA SUPERFUND § 301(E) STUDY GROUP REPORT, *supra* note 170, at 190-91.

litigation.¹⁷² Several commentators have offered creative suggestions for creating causation presumptions in various toxic torts contexts.¹⁷³

1. The Berger Proposal

In a perceptive essay on general causation in toxic torts, Professor Margaret Berger criticizes the tendency of the lower courts to apply *Daubert* strictly to exclude testimony on general causation when the underlying science is unclear.¹⁷⁴ In her view, proponents of the strict approach and reformers who would lodge decisionmaking power in entities other than the jury "appear unconcerned with protecting persons from harm while there is insufficient or indeterminate knowledge regarding the potential adverse effects of a product, or with appraising the need for interim protective measures while uncertainty persists."¹⁷⁵ Because "[c]ausation knocks out the link between culpability and liability,"¹⁷⁶ the plaintiff's burden of proving causation in toxic tort cases prevents the liability regime from discouraging negligent corporate conduct. Professor Berger therefore proposes a "new tort that conditions culpability on the failure to develop and disseminate significant data needed for risk assessment."¹⁷⁷ She would accomplish this by expanding the scope of personal liability for corporate officers and by holding a company liable to "those put at risk by its action, without regard to injuries that eventually ensue."¹⁷⁸

As a practical matter, Professor Berger's proposal would impose liability upon products liability defendants without requiring plaintiffs to

¹⁷² For example, an appropriate sanction for unauthorized document destruction or similar discovery abuse might be an order reversing the burden of proof on a particular issue in the trial. See, e.g., *Sweet v. Sisters of Providence*, 895 P.2d 484, 490-92 (Alaska 1995) (addressing missing medical records resulting from defendant's misconduct by shifting the burden of proof on negligence and causation to defendant); see also RALPH NADER ET AL., *NO CONTEST: CORPORATE LAWYERS AND THE PERVERSION OF JUSTICE IN AMERICA* 138-39 (1996) (raising the burden of proof as sanction for document destruction).

¹⁷³ Finley, *supra* note 8, at 367 n.102 (describing suggestions of three scholars for shifting the burden of proof on causation in toxic torts contexts).

¹⁷⁴ Berger, *supra* note 7.

¹⁷⁵ *Id.* at 2131.

¹⁷⁶ *Id.* at 2134.

¹⁷⁷ *Id.* at 2140.

¹⁷⁸ *Id.* at 2134.

prove general causation.¹⁷⁹ A court would impose liability on the manufacturer of a product if it was negligent in failing “to provide substantial information relating to risk.”¹⁸⁰ Conversely, a defendant would be relieved of liability if it “met the required standard of care for developing and disseminating information relevant to risk.”¹⁸¹ In addition, a defendant could avoid liability by proving that the product did not cause the plaintiff’s disease, and it could reduce liability by proving that the plaintiff’s disease was at least partially attributable to some other toxic exposure.¹⁸² Finally, actual damages (perhaps excluding compensation for pain and suffering) would be awarded through an accompanying administrative regime,¹⁸³ and punitive damages would not be allowed at all.¹⁸⁴

Although the Berger proposal represents a significant improvement upon the existing post-*Daubert/Joiner* regime, it may not be the best general solution to the causation problem in toxic tort cases. First, the scope of Professor Berger’s proposal is necessarily limited to products liability litigation. Its primary purpose is to use tort law to motivate corporations to collect and disseminate risk-related information about the products that they market. It does not speak at all to corporate responsibility for the by-products that they release into the environment.

Second, the Berger proposal presumes that there is some agreed-upon standard for the kind and amount of risk-related testing that a manufacturer should employ in the case of any given product. The clearest cases of properly imposed liability for failure to test would be situations in which a company conducted testing but did not report the results to relevant regulatory agencies or the public and situations in which a company initiated testing but terminated it in anticipation of receiving bad results.¹⁸⁵ In both of these instances, however, the corporation has in a sense defined its own benchmark and failed to come up to that standard. A testing regime prescribed by an administrative agency could provide the needed benchmark, but regulatory agencies rarely specify in detail all of the tests that must be performed, preferring instead to make such

¹⁷⁹ *Id.* at 2143.

¹⁸⁰ Berger, *supra* note 7, at 2143.

¹⁸¹ *Id.*

¹⁸² *Id.* at 2145.

¹⁸³ *Id.*

¹⁸⁴ *Id.*

¹⁸⁵ Berger cites RJR Corporation’s dismantling of a rabbit testing laboratory. *Id.* at 2142.

determinations on a "tiered" or case-by-case basis.¹⁸⁶ Thus, in cases in which the manufacturer did not define its own benchmark, the jury would be in the position of determining the proper testing regime for toxic substances, no doubt with the aid of *Daubert*-reliable expert witnesses.

Third, Professor Berger's plan is vague on how the courts would determine which diseases are compensable. Surely, she does not mean to allow any person with any disease to draw funds from a culpable entity merely upon a showing that he or she was exposed to the product at issue. Yet without adequate testing and/or epidemiological data, it will be difficult to know what diseases are associated with exposures to the product at issue. It may be that the accompanying administrative regime will adequately address this problem, but the scope of the proposal's applicability is a question that its implementers will need to address.

Fourth, while Professor Berger makes a plausible case for the proposition that eliminating general causation is a fair trade-off for a similar elimination of punitive damages, the absence of the punitive damages allure may discourage plaintiffs' attorneys from expending the resources that are required to conduct discovery and put on a trial relevant to the defendant's misconduct.¹⁸⁷ In the context of toxic torts involving exposure to a by-product, one would expect the courts in fairness to potential defendants to demand at least a showing that the plaintiff was exposed to the defendant's by-product. Such a showing would presumably still be required as a matter of the plaintiff's burden of demonstrating specific causation. As Jan Schlichtmann's well-documented experience with the contamination of water wells in Woburn, Massachusetts demonstrates,¹⁸⁸ this frequently requires additional expensive proof that trial lawyers will be reluctant to undertake in the absence of a promise of a high payout by way of punitive damages.¹⁸⁹

¹⁸⁶ See Thomas O. McGarity, *Politics by Other Means: Law, Science, and Policy in EPA's Implementation of the Food Quality Protection Act*, 53 ADMIN. L. REV. 103, 140-41 (2001).

¹⁸⁷ See Berger, *supra* note 7, at 2149-50 (recognizing that "[i]t would, of course, be costly and time consuming to establish a defendant's failure to exercise due care in obtaining and disseminating substantial information about risk").

¹⁸⁸ See HAAR, *supra* note 14.

¹⁸⁹ The fear that out-of-control juries will arbitrarily award punitive damages seems misplaced. A recent study of judges and juries concluded that juries do not differ greatly from judges in determining whether punitive damages should be allowed and in determining the amount of punitive damages that should be awarded. See William Glaberson, *A Study's Verdict: Jury Awards Are Not Out of Control*, N.Y. TIMES, Aug. 6, 2001, at A9.

2. The *Broin* Settlement

The omnibus settlement in the *Broin* litigation between a large class of airline flight attendants and the tobacco industry adopted a presumption/rebuttal approach that was consciously designed to provide a substantive advantage to plaintiffs in the individual actions that followed the omnibus settlement. Although the tobacco company defendants agreed to pay the class plaintiffs' attorneys' fees and establish a \$300 million fund for research ETS,¹⁹⁰ the settlement did not finally resolve any individual claims for damages attributable to ETS exposure. Individual members of the class retained the right to bring individual claims for compensatory (but not punitive) damages against the defendant tobacco companies based on any theory other than fraud, misrepresentation, conspiracy, or intentional tort. With respect to any claim seeking damages for lung cancer, chronic bronchitis, emphysema, chronic obstructive pulmonary disease, or chronic sinusitis, the burden of proof on the question of general causation (i.e., whether ETS "can cause one of the above-described diseases")¹⁹¹ would fall on the defendant and the jury would be so instructed. The settlement then provided that "[i]n all other respects, including the issue of whether an individual plaintiff's disease was caused by ETS ('specific causation'), the ordinary burdens of proof" would remain unaltered.¹⁹²

By the time that the extended limitations period provided for in the settlement agreement had expired, approximately 3125 individual flight attendants had filed individual claims.¹⁹³ Prior to the first trial, the original *Broin* trial judge issued an interpretation of the agreement that limited the issues at trials involving plaintiffs with one or more of the five named diseases (lung cancer, chronic bronchitis, emphysema, chronic obstructive pulmonary disease, and chronic sinusitis) to the questions of specific causation and the extent of damage. The court read the settlement agreement to waive the plaintiff's otherwise applicable burden of proving a breach of the relevant standard of care and proximate causation.¹⁹⁴ This

¹⁹⁰ *Broin v. Philip Morris Cos.*, Case No. 91-49738CA(22), ¶¶ 7, 10 (Fla. Cir. Ct. Oct. 9, 1997) (Settlement Agreement).

¹⁹¹ *Id.* ¶ 12(d).

¹⁹² *Id.*

¹⁹³ See Appellants' Initial Brief at 1, *Philip Morris Cos. v. Jett* (3d Fla. Dist. Ct. App. 2001) (Civ. No. 3D00-3189).

¹⁹⁴ See *id.* at 9.

holding was appealed and most cases were held in abeyance pending the outcome of the appeal.¹⁹⁵

One case involving a dying plaintiff, however, was allowed to go forward. In that case, brought by flight attendant Marie Fontana, the jury at the end of a three-week trial held in favor of the four defendant tobacco companies.¹⁹⁶ The *Fontana* case may have been unique, because the plaintiff's primary lung-related ailment was an affliction called sarcoidosis, an ideopathic disease that scientists have thus far not associated with cigarette smoking.¹⁹⁷ The plaintiffs had hoped to prove to the jury that the plaintiff also suffered from emphysema and chronic obstructive pulmonary disease, two diseases which (according to the terms of the *Broin* settlement) were presumptively caused by environmental tobacco smoke, and that the sarcoidosis was exacerbated by those diseases.¹⁹⁸ Although the defendants put on expert testimony to rebut the general causation presumption, the court held as a matter of law that the testimony had not rebutted that presumption.¹⁹⁹

¹⁹⁵ The defendants argued on appeal that the court's holding is difficult to square with the language in paragraph 12(d) of the settlement agreement that says: "in all other respects, including the issue of whether an individual plaintiff's disease was caused by ETS ('specific causation'), the ordinary burdens of proof applicable to any Retained Claims shall remain unaltered." *Id.* at 14. The plaintiffs argued on appeal that the intent of the parties in settling the *Broin* litigation was to bring Stage I of that trial to an end and to allow Stage II to proceed on a case-by-case basis with the issues being limited to those concerning the individual plaintiffs. Appellee's Answer Brief at 4, Philip Morris Cos. v. Jett (3d Fla. Dist. Ct. App. 2001) (Civ. No. 3D00-3189). Since issues common to all of the plaintiffs had been excluded from Stage II of *Broin*, the parties intended for such issues to be excluded from each of the individual trials that were governed by the settlement. *Id.* They further noted that the trial court and the court of appeals would never have approved the *Broin* settlement if it had required the plaintiffs to prove all of the elements of their toxic tort claims except for general causation. *Id.* at 12-15.

¹⁹⁶ *Fontana v. Philip Morris Cos.*, Civ. No. 00-01731CA (09) (Fla. Cir. Ct. Mar. 19, 2001).

¹⁹⁷ See Terri Somers, *Experts: Tobacco Lawsuits Still Viable; Secondhand Smoke Cases Will Be Pursued*, FORT LAUDERDALE SUN-SENTINEL, Apr. 8, 2001, at B1.

¹⁹⁸ Myron Levin, *Tobacco Wins First In Series Of Secondhand Smoke Suits*, L.A. TIMES, Apr. 6, 2001, at C3; see also Terri Somers, *Jury Clears Big Tobacco*, FORT LAUDERDALE SUN-SENTINEL, Apr. 6, 2001, at A1.

¹⁹⁹ The trial court's initial jury instruction made it clear that "there is a rebuttable presumption that exposure to secondhand smoke, or environmental tobacco smoke, is harmful to one's health and can cause chronic bronchitis, emphysema, chronic sinusitis and chronic obstructive pulmonary disease in healthy nonsmokers," but "it is the plaintiff's burden to prove that, 1, she has one of the diseases enumerated above or an aggravation of an existing condition and, 2, that her exposure to secondhand smoke was

The plaintiff's counsel were apparently unable to persuade the jury that ETS was the specific cause of one of the diseases named in the *Broin* settlement and that her sarcoidosis was exacerbated by that disease. This was no doubt attributable to the fact that the plaintiff's treating physician's original diagnosis had not included one of the settlement diseases and the fact that the defendant's expert testified that the plaintiff did not suffer from any of those diseases.²⁰⁰ Juror Melvin Raul Galeano, in post-trial comments to the press, related that "[w]e just agreed that there was not enough evidence to swing us to her favor."²⁰¹ He also complained that the only testimony that the plaintiffs elicited from a treating physician came as a rebuttal to a chorus of defense witnesses.²⁰²

Finally, although the plaintiff's counsel in *Fontana* did introduce limited evidence of the tobacco companies' knowledge of the harmful effects of ETS, the defendants' culpability was not a major focus of the trial because the plaintiff adhered to the position taken by all of the other plaintiffs that they were not required to put on evidence of culpability. The plaintiff, therefore, did not produce anything of the caliber of the "smoking gun" memos that characterized the plaintiffs' well-publicized victory in the same court in the *Engel* direct smoking trial.²⁰³

Because of the opening that it provided for the defense counsel to focus the jury's attention on the fact that the plaintiff's primary life-threatening disease was not associated with smoking, the *Fontana* case is probably not a good case for testing the viability of a burden-shifting device as a tool for punishing culpable conduct on the part of companies who subject the public to environmental risks. Whether the *Broin* settlement claims will provide a crucible for testing the workability of a general causation presumption may also depend upon the outcome of the

the specific cause of the disease or aggravation claimed by her." Record at 400, *Fontana* (Civ. No. 00-01731CA (09)). At the end of the trial, the court instructed the jury "as a matter of law," that ETS "does cause chronic bronchitis, emphysema and/or chronic obstructive pulmonary disease ("COPD") in health nonsmokers." *Id.* at 3077.

²⁰⁰ *Id.* at 2388-2424.

²⁰¹ Somers, *supra* note 198, at A1.

²⁰² One of the attorneys for the plaintiffs explained that they did not call the primary treating physician because he had not included chronic obstructive pulmonary disease as one of the plaintiff's afflictions in his original report, and the plaintiff's attorneys did not want to risk embarrassing one of their own witnesses. *Fontana v. Philip Morris Cos.*, Civ. No. 00-01731CA (09) (Fla. Cir. Ct. Mar. 19, 2001).

²⁰³ See Maya Bell, *\$145 Billion: Tobacco Giants Get Burned*, ORLANDO SENTINEL, July 15, 2000, at A1; see also Rick Bragg, *Tobacco Lawsuit in Florida Yields Record Damages*, N.Y. TIMES, July 15, 2000, at A1.

appeal. If counsel for the remaining three thousand flight attendants must prove negligence and proximate causation, as well as rebut the defendants' proof on cause-in-fact, then the trials may take considerably longer than three weeks to reach completion, and each individual case will consume considerably greater resources.

Even if the plaintiffs prevail in the appeal, several potential problems remain with the *Broin* presumption-rebuttal approach. First, like Professor Berger's approach, the *Broin* settlement approach begs the question of the range of diseases to which it is applicable. In *Broin*, the settlement itself defined the scope of its applicability. The list of compensible diseases grew out of a very lengthy trial in which a great deal of expert evidence was presented about the diseases potentially caused by ETS exposure. This solution, however, probably cannot be extrapolated to all toxic torts. Unless the regime is willing to allow any exposed plaintiff who complains of any disease to shift the burden of proving general causation to the defendant, then some entity will have to determine which diseases do raise the presumption. This is by no means an insurmountable difficulty, but it must be addressed in any regime that cannot depend upon the parties themselves to decide which diseases are in this sense "compensible."

Second, the absence of punitive damages as a lure to plaintiffs' attorneys may stand in the way of the *Broin* settlement approach as a general solution to the causation problem in toxic torts cases. The seven law firms that have agreed to represent the more than 3000 flight attendants in the litigation provided for in the *Broin* settlement are apparently prepared to put on hundreds of cases on specific causation in an assembly-line fashion. The hope is, however, that once a large enough number of cases are decided in favor of plaintiffs, the defendants will be willing to settle the remaining cases on terms acceptable to the law firms and the remaining plaintiffs. Given a guaranteed flow of cases with a high probability of success, the assembly line approach may provide a sufficient inducement for attorneys to put resources into trials without the hope of receiving punitive damage awards. The prospect of punitive damages may be necessary in more run-of-the-mill situations to attract plaintiffs' attorneys.

Finally, the *Broin* settlement, as currently interpreted by the district judge, assigns a very small role, if any, to the defendant's culpability. The defendants' misconduct was presumably one of the reasons that the original attorneys for the *Broin* class plaintiffs were able to extract concessions from the defendants during the settlement negotiations. If the plaintiffs prevail on appeal, then the coupling of culpability with causation

will have occurred during the settlement process. Culpability may have to play a larger and more clearly defined role in a more general tort regime that does not grow out of a particular settlement.

3. The Superfund Section 301(e) Study Group Proposal

The Comprehensive Environmental Response, Compensation, and Liability Act of 1980 ("CERCLA") created a Study Group to examine alternative reparations regimes for compensating victims of hazardous wastes.²⁰⁴ The Study Group, which was composed of law professors and attorneys from the plaintiff and defense bars, recommended that Congress enact legislation establishing a two-tiered system for compensating persons injured by exposure to hazardous wastes.²⁰⁵ Modeled on many state workers' compensation regimes, the first tier would be administered by a federal agency and would provide rapid reimbursement of medical expenses and limited compensation (\$2000 per month cap) for loss of earnings. Plaintiffs could elect, however, to pursue the second tier, which would consist of a common law trial under slightly modified procedural rules.

The Study Group's Tier One compensation regime would be managed by a federal agency created by Congress and funded through a special tax on petroleum and the generation of hazardous wastes.²⁰⁶ It would compensate persons who suffered injury due to non-occupational exposure to hazardous wastes for their medical expenses and provide limited compensation for loss of earnings. In order to be entitled to recovery, a claimant would have to show that he or she was exposed to a hazardous waste that was known to cause injury, that the plaintiff suffered disease or injury of the sort that the hazardous waste was known to cause, and that the exposure caused the disease or injury. In this regard, the claimant could refer to a "toxic substances document" prepared by a federal agency and based upon "scientific data," including information on particular hazardous wastes or substances accumulated during the "exercise of agency responsibilities" and "evidence collected in connection with past or pending cases."²⁰⁷ A claimant who showed that he or she had

²⁰⁴ 42 U.S.C. § 9651(e) (2001).

²⁰⁵ See CERCLA SECTION 301(E) STUDY GROUP REPORT, *supra* note 170.

²⁰⁶ *Id.* at 207.

²⁰⁷ *Id.* at 176-77. Toxic substances documents would be prepared by a suitable federal agency and published in the Federal Register, along with supporting documents and

been exposed to a hazardous waste would be entitled to a rebuttable presumption that an injury or disease was caused by that exposure if, according to a "toxic substances document," the injury or disease was "known to result" from exposure to that hazardous waste.²⁰⁸

A claimant could, however, opt out of the Tier One administrative regime and pursue a Tier Two claim in court under relaxed procedural rules governing the time of accrual of actions, joinder of parties, apportionment, and proof of causation.²⁰⁹ The Study Group stopped short, however, of recommending that state courts apply the causation presumption based upon agency-prepared "toxic substances documents."²¹⁰ A majority of the Study Group concluded that while such a presumption would be warranted in an administrative regime in which speedy decisionmaking was important and in which damages were limited, it should not be extended to ordinary tort litigation.²¹¹ Some members feared that requiring such a presumption in plenary tort actions would encourage claimants to forego the administrative regime in favor of the potentially larger final award in the tort action.²¹² At the same time, the Study Group concluded that the "toxic substance documents" might be freely introduced as evidence in Tier Two jury trials in the same manner as learned treatises and other authoritative works without giving rise to a rebuttable presumption.

The Superfund Study Group's recommendations go a long way toward solving the scope of applicability problem by assigning that role to a federal agency. The agency-prepared "toxic substances document" would identify those diseases with which exposures to a particular toxic substance were associated. Claimants in the Tier One administrative regime would then benefit from a presumption of general causation for the diseases identified in the documents. The proposal is quite vague, however, on the role that the defendant's culpability would play in the Tier One compensation regime. Apparently, the plaintiff would be required only to prove that the defendant contributed to the hazardous waste to which the plaintiff was exposed in order to enter the Tier One process.

references to published scientific studies, and the agency would consider public comments prior to finalizing the documents. *Id.*

²⁰⁸ See *id.* at 176, 179. Either the claimant or the fund could obtain judicial review of the compensation award. See *id.* at 214.

²⁰⁹ *Id.* at 217.

²¹⁰ *Id.* at 225.

²¹¹ CERCLA SECTION 301(E) STUDY GROUP REPORT, *supra* note 170, at 226.

²¹² *Id.*

While it may be socially desirable to hold disposers of hazardous wastes strictly liable, that policy may not apply in other toxic tort contexts. In addition, by limiting Tier One claimants' compensation to medical expenses and lost wages the regime has the great disadvantage of rendering typical cases unattractive to plaintiffs' attorneys. A plaintiff could opt into the Tier Two common law regime, but would no longer be able to invoke a presumption based upon the "toxic substances document."

B. *A Proposal for a Mixed Regime*

The Superfund Study Group's proposal has the considerable advantage of assigning a powerful role in determining general causation to an independent federal agency with expertise in assessing the human health risks posed by toxic substances in the environment. It offers the possibility of developing "new institutional arrangements that adapt the best features of both courts and the administrative process to deal with the problems of toxics in the environment."²¹³ I would like to carry that proposal a step farther to suggest a common law-like reparations regime that assigns an important scientific assessment role to a federal agency and an equally important moral assessment role to the jury. The toxic tort regime described below would explicitly allow juries to commingle culpability and causation, but would confine its role (and that of the trial judge) as a factfinder on the question of general causation to one that better fits lay decision makers.

The starting point in my proposal is a formal hazard assessment document prepared by a federal agency.²¹⁴ This suggestion reflects the understanding of the Superfund Study Group and other scholars that federal agencies have a great deal to offer the common law courts by way of evaluating the strengths and weaknesses of scientific information for

²¹³ E. Donald Elliott, *The Future of Toxic Torts: Of Chemophobia, Risk as a Compensable Injury and Hybrid Compensation Systems*, 25 Hous. L. Rev. 781, 783 (1988). Professor Elliott recognized early on that common law courts may not be the most appropriate institution to resolve the complex and uncertainty-dominated science/policy questions that are at the heart of the cause-in-fact question in toxic tort litigation. See also Elliott, *supra* note 11.

²¹⁴ There is no reason in theory why the risk assessor could not be a state agency. Since the proposal is offered as a solution to a problem caused largely by the federal judiciary's overly aggressive application of a federal rule of evidence, the institution will probably need to be federal in nature.

policy relevant purposes.²¹⁵ The federal agency charged with the hazard assessment task could be the EPA, the National Institute for Occupational Safety and Health, the Centers for Disease Control, the Agency for Toxic Substances and Disease Registry ("ATSDR"), or some other agency designated for this purpose.²¹⁶ The task must, however, be assigned to a single entity to avoid the possibility of "dueling assessments." In order to lend the necessary degree of legitimacy to the hazard assessment enterprise, it should be open to the public. The agency would maintain an administrative record containing scientific studies, comments on those studies by interested persons, and the minutes of any meetings between the relevant agency personnel and outsiders. The agency could empanel scientific advisory committees, elicit the views of "peer reviewers," and make inquiries of companies and scientists who had conducted relevant studies. The final hazard assessment document would be subject to judicial review in a court of appeals under the Administrative Procedure Act's familiar "arbitrary and capricious" test.²¹⁷

The agency would be required to evaluate existing scientific information on the hazard posed by the substance at issue using a "weight-of-the-evidence" evaluation of that information. The agency could determine that a study was so poorly conducted that it was entitled to no weight at all, or it could consider flawed studies to the extent that they provided support for conclusions suggested by better designed and executed studies. The agency would, of course, be allowed to consider animal studies to the extent that they were appropriately conducted and were scientifically relevant. The agency would be asked to assign the substance to one of four categories based upon its evaluation of the weight of the evidence. Depending upon the strength of association that the agency found between exposure to the substance and disease, the agency document would have a predetermined procedural effect on the jury's deliberations after an initial jury finding on the issue of the defendant's culpability.

1. The Culpability Determination

²¹⁵ See Elliott, *supra* note 38, at 505-07; see also Elliott, *supra* note 11, at 1358-60.

²¹⁶ Professors Elliott and Brennan have expressed some confidence in ATSDR as a risk evaluator. Brennan, *supra* note 43, at 50; Elliott, *supra* note 213, at 799.

²¹⁷ See *Motor Vehicle Mfrs. Ass'n of the U.S. v. State Farm Mut. Auto. Ins. Co.*, 463 U.S. 29, 42 (1983).

Like the administrative agency's hazard assessment, the defendant's culpability would play a carefully defined role in determining liability. Given the potentially enormous stakes involved, proof that the defendant was evil in some regard would not be enough to provide a procedural advantage to the plaintiff. The culpability would have to be causally tied to the alleged injury in the broad sense that the defendant's culpable conduct played a role in the plaintiff's exposure to the defendant's agent. For purposes of the mixed regime suggested here, "culpability" would consist of: (1) a significant violation by the defendant of existing state or federal regulatory requirements governing the sale, distribution, use or disposal of the agent; (2) a serious attempt to manipulate inappropriately a state or federal agency risk assessment or standard setting process applicable to the agent;²¹⁸ or (3) a successful attempt to mislead at-risk members of the public (including the plaintiff) with respect to the nature and magnitude of the risk posed by the agent. Attorneys for plaintiffs would have full discovery rights for the purpose of ascertaining culpability. Following the culpability determination, the jury would make general and specific causation determinations in accordance with the following procedural approach.

2. No Association Between Exposure and Plaintiff's Disease

For cases in which the administrative assessment found no association between exposure to the defendant's agent and a disease of the sort suffered by the plaintiff, the plaintiff would take nothing, no matter how culpable the jury believed the defendant's conduct to be. This would be the case for substances in which the agency found no association between exposure to the defendant's substance and any disease and in situations in which the jury found that the plaintiff did not suffer from one of the diseases for which the agency found an association. The former situation would be an appropriate occasion for granting a defendant's motion of summary judgment. For example, silicone gel breast implants could well wind up in this category. Even though much evidence exists of gross misconduct on the part of the manufacturers, the relevant agency could determine that the weight of the scientific evidence does not indicate

²¹⁸ This would include funding bogus science, screening and hiding negative studies, and stopping ongoing studies when they appeared to be going the wrong way. For this purpose, the "regulatory process" should be viewed broadly to include attempts to manipulate White House or congressional personnel with false or misleading data or other information in an attempt to bring pressure to bear on the relevant administrators.

even a weak association between implants and connective tissue disorders.²¹⁹ If so, then plaintiffs would receive no compensation, and any punishment for culpable conduct would have to come directly at the hands of the government.²²⁰

3. Weak Association Between Exposure and Plaintiff's Disease

A conclusion in the hazard assessment documents that the weight of the evidence demonstrated a "weak" association between exposure to the substance at issue and the plaintiff's disease would give the plaintiff a modest procedural advantage in the jury trial. Although opinions might vary about what kinds of evidence should support a conclusion of a "weak" association, I would suggest that it could be based upon: (1) multiple animal studies supported by pharmacodynamics studies demonstrating the relevance of the tested species to the human exposures; (2) a single well-conducted epidemiological study or meta-analysis of multiple studies demonstrating a relative risk of 1.4 or greater; or (3) a combination of animal studies and one or more epidemiological studies demonstrating a relative risk of 1.3 or greater.

Although others might suggest different criteria for the "weak" association category, the defining criteria outlined above appear consistent with general practice. For example, EPA concluded that ETS was a Group A human carcinogen based upon a meta-analysis of epidemiological studies demonstrating a relative risk of approximately 1.42 for lung cancer.²²¹ According to EPA's criteria, a Group A classification "is used only when there is sufficient evidence from epidemiologic studies to support a causal association between exposure to the agents and cancer."²²² EPA's guidelines allow the agency to conclude that a chemical is a "Group B—Probable Human Carcinogen" on the basis of "limited" epidemiological data combined with "sufficient" animal studies demonstrating evidence of carcinogenicity.²²³

²¹⁹ See Nagareda, *supra* note 41, at 1146-47.

²²⁰ At this point, no punishment has been meted out to the manufacturers of silicone gel breast implants, but the FDA has imposed a moratorium on their sale, and they are no longer being produced. See *id.* at 1140.

²²¹ See FINAL EPA ETS REPORT, *supra* note 95, at 5-2.

²²² See Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17,960 (Apr. 23, 1996).

²²³ *Id.*

When the agency report concluded that a “weak” association existed between exposure and the plaintiff’s disease, the jury would be permitted to make a finding of general causation without any additional evidence, but there would be no presumption in favor of a general causation finding. Both the plaintiff and the defendant could introduce *Daubert*-reliable expert testimony on the issue of general causation, but the plaintiff would not be required to present *Daubert*-reliable expert testimony on that issue and could instead rely upon the report.

If the plaintiff proved to the jury’s satisfaction that the defendant was culpable, however, a presumption would arise that the substance was capable of causing the plaintiff’s disease. The plaintiff would prevail on the general causation issue unless the defendant proved with *Daubert*-reliable expert testimony that the substance was incapable of causing the disease. In either case, a plaintiff prevailing on the general causation issue would have the burden of proving specific causation through direct testimony and, if necessary, through *Daubert*-reliable expert testimony. As previously discussed, this would require at least a showing that the plaintiff received a sufficient exposure to cause the plaintiff’s disease.

4. Strong Association Between Exposure and Disease

If the agency’s report concluded that a “strong” association existed between exposure to the substance and the plaintiff’s disease, the plaintiff would benefit from more powerful presumptions. A strong association could be established by a single epidemiological study deemed adequate to the agency demonstrating a relative risk of 2.0 or greater or a well conducted epidemiological study demonstrating a relative risk of 1.5 or greater with more than one supporting animal study. Although a relative risk of less than 2.0 does not, standing alone, indicate that the exposure was “more likely than not” the cause of the disease, a relative risk of 1.5-2.0 is nevertheless a stronger association than required by EPA to classify a chemical as a Group A carcinogen. When combined with confirming animal studies such a strong association is usually enough to persuade regulatory agencies to take action to protect the public, even though other agents, activities, or even genetic makeup may also be a cause of the observed disease.²²⁴

²²⁴ See, e.g., *Chemical Mfrs. Ass’n v. EPA*, 28 F.3d 1259 (D.C. Cir. 1994) (EPA list of high risk hazardous air pollutants); see also *Int’l Fabricare Inst. v. EPA*, 972 F.2d 384 (D.C. Cir. 1992) (drinking water standards for perchloroethylene); *Int’l Union, United*

In cases in which the administrative hazard assessment found a strong association between exposure and the plaintiff's disease, the report itself would give rise to a presumption in favor of general causation without proof of culpability. Proof of culpability would raise an additional presumption that the defendant's agent was the specific cause of the plaintiff's disease. The defendant could rebut both presumptions by proving to the jury with *Daubert*-reliable expert witnesses that the plaintiff's disease was probably not caused by exposure to the defendant's agent. The defendant could also prevail if it proved to the jury that some other agent was more likely than not the cause-in-fact of the plaintiff's disease.

5. Dominant Among Multiple Causes

Occasionally the epidemiological data demonstrates a statistical association so powerful that the exposure to the toxic substance appears to be the dominant cause among many possible causes of the disease. Such strong associations occur more frequently in the context of rare diseases, because the denominator in the relative risk ratio (representing the incidence in the unexposed population) is already quite small. Although a single study demonstrating a relative risk of 2.0 suggests that any given individual incidence in the exposed population was probably caused by the exposure to the defendant's agent, multiple epidemiological studies demonstrating a relative risk of greater than 2.0 should be required to demonstrate that the defendant's agent was the dominant among multiple causes. A single epidemiological study demonstrating a relative risk of 3.0 or greater when supported by well-conducted animal studies should also suffice.

In such cases, the administrative report would give rise to a rebuttable presumption in favor of the plaintiff on both general and specific causation without proof of culpability. The defendant would be permitted to rebut the presumption by attacking the validity of the agency's conclusions with *Daubert*-reliable expert testimony. If the defendant's proof undermined the agency's conclusions and the jury concluded that the defendant's agent was probably not the cause of the plaintiff's disease, the defendant would escape liability. Proof of culpability would, however, prevent the defendant from pointing to other possible causes of the plaintiff's affliction in an effort to rebut the

presumption. If a culpable defendant's agent is statistically the dominant among multiple causes, then the mere possibility that something else might have caused the plaintiff's disease should not relieve the defendant of liability.

C. *The Virtues of Coupling Culpability with Causation*

The combined approach outlined above should improve the existing common law regime in several important regards. First, it should provide adequate compensation for persons who are harmed by exposure to toxic agents and who would otherwise have to rely upon their own or collective societal resources. Because culpability should play a powerful role in liability determinations, certainly a more direct role than it plays in much current products liability litigation, the shift in resources should come primarily at the expense of defendants who cannot claim to be innocent victims of an out-of-control litigation machine.

Second, by assigning the primary hazard assessment role to an administrative agency, the proposal provides a better match between assigned institutional roles and institutional competence than the current regime. A presumably objective administrative agency with expertise in epidemiology and toxicology will play a prominent role in assessing the validity of scientific studies and in drawing scientific conclusions. Although administrative agencies may be somewhat more susceptible to the prevailing political winds than a judge or a jury, they can bring expertise to bear on complex science-policy questions in a way that is generally credible and therefore acceptable to the general public.²²⁵ Making a single hazard assessment available for all lawsuits involving the same toxic agent should enhance litigative efficiency. At the same time, the proposal divests trial judges of the wholly inappropriate scientific gatekeeper role that *Daubert/Joiner* has forced them to play.²²⁶ The appellate judiciary would still play a modest role of ensuring the rationality of the agency's hazard assessment process when it reviews the administrative hazard assessments under the "arbitrary and capricious" test.²²⁷ The trial and appellate judges in individual cases would not, however, be permitted to nit-pick the scientific evidence under the corpuscular approach followed by many current courts.

²²⁵ Elliott, *supra* note 213.

²²⁶ See Berger, *supra* note 7, at 2122.

²²⁷ See Elliott, *supra* note 11, at 1374.

Third, the proposal would assign a prominent role to the jury in the decision-making process while recognizing that lay juries need expert help in resolving difficult science-policy issues. The *Daubert* Court's implicit conclusion that the jury cannot be trusted to separate junk science from the real thing may well be correct. The courts have, however, applied *Daubert* aggressively to divest the jury of a critical decisionmaking role in toxic tort litigation, an outcome that is not easy to square with the plaintiff's Seventh Amendment right to a jury trial in civil cases.²²⁸ Although my proposal does not make the jury the exclusive factfinder, it allows the jury to determine whether the presumption of causation has been rebutted, a task for which it is better suited than that of evaluating the scientific data and inferences in the first instance. Moreover, my proposal will assign to the jury the critical role of determining culpability, a role for which the jury is especially well-suited.²²⁹ With the aid of an agency's hazard assessment report, appropriate presumptions, and competent expert testimony, juries can be trusted to evaluate technically difficult information.²³⁰

Fourth, the strong incentive that the proposal provides to plaintiffs to prove culpability should help smoke out more evidence of corporate malfeasance than the existing federal administrative process. One of the considerable advantages of a tort reparations regime is its capacity to get to the truth of the matter in ways that are largely unavailable to regulatory agencies engaged in traditional rule-making and enforcement.²³¹ Regulatory agencies ordinarily do not have a strong interest in culpability per se, because their primary goal is to get dangerous products off the market and dangerous contaminants out of the environment as quickly as possible, regardless of who is at fault. Private attorneys, by contrast, are adept at uncovering evidence of culpability in the discovery that precedes common law trials, and they are willing to spend the resources necessary to copy and organize documents, take depositions, and fight the company's

²²⁸ See Gottesman, *supra* note 8, at 759.

²²⁹ See Berger, *supra* note 7, at 2151 (arguing that "decisions about whether particular behavior warrants liability" are "well-suited to the jury's role as a representative of the community").

²³⁰ See Christopher H. Buckley, Jr., *Toxic Tort Cases: Risk Assessment and Junk Science*, 9 KAN. J.L. & PUB. POL'Y 487, 499 (2000) (concluding that "juries understand good science even if it is complicated").

²³¹ See Berger, *supra* note 7, at 2150 (citing case studies that "amply demonstrate the legal system's ability to ferret out the 'smoking guns' that would establish negligence").

efforts to resist discovery.²³² The tobacco litigation has underscored the value of document discovery and compelled testimony under oath, and my proposal takes full advantage of that potential.

Fifth, by focusing the culpability inquiry at least partially on attempts to manipulate the regulatory process, the tort regime should complement the regulatory regime by indirectly enhancing its integrity. The culpability-based presumptions should send a message to regulated entities that quiet attempts to manipulate the regulatory programs will probably be uncovered if something goes wrong and someone is injured. The tort regime should therefore discourage manipulation and deceit in interactions between regulators and regulatees. If culpability is defined more broadly to include fraudulent or misleading manipulation of public perceptions, the approach suggested here will also encourage truthfulness in advertising and public relations initiatives. Similarly, by making substantial noncompliance with state and federal regulations a basis for a culpability finding, my proposal should encourage compliance with those regulations. Finally, the knowledge that a federal agency will evaluate all of the available information in assessing the hazards posed by a chemical should enhance the incentive of the manufacturer to conduct adequate testing. At the very least it should take away the “perverse disincentive” that the current post-*Daubert* law provides to forego testing altogether.²³³

D. *Objections to the Proposal*

There are, of course, legitimate reasons for objecting to the proposal outlined above. First, some will no doubt argue that it assigns too large a role to a remote federal bureaucracy. Many potential defendants are also regulatees and have therefore interacted on a regular basis with federal regulatory agencies. Depending upon the nature of that interaction, they may not be inclined to place much faith in federal bureaucrats to come up with fair and accurate hazard assessments. One answer to this objection is that the proposal allows affected companies to participate vigorously in the

²³² See GREEN, *supra* note 21, at 15 (detailing how attorneys for plaintiffs in asbestos information obtained information on wrongdoing); Nagareda, *supra* note 44, at 923 (reporting that plaintiffs’ attorneys in the breast implant litigation “invested several million dollars toward the collection and organization of documents bearing upon the defendant manufacturers’ knowledge of potential product risks”).

²³³ See Beecher-Monas, *supra* note 8, at 1090.

hazard assessment process and thereby attempt to affect its outcome.²³⁴ Another answer may be to assign the task to an agency that can command the trust of most affected parties or to create such an agency.

Second, the combined administrative-common law regime suggested here may exacerbate an unavoidable timing issue. One important aspect of toxic tort claims is the typically long latency period between the exposure to the toxic substance and the manifestation of a disease caused by that exposure.²³⁵ At any given time, some victims may have contracted a disease associated with exposure while others have been put at risk for contracting diseases at some point in the future.²³⁶ Plaintiffs' attorneys have typically attempted to address this problem by including both past and potential victims in a single lawsuit and structuring the damage award so that past victims are compensated immediately and future victims are compensated through a quasi-administrative arrangement.²³⁷ Although not necessarily inconsistent with an omnibus remedy that resolves present and future liability, the approach suggested here encourages the agency to amend the hazard assessment as more knowledge becomes available. If the revised hazard assessment indicated that the association was stronger or weaker than previously determined, the presumptions could be adjusted accordingly for application in future cases. Although it would not affect pre-existing remedies, this possibility might discourage parties from entering into omnibus settlements that resolve present and future liabilities. Whether the increased accuracy of a regime that changes with changing information is preferable to the increased efficiency of a regime that encourages omnibus remedies is admittedly a question about which reasonable minds may differ. In my view, it is unlikely that parties will be motivated to enter into large omnibus settlements until the available scientific information is sufficiently robust that the probability of a significant modification of any risk assessment is fairly low.

Third, one may raise legitimate policy concerns about a reparations regime that encourages juries to commingle culpability with causation.

²³⁴ The tobacco industry participated exceedingly vigorously in EPA's attempt to assess the risks posed to nonsmokers by environmental tobacco smoke, and they successfully challenged the risk assessment in district court. See *Flue-Cured Tobacco Coop. Stabilization Corp. v. EPA*, 4 F. Supp. 2d 435 (M.D.N.C. 1998).

²³⁵ See Nagareda, *supra* note 10, at 1787 ("Mass torts characteristically involve injury in the form of disease for which there is a substantial gap in time between exposure to the product in question and the onset of impairment.").

²³⁶ See *id.* at 1786-87.

²³⁷ See *id.* at 1782, 1785, 1787-88, 1798.

Professor Nagareda, for example, suggests that jurors who commingle culpability with causation "are putting into effect their moral intuitions through channels wildly unsuited to their implicit goal."²³⁸ Under the United States Constitution, criminal sanctions are not to be lightly imposed by the state, and the law of criminal procedure provides many protections to criminal defendants that are unavailable in common law tort regimes.²³⁹ The typical "preponderance of the evidence" standard of proof and the burden shifting devices suggested above place an unaccustomed burden of proof on the defendant (though not on the question of "innocence"). Nevertheless, the American legal system has until now tolerated considerable intrusion by civil law into territories that are ordinarily within the domain of the criminal law. The state itself may punish unlawful conduct through civil penalties that are equally burdensome to the pocketbook without proving the facts beyond a reasonable doubt and without protections against compelled self-inculcating testimony.²⁴⁰ And the harshest penalty that the tort law can inflict against a corporation, bankruptcy, is so frequently invoked these days by tort defendants as a strategic tool that none of the stigma of criminal liability attaches to that result.²⁴¹

Finally, many fear that a reparations system that permits commingling of culpability with causation will allow plaintiffs to file undeserving claims against defendants that did not cause any harm based solely upon an uninformed "sense of moral outrage."²⁴² For many observers, "corrective justice" should be the primary goal of a tort regime,²⁴³ and a fundamental requirement of corrective justice is that the entity that the law requires to adjust its position for the benefit of some

²³⁸ Nagareda, *supra* note 41, at 1174.

²³⁹ The requirement of proof beyond a reasonable doubt, the privilege against self-incrimination, and protections against double jeopardy are just a few of the procedural protections that are available to defendants when the state attempts to assign blame through the criminal law.

²⁴⁰ See *U.S. v. Ward*, 448 U.S. 242 (1980).

²⁴¹ In this regard, I am inclined to disagree with Professor Nagareda's assessment of Chapter 11 bankruptcy as a stigma-providing substitute for the criminal law. See Nagareda, *supra* note 41, at 1146-47.

²⁴² *Id.* at 1126.

²⁴³ According to the theory of "corrective justice," the primary purpose of tort law is to place two parties in the same position they were in prior to some unlawful "transaction." See, e.g., Klein, *supra* note 43, at 1189-90.

other person has caused loss to the other person.²⁴⁴ Professor Schroeder, however, persuasively argues that allowing persons placed at risk to recover from risk-producing entities without proving that the activity caused a particular injury is consistent with corrective justice because it provides ex ante incentives to potential risk producers to take the interests of potential victims into account at the time they take risk-producing action and compensates to some degree those persons put at risk.²⁴⁵

However one resolves the debate over corrective justice and risk-based compensation, the proposal outlined above has a much lower potential to violate corrective justice principles. First, it does not abandon cause-in-fact altogether; it is merely willing to accept proof of general causation on the basis of statistical evidence short of 50-50 probability in cases involving culpable defendants. Second, since only a culpable defendant can be held liable on proof of less than 50-50 probability, the primary injustice inherent in the regime is its potential to punish wrongdoers by way of providing windfalls for undeserving plaintiffs. Professor Nagareda notes that "the conventional conception of tort law offers no justification for the compelled transfer of money to [an unharmed] individual, no matter how much good one might produce in the future by so doing."²⁴⁶ One justification, however, may be that since the regulatory regime is incapable of punishing many such wrongdoers, a modest change in the tort regime may be warranted in the interests of general deterrence and, perhaps more importantly, to fill a legitimate societal need for retribution and closure.²⁴⁷

IV. CONCLUSIONS

²⁴⁴ Christopher H. Schroeder, *Corrective Justice and Liability for Increasing Risks*, 37 UCLA L. REV. 439 (1990). For other thoughtful arguments in support of liability for increased risk, see Glen O. Robinson & Donald D. Elliott, *Probabilistic Causation and Compensation for Tortious Risk*, 14 J. LEGAL STUD. 779, 783-99 (1985); David Rosenberg, *The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System*, 97 HARV. L. REV. 849 (1984).

²⁴⁵ Schroeder, *supra* note 244, at 465-66.

²⁴⁶ Nagareda, *supra* note 41, at 1176.

²⁴⁷ Plaintiffs' lawyers in toxic tort litigation soon discover, sometimes to their dismay, that for their clients the lawsuit is not so much about the money as it is about the desire to force a callous company to acknowledge and/or pay for its wrongdoing in the absence of any effective government-imposed sanctions. See HAAR, *supra* note 14.

Responding to the chemical industry's resistance to a victim compensation measure that he had hoped to include in a pending Superfund Reauthorization bill, Rep. James Florio warned in 1984 that the lax environmental policies of the Reagan Administration would "produce a backlash" that would soon change the industry's mind.²⁴⁸ The backlash did in fact occur,²⁴⁹ but the industry has never supported an administrative compensation regime of the sort that Rep. Florio proposed. Perhaps for that reason, Congress has rejected suggestions for such administrative regimes in all but a few limited contexts.²⁵⁰ The coming accountability crisis should offer new opportunities for advocates of past and future victims of modern technologies to persuade Congress to enact fundamental changes in the way that courts go about determining liability for harm inflicted by poorly regulated toxic agents. The next round of hearings on "tort reform" should at the very least examine the legitimacy of the Supreme Court's assignment of a scientific gatekeeping role to marginally qualified trial judges, and the next round of proposed tort reform legislation should include proposals for combining culpability with causation in a mixed administrative/common law tort regime.

²⁴⁸ *Chemical Industry Ultimately Will Request Victim Compensation System, Florio Predicts*, 15 Env't Rep. (BNA) 575, 575-76 (1984) (quoting Rep. James J. Florio (D-NJ)).

²⁴⁹ See, e.g., JONATHAN LASH ET AL., *A SEASON OF SPOILS* (1984).

²⁵⁰ See Brinker, *supra* note 42, at 1324.