



Washington Legal Foundation
Advocate for freedom and justice®
2009 Massachusetts Avenue, NW
Washington, DC 20036
202.588.0302

**CAUSATION IN COURT:
WORKING PRINCIPLES FOR
TOXIC TORT CASES**
by
Antony Klapper
Reed Smith LLP



Washington Legal Foundation
CONTEMPORARY LEGAL NOTE Series
Number 61
October 2008
Visit WLF's online legal studies library
www.wlf.org/Publishing

ABOUT WLF'S LEGAL STUDIES DIVISION

The Washington Legal Foundation (WLF) established its Legal Studies Division to address cutting-edge legal issues by producing and distributing substantive, credible publications targeted at educating policy makers, the media, and other key legal policy outlets.

Washington is full of policy centers of one stripe or another. But WLF's Legal Studies Division has deliberately adopted a unique approach that sets it apart from other organizations.

First, the Division deals almost exclusively with legal policy questions as they relate to the principles of free enterprise, legal and judicial restraint, and America's economic and national security.

Second, its publications focus on a highly select legal policy-making audience. Legal Studies aggressively markets its publications to federal and state judges and their clerks; members of the United States Congress and their legal staffs; government attorneys; business leaders and corporate general counsel; law school professors and students; influential legal journalists; and major print and media commentators.

Third, Legal Studies possesses the flexibility and credibility to involve talented individuals from all walks of life - from law students and professors to sitting federal judges and senior partners in established law firms - in its work.

The key to WLF's Legal Studies publications is the timely production of a variety of readable and challenging commentaries with a distinctly common-sense viewpoint rarely reflected in academic law reviews or specialized legal trade journals. The publication formats include the provocative COUNSEL'S ADVISORY, topical LEGAL OPINION LETTERS, concise LEGAL BACKGROUNDEERS on emerging issues, in-depth WORKING PAPERS, useful and practical CONTEMPORARY LEGAL NOTES, interactive CONVERSATIONS WITH, law review-length MONOGRAPHS, and occasional books.

WLF's LEGAL OPINION LETTERS and LEGAL BACKGROUNDEERS appear on the LEXIS/NEXIS[®] online information service under the filename "WLF" or by visiting the Washington Legal Foundation's website at www.wlf.org. All WLF publications are also available to Members of Congress and their staffs through the Library of Congress' SCORPIO system.

To receive information about previous WLF publications, contact Glenn Lammi, Chief Counsel, Legal Studies Division, Washington Legal Foundation, 2009 Massachusetts Avenue, NW, Washington, D.C. 20036, (202) 588-0302. Material concerning WLF's other legal activities may be obtained by contacting Daniel J. Popeo, Chairman.

ABOUT THE AUTHOR

Antony Klapper is a Partner in the Washington, D.C. office of Reed Smith, LLP. He is a commercial litigator, but focuses most of his time on toxic tort and other products liability cases, frequently serving as national science counsel for various defendants. Although Mr. Klapper principally represents defendants, his views here are independent of those representations and do not necessarily reflect those of Reed Smith LLP or its clients.

The author wishes to thank Eric Alexander, Esq. and Carol Ward, Ph.D., for their helpful comments and contributions.

CAUSATION IN COURT: WORKING PRINCIPLES FOR TOXIC TORT CASES

by
Antony Klapper
Reed Smith LLP

INTRODUCTION

We believe that it is humane to monetize welfare losses associated with grief, pain and suffering, humiliation, mental anguish, and other intangible injuries so that we can make plaintiffs whole. What we do not do, again for reasons grounded in humanity, is force a defendant to compensate a plaintiff if the plaintiff does not show that the defendant has probably done something to him.¹

Law is infused with uncertainties. Arguments fill briefs, courtrooms and judge's chambers as the legally trained try to interpret and apply often squishy legal principles to an endless array of fact patterns. Black and white is rarely found. Gray predominates.

The element of causation is no exception to this legal world of gray. While questions of causation are addressed daily in criminal cases, civil tort case and even contract cases, litigants, judges and juries are rarely afforded clear guidance on what this legal element means, let alone how it should be applied in a principled and consistent fashion. What do the expressions "proximate cause,

¹*Wright v. Willamette Indus. Inc.*, 91 F. 3d 1105, 1108 (8th Cir. 1996).

“substantial factor,” “contributing factor,” and “de minimis contribution” really mean? And do the definitions square with both scientific and legal principles?

This CONTEMPORARY LEGAL NOTE offers some perspectives and six working principles that may render “causation” a less mysterious element to understand and apply. Those principles are:

1. Causation in science is not synonymous with causation in law, but the gap has closed.
2. Proof of general causation requires, at a minimum, reliable epidemiology and a statistically significant estimated relative risk of more than 2.0.
3. Proving causation does not end with the general causation inquiry. Proof of specific causation is absolutely essential before any causal conclusions can be drawn.
4. Risk assessment is the best tool available to answer questions of causation.
5. Although risk assessment is the best tool available, regulatory rules for implementing risk assessments should not be used and too often are abused.
6. Where there are multiple exposure sources for the same toxin, a more principled, objectively reliable methodology should be used to answer questions of causation. Concepts like “substantial contributing” cause should be jettisoned.

The focus here is on toxic substances and disease causation, a subset of tort law that typically creates more confusion than other areas of law, due largely to

ambiguity surrounding the often uncertain relationship between exposure and injury.²

PRINCIPLE 1:
CAUSATION IN SCIENCE IS NOT
SYNONYMOUS WITH CAUSATION IN LAW,
BUT THE GAP HAS CLOSED

In a toxic tort case, the plaintiff has the burden of proving that the defendant more likely than not caused her injury. To do so, she can introduce the expert testimony of toxicologists, epidemiologists, pathologists, and/or medical doctors, who are each asked to answer scientific questions of causation filtered through a series of legal evidentiary rules.

Problems arise when the scientific principles to which these experts ascribe do not mesh well with these evidentiary rules. For example, rarely does the medical doctor ask whether a toxic agent or a genetic disorder was the cause of her patient's disease. Instead, her principal objective is to diagnose her patient's disease and find a cure. Or, take the epidemiologist. Rarely does she describe the relationship between a disease and some toxic agent in "more likely than not" terms. Instead, she uses terms like confidence intervals, relative risk and statistical significance. And what expert in the normal course of her non-forensic

²By toxic tort, the author includes occupational exposures to chemicals, environmental exposures to chemicals and consumer exposures to chemicals, including pharmaceuticals. Torts that involve traumatic injuries typically involve less difficult causation analyses and thus are not the focus of this article.

activities actually uses the expression “to a reasonable degree of scientific certainty”?

But even accepting these ill-fitting components of science and law, more similarities than differences exist. And, in many ways, the Supreme Court’s seminal *Daubert* decision brought these worlds together. For in that decision, the Court acknowledged the evolving nature of science and law, and highlighted the central importance of working principles, sound methodology, and reliability in legal and scientific assessments:

The subject of an expert’s testimony must be ‘scientific ... knowledge.’ The adjective ‘scientific’ implies a grounding in the methods and procedures of science. Similarly, the word ‘knowledge’ connotes more than subjective belief or unsupported speculation. The term ‘applies to any body of known facts or to any body of ideas inferred from such facts or accepted as truths on good grounds.’ Webster’s Third New International Dictionary 1252 (1986). Of course, it would be unreasonable to conclude that the subject of scientific testimony must be ‘known’ to a certainty; arguably, there are no certainties in science. *See, e.g.*, Brief for Nicolaas Bloembergen et al. as *Amici Curiae* 9 (‘Indeed, scientists do not assert that they know what is immutably ‘true’-they are committed to searching for new, temporary, theories to explain, as best they can, phenomena’); Brief for American Association for the Advancement of Science et al. as *Amici Curiae* 7-8 (‘Science is not an encyclopedic body of knowledge about the universe. Instead, it represents a *process* for proposing and refining theoretical explanations about the world that are subject to further testing and refinement’ (emphasis in original)). But, in order to qualify as ‘scientific knowledge,’ an inference or assertion must be derived by the scientific method. Proposed testimony must be supported by appropriate validation- *i.e.*, ‘good grounds,’ based on what is known.

In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability.³

In sum, compared to the *Frye* rule, the Supreme Court's explicit adoption of evidentiary standards that reflect well-tooled scientific methodologies demonstrates that the disciplines of law and science are not that far apart.

PRINCIPLE 2:
PROOF OF GENERAL CAUSATION REQUIRES,
AT A MINIMUM, RELIABLE EPIDEMIOLOGY
AND A STATISTICALLY SIGNIFICANT ESTIMATED
RELATIVE RISK OF GREATER THAN 2.0

In determining whether a toxic agent can cause harm, the tool of choice is epidemiology.

Epidemiology is the study of disease in populations. Stated in its most basic terms, it involves a comparison of the incidence of disease in a group exposed to an agent with the incidence of disease in an unexposed group (also known as the control population). This comparison is often represented as a ratio of the rate of disease in the exposed population as compared to the rate of disease in the unexposed population. This value is often described as the estimate of relative risk (or RR).⁴ Although some random sampling error will

³*Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 589-90 (1993).

⁴Depending on the type of study, risk may be depicted as "relative risk" or as an "odds ratio" (OR). Case control studies use odds ratios; clinical and cohort studies use relative risk measures. As a convention, epidemiologists and statisticians generically refer to risk ratios as "estimated relative risks" and that is how this author is using the expression.

invariably impact the estimated relative risk ratio, if the range (also known as the confidence interval) within which the actual risk value lies is above an estimated relative risk of “1.0”, the study results are deemed statistically significant. If such a study exists and after excluding potential confounding or bias in the study (discussed below), an epidemiologist can fairly conclude that there is a likely chance of an association between the agent and the disease.

As applied in a court of law, if the decisional standard is that the substance more probably than not caused disease, an estimated relative risk of greater than “2.0” should be required. “When the relative risk reaches 2.0, the agent is responsible for an equal number of cases of disease as all other background causes. Thus, a relative risk of 2.0 (with certain qualifications noted below) implies a 50% likelihood that an exposed individual’s disease was caused by the agent.”⁵ Although far from universally subscribed to, several courts have required for proof of causation, reliable epidemiologic evidence demonstrating more than a doubling risk.⁶

Assuming this threshold of proof is established, the analysis does not end there. A single epidemiologic study only establishes an association between agent and disease. To prove causation, more evidence is needed.

⁵Fed. Judicial Ctr., REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 333, 384 & n. 140 (2d ed. 2000). “If genetics are known to be responsible for 50% of the incidence of a disease independent of exposure to an agent and genetics [or other confounding factors] can be ruled out of an individual’s case, then a relative risk greater than 1.5 might be sufficient to support an inference” of a causal relationship. *Id.* at 386.

⁶*Id.* at 386 n. 140.

First, multiple positive, consistent, and statistically significant epidemiological studies are needed in order to have some reliable basis for concluding that exposure to a substance can cause a particular adverse health outcome. Any one study can suffer from various system errors, such as selection bias, recall bias, as well as unaccounted for confounders, and random sampling errors. But if a number of studies consistently demonstrate a statistically significant association between agent and disease, and the studies themselves are methodologically sound, these biases and errors diminish in importance. Reproducibility of results is the hallmark of good science.

Second, consideration must be given to what are commonly referred to as the Bradford Hill criteria.⁷ These criteria include, among other things, a temporal relationship between the agent and disease (that the disease follow exposure to the agent); that as the amount of exposure to the agent goes up, the risk of disease does as well; and that the relationship between the agent and the disease makes biological sense. Assuming these, or some of these, criteria are met, the ultimate question of whether an agent causes a particular disease still boils down to scientific judgment. But whose?

Oftentimes, expert scientific bodies come together to evaluate whether a substance should be deemed a cause of disease. Consensus bodies like the Agency for Toxic Substances and Disease Registry (ATSDR), World Health

⁷A.B. Hill, "The Environment and Disease: Association or Causation?," 58 PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE 295-300 (1965).

Organization (WHO), National Toxicology Program (NTP) and the International Agency for Research on Cancer (IARC) evaluate various chemicals and make probabilistic determinations based on epidemiology, toxicology and mechanistic data as to whether those chemicals cause disease.⁸ The ability of a chemical to cause disease is classified by a hierarchy of essentially evidentiary proof: Known, probable, possible, probably not (or similar variants).⁹ Translated into legal evidentiary proof, chemicals classified as known or probable causes of a particular disease fit the “more likely than not” evidentiary paradigm that drives causation decisions in civil courts of law. Conversely, chemicals classified as “possible” and “probably not” causes of a particular disease fail to meet that evidentiary standard. This hierarchy presents a potentially useful heuristic for litigators, judges and juries.

Unfortunately, these consensus bodies have only made these types of evaluations for a small subset of the chemicals that humans are exposed to and they do not always agree. What happens when the plaintiff claiming injury has no (or inconsistent) conclusions from NTP, IARC or WHO to turn to, or no epidemiologic evidence at all? Should that case be dismissed outright or should the plaintiff be allowed to marshal “other” evidence to prove up causation? In

⁸Joseph Rodricks, “Evaluating Disease Causation in Humans Exposed to Toxic Substances,” 14 J.L & POL’Y 41, 48-49 (2006).

⁹See, e.g., IARC Monographs Preamble 22-23 (2006). IARC provides that an agent is “probably” a carcinogen only if there is at least some limited evidence of carcinogenicity due to an observed positive association between exposure to the agent and cancer in humans. *Id.* at 19 & 22.

these situations, some courts have allowed *other data* to establish general causation.¹⁰ Typically, this happens in cases where there is very good mechanistic data, a short latency period and a good explanation for why epidemiological evidence is wanting (for example, because the background incidence of the disease is so low, or the number of individuals exposed is too small).¹¹ And some commentators contend that without epidemiology, regardless of the reason, proof of causation should still be allowed.¹² For several reasons, this author disagrees.

First, it was not long ago that legal scholars and courts were chastising the use of epidemiology in establishing causation, calling instead for more demanding and direct mechanistic data – biological data showing agent A causes

¹⁰See RESTATEMENT (THIRD) TORTS: LIABILITY FOR PHYSICAL HARM § 28 comment c(3) (listing numerous courts that have concluded that epidemiology is not required).

¹¹See, e.g., *Lakie v. Smithkline Beecham*, 965 F. Supp. 49, 56 (D.D.C. 1997) (“The absence of epidemiological studies, however, while important, is not dispositive as long as the methodology employed by the expert is sound . . . This is especially true when the disease is an extremely rare disorder like MDS 5 q-minus.”); see also Joseph Sanders & Julie Machal-Fulks, “The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law,” 64 LAW AND CONTEMP. PROBL. 107, 131 (2001) (discussing cases).

¹²See, e.g., Michael D. Green, “Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of Agent Orange and Bendectin Litigation,” 86 NW. U. L. REV. 643, 680-81(1992) (“The point is that plaintiffs should be required to prove causation by a preponderance of the available evidence, not by some predetermined standard that may require nonexistent studies. This means that in every case involving an alleged toxic agent for which a mature epidemiologic record does not exist, analysis of the sufficiency of plaintiff’s evidence would begin by considering the universe of available evidence of toxicity.”). Professor Green concedes, however, that “opening the courthouse doors to plaintiffs entering with such thin and attenuated evidence and rendering a decision on such a record is discomfiting and unfortunate.” *Id.* at 681.

disease B.¹³ In the larger historical sense, then, requiring reliance on epidemiologic evidence – indirect inferential data – actually affords plaintiffs today greater opportunities for recovery than when the courts deemed epidemiology insufficient for causal proof. Requiring epidemiologic data strikes the right equitable balance and is consistent with the approach of most scientific bodies.

Second, requiring sound epidemiologic evidence ensures that the courtroom is not where unproven scientific hypotheses are tested. As Judge Posner once wrote: “the courtroom is not a place for scientific guesswork, even of the inspired sort. Law lags science; it does not lead it.”¹⁴ If the scientific community has not yet decided that exposure to an agent actually causes a particular disease and at what levels of exposure, a judge’s or jury’s decision that the agent does cause disease would rest on a foundation of pure speculation.

Third, it would be an odd decisional scheme that allows a plaintiff to prove general causation when epidemiologic evidence is unavailable, but denies proof of general causation when the epidemiology is equivocal.

And, fourth, allowing proof of causation with scientific tools that are inferior to epidemiology (such as animal studies) is inconsistent with our legal

¹³See, e.g., Michael Dore, “A Commentary on the Use of Epidemiologic Evidence in Demonstrating Cause-in-Fact,” 7 HARV. ENVTL. L. REV. 429, 436 (1983); Troyen A. Brennan, “Causal Chains and Statistical Links: The Role of Scientific Uncertainty in Hazardous Substances Litigation,” 73 CORNELL L. REV. 469, 489-93 (1988).

¹⁴*Rosen v. Ciba-Geigy Corp.*, 78 F. 3d 316, 319 (7th Cir. 1996).

system's basic evidentiary scheme – a scheme that eschews speculation and requires proof of causation by a “more probable than not” standard.

PRINCIPLE 3:
**PROVING CAUSATION DOES NOT END WITH
THE GENERAL CAUSATION INQUIRY. PROOF OF
SPECIFIC CAUSATION IS ABSOLUTELY ESSENTIAL
BEFORE ANY CAUSAL CONCLUSIONS CAN BE DRAWN**

Merely proving a general causal relationship between a toxin and a particular disease does not translate into proving that an individual who had some exposure to the toxin and had the relevant disease got that disease because of the exposure to the toxin. As the authors of the *Restatement (Third) of Torts* described it:

[T]he extent to which the group-study outcome reflects the increased risk to the plaintiff depends on the plaintiff's similarity to those included in the group study. Relevant differences include whether (a) the plaintiff was exposed to a comparable dose; (b) the plaintiff was not differentially exposed to other potential causes of the disease; (c) the plaintiff has individual characteristics that might also bear on the risk of disease, such as age, gender, or general health, comparable to those in the study group.¹⁵

These three factors are critical to the specific causation inquiry.

The first factor relates to whether the individual plaintiff mirrors the group of exposed individuals for whom the epidemiologic studies evidenced the requisite minimum doubling of the risk of disease. If the plaintiff does not fit that

¹⁵RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL HARM § 28 cmt. C(4).

portrait and was exposed to less of the toxin than those in the studies, causation cannot be established. Of course, implicit in this requirement is the need for exposure data. It is not enough for plaintiffs to merely say they were exposed to the agent; they must offer credible evidence quantifying that exposure. As the Fifth Circuit has observed, “[s]cientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are *minimal* facts necessary to sustain the plaintiffs’ burden in a toxic tort case.”¹⁶

The second and third factors are commonly referred to as the “differential diagnosis” analysis (more correctly called the “differential etiology” analysis). This analysis is critical in situations where several known causes of a particular disease exist. In some instances, the epidemiological studies that support general causation may have controlled for these “other factors.” But when they have not, an assessment needs to be made on whether the individual plaintiff has or has not been affected by these other factors. If factors other than the toxin in question could have independently caused the harm, and the plaintiff cannot rule out these other factors, the plaintiff cannot prove the toxin caused the harm.¹⁷

¹⁶*Allen v. Pa. Eng’g Corp.*, 102 F.3d 194,199 (5th Cir. 1996) (emphasis added); *see also Wright v. Willamette Indus. Inc.*, 91 F. 3d 1105, 1106 (8th Cir. 1996) (reversing the judgment of the district court, the appellate court held that “a plaintiff in a toxic tort case must prove the levels of exposure that are hazardous to human being generally as well as the plaintiffs’ actual level of exposure to the defendant’s toxic substance before he or she may recover.”)

¹⁷*Cf.* RESTATEMENT (THIRD) TORTS: LIABILITY FOR PHYSICAL HARM § 26(I) (illustration 5) (describing situation where an infection and an adverse drug reaction are factors that independently could explain a child’s death; if plaintiff cannot rule one out or show that the
(Continued on following page)

Alternatively, if the other causes combine with the toxin to cause the harm, then the manufacturer of that toxin should shoulder some of the responsibility.

PRINCIPLE 4:
RISK ASSESSMENT IS THE BEST TOOL AVAILABLE
TO ANSWER QUESTIONS OF CAUSATION

“The framework outlined for evaluating general and specific causation consists of elements that parallel those contained in the risk assessment framework used by regulatory agencies.”¹⁸ Risk Assessment as a scientific discipline gained traction in 1983 when the National Academy of Sciences published the *Red Book*.¹⁹ The *Red Book* outlines a four-step process for evaluating risk:

Step one involves the determination of whether a particular agent is or is not causally linked to a particular health effect. This is the same question as the “general causation” inquiry and principally involves consideration of the epidemiology and the Bradford Hill criteria.

(Continued from previous page)

adverse drug reaction is the more likely cause of disease, the plaintiff cannot recover); *Coleman v. Danek Med., Inc.*, 43 F. Supp. 2d 637, 650 n. 23 (S.D. Miss. 1999) (stating that “in reaching his conclusion that these plaintiffs were injured by Danek’s product, Dr. Aldreti did not rule out other causes of their alleged injuries. Thus, his conclusion that their injuries were caused by Danek’s product is based on pure speculation – and is not a valid differential diagnosis.”)

¹⁸Joseph Rodricks, “Evaluating Disease Causation in Humans Exposed to Toxic Substances,” 14 J.L & POL’Y 41, 62 (2006)

¹⁹NATIONAL RESEARCH COUNCIL, RISK ASSESSMENT IN THE FEDERAL GOVERNMENT: MANAGING THE PROCESS (1983).

Step two involves using the epidemiology to understand the relationship between the amount of exposure to the toxic agent (the dose) and the resulting risk (or probability) of disease (the dose-response relationship).

Step three involves assessing how much an individual or group of individuals are exposed to a particular toxin. This, of course, requires accurate exposure data and work histories.

Lastly, step four involves characterizing the risk. This requires plotting the exposure levels (determined in step three) along the dose-response curve (established in step two), and identifying a corresponding risk of disease, along with an uncertainty measurement (the confidence interval). To the extent there are unique features of the individual plaintiff, quantitative or qualitative adjustments can be made to the dose-response curve, which will affect where the individual plaintiff will fall on that curve. With this information in hand, one can ask “what is the probability that the plaintiff’s exposure to agent A caused disease B and is the probability sufficiently high to meet legal requirements of causation?”

Although this approach is not terribly novel (after all, it simply integrates the core components of general and specific causation that most courts adhere to), few courts, if any, have explicitly adopted the risk assessment paradigm for this purpose. Part of the problem is that risk assessments are most commonly known for their use as regulatory tools and the decisional rules in the regulatory context are different from those used in litigation. The rest of the problem is that

employing a risk assessment framework to answer questions of causation (and not simply risk) can result in misuse, if not abuse, of that paradigm – thus, necessitating principle #5.

PRINCIPLE 5:
ALTHOUGH RISK ASSESSMENT IS THE BEST
SINGLE TOOL AVAILABLE TO ANSWER LITIGATION-BASED
CAUSATION QUESTIONS, ITS USERS MUST
STICK WITH OBSERVATION, NOT SPECULATION

Regulators are faced with the statutory or regulatory charge of protecting the population from exposures to toxic agents. Oftentimes, agencies like OSHA are tasked with setting exposure levels for various chemicals so that the corresponding risk associated with such exposure levels is very small. But, typically such risks are not observable due to the fact that epidemiology studies are not sensitive enough to capture the effect of low level exposure to a toxic agent. Accordingly, regulators must extrapolate from *observed* risks found at higher exposure levels and inferentially draw conclusions about what the risk might be at lower levels of exposure. Regulators often assume that the dose-response curve has no threshold; meaning even at low levels of exposure where risk is not statistically observed, regulators assume some measure of risk nonetheless exists. This is oftentimes expressed as a mathematical model called the linear, no-threshold model. The assumption that “risk” exists, even if it cannot be observed, is premised on the core toxicological principle that the more

one is exposed to a chemical substance, the greater the risk of having some adverse health effect.

While appropriate in the public health context where policy decisions oftentimes are predicated on being cautious and speculation often takes a back seat to understandably risk averse regulators, in the court of law such speculative extrapolations have no place.²⁰

Unfortunately, some litigants have used the regulatory model to argue that there is no safe level of exposure to a toxic agent and to further argue that any amount of exposure can increase one's risk of harm. As a theoretical construct, the regulatory risk model supports such claims. But the model is just that, a model. In the *unobserved* range, conclusions about risk and harm are not based on empirical data. Unless there is actual data available, one cannot draw reliable conclusions about risk and probability of harm at very low levels of exposure. The absence of evidence of 'no harm' is not the same as evidence of harm. Even the regulators will tell you that.²¹ And at vanishingly low levels of exposure, drawing conclusions about risk defies common sense.

²⁰See, e.g., *Castellow v. Chevron, USA*, 97 F. Supp. 2d 780, 792 (S.D. Tex. 2000) (rejecting expert testimony where even if exposure assessment of 177 ppm/years of benzene was correct, the "relevant scientific/medical literature is conclusive only in documenting [plaintiff's condition] in human beings after benzene exposure levels reach beyond 200 ppm/years").

²¹See, e.g., Environmental Protection Agency, *The Risk Assessment Guidelines of 1986 at 1-9* (Aug. 1987) ("It should be emphasized that the linearized multistage procedure leads to a plausible upper limit to the risk that is consistent with some proposed mechanism of carcinogenicity. Such an estimate, however, does not necessarily give a realistic prediction of the risk. The true value of the risk is unknown, and may be as low as zero.")

Courts that have been thoughtful about this analysis have rejected the assumption that high-dose studies can be used to estimate low-dose disease. In *In re Toxic Substances Cases*, for example, Judge Colville ruled that:

The fallacy of the ‘extrapolation down’ argument is plainly illustrated by common sense and common experience. Large amounts of alcohol can intoxicate, larger amounts can kill; a very small amount, however, can do neither. Large amounts of nitroglycerine or arsenic can injure, larger amounts can kill; small amounts, however, are medicinal. . . . In short, the poison is in the dose.²²

The misuse of the regulatory risk assessment model is most apparent in the asbestos context. There, some experts have successfully espoused the view that even exposure to a single fiber of asbestos increases one’s risk of contracting mesothelioma.²³

Thankfully, some recent court decisions have rejected the one fiber theory and have helped stem the tide, reaffirming basic principles of toxicology and putting regulatory risk assessment models in their proper place.²⁴

²²2006 WL 2404008 at *7 (Pa. Ct. Com. Pl. Aug. 17, 2006). A federal bankruptcy court rendered a similar decision recently, holding that the “use of the no safe level or linear ‘no threshold’ model for showing unreasonable risk ‘flies in the face of the toxicological law of dose-response.’” *In re W.R. Grace & Co.*, 355 B.R. 462, 476 (Bankr. D. Del. 2006) (quoting Federal Judicial Center’s REFERENCE MANUAL ON SCIENTIFIC EVIDENCE).

²³*See, e.g., Jones v. John Crane, Inc.*, 35 Cal. Rptr. 3d 144 (Ct. App. 2005) (holding that regardless of amount of exposure, asbestos exposure is sufficient to establish causation). In fact, this approach has been used by lawyers to cast an ever wider net of putative defendants who are alleged to have caused at least one fiber’s worth of exposure. *See, e.g., Chavers v. Gatke Corp.*, 107 Cal. App. 4th 606, 610 (2003) (plaintiff “joined as defendants scores of manufacturers, suppliers and distributors of friction brake products containing asbestos – 59 named defendants and 800 ‘Doe’ defendants”).

²⁴*See, e.g., In re W.R. Grace & Co.*, 355 B.R. 462 (Bankr. D. Del. 2006).

PRINCIPLE 6:
**WHERE THERE ARE MULTIPLE EXPOSURE SOURCES
FOR THE SAME TOXIN, A MORE PRINCIPLED,
OBJECTIVELY RELIABLE METHODOLOGY SHOULD BE
USED TO ANSWER QUESTIONS OF CAUSATION.
CONCEPTS LIKE “SUBSTANTIAL CONTRIBUTING”
CAUSE SHOULD BE JETTISONED**

According to the *Restatement*, “tortious conduct must be a factual cause of physical harm for liability to be imposed. Conduct is a factual cause of harm when the harm *would not have occurred absent the conduct.*”²⁵ The notion that the harm would not have occurred absent the conduct is commonly referred to as the “but for” test. Stated differently, and in the toxic tort context, the “but for” test requires that exposure to the manufacturer’s product was necessary for the individual to become sick. In many instances, this test means that only significant actual exposures are deemed the legal cause of a disease outcome. But as demonstrated below, if applied in the strictest sense, the “but for” test can result in inequities for plaintiffs and defendants.

To illustrate this point, take, for example, exposure to the toxin asbestos and assume that the dose of asbestos needed to double the risk of mesothelioma is 20 units of exposure. If an asbestos worker is exposed to 9 units of asbestos from one manufacturer’s product and 11 units from another manufacturer’s product, both the 9 and 11 unit exposures would be necessary (“but for”)

²⁵RESTATEMENT (THIRD) TORTS: LIABILITY FOR PHYSICAL HARM § 26 (emphasis added).

conditions of that plaintiff's disease. If the plaintiff had been exposed to only one product (let's say the product that generated 9 units of exposure), that individual would not be able to prove as a matter of law and science that her exposure caused her disease, as she would have fallen well short of the 20 units necessary to prove causation. But, in the scenario where the plaintiff had exposures to both products and received the needed 20 units, it would not be unreasonable to conclude that each of the two manufacturers contributed in some significant way to the plaintiff's disease. An equitable approach under that scenario would be for each manufacturer to pay a proportionate share of the damages resulting from the injury.

Now assume there were three manufacturers and three exposure sources, each resulting in the following units of exposure: 9, 11, and 12 units. Any combination of *two* of these exposures would be sufficient to cause plaintiff's disease (assuming, again, 20 units are necessary to reach the "more probable than not" threshold – or doubling risk threshold). Accordingly, whatever the third exposure is would be technically unnecessary to cause harm. It would not be a "but for" cause. Nevertheless, under this scenario, each manufacturer should be held liable – not because its conduct will always be the "but for" cause, but rather because under certain non-excludable circumstances (i.e., circumstances that cannot be ruled out), the conduct *could be* a "but for" cause.²⁶

²⁶*Cf.* RESTATEMENT (THIRD) TORTS: LIABILITY FOR PHYSICAL HARM § 27 (comment g) (citing *Eagle-Picher Indus. Inc. v. Balbos*, 604 A.2d 445, 459 (Md. 1992)).

Take a third scenario: The plaintiff is exposed to just two exposure sources: 26 units of exposure from one source and 9 units from another. Under this scenario, a rational outcome would allow the plaintiff to recover from the manufacturer causing 26 units of exposure, but not the manufacturer who caused 9 units. The 9 unit exposure is neither a necessary, nor sufficient cause of the plaintiff's disease.

A fourth scenario is the sufficient, but not necessary, scenario. Here, the plaintiff is exposed to 26 units of exposure from one manufacturer and 26 units of exposure from another manufacturer. Under this scenario, both exposures are sufficient, but neither are necessary. As described by the *Restatement*, plaintiff's harm is "overdetermined." While neither exposure technically satisfies the "but for" test because neither exposure was necessary to cause harm, most would agree that both (or either) manufacturer should shoulder the responsibility of compensating the plaintiff.²⁷ Of course, only one manufacturer should shoulder the burden if it could be shown that the exposure to 26 units from one manufacturer predated the exposure to the additional 26 units from another manufacturer and the first 26 units were sufficient to cause the harm. This scenario is similar to what the *Restatement* defines as "preemptive causes" –

²⁷*Id.* § 27 cmt. A ("court has long imposed liability when a tortfeasor's conduct, while not necessary for the outcome, would have been a factual cause if the other competing cause had not been operating").

where hunter X negligently shoots and kills hiker Y; and then afterwards hunter Z negligently shoots the already dead hiker Y.²⁸

As is readily apparent from just these four scenarios, the “but for” test, strictly construed and without modification, does not always sensibly apply to factual scenarios found in the real world. And, sometimes even the same level of exposure is treated very differently depending on the factual scenarios at play.

Take a fifth scenario: Assume the plaintiff is exposed to 1 unit, 3 units, 4 units, 5 units, 7 units, 8 units and 9 units; what then? Under this fact pattern, the plaintiff would have been exposed to a total of 37 units, 17 more than needed to prove legal cause under our hypothetical. Under most circumstances, the 1 unit of exposure is neither sufficient nor necessary to cause disease. In fact, the *Restatement (Third) of Torts* would likely describe the 1 unit in this context as a “trivial contribution” and thus liability likely would not apply.²⁹

But what if the 1 unit of exposure was necessary to cause disease? Imagine the plaintiff is exposed to the following units of exposure from different manufacturing sources: 1 unit, 3 units, 4 units, 5 units, and 7 units. Totaled, this equals 20 units of exposure. Assuming, again, that the plaintiff could demonstrate through epidemiology that her 20 units of exposure caused her

²⁸*Id.* at § 26(k). *Cf.* § 28(b) (providing that when more than one defendant could have been the cause of the harm, but plaintiff cannot determine which actually caused the harm, the burden of production and persuasion shifts to the defendants).

²⁹“When an actor’s negligent conduct constitutes only a trivial contribution to a causal set that is a factual cause of physical harm under § 27, the harm is not within the scope of the actor’s liability.” *Id.* § 36.

disease, what do we say about the manufacturer who caused the plaintiff to be exposed to just 1 unit of exposure; 1/20 of the total deemed necessary to prove causation in a court of law? Should that manufacturer pay? Pay 1/20 of the liability?³⁰ In the 20 unit example, there are no multiple sufficient causes, which would otherwise render the 1 unit superfluous. In the 20 unit example, the 1 unit, while trivial, is still necessary for the harm to occur; it is the proverbial “straw that breaks the camel’s back.”

The same would also hold in the 37 unit example. One can envision a situation where even when there are multiple sufficient causes, the extra 1 unit of exposure nevertheless plays a significant, non-trivial, role. For example, in the 37 unit scenario, what if the extra 1 unit of exposure occurred after the plaintiff was exposed to 19 other units of exposure? Though it would be impossible to prove in most situations, the 1 unit would have actually sealed the individual’s fate. The 17 units that came later may not have played any role in disease causation.

In sum, one can come up with a myriad of different factual scenarios that render the same unit of exposure (the 1 unit example) meaningless, insignificant, moderately important, or very important. While much of tort law is premised on finding the victim as he or she appears (the so-called thin skull or “eggshell plaintiff” rule), there is something unsettling about having a manufacturer’s

³⁰*Id.* § 36(b) (“The exception applies only when there are multiple sufficient causes and the tortious conduct at issue constitutes a trivial contribution to any sufficient causal set. . . . The limitation on the scope of liability provided in this Section is not applicable if the trivial contributing cause is necessary for the outcome; this Section is only applicable when the outcome is overdetermined.”).

liability (such as the 1 unit manufacturer's liability) vary so fundamentally depending on factors that are outside of that manufacturer's control.

One solution is to only require the manufacturer to pay if plaintiff can demonstrate that exposure to its product is alone sufficient to cause disease. Under this decisional standard, regardless of the exposure from other sources, if the exposure to any manufacturer is anything less than 20 units, that manufacturer assumes no liability. Under this solution, a manufacturer causing 21 units of exposure would be fully responsible; whereas, a manufacturer of 19 units to the same plaintiff would have no liability. Certainly for the manufacturer of 21 units this is an unpalatable resolution. For the manufacturer causing 19 units of exposure, an observer could reasonably conclude that it "escaped" liability.

Another solution would be to allow all exposures that may have been a "but for" cause to be actionable. Many jurisdictions do just that because it is often very difficult to separate the true cause of disease from multiple possible causes. But such a decisional rule could render even the most *de minimis* exposures a "but for" cause. The mere fact that a manufacturer could be liable for such *de minimis* exposure would incentivize plaintiffs to name every possible exposure source, regardless of the amounts of exposure. And, in jurisdictions that still have strict joint and several liability rules, this could result in a grave injustice for several of the marginal manufacturers. Some suggest this problem is cured by requiring the plaintiff to show the manufacturer made a "substantial

contribution” in exposures, such that more than *de minimis* exposures would be required.³¹ But the so-called “substantial contribution” test offers no predictability or consistency because it has no defined terms or boundaries. It is what litigants and ultimately judges and juries wish it to be, as opposed to what science requires.³²

A third solution and the one the author offers here would involve using the tools of risk assessment. Assuming the epidemiology was valid and a causal association established, and a plaintiff was exposed to a sufficient number of total units of cumulative exposure above that established as causal (say 20 units), the dose response curve could be used, along with its confidence intervals, to identify how much additional exposure is need to actually *observe* a measurable increase in risk. An increment of exposure less than the 20 units would not result in the assignment of liability to a individual manufacturer responsible for that increment unless two elements were satisfied. First, the exposure would need to be a “but for” cause in the strictest sense. In other words, the exposure would have to be necessary to cause the disease, but need not be sufficient. Second, although the exposure need not be sufficient to cause the disease, unless the

³¹See, e.g., *Laney v. Celotex Corp.*, 901 F.2d 1319, 1320-21 (6th Cir. 1990) (defendant was allowed to introduce evidence of other asbestos exposures to show that its product was not a substantial contributor in causing plaintiff’s asbestos disease).

³²The substantial factor test originated in the Restatement of Torts §§ 431-32 and was included again in the Restatement (Second) of Torts §§ 431-32. According to the Restatement (Third), the test has “not, however, withstood the test of time, as it has proven confusing and been misused.” RESTATEMENT (THIRD) TORTS: LIABILITY FOR PHYSICAL HARM § 26(j).

additional contribution of exposure was itself sufficient *to increase, in an observable way, the estimated relative risk of harm beyond the upper bound on the confidence interval*, there would be no legal proof of causation.

This approach balances the equities and is grounded in the very scientific principles that make up causation and risk analysis. While the plaintiff can recover a proportionate share from a manufacturer who, in isolation, could not have caused the plaintiff's disease, it only asks the manufacturer to pay if the exposure amounts are *observed* to be significant contributors to total risk as defined by the dose-response model itself and not the vagaries of semantics.

WLF LEGAL POLICY ADVISORY BOARD

The Honorable Dick Thornburgh

*Kirkpatrick & Lockhart Preston
Gates Ellis LLP
Washington, D.C.*

CHAIRMAN OF THE BOARD

Frank J. Fahrenkopf, Jr., Esq.
Hogan & Hartson LLP

The Honorable Richard K. Willard

Step toe & Johnson LLP

CHAIRMEN EMERITUS

Joseph A. Artabane, Esq.
Artabane & Belden, P.C.

Mary Azcuenaga, Esq.
Heller Ehrman White & McAuliffe

Professor Thomas E. Baker
Florida International University
College of Law

Mark A. Behrens, Esq.
Shook, Hardy & Bacon L.L.P.

Thomas Hale Boggs, Jr., Esq.
Patton Boggs

Gary B. Born, Esq.
Wilmer Hale

Michael K. Brown, Esq.
Reed Smith LLP

George M. Burditt, Esq.
Bell, Boyd & Lloyd

The Hon. James H. Burnley, IV
Venable LLP

Henry N. Butler
Director, Judicial Education
Project, AEI-Brookings Joint
Center for Regulatory Studies

Ralph J. Caccia, Esq.
Powell Goldstein LLP

The Honorable Tom Campbell
Dean, Haas School of Business
University of California, Berkeley

Michael A. Carvin, Esq.
Jones Day

Charles Cooper, Esq.
Cooper & Kirk

Joseph E. diGenova, Esq.
diGenova & Toensing

Professor Viet D. Dinh
Georgetown University Law Center

The Honorable John Engler
President and CEO
National Association of
Manufacturers

Richard L. Frank
*Olsson Frank Weeda Terman
Bode Matz PC*

The Honorable Harold Furchtgott-Roth
Furchtgott-Roth Economic
Enterprises

Kenneth S. Geller, Esq.
*Mayer, Brown, Rowe & Maw
LLP*

Stuart M. Gerson, Esq.
Epstein Becker & Green, P.C.

Albert Gidari, Esq.
Perkins Coie LLP

Benjamin L. Ginsberg, Esq.
Patton Boggs LLP

Steven B. Hantler
Chairman
American Justice Partnership

Professor William F. Harvey
Dean Emeritus & Carl M. Gray
Emeritus Professor of Law
Indiana University School of
Law

Professor Maurice J. Holland
University of Oregon
School of Law

The Honorable Troy King
Attorney General
State of Alabama

Clarence T. Kipps, Jr., Esq.
Miller & Chevalier

Philip A. Lacovara, Esq.
*Mayer, Brown, Rowe & Maw
LLP*

Susan W. Liebeler, Esq.
Lexpert Research Services

Arvin Maskin, Esq.
Weil, Gotshal & Manges

The Honorable Robert F. McDonnell
Attorney General
Commonwealth of Virginia

The Honorable Rob McKenna
Attorney General
State of Washington

John A. Merrigan, Esq.
DLA Piper

Professor John Norton Moore
University of Virginia
School of Law

Mark C. Morrill
Senior Vice President & Deputy
General Counsel
Viacom Inc.

The Honorable Gerald J. Mossinghoff
*Oblon, Spivak McClelland, Maier
& Neustadt, P.C.*

Glen D. Nager, Esq.
Jones Day

The Honorable Theodore B. Olson
Gibson, Dunn & Crutcher LLP

W. Hugh O'Riordan, Esq.
Givens, Pursley & Huntley

Robert D. Paul, Esq.
White & Case

Professor Stephen B. Presser
Northwestern University
School of Law

Professor George L. Priest
John M. Olin Professor of
Law and Economics
Yale University

Charles F. (Rick) Rule, Esq.
*Cadwalader, Wickersham &
Taft LLP*

The Honorable Mark Shurtleff
Attorney General
State of Utah

The Honorable Kenneth W. Starr
Dean, Pepperdine University
School of Law

The Honorable Don Stenberg
Former Attorney General
State of Nebraska

The Honorable Hal Stratton
Dykema Gossett PLLC

Ambassador Robert S. Strauss
*Akin, Gump, Strauss, Hauer &
Feld*

The Honorable Eugene R. Sullivan
CEO, Gavel Consulting Group

George J. Terwilliger, Esq.
White & Case

Daniel E. Troy, Esq.
Sidley Austin LLP

The Honorable William F. Weld
McDermott Will & Emery

Joe D. Whitley, Esq.
Greenberg Traurig, LLP

Wayne Withers
Former Senior Vice President
& General Counsel
Emerson Electric Co.