

**THE CAUSAL CONUNDRUM:
EXAMINING THE MEDICAL-LEGAL
DISCONNECT IN TOXIC TORT CASES
FROM A CULTURAL PERSPECTIVE
OR
HOW THE LAW SWALLOWED THE
EPIDEMIOLOGIST AND GREW
LONG LEGS AND A TAIL**

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ABSTRACT:

The literature regarding the law-epidemiologic disconnect in causal proof is vast, non-conclusive and partisan. Some favor weakening the plaintiff's burden in cases of ambiguous causation (when we do not have enough objective scientific proof), even if bastardizing scientific requisites is necessary. Scientific purists, of course, object. Some suggest crafting novel legal causes of action, such as proportionate liability. Legal purists reject this approach. The conflict has been raging for decades and we are no closer to resolving the issue than when it was first raised some thirty years ago.

This research takes a novel approach at resolving the dilemma, trying to understand the divide from a cultural, or "poetics," perspective before even attempting to reconcile the disconnect. After discussing five conflicting decisions reaching contrary scientific conclusions regarding 'specific' and 'general' causation, and different legal resolutions regarding admissibility of expert testimony, I set forth a detailed discussion of the elements of "poetics," a method of examining how meanings get mangled across the communication divide. Using this methodology, I examine the cultural and linguistic dissonance of tort law and science. Then, with these tools in hand, I re-visit the poetics of

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law, epidemiology, and medicine, only to discover not only “poetic failure” among the three disciplines, but internal discord within the field of epidemiology itself. Exposing these faults within the foundational underpinnings of epidemiology highlights difficulties faced by the field, which, when incorporated into legal discourse, compromises legal outcomes. Once the flaws were exposed, options for legally reliable and scientifically valid resolution became apparent.

However, not until courts appreciate the inherent discord within epidemiology itself and the resultant limitations in addressing multi-causation, will we reach a point where far-reaching resolution is even on the event horizon, and we are doomed to generating ever conflicting opinions.

I. INTRODUCTION

[I]t would be a dangerous undertaking for persons trained only to the law to constitute themselves the final judges of the worth of [a work], outside of the narrowest and most obvious limits.¹

—Oliver Wendell Holmes

Rudyard Kipling’s *Just So Stories* have the theme of a particular animal being modified from its original form to its current state by acts of humans or some magical being. No description could come closer to the morphed state epidemiology has assumed in legal parlance than the narrative of Kipling’s *How the Kangaroo Got its Legs*.²

To paraphrase Kipling’s opening lines,³ “Not always was the Epidemiologist as now we do behold him, but a different animal with four long legs and a long tail.”⁴ The statement is apt. *Tail* is an epidemiological term that refers to the skewness of a population distribution; if the test population is not normally distributed, it provides a poor basis for extrapolation, either to the plaintiff or the public. *Long legs* is a

1. *Bleistein v. Donaldson Lithographing Co.*, 188 U.S. 239, 251 (1903) (“At the one extreme, some works of genius would be sure to miss appreciation. Their very novelty would make them repulsive until the public had learned the new language in which their author spoke.”).

2. RUDYARD KIPLING, *THE JUST SO STORIES* (1895).

3. *See id.*

Not always was the Kangaroo as now we do behold him, but a different animal with four short legs and a tail. He was grey and he was woolly, and his pride was inordinate: he danced on an outcrop in the middle of Australia, and he went to the Little God Nqa. He went to Nqa at six before breakfast, saying, “Make me different from all other animals by five this afternoon.”

Id.

4. A long tail is the portion of the distribution having a large number of occurrences far from the “head” or central part of the distribution. B2Bwhiteboard, *Long Tail (Statistics)-Explained*, YouTube (Oct 17, 2013), <https://www.youtube.com/watch?v=G0Vkitqv8Ug>; RICHARD C. DICKER, *PRINCIPLES OF EPIDEMIOLOGY IN PUBLIC HEALTH PRACTICE, AN INTRODUCTION TO APPLIED EPIDEMIOLOGY AND BIostatISTICS* (3d ed. 2012).

colloquial term referring to *staying power*, such as the hold the present application of epidemiology has had on legal causation, even when perhaps it is unsuited for the task.

Kipling's story ends with the following verse, eerily foreshadowing problems inherent in proving legal culpability: "So they were left in the middle of Australia, Old Man Kangaroo and Yellow-Dog Dingo, and each said, 'That's your fault.'"⁵

II. BACKGROUND

Numerous attempts at reconciling disconnects between epidemiologic and legal proof have been attempted⁶ and proven wanting.⁷ Some commentators have "reconfigured science" for legal use, or diluted conventionally accepted scientific standards to ease the plaintiff's burden⁸ where traditional scientific proof is unavailable.⁹ These approaches have been met with resistance by purists of both the scientific and legal professions.¹⁰

Before trying to resolve problems in "causal ambiguity" (which I define as instances when enough definitive science to meet current legal standards is unavailable),¹¹ I attempt to understand the medico-legal divide using a novel approach, assessing the issue from a cultural perspective. In so doing, I propose that the causal conundrum arises from dissimilar histories, linguistic misunderstandings and cultural conflicts. Using case studies and a comparative discipline analysis, I demonstrate a cultural and linguistic mismatch, a "poetic failure"¹² between the disciplines. In other words, judges, lawyers, cli-

5. KIPLING, *supra* note 2, at 2.

6. See, e.g., RICHARD GOLDBERG, PERSPECTIVES ON CAUSATION (2011).

7. See Sana Loue, *Comparing Epidemiological and Legal Causation Without the Benefit of Poetics*, in STATISTICAL SCIENCE IN THE COURTROOM 264 (Joseph L. Gastwirth ed., 2000).

8. CARL F. CRANOR, TOXIC TORTS, SCIENCE, LAW AND THE POSSIBILITY OF JUSTICE 283-86 (2006). *But see* Nathan Schactman, *Desultory Thoughts on Milward v. Acuity Specialty Products*, RESEARCHGATE (Oct. 2015), available at <https://www.researchgate.net/publication/282816421> (providing a scathing assessment).

9. See FH 4693/05 Carmel Haifa Hosp. v. Malul, 2010 IsrLR 1 (2010) (Isr.), available at <http://versa.cardozo.yu.edu/sites/default/files/upload/opinions/Carmel%20Haifa%20Hospital%20v.%20Malul.pdf>.

10. See generally Nathan Schactman, *How Does the New Reference Manual on Scientific Evidence Treat Statistical Evidence?*, SCHATMAN L. (July 8, 2012, 9:58 AM), <http://schactmanlaw.com/reference-manual-on-scientific-evidence-3d-edition-on-statistical-significance/> (Schactman replies "[i]nconsistently and at times incoherently"); see also Schactman, *supra* note 8 (The reader is referred generally to Schactman's superb and insightful series of articles on *Daubert* accessible via his website).

11. See Barbara P. Billauer, et al., *The Toxic Tort is Ill: Deficiencies in the Plaintiff's Case and How to Prove Them*, in CHEMICAL CONTAMINATION AND ITS VICTIMS 74-77 (1989); see also ARIEL PORAT & ALEX STEIN, TORT LIABILITY UNDER UNCERTAINTY (2001).

12. Shulamit Almog, *Creating Representations of Justice in the Third Millennium: Legal Poetics in Digital Times*, 32 RUTGERS COMPUTER & TECH. L.J. 183, 211 (2006).

nicians (treating physicians) and epidemiologists simply do not understand each other, not surprisingly generating confusion and legal conflicts.

Instead of crafting an artificial middle ground or imposing additional obstacles to address perceived scientific deficiencies,¹³ I suggest that resolving the issue first requires recognizing the impasse. Further, I suggest that the clinician's view of epidemiology is far different than, and perhaps more relevant to, proving legal causation than the current "public-health epidemiology" paradigm.¹⁴ I begin the inquiry with a case-study analysis of five cases.

A. SPECIFIC V. GENERAL CAUSATION: WHY DO THE RESULTS DIFFER So?

In the pivotal case of *Parker v. Mobil Oil Corp.*,¹⁵ the New York Court of Appeals held that the factors needed to prove causation in toxic tort cases¹⁶ are: (1) exposure, (2) general causation,¹⁷ and (3) specific causation.¹⁸ Exposure addresses whether the amount of toxin to which the plaintiff was exposed¹⁹ was sufficient to cause the disease in question,²⁰ a standard arising as early as 1988.²¹ General causation asks whether a substance *can* cause the disease. Specific

13. Barbara Pfeffer Billauer, *Admissibility of Scientific Evidence Under Daubert: The Fatal Flaws of 'Falsifiability' and 'Falsification'*, 22 B.U. J. SCI. & TECH. L. 27-29, 28, 42, 43 (2016).

14. See Alexander A. Reinert, *What is Epidemiology and How is it Being Misused by the Courts*, AM. JUDICATURE SOC'Y (2003); see also Nancy Krieger, *Questioning Epidemiology: Objectivity, Advocacy and Socially Responsible Science*, 89 AM. J. PUB. HEALTH 1151, 1151-53 (1999).

15. 857 N.E.2d 1114 (N.Y. 2006). Eight amicus briefs were submitted, highlighting the multiple interest groups concerned.

16. *Mancuso v. Consol. Edison Co. of N.Y., Inc.*, 967 F. Supp. 1437 (S.D.N.Y. 1997); *Mancuso v. Consol. Edison Co. of N.Y., Inc.*, 56 F. Supp. 2d 391, 399 (S.D.N.Y. 1999) (alleging harm from PCB exposure from contaminated land).

17. See *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1241 (11th Cir. 2005).

18. *Wright v. Williamette Indus., Inc.*, 91 F.3d 1105, 1106 (8th Cir. 1996) ("[A] plaintiff in a toxic tort case must prove the levels of exposure that are hazardous to human beings generally as well as the plaintiff's actual level of exposure to the defendant's toxic substance before he or she may recover."). See also *Wade-Greux v. Whitehall Labs*, 874 F. Supp. 1441 (D.V.I. 1994).

19. I.e., route of exposure affecting the critical dose to the target organ: inhalation, ingestion, or dermal.

20. See generally David Faigman, et al., *Group to Individual (G2i) Inference in Scientific Expert Testimony*, 81 U. CHI. L. REV. 417 (2014) (noting the general versus specific causation issue "Group to Individual Inference"). He proposes two types of expert evidence: framework evidence describes general scientific propositions; and diagnostic evidence applies these to individual cases, but gives little insight into how conflicts arose or how to reconcile the vastly differing case decisions. See also *McClain*, 401 F.3d at 1241.

21. *Sterling v. Velsicol Chem. Corp.*, 855 F.2d 1188, 1200 (6th Cir. 1988).

causation²² asks whether the substance *did* cause the disease in this plaintiff,²³ seemingly simple issues to understand,²⁴ if not prove.

This three-fold legal inquiry has international acceptance.²⁵ Nevertheless, individual court decisions appear outcome-determinative, societal expectations and outcry seem influential, and contradictions in conceptualization and understanding run rampant. The illustrative cases discussed in the next section, *Parker, Milward v. Acuity Specialty Products Group Inc.*,²⁶ the two Israeli *Kishon* cases, and the *DuPont C-8 cases*, highlight the confusion. The disparate results and reasoning are surely harbingers of United States Supreme Court intervention.

One issue driving the disparity emerges from the exposure element—i.e., determining under which rubric we assess whether the plaintiff was sufficiently exposed. A second problem emerges from determining the competency of experts to testify, which drives the decision.²⁷ Thus, in comparison with medical malpractice cases where many states allow licensed physicians to testify regardless of specialty,²⁸ toxic tort cases are more restrictive.²⁹ Further, admissibility of epidemiological testimony on specific causation has proven difficult, mainly because of a misguided belief regarding its inapplicability to individual diagnosis misconceptions, which I demonstrate, were seeded by epidemiologists themselves.

22. William O. Dillingham et al., *Blueprint for General Causation Analysis in Toxic Tort Litigation*, FED’N DEF. COUNS. Q. (Oct. 1, 2003).

23. Faigman et al., *supra* note 20.

24. FED. JUDICIAL CTR., FEDERAL MANUAL ON SCIENTIFIC EVIDENCE 444 (2d ed. 2000).

25. (3.11.2013, פורסם בנבו, ת”א (חי’) 01/732 זולי נ’ היפה כימיקלים בע”מ (פורסם בנבו, 24.9.2015). ע”א, ת”א (חי’) 13/6102 עצמון נ’ היפה כימיקלים בע”מ (פורסם בנבו, 1.10.2015). נ’ היפה כימיקלים 414.04 אבנ, אורי ת”א (א) 13/6102 עצמון נ’ היפה כימיקלים בע”מ (פורסם בנבו, 1.10.2015). In the Israeli case of *Atzman v. Haifa Chemicals*, [hereinafter the *Diver’s Case*], Judge Zarankin wrote:

Accordingly when the question arises of potential factual causal connection, the plaintiff must prove not only potential causal facts (exposure capable of causing disease) but also the specific factual causal connection (exposure associated with the defendant caused the disease in the plaintiff’s body considering his personal characteristics including other risk factors).

Diver’s Case.

26. 664 F. Supp. 2d 137 (D. Mass. 2009).

27. See Joe Cecil, *Ten Years of Judicial Gatekeeping Under Daubert*, 95 AM. J. PUB. HEALTH 574, 576-79 (2005).

28. See Steve Babitsky, *State Specific Rules Governing Testifying as an Expert Witness in Medical Malpractice Case*, SEAK (July 31, 2014), <http://www.testifyingtraining.com/state-specific-rules-governing-testifying-as-an-expert-witness-in-medical-malpractice-case/> (showing that as of 2014, twenty-three states had few or no rules governing the specialty of a medical expert allowed to testify in malpractice cases).

29. See FED. R. EVID. 702, 703.

1. *Parker v. Mobil Oil Corp.*

The *Parker* case concerned a gas station attendant who claimed his seventeen-year benzene exposure via gasoline inhalation caused his acute myelogenous leukemia (“AML”).³⁰ The court ruled there is no dispute that benzene is a known carcinogen.³¹ The issue, however, involved whether gasoline, a component of which is benzene, is carcinogenic because no significant association has been found between gasoline exposure and AML via epidemiologic study.³² The court ruled that regulatory standards were inadequate to demonstrate legal causation,³³ did not allow extrapolation of the benzene studies³⁴ to gasoline-containing-benzene exposure,³⁵ and refused to acknowledge the possibility of synergism.³⁶

Another key issue in *Parker* was lack of exposure measurement. Without curing this infirmity, the court ruled that specific causation could not be established.³⁷ Thus, the court rejected the plaintiff’s expert vague exposure assessment³⁸ because his characterization was adjectival. The court held the words “frequently” and “excessive” were speculative,³⁹ not based on reliable methodology, and hence unscientific.⁴⁰ Although the court recognized alternative means of scientific exposure methods,⁴¹ these were not used.

30. *Cancer Stat Facts: Acute Myeloid Leukemia*, NAT’L CANCER INST., <https://seer.cancer.gov/statfacts/html/amyl.html> (last visited Jan. 7, 2018) (AML is a rare type of cancer of the white blood cells, with an incidence of about four cases per 100,000 per year).

31. *Parker v. Mobil Oil Corp.*, 857 N.E.2d 1114 (N.Y. 2006).

32. *See Burst v. Shell Oil Co.*, 104 F. Supp. 3d 773 (E.D. La. 2015) (holding plaintiff’s expert’s testimony was unreliable because he had not evaluated studies on gasoline exposure).

33. *Id.* *See also* *Milward v. Rust-Oleum Corp.*, 820 F.3d 469 (1st Cir. 2016).

34. *See* MYRON A. MEHLMAN, *RISK ASSESSMENT AND RISK MANAGEMENT OF INDUSTRIAL AND ENVIRONMENTAL CHEMICALS* 17 (C. Richard Cohen, et al., eds. 1988) (“The EPA believes that the human and animal evidence provides an adequate basis for classifying benzene as a human carcinogen . . . [and] . . . that ambient exposures [to benzene] may constitute a cancer risk . . .”).

35. *See Parker*, 857 N.E.2d at 1116 (benzene gasoline is allowed by the EPA up to an average of .62% by volume with a maximum of 1.3%).

36. *See* FRANK C. LU, *BASIC TOXICOLOGY, FUNDAMENTALS, TARGET ORGANS, AND RISK ASSESSMENT* 65 (3d ed. 1996).

37. *Parker*, 857 N.E.2d at 1116.

38. *See id.* (“It is not always necessary for a plaintiff to quantify exposure levels precisely . . . provided whatever methods an expert uses to establish causation are generally accepted in the scientific community . . .”).

39. *See id.* at 1122 (“As with any other type of expert evidence, we recognize the danger in allowing unreliable or speculative information [‘junk science’] to go before the jury with the weight of an impressively credentialed expert behind it.”).

40. *Id.*

41. *See, e.g.*, Andrea Spinazze et al., *Accuracy Evaluation of Three Modelling Tools for Occupational Safety Assessment*, in 61 *ANNALS OF WORK EXPOSURES AND HEALTH* 284-98 (2017); *see also* Anila Bello et al., *Retrospective Assessment of Occupation Expo-*

Thus, while the defendants' epidemiologist "acknowledged that there is an increased risk of AML for service station employees exposed to large amounts of benzene⁴² over an extended period of time," he concluded that the low levels of benzene exposure typically resulting from gasoline service station work were "below the practical threshold for the dose necessary to initiate the leukemia process"

Nevertheless, the impact of the plaintiff's exposure to cigarettes (of which benzene is a component)⁴³ as adding to his benzene-burden was not raised or considered. Thus, the subsequent exposure to the benzene in the gasoline may have added to his cumulative benzene exposure, exacerbating benzene-related cigarette smoking injuries.⁴⁴ Alternatively, the gasoline containing benzene may have acted as a cumulative or joint causal agent. These approaches do not seem to have been raised by the plaintiff and were not considered by the court.⁴⁵

2. Milward v. Acuity Specialty Products Group, Inc.

The benzene-causation saga continues in *Milward*, where Brian Milward sought redress for his Acute Promyelocytic Leukemia ("APL"),⁴⁶ an even rarer form of cancer than AML.

Following his thirty-year exposure as a refrigerator technician and pipefitter, Mr. Milward was diagnosed with APL. The trial ruled that Milward needed to show that exposure to benzene can cause APL (general causation), and that exposure to benzene was, in fact, a substantial factor in the development of Milward's APL (specific causation).⁴⁷ The first phase of the first trial concerned whether the plaintiff's expert opinion on "general causation" was admissible under Federal Rule of Evidence 702. If proven, the second phase would have considered negligence, exposure, and "specific causation." The trial court rejected plaintiff's general causation expert, a toxicologist who

tures for the GENEVA Study of ALS (Amyotrophic Lateral Sclerosis) Among Military Veterans, in 61 ANNALS OF WORK EXPOSURES AND HEALTH 298-310 (2017).

42. "[T]ypically over 100 PPM TWA." See *Parker*, 857 N.E.2d at 1116.

43. See J.E. Korte et al., *The Contribution of Benzene to Smoking-Induced Leukemia*, 108 ENVTL. HEALTH PERSP. 333-39 (2000).

44. Of course, exacerbation of a pre-existing injury generally needs to be raised in the initial pleadings, perhaps explaining why this approach was not raised at trial. Further, the joint exposure to benzene from both the cigarettes and the gasoline might have rendered the gasoline a joint or concurrent cause, again approaches which were not considered by the court and do not seem to have been raised by the plaintiffs.

45. The full exposition of this approach is outside the scope of this Article and awaits further research.

46. See NAT'L CANCER INST., *supra* note 30. Acute promyelocytic leukemia is a subtype of acute myelogenous leukemia.

47. *Milward v. Acuity Specialty Prods. Grp., Inc.*, 664 F. Supp. 2d 137, 148 (D. Mass. 2009).

relied on epidemiological evidence, because the data did not reach statistical significance.⁴⁸ The appellate court reversed,⁴⁹ refusing to exclude plaintiff's expert testimony merely because the results were not statistically significant.⁵⁰ To do otherwise, it held, would deny plaintiff his day in court,⁵¹ a holding criticized by many as both scientifically and legally untenable.⁵²

a. Exposure—A General or Specific Causation Issue?

Following the appellate ruling that general causation existed between benzene and APL,⁵³ most parties settled, leaving Rust-Oleum the lone defendant and specific causation the sole issue, i.e., did Milward's exposures to benzene cause or constitute a substantial contributing factor of his leukemia.⁵⁴

Seeking to avoid the exposure quantification problems of *Parker*, Milward presented testimony of an industrial hygienist who quantified his exposure. They also proffered Sheila Butler, an occupational physician,⁵⁵ who was described as:

[M]ore than an ordinary clinician who diagnoses and treats sick individuals, [Butler is] . . . board certified in occupational medicine, anatomic pathology, clinical pathology and hematology with over ten years of experience as a practicing diagnostic hematopathologist, and as a consultant on occupationally-related malignancies and . . . figuring out the causes of chronic illnesses in patients exposed to toxic substances is what she does day in and day out.⁵⁶

48. See SIR AUSTIN BRADFORD HILL, A SHORT TEXTBOOK OF MEDICAL STATISTICS 117 (1977) (defining statistical significance as the likelihood results were not due to chance, a conventional practice in the field that strict *Daubert* adherence should require).

49. *Milward v. Acuity Specialty Prods. Grp., Inc.*, 639 F.3d 11, 14 (1st Cir. 2011).

50. *Milward*, 639 F.3d at 15.

51. *Kennedy v. Collagen Corp.*, 161 F.3d at 1453, 1459 (9th Cir.1998), *cert. denied*, 526 US 1099 (1999) (finding the district court placed too much emphasis on lack of epidemiological studies where such studies would be almost impossible to perform).

52. See *In re Meridia Prods. Liab. Litig.*, 328 F. Supp. 2d 791, 800 (N.D. Ohio 2004) ("As long as the basic methodology employed to reach such a conclusion is sound . . . [,] products liability law does not preclude recovery until a 'statistically significant' number of people have been injured . . ."); see generally *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993) (allowing relevant scientific testimony to be admitted, so long as it was reliable, overruling the *Frye* standard); *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923) (requiring proffered scientific testimony to conform with the consensus in the scientific community).

53. See generally *Matsuyama v. Birnbaum*, 890 N.E.2d 819 (Mass. 2008).

54. *Milward v. Rust-Oleum Corp.*, 820 F.3d 469 (1st Cir. 2016).

55. *Rust-Oleum*, 820 F.3d at 469 ("Occupational medicine is the field of diagnosing and treating workplace diseases. It relies heavily on patient history of past exposure and knowledge of epidemiological evidence connecting exposure to disease.")

56. *Id.*

Butler opined that there was a reasonable medical probability that Milward's benzene exposure was a cause-in-fact of his APL, basing her opinion "on toxicology . . . what I know about the biology, the pathophysiology . . . the disease process,"⁵⁷ the temporal relationship between exposure and Milward's leukemia and selected epidemiological studies showing increased relative risk following low average dose exposures to benzene.⁵⁸ Testimony that Milward had a cumulative exposure of 25.6 ppm [particle per million] years were compared with a peer-reviewed study finding that workers exposed to benzene at far lower levels were seven times more likely than controls to develop leukemia, supported her opinion.⁵⁹ A later-filed affidavit included a differential diagnosis in which Butler eliminated other possible causes of Milward's leukemia.

b. Differential Diagnoses

Through the differential diagnostic technique (essentially a process of elimination), Dr. Butler "ruled out" the more common factors associated with APL, including obesity and smoking. She then determined that since benzene exposure was a potential cause, she could "rule out" idiopathic causes (diagnoses without a known cause), and, since benzene was the only significant potential cause remaining, it was likely the culprit.⁶⁰ While the court noted "differential diagnosis" is a useful and accepted means of assessing causation,⁶¹ particularly where an expert cannot provide epidemiological studies,⁶² it rejected

57. See *Schultz v. Akzo Nobel Paints, LLC*, 721 F.3d 426 (7th Cir. 2013) (indicating that the trial court rejected plaintiff's expert because he subscribed to no threshold theory). The appellate court reversed, focusing on the multi-stage complex process of carcinogenesis, "where many factors work together to contribute to the ultimate emergence of a full-blown malignancy . . . [each of which] must properly be considered a cause of the ultimate cancer and a substantial factor in bringing it about." Regarding specific causation analysis, the court held that:

[T]he mere fact that genetics and/or other environmental risk factors have been identified as probable causes of a particular case of cancer . . . in no way refutes the possibility that chemical exposure[s] being investigated have also played a substantial contributing role at one or more stages of the development of that person's cancer

Schultz, 721 F.3d 426.

58. *In re Breast Implant Litig.*, 11 F. Supp. 2d 1217, 1224 (D. Colo. 1998).

59. See generally FED. JUDICIAL CTR., *supra* note 24, at 611-12 (discussing the doubling of relative risk to establish specific causation).

60. *Milward*, 820 F.3d at 469.

61. See *In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 755 (3d Cir. 1994); see also *Baker v. Dalkon Shield Claimants Tr.*, 156 F.3d 248, 253 (1st Cir. 1998).

62. *Hardyman v. Norfolk & W. Ry. Co.*, 243 F.3d 255, 262 (6th Cir. 2001). See also *In re Heparin Prods. Liab. Litig.*, 803 F. Supp. 2d 712 (N.D. Ohio 2011) (allowing plaintiffs' case to proceed on a differential diagnosis basis rather than epidemiology).

its use here relying on the Restatement (Third) of Torts,⁶³ stating Butler's reasoning was circular; i.e., that she "ruled out" an idiopathic APL by "ruling in" benzene, but failed to provide a scientifically reliable method of "ruling in" benzene in the first place. Even if Butler could rule out smoking and obesity, the court said differential diagnosis provided little information because of the high percent of cases of APL without identifiable causes.

c. Relative Risk

The court dismissed Butler's reliance on selected epidemiological studies because she did not weight them against others.⁶⁴ While acknowledging the studies that she did rely on were published in peer-reviewed journals (which by itself should have allowed it to pass *Daubert*⁶⁵ muster),⁶⁶ the court discounted the significance, noting that "an article does not reach the dignity of a 'reliable authority' merely because some editor, even a most reputable one, sees fit to circulate it"⁶⁷ and mere publication cannot make them an automatically reliable authority.⁶⁸ In essence, the court required Butler to dissect and distinguish studies at odds with her position—a function usually reserved for cross-examination and/or proffer of the defendant's expert⁶⁹—in effect requiring her to cross-examine herself.⁷⁰ The court's objection was manifold: Butler was not an epidemiologist, she did not evaluate all the epidemiologic literature, her interpretation of relative risk evidence was not "scientific," and her use of differential diagnosis

63. See Restatement (Third) of Torts § 28 cmt. c(4) (Am. Law Inst. 2010) ("The underlying premise [of differential etiology] is that each of the known causes is independently responsible for some proportion of the disease in each population. When the causes of a disease are largely unknown . . . [,] differential etiology is of little assistance.").

64. See *Rust-Oleum*, 820 F.3d 469 (citing 3 DAVID FAIGMAN, MODERN SCIENTIFIC EVIDENCE § 23:27 (2014-2015 ed.)) (discussing the use of the relative risk in establishing specific causation).

65. *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993).

66. *Daubert*, 509 U.S. 579. The Court assigned the judge the role of gatekeeper to ascertain that only scientifically reliable evidence would be admitted and suggested five non-binding tests, including publication in a peer-reviewed journal. *Id.*

67. See *Rust-Oleum*, 820 F.3d at 475 n.4 (quoting *Meschino v. N. Am. Drager, Inc.*, 841 F.2d 429, 434 (1st Cir.1988)) ("We have noted though, an article does not reach the dignity of a 'reliable authority' merely because some editor, even a most reputable one, sees fit to circulate it . . . [and] [m]ere publication cannot make them automatically reliable authority. . . . Given the need for some evidence establishing the reliability of the studies invoked, the court likewise did not err in refusing to take judicial notice of their reliability."); see also FAUST F. ROSSI, EXPERT WITNESSES 142 (1991); *Meschino*, 841 F.2d at 434; *Kace v Liang*, 36 N.E.3d 1215 (Mass. 2015).

68. *Kace*, 36 N.E.3d 1215. See also *Milward*, 639 F.3d at 15.

69. *Milward*, 639 F.3d at 15.

70. See *Hollander v. Sandoz Pharm. Corp.*, 289 F.3d 1193 (10th Cir. 2002) (holding the district court abused its discretion in excluding expert opinion based on differential diagnosis when the diagnosis was supported by the scientific literature).

was also unscientific because too many unknown causes of APL exist to be sure these (unknown) causes were not the actual causal culprit—and dismissed the case.

The dissent was livid. Unsparing in its criticism, the dissent noted that an opinion does not have to conclusively prove causation to be admissible, stating that medical knowledge, we can all agree, is often uncertain:

The human body is complex, etiology is often uncertain, and ethical concerns often prevent double-blind studies calculated to establish statistical proof.⁷¹ [But that] does not preclude the introduction of medical expert opinion testimony when medical knowledge permits the assertion of a reasonable opinion [The plaintiffs] do not necessarily have the burden of disproving a study championed by the other side—that is what a case the majority relies on says The proponents must show that [their expert] arrived at [her] contrary opinion in a scientifically sound and methodological fashion. And if they do, the question becomes one for the jury to decide.⁷²

The dissent remains—the dissent.

C. THE DUPONT C-8 CASES

The pending cases against DuPont, whose perfluorooctanoic acid (“C-8”)⁷³-contaminated drinking water in six water districts in West Virginia⁷⁴ affected 80,000 residents,⁷⁵ further illustrate the problem. In a non-precedential agreement (“Leach Agreement”),⁷⁶ DuPont con-

71. *Milward*, 639 F.3d at 15.

72. *Id.*

73. Elizabeth V. Young, *Outsourcing the Jury: Bartlett v. DuPont and the Role of Alternative Adjudication in Preserving Jury “Fairness” in Complex Scientific Litigation*, 77 OHIO ST. L.J. 15 (2015).

74. See *In re E.I. DuPont De Nemours & Co. C-8 Pers. Injury Litig.*, Case No. 2:13-md-2433 (S.D. Ohio Apr. 9, 2013).

75. Jack Kaskey, *DuPont’s W.Va., Ohio Neighbors Worry Chemours Won’t Pay C8 Bills*, CHARLESTON GAZETTE-MAIL (May 7, 2015), https://www.wvgazette.com/business/dupont-s-w-va-ohio-neighbors-worry-chemours-won-t/article_b4811868-9a81-5c75-9409-d55f9eea0f74.html. See also *DuPont C8 Cases*, slip op. (S.D. Ohio Apr. 9, 2013); *The Brookmar C8 Health Project (CO)*, HILL PETERSON, CARPER, BEE & DEITZLER, PLLC, <https://www.hpcbd.com/Personal-Injury/DuPont-C8/The-Brookmar-C8-Health-Project-CO.shtml> (last visited Jan. 7, 2018).

76. “Based on the findings of the C8 Science Panel, Dupont is precluded from denying that C8 can cause the six linked disease in any trials that arise from claims made by Class Members (as defined within the Leach class action litigation).” *C-8 Settlement Project*, HILL, PETERSON, CARPER, BEE & DEITZLER, PLLC, <https://www.hpcbd.com/> (last visited Jan. 7, 2018). See also David Andrews & Bill Walker, *Poisoned Legacy*, ENVTL. WORKING GROUP, Apr. 2015, at 4, <https://www.ewg.org/research/poisoned-legacy#WjczVFQ-fBI>. In pre-trial maneuvering, DuPont tried to renege on a key promise it made in the 2005 settlement: that in the case of any resident who drank contaminated water and sued over a disease the science panel determined has a probable link to

ceded “general causation”⁷⁷ if a special epidemiological study proved that it is more likely than not that there is a link between C-8 exposure and the class members’ diseases.⁷⁸ Presumably, DuPont’s high-priced lawyers⁷⁹ understood what “general causation” meant when they signed on. Seven years later, a world-class epidemiological study⁸⁰ concluded that, indeed, “it is more likely than not that there is a link between exposure to C-8 and six different diseases among [3,500] members”⁸¹ who lived in the affected vicinity for at least a year.⁸² The actionable diseases were high cholesterol, thyroid disease, testicular cancer, kidney cancer, ulcerative colitis, and pregnancy-induced hypertension (pre-eclampsia),⁸³ diseases with little or no obvious biological relationship.

Difficulty arose when it came time to determine what the ostensibly simple phrase “general causation” meant. The defendants claimed their concession was only that the chemicals—*at a certain threshold exposure*—to be later determined, can cause these diseases.⁸⁴ The

C-8 exposure, DuPont would concede that C-8 could cause the disease in that group of people. The trial judge ruled against DuPont. *Id.*

77. See *In re E.I. DuPont De Nemours & Co. C-8 Pers. Injury Litig.*, No. CV 2:13-md-2433, 2016 WL 2946195, at *1 (S.D. Ohio May 19, 2016). The court stated:

Class [members] who allege they suffer or suffered from one or more Linked Diseases [may] . . . pursue . . . claims “for personal injury and wrongful death . . . and any other damages whatsoever associated with such claims that . . . relate to exposure to C-8 of Class Members[,]” and DuPont agreed not to contest general causation in those actions. . . . DuPont retained the right to contest specific causation and to assert any other defenses not barred by the Leach Settlement Agreement. For a full background of the Leach Case, see Dispositive Motions Order No. 12. (ECF No. 4306).

Id. (internal citations omitted) (emphasis added).

78. See *Leach v. E.I. DuPont de Nemours & Co.*, No. 01-C-608, 2002 WL 1270121 (W. Va. Cir. Ct., Apr. 10, 2002).

79. The average partner hourly billing rate of Squire Patton Boggs, who represented DuPont in this case is, \$665.00. See NAT’L LAW JOURNAL, www.nationallawjournal.com/id=1202636785489/Billing-Rates-Across-the-Country.

80. The panel, consisting of Dr. Tony Fletcher of the London School of Hygiene and Tropical Medicine, Dr. David Savitz of Mt. Sinai School of Medicine, and Dr. Kyle Steenland of Emory University, produced reports that can be found at <http://www.hpcbd.com/Personal-Injury/DuPont-C8/The-Science-Panel.shtml>, and public presentations. See, e.g., Stephanie J. Frisbee, et al., *The C8 Health Project: Design, Methods, and Participants*, ENVTL. HEALTH PERSP. (July 13, 2009); see also Tony Fletcher, *The Health Risks of PFOA and PFOS: Biomarkers vs. Exposure Assessment* (July 8, 2015).

81. See G.W. Olsen, *Epidemiologic Assessment of Worker Serum Perfluorooctanesulfonate (PFOS) and Perfluorooctanoate (PFOA) Concentrations and Medical Surveillance Examinations*, 45 J. OCCUPATIONAL ENVTL. MED. 260-70 (2003).

82. *In re E.I. DuPont De Nemours & Co. C-8 Pers. Injury Litig.*, 2016 WL 2946195, at *3 n.1 (S.D. Ohio May 19, 2016) (citing Leach Settlement Agreement § 1.49).

83. Young, *supra* note 73.

84. See generally Joint Amicus Brief of the U.S. Chamber of Commerce, Am. Tort Reform Ass’n & Am. Chem. Council, *In re DuPont De Nemours & Co. C8 Pers. Injury Litig.*, No. 16-3310, 2016 WL 34115291 (6th Cir. June 20, 2016); *In re E. I. DuPont De Nemours*, Case No.: 2:13-CV-170, 2016 WL 659112, at *1-3 (S.D. Ohio Feb. 17, 2016).

plaintiffs argued, and the lower court agreed, that the agreement subsumed the exposure requirement for all plaintiffs in the class,⁸⁵ limiting the defendants to a “differential diagnosis” defense, i.e., that the plaintiffs’ disease came from some other cause. After losing at the trial level, the defendants appealed.

DuPont clearly reserved the right to argue specific causation.⁸⁶ But what, exactly, does *that* mean? The defense argued plaintiffs must prove they had sufficient exposure. They further claimed “[t]he district court’s error short-circuited this process for determining causation issues by essentially directing a verdict for Bartlett. No rational defendant would agree to delegate away its *entire* causation defense to an independent panel in this manner.”⁸⁷ Of course, the *entire* causation defense was never part of the agreement; the defendants always had the *Milward* defenses available—i.e., did other exposures cause the disease? The defense’s mischaracterization of the decision implies they did not understand what they were doing. Their argument certainly raises eyebrows. It is hard to imagine the parties agreeing to the delay and expense of an epidemiological study, merely to show that theoretically C-8 *can* cause cancer. That information could have been obtained via animal studies or simpler/shorter studies.

And therein lies the rub. What exactly does the simplest of phrases, “general causation,” really mean? To whom?⁸⁸ Who decides? On what basis? And which experts can testify about it?

One amicus brief claims the Leach Agreement defined general causation according to “the accepted understanding of general causa-

85. See generally, *Chronology*, HILL, PETERSON, CARPER, BEE & DEITZLER, PLLC, <http://www.hpcbd.com/Personal-Injury/DuPont-C8/C8-Health-Effects-Chronological.shtml>.

86. See *In re E.I. DuPont De Nemours & Co. C-8 Pers. Injury Litig.*, 2016 WL 2946195, at *1 (S.D. Ohio May 19, 2016).

87. Joint Amicus Brief of the U.S. Chamber of Commerce, Am. Tort Reform Ass’n & Am. Chem. Council, *supra* note 84 (emphasis added). Since the issue arose from a non-precedential agreement that bound only DuPont, the keen interest of outsiders is perplexing.

88. See *In re E.I. DuPont De Nemours & Co. C-8 Pers. Injury Litig.*, No 2:13-CV-1103, slip op. (S.D. Ohio Apr. 9, 2013). One amicus brief argued:

[T]he court erroneously transformed the Probable Link Finding into a finding that the plaintiff’s exposure to a very low level of C8 more likely than not did cause her kidney cancer. This error contradicted the plain text of the Leach Agreement, departed from settled causation analysis, and deprived DuPont of a bargained-for specific causation defense A key issue at trial thus should have been whether Bartlett’s exposure to C8 at the lowest level reliably measured could have materially increased her risk of kidney cancer.

Joint Amicus Brief of the U.S. Chamber of Commerce, Am. Tort Reform Ass’n & Am. Chem. Council, *supra* note 89. The plain meaning, however, is that the court only held that C-8 can cause the plaintiff’s kidney cancer at the exposure levels she suffered—whether it in fact did was for the jury determine. *Id.*

tion [that] does not tie the concept to any particular dose or exposure level; it concerns whether a substance is capable at all of causing a given condition.”⁸⁹ However, there is no generally accepted understanding of either general or specific causation, either in science or law, other than what the plain words convey to the listener or reader—which differs depending on the listeners or readers. The confusion over meaning is quite apparent in amici briefs, one claiming:

General causation is usually shown by applying complex statistical methods to large-scale public health data sets This inquiry often involves complicated and competing expert testimony about dose-response curves, regression analysis, and the strength of statistical associations⁹⁰

Notably, the *Milward* court did not follow this formula, allowing a toxicologist to establish general causation, and leaving specific causation to be determined via epidemiological evidence and/or a differential diagnosis that ruled out competing causes. The Leach Agreement is silent on the issue and the United States Court of Appeals for the Sixth Circuit has yet to decide.

D. “THE KISHON AFFAIR”⁹¹

The causal quagmire is further exemplified by two Israeli cases concerning pollution of the Kishon river, colloquially known as “The Kishon Affair.”⁹² The cases arose from exposures to the same chemical soup of pollutants: arsenic, nickel, chromium, cadmium, lead,⁹³ and benzene. One case was brought by an elite cadre of Naval commandos, another by a group of fishermen. Both groups alleged multiple injuries from their exposures. The courts, recognizing the difficult

89. Joint Amicus Brief of the U.S. Chamber of Commerce, Am. Tort Reform Ass’n & Am. Chem. Council, *supra* note 84 (indicating not many courts define general causation other than whether the substance could cause cancer). One court that did define general causation was the court in *Wade-Greux v. Whitehall Labs., Inc.*, 874 F. Supp. 1441 (D.V.I. 1994), *aff’d*, 46 F.3d 1120 (3d Cir. 1994) (“[G]eneral causation concerns whether the agent is capable of causing [disease] in humans at therapeutic dose levels.”).

90. FED. JUDICIAL CTR., *supra* note 24, at 599-600 (internal citations omitted).

91. The *Diver’s Case* was decided by Judge Adi Zarankin: אביטל עצמון ואח’נ’ היפה 972-00,414-04,577-04,670-04 17/07/2011 *Levy Tuli v. Haifa Chemicals Ltd.* was decided by Judge Shapira 732-01 455-02 03/11/2013 ת”א (חי’) (3.11. 2013). פסק-דין בתיק ת”א 01/732 01/732 תולד’נ’ היפה כימיקלים בע”מ (פורסם בגבו, 2013). [hereinafter *Fisherman’s Case*]. The cases were appealed and counter-appealed to the Supreme Court sitting as the Court of Civil Appeals, coming before Justice Y. Amit, Judge Z. Gilberta, Justice N. Solberg: 24.9.2015, CA 2880/14Y ע”א 13/6102 עצמון נ’ היפה כימיקלים בע”מ (פורסם בגבו, 2015) [hereinafter the *Kishon Cases*].

92. See generally *Diver’s Case; Fisherman’s Case; Kishon Cases*.

93. See ALICE HAMILTON, *EXPLORING THE DANGEROUS TRADES* 128-62 (1943). The *Kishon* court’s exposure assessment neglected older, historic occupational exposure situations that would have alerted them to the importance of lead dust *inhalation*. *Id.*

questions of causal proof in these cases, noted the American experience⁹⁴—requirements for general and specific causation (along with exposure) and relied on American law⁹⁵ for determining evidentiary admissibility⁹⁶ of medical evidence.⁹⁷ But Israeli courts typically impose an additional obstacle: “legal causation.”⁹⁸ Unlike the American concept, which is essentially policy driven, the Israeli version relates back to evidentiary admissibility and involves identifying a logical nexus between the facts and conclusion via three different tests: a risk (probability) test, a test of expectations, and a test of common sense.⁹⁹ Absent direct evidence of a causal link, the court will consider epidemiological evidence to be probative, albeit circumstantial evidence, if it is logically connected to the facts.¹⁰⁰

While the evidence in the two lower *Kishon* courts differed, the results were the same: both judges held general and specific causation were not proven (upheld in a joint appeal). Nevertheless, the ultimate outcome could not have been more different. The commandos eventually received redress, albeit under a unique administrative remedy crafted to address the unprecedented national concerns sparked by naval commandos exposed to toxins during their required military service.

94. Judge Zarankin stated: “In the United States, such claims . . . have become so widespread ‘that they have been given a definition of their own—Toxic-Torts.’” *Diver’s Case*.

95. “Israeli courts when asked about the criteria for obtaining scientific evidence, relied, *inter alia*, on the Daubert standard, and the tests prescribed in it. See 7093/10 State of Israel v. Orna Dreazen (July 1, 2012); see also CrimA 9724/02, Abu Hamad v. State of Israel (2003).71 (1) מ"י פ"ד נח 02/9724 ע"ש *Fisherman’s Case*; *Diver’s Case*.

96. “The matter of Daubert . . . is important in our eyes.” *Kishon Cases*, *supra* note 91.

97. See *Diver’s Case*, *supra* note 25, at 18. The court stated:

A. Proof that the harmful substances were found, at the times relevant to the claim, in the waters of the *Kishon*; B. Scientific evidence according to which exposure to these substances can cause illness exhibited by each plaintiff; C. Actual Proof demonstrating that the activity of each plaintiff in the waters of the *Kishon* exposed him to the alleged harmful substances; D. Proof that the alleged exposure, is, in fact, that which caused the disease.

Id.

98. See *generally id.* The court provided:

After proving the existence of a potential factual link, both general and specific, between the alleged negligence and the damage, there still stands before the plaintiff the additional hurdle of proving the legal connection between the negligence and the damage, namely the existence of a legal causal connection between the two.

Id. (author’s translation).

99. *Id.*

100. The *Kishon* court’s decision formalizes the *ipse dixit* ruling of Gen. Elec. Co. v. Joiner, 522 U.S. 136 (1997).

E. EXPERT-DETERMINATIVENESS

In the *Diver's Case*, Judge Zarankin ruled the plaintiffs “failed miserably to prove their causal claim from both a scientific and factual perspective,”¹⁰¹ accepted the defendants’ occupational health physician-expert’s views on causation, and snidely dismissed the plaintiffs’ chief expert, Professor Hod, calling him a veterinarian whose claimed deficits included “not having an expertise in oncology, or any clinical or clinical research experience.”¹⁰² The appellate court sustained this finding, calling Dr. Hod’s expertise in cancer “self-proclaimed.” While Dr. Hod indeed lists his professorship of veterinary medicine on his curriculum vitae, his qualifications are far more impressive and relevant than the judges would have people believe.¹⁰³ Discounting Professor Hod’s expertise in cancer research because it was not performed on humans,¹⁰⁴ the appellate court demonstrates its ignorance in cancer research.¹⁰⁵ But it gets worse.

F. GENERAL AND SPECIFIC CAUSATION: CARCINOGENESIS AT THE BAR

To put the issues in starkest relief, this article focuses on rulings relating to benzene alleged to have caused several blood and lymph cancers in twelve Kishon plaintiffs. Nine of the cases (i.e., nine plaintiffs manifesting three types of cancer) are amply supported by the medical literature as having a causal connection to benzene.¹⁰⁶

101. *Diver's Case*, *supra* note 25.

102. In addition to his full professorship at Hebrew University, per his online curriculum vitae, Dr. Hod is a medical doctor (MD) with a PhD who has published on oncology and vascular diseases and expertise in, *inter alia*, comparative medicine, malignant occupational diseases, and diagnosis of neoplastic diseases.

103. *Id.*

104. The appellate court wrongly stated that “Professor Hod has no expertise or experience in the field of medical causal connection, nor occupational medicine.”

105. The failure of plaintiff’s lawyers to bring to the court’s attention the positive benzene studies (as well as the dangers of lead dust *inhalation*) may be due to cultural differences in the practice of law between Israel and the United States where lawyers are more intricately involved in developing scientific proof. Indeed, one comprehensive list of scientific causal literature was found on the website of an American law firm, as were others located by this author.

106. See, e.g., Jelle Vlaanderen, et al., *Occupational Benzene Exposure and the Risk of Lymphoma Subtypes: A Meta-analysis of Cohort Studies Incorporating Three Study Quality Dimensions*, ENVTL. HEALTH PERSP. 159–67 (2011). Vlaanderen states:

The International Agency for Research on Cancer (IARC) classified benzene as a group 1 carcinogen (carcinogenic to humans) in its 1982 and 1987 evaluations . . . [,] particularly . . . acute myelogenous leukemia (AML). Recently, . . . IARC determined . . . there was . . . limited evidence that benzene causes acute lymphocytic leukemia (ALL), chronic lymphocytic leukemia (CLL), non-Hodgkin’s lymphoma (NHL), and multiple myeloma (MM) in humans.

Id. See also M.T. Smith, *Benzene Exposure and Risk of non-Hodgkin Lymphoma*, CANCER EPIDEMIOLOGY BIOMARKERS & PREVENTION 385-91 (2007) (“Exposure to benzene, . . . [a] component of gasoline, is a widely recognized cause of leukemia . . . [,] and] we conclude that, overall, the evidence supports an association between occupational benzene

The court noted the plaintiffs tried to establish their claim without “epidemiological-statistical evidence,”¹⁰⁷ failed to rule out other exposures or lifestyle factors,¹⁰⁸ that cancer is common in the population regardless of unusual exposure to hazardous substances, and that most “of the population of Western men suffer cancer at one point or another” In a 180 degree about face with the *Parker*¹⁰⁹ holding, Judge Zarankin opined that the absence of confirmatory studies in the petrochemical or gas stations companies was determinative of no benzene-disease causation. He further added that “all of us are exposed to substances known to be cancer risk factors, without becoming ill and that multiple cancers cannot be attributed to one causal agent,” plainly contradicting the epidemiological panel in the *DuPont C-8 cases*, the scientific literature, and *subjectively* minimizing pollution risks.

Unlike the *Diver’s Case*, the plaintiffs in the *Fishermen’s Case* used epidemiology—which was rejected.¹¹⁰ Here, too, vilified the testimony of plaintiffs’ expert, a noted epidemiologist:

[T]he plaintiffs abandoned the field of epidemiology and sought to base themselves on “qualitative epidemiology” . . . epidemiology deals with the assessment of the risk of disease, and by its very nature, its definition is quantitative science, and it has not been proven that there is an accepted theory of qualitative epidemiology in the scientific world.¹¹¹

exposure and NHL.”). Other literature suggests cumulative exposure from different routes increases the risk. See JOSEPH V. RODERICKS, *CALCULATED RISKS: THE TOXICITY AND HUMAN HEALTH RISKS OF CHEMICALS IN OUR ENVIRONMENT* 57 (1992). Three plaintiffs suffered Hodgkins’ Leukemia, for which no causal association is noted in the literature. Their inclusion in the class reflects lawyers who did not research the medicinal causation issues—or poor litigation strategy. *Id.*

107. At least one epidemiological study, however, did determine a causal relationship existed in the *Kishon* matter. See Elihu Richter, et al., *Cancer Risks in Naval Divers with Multiple Exposures to Carcinogens*, 111 ENVTL. HEALTH PERSP. 609–17 (2003) (indicating this research was not mentioned in the decision). Its findings also were at odds with the Shamgar Commission Panel, comprising of toxicologist Meir Wilchek and epidemiologist Gad Renner. See Tal Golan, *The Fall and Rise of the Kishon River*, 8 WATER 283 (2016) (noting some questioned Richter’s exposure assessment); Yona Amitai et al., *Cancer Risk to Navy Divers Questioned*, 111 ENVTL. HEALTH PERSP. 1571-76 (2003); see also Adi Oren, Zeev Aizenshtat, & Benny Chefetz, *Persistent Organic Pollutants and Sedimentary Organic Matter Properties: A Case Study in the Kishon River, Israel*, 141 ENVTL. POLLUTION 265-74 (May 2006).

108. The possibility of multiple causation is not addressed. See *Schultz v. Akzo Nobel Paints, LLC*, 721 F.3d 426 (7th Cir. 2013) (acknowledging, but ultimately rejecting, synergistic causation of the various substances).

109. 857 N.E.2d 1114 (N.Y. 2006).

110. The judge plainly did not understand that *Daubert* allows scientific testimony that is not generally accepted by the scientific community.

111. See *Fishermen’s Case*, *supra* note 91, at 40.

Rejecting general causation of benzene,¹¹² the court dismissed findings of the International Agency for Research on Cancer¹¹³ and an NIH study. The appellate opinion also ratified the importance of excluding other risk factors (e.g., smoking, sun exposure, genetics)—impliedly believing causation must be but/for (or single-agent), based on the notion that different cancers must have different causes.¹¹⁴

III. INVESTIGATING THE CASES FROM A CULTURAL PERSPECTIVE

A. INTRODUCTION

The “Kishon Affair” raises yet a new problem: legal discord in carcinogenesis. For the sake of legal consistency, one scientific principle should govern all related causal decisions. Thus, one substance either can cause multiple diseases, (as in the *DuPont C-8 cases*), or it cannot (as the *Kishon Cases* judges opined, relying on unspecified “conventional wisdom”); either benzene can cause certain leukemias (*Milward*¹¹⁵ and *Parker*¹¹⁶), or it cannot (*Kishon*); either “exposure” decisions are part of general causation, (*DuPont C-8 cases*) or specific causation (*Milward*)—but not both (*Kishon*).

That there are different answers to these questions sounds a red alert that there is a causal schism between law and science; and within science, there is a disconnect between clinicians and epidemiologists (and lawyers who rely on them). In sum, courts, judges, doctors, epidemiologists, and lawyers are at linguistic loggerheads.¹¹⁷ As stated earlier, rather than trying to reconcile these discordant holdings, this article attempts to decipher them from a cultural perspective, using the vehicles of poetics¹¹⁸ and social epistemology.

112. See *Kishon Cases*, *supra* note 91.

113. Norman Breslow & Nick Day, *Innovation in Statistical Methods*, INT’L AGENCY FOR RES. ON CANCER, https://www.iarc.fr/en/publications/books/iarc50/IARC_Ch4.1.2_web.pdf (last visited Jan. 9, 2018) (“Epidemiological studies aimed at investigating causes of cancer were—and are—at the core of IARC’s research.”).

114. See *Kishon Cases*, *supra* note 91 (“Indeed, in the language of the people, cancer is seen as a single disease, manifested in the uncontrolled distribution of cells. But conventional wisdom is that cancer should not be seen as a single disease, but as a variety of diseases that have different causes, different development, and different target organs. Not every substance known as cancer is a risk factor for all cancers, and there are cancerous diseases whose risk factors are not known.”).

115. 664 F. Supp. 2d 137 (D. Mass. 2009).

116. 857 N.E.2d 1114 (N.Y. 2006).

117. JAMES WHITE, *HERCULES’ BOW: ESSAYS ON THE RHETORIC AND POETICS OF THE LAW* 126 (1989) (“Language is continuous with culture for it provides the social and factual description of motive and value that makes our culture what it is.”).

118. See SHULAMIT ALMOG, *HOW DIGITAL TECHNOLOGIES ARE CHANGING THE PRACTICE OF LAW* 81 (2007).

B. POETICS

Poetics is a system of rules that delineates how meanings are generated in a particular field¹¹⁹ and why they are accepted as meaningful in a particular way,¹²⁰ i.e., “understanding . . . how a text’s different elements come together and produce certain effects on the reader.”¹²¹ Today, “poetics” is applied to any field, not just the literary domain.¹²² A scientific inquiry into poetics, or the transmission of information such that it is received in the manner intended, i.e., a shared understanding of events,¹²³ includes investigating the related history,¹²⁴ culture, and language—each alone being insufficient.¹²⁵

“Poetic failure occurs when an expression fails to convey its intended meaning. It is an expression that does not make sense or that makes sense other than the one it was intended.”¹²⁶ “Poetic failure [can occur] . . . from a lack of coherence and consistency . . . [or] because it pretends to link non-linkable parts”¹²⁷ The disconnects illustrated above indicate a system gone awry—i.e., “poetic failure.”¹²⁸

Poetic failure may occur not only by errors in transmission, but problems in reception.¹²⁹ And in this second aspect, the law bears responsibility. Part is a language problem, where lawyers use the same words differently than scientists, or make up their own language, e.g., “differential etiology”¹³⁰ to describe their understanding of a medical process. Part is cultural, and here, too, poetics provides assistance:

[Poetics] provides a basis for comparing not cultures themselves, which are in a practical sense incomparables—no one

119. See J. Gassner, *Introduction*, in *ARISTOTLE’S THEORY OF POETRY AND FINE ART* (1951).

120. Shulamit Almog, *When Meaning Matters*, 32 *RUTGERS COMPUTER & TECH. L.J.* 183, 195 (2006).

121. JONATHAN CULLER, *LITERARY THEORY: A VERY SHORT INTRODUCTION* 145 (1997).

122. Almog, *supra* note 120, at 183, 196. See also Anthony K. Webster, *Cultural Poetics (Ethnopoetics)*, in *OXFORD HANDBOOK* (2015); T. BROGAN, *THE NEW PRINCETON HANDBOOK OF POETIC TERMS* (1994).

123. WHITE, *supra* note 117, at 19-20 (1989).

124. David Nelken, *Law, Liability and Culture*, in *FAULT LINES: TORT LAW AS CULTURAL PRACTICE* (Engel & McCann eds., 2009) (“Legal culture, like all culture is a product of the contingencies of history and is always undergoing change.”). See also OLIVER WENDELL HOLMES, *THE COMMON LAW* (1881) (“The history of what the law has been is necessary to the knowledge of what the law is.”).

125. See generally SEHNAZ TAHIR GURCAGLAR, *THE POLITICS AND POETICS OF TRANSFORMATION OF TURKEY 1923-1960* (2008); see also Robert Sheppard, *The Necessity of Poetics*, BBC, <http://www.pores.bbk.ac.uk/1/Robert%20Sheppard,%20The%20Necessity%20of%20Poetics'.htm> (last visited Jan 10, 2018).

126. See ALMOG, *supra* note 118.

127. *Id.*

128. Almog, *supra* note 120.

129. *Id.*

130. See *FED. JUDICIAL CTR.*, *supra* note 24, at 443 (admitting to the non-medical nature of the term).

culture can turn itself into another, however much its members might think they wish to do so. But comparing responses to the cultures: ways in which the patterned resources of meaning that define all cultures, and their limits, are remade. These *are* comparable, for all of us share the problem that our language and the culture it defines are inadequate¹³¹

Poetic failure thus can occur at two ends of the communication spectrum:

At the initiating site, the speaker or writer's inappropriate selections may result in expressions that fail to evoke the desired affect: . . . [But] even a poetically flawless articulation might fail to convey its intended meaning because of the addressee's inability to receive it The addressee's capacity to grasp and comprehend the representation is as important as the skills of its creator. These two factors must work in tandem in order to produce expressions that convey intended meaning and effects.¹³²

C. POETICS IN THE LAW

"Law, in fact, has its own poetics"¹³³ It, too, can be seen at once as a language, as a culture, and as a community.¹³⁴ Because legal poetics brings the capacity to acknowledge the existence of overlapping cultural domains,¹³⁵ its analysis bears relevance to this Article's inquiry: which of the mechanisms of proof: medical (qualitative) or mathematical (statistical/epidemiological), are culturally better "fits" with the toxic tort causal paradigm?

Another characteristic of legal poetics is that cases must "fit together to form a more or less coherent whole,¹³⁶ with a shape and a history The most basic rule of logic (the rule of non-contradiction) and the basic rule of justice require consistency of meaning."¹³⁷ When disparate holdings such as described earlier present themselves, something is amiss,¹³⁸ i.e., we encounter "poetic failure." The poetics

131. WHITE, *supra* note 117, at 130.

132. ALMOG, *supra* note 118, at 82, 83.

133. Almog, *supra* note 120.

134. WHITE, *supra* note 117.

135. Shulamit Almog, *One Young and the Other Old—Halakhah and Aggadah as Law and Story*, 18 CAN. J. L. & SOC'Y 27-43 (2003).

136. See Nelken, *supra* note 124.

137. WHITE, *supra* note 117, at 68, 111.

138. See Ann Scales, *Nobody Broke It, It Just Broke: Causation as an Instrument of Obfuscation and Oppression*, in *FAULT LINES: TORT LAW AS CULTURAL PRACTICE* 273-74 (Engel & McCann eds., 2009) ("Justice demands clarity [about societal] habits, [Clarity would also be the result of giving up the graven worship of quasi-science which she seems to label epidemiology].").

of law¹³⁹ also has the capacity “to convince society that legal institutions uniquely control the activation of justice.”¹⁴⁰ “At the end of the process, the legal speaker is required . . . to express his or her judgment in the most simple binary terms No third possibility is admitted.”¹⁴¹

Since clinical medicine and epidemiology have different mechanisms of proof and languages, only one can be “best suited” to resolve causation in the legal arena; “no third possibility is admitted.” Because the “law as an intellectual and cultural activity is something we do with words¹⁴² [not equations], with our minds [not with experiments] and with each other,”¹⁴³ not in a lab or test-tube, this article suggests epidemiology is less suited to the legal world than clinical medicine, which relies on words, not equations, clinical judgment, not experiments, and doctor-patient relationships, not population statistics.

Complicating the evaluation is that “certain features of legal discourse make it peculiarly difficult for the non-lawyer to understand and to speak.”¹⁴⁴ Nevertheless, it should be recalled that “it is not the vocabulary of the legal language that is responsible for its obscurity and mysteriousness, but its ‘cultural syntax,’ the invisible expectations governing the way words are used.”¹⁴⁵ In these cases, often it is the expectation that but/for or single-source causation is the sole avenue for tortious causal proof.

IV. OF TORTS, TORAH, AND CAUSATION: THE POETICS OF TORTS

A. INTRODUCTION

“Tort law is a cultural phenomenon.”¹⁴⁶

Professors David Engel and Michael McCann claim that “tort law and culture are inseparable dimensions of a single domain in which risk, injury, liability, compensation, deterrence, and normative pronouncements about acceptable behavior are crucial features” Because tort law reflects culture, and just as culture is neither static nor

139. Moshe Gorali, *Letters of the Law: Some Judges' Literary Leanings Make for Long-Winded Decisions and Delays in Court Rulings*, HAARETZ (Feb 11, 2002, 12:00 AM), <https://www.haaretz.com/israel-news/culture/leisure/letters-of-the-law-1.53269>.

140. Almog, *supra* note 120.

141. WHITE, *supra* note 117.

142. See Shulamit Almog, *From Sterne and Borges to Lost Storytellers: Cyberspace, Narrative, and Law*, 13 FORDHAM INTELL. PROP., MEDIA & ENT. L.J. 1 (2002).

143. WHITE, *supra* note 117, at 49.

144. *Id.* at 61, 73.

145. *Id.* at 72.

146. Nelken, *supra* note 124.

singular, Engel and McCann contend that the “culture” reflected in tort law should periodically be “[re]-examined in terms of its multiplicity and variability across time as well as space,”¹⁴⁷ a premise they claim has been largely ignored.¹⁴⁸ By this caveat, legal use of epidemiology, once viewed as the essential determinant of toxic tort causation, should be subjected to periodic re-evaluation. Just as legal principles are recodified based on societal trends,¹⁴⁹ similarly, social responses to injuries change over time¹⁵⁰ and require different responses.

That tort law is reflective of local culture and the times also may help explain variations in verdicts between countries.¹⁵¹ But this maxim is of little help in resolving the discrepancy between the *Kishon Cases* and the American cases. Other than the lack of a jury system, Israel is quite comfortable lifting legal doctrines and practices from other countries,¹⁵² not only British common law, which is explicitly incorporated in their tort statute,¹⁵³ but American law, as evidenced by the specific reliance on *Daubert*. Hence, we need to look deeper to find the source of the discordant resolutions of the causal conundra.¹⁵⁴

Causation, of course, is one of four elements of the tort of negligence,¹⁵⁵ and “is thus more than a method of measuring damages,¹⁵⁶ but more like a connecting bridge between negligence and harm that

147. *Id.* at 9.

148. *Id.* at 3. See also Ken Oliphant, *Cultures of Tort Law in Europe* 3 J. EUR. TORT L. 147 (2012) (asserting that, at least as of 2012, the field of the culture of law was still in its infancy).

149. The “proximate cause” of *Palsgraff v. Long Island R.R.*, 162 N.E. 99 (N.Y. 1928) has been transmogrified into legal causation, a policy issue.

150. Nelken, *supra* note 124.

151. See, e.g., Lynn Mather, *Lawyers and Solicitors Separated by a Common Legal System*, in *FAULT LINES: TORT LAW AS CULTURAL PRACTICE* (Engel & McCann eds., 2009).

152. Barbara Pfeffer Billauer, *Primacy in Products Liability: A Comparison of Israeli and American Law*, 51 TORT TRIAL & INS. PRAC. L.J. 3 (2016).

153. *Tort Ordinance (New Version)-Updated to March 2015*, LEVITAN SHARON & CO.: ADVOCATES & NOTARIES (Aug. 20, 2016), <http://www.israelinsurancelaw.com/tort-ordinance-new-version-updated-to-march-2015/>. The ordinance states:

Chapter One: Interpretation 1. Subject to the Interpretation Ordinance, this Ordinance will be interpreted in accordance with the principles of legal interpretation obtaining in England, and expressions used in it will be presumed, so far as is consistent with their context, and except as may be otherwise expressly provided, to be used with the meaning attaching to the corresponding expressions in English law and will be construed in accordance therewith.

Id.

154. Mather, *supra* note 151, at 202.

155. *Rouleau v. Blottner*, 152 A. 96 (N.H. 1931)

156. See *Keene Corp. v. Ins. Co. of N. Am.*, 513 F. Supp. 47 (D.D.C. 1981) (“Damage” now includes both general harm and injury at a cellular level.”).

gives rise to the cause of action.”¹⁵⁷ “Putting it another way, there must be negligence and harm and they must have a causal connection.”¹⁵⁸ “[C]ausation has been the justifying glue that sticks a defendant to a plaintiff. *Causation* particularizes the injury by singling out this plaintiff; *wrongdoing* particularizes this defendant against the background of the totality of the injury’s causes.”¹⁵⁹

B. THE NARRATIVE: A VEHICLE FOR CAUSAL PROOF

Proof of the causal element, however, is not a tort issue, but an evidentiary one, dependent on the persuasive “giving over” (or rhetoric) for the causal connection as set forth in the trial narrative. Thus, “assignment of causal responsibility involves a short *chain* of causal explanation, [and] a simple narrative linking a harmful result to a blameworthy character [C]ausation [therefore] is merely an acceptable deduction from evidential facts,”¹⁶⁰ and the trial narrative¹⁶¹ is the platform on which the evidence is set forth.¹⁶²

This expectation dates to biblical times. Thus, we find in Exodus a scenario depicting a man who dug a pit and left it uncovered, whereupon a wandering bull or ox or child stumbles upon it, falls in, and is injured.¹⁶³ Since but/for the pit’s being uncovered, the ox or bull or potential plaintiff would not have fallen in—one discerns the open pit caused the harm, and the man who dug it is responsible for compensation.¹⁶⁴

C. VERISIMILITUDE IN THE SCIENTIFIC AGE

Conventionally, scholars believed we make causal assessments based on our senses—what we see or hear, or what we think we see and hear. Recognizing “the undeniable fact that neither judge nor jury actually had an unmediated encounter with the event they are being asked to evaluate”¹⁶⁵ Legal poetics addresses the issue via the concept of “legal verisimilitude,” the legal system’s version of pro-

157. *Overseas Tankship (U.K.) Ltd. v. Morts Dock & Eng’g Co. (Wagon Mound. No. 1)*, Privy Council (1961).

158. *White v Schnoebelen*, 18 A.2d 185 (N.H. 1941).

159. Nelken, *supra* note 124, at 279.

160. *Id.* at 253.

161. Christian Metz describes the doubly temporal sequence of the narrative; there is the time of the thing told and the time of the telling. See JESPER JUUL, *A CLASH BETWEEN GAME AND NARRATIVE* (1998).

162. Nelken, *supra* note 124, at 253.

163. *Exodus* 33:34.

164. See Richard Wright, *The Efficiency Theory of Causation and Responsibility: Unscientific Formalism and False Semantic*, 63 CHI.-KENT L. REV. 553, 209 (1987).

165. *Id.*

ducing a readily recognizable reenactment of reality.¹⁶⁶ When sensual evidence is unavailable, the causal dilemma begs for another solution.

In toxic torts, for example, causation occurs at the sub-visual level.¹⁶⁷ Even before the toxic tort age, recreating an event exactly and conveying it so it is received as delivered is a demand “that no human can literally fulfill.”¹⁶⁸ Legal poetics strives to achieve this verisimilitude through the creation of temporal and spatial distinctiveness. “However, verisimilitude becomes almost unattainable in our current, simulacra-saturated environment,”¹⁶⁹ states Professor Shulamit Almog. The same phenomenon Professor Almog attributes to digital technology well describes the toxic tort arena requiring an understanding biological mechanisms to prove causation.

D. THE TEMPORAL CONSTRAINT AND BUT/FOR CAUSATION

Professor Almog adds another element to legal poetics, a temporal component which relates different facets of time, linking the past with what exists in another temporal dimension, i.e., beginnings and ends.¹⁷⁰ In tort law, this is reflected in an expected linearity of causal expectations, “an ingrained-thinking-habit,” which suggests that “ontological fuzziness is reason to discredit any explanation,”¹⁷¹ thereby explaining why some expert testimony may be rejected by courts where it does not have the crispness of quantitative measures.

In torts, the linear process usually manifests as the expectation of a single action necessarily resulting in a single event: in law-speak—but/for causation,¹⁷² (loosely an analog of *ceteris paribus*).¹⁷³ This expectation invites cognitive dissonance when judges are confronted with multiple diseases claimed to be caused by one agent. Further, legal poetics requires that each term in a legal rule convey a range of possible meanings among which choices will have to be made.¹⁷⁴ Hence, one must assess whether but/for causation is the sole key to unlocking the legal causation door, or whether we need to broaden the

166. See Almog, *supra* note 118.

167. *Keene v. Ins. Co. of N. Am.*, 597 F. Supp. 946 (D.D.C. 1984).

168. See JANET MALCOLM, *THE CRIME OF SHEILA MCGOUGH* 3-4 (1999).

169. Almog, *supra* note 12, at 191-92.

170. Almog *supra* note 135 (“Even when we deal with a non-linear narrative . . . [.] such as deviations and retreats, the awareness of some kind of temporal axis is preserved.”).

171. Nelken, *supra* note 124, at 270.

172. *Barnett v. Chelsea & Kensington Hosp. Mgmt. Comm.*, 1 QB 428 (1969) (Eng.).

173. *Ceteris paribus*, STANFORD ENCYCLOPEDIA OF PHILOSOPHY (2011), <http://plato.stanford.edu/entries/ceteris-paribus/>; NANCY CARTWRIGHT, *THE LARGE, THE SMALL AND THE HUMAN MIND* 166 (Roger Penrose ed., 1997).

174. WHITE, *supra* note 117, at 66.

legal mindset to encompass multi-causation.¹⁷⁵ But “Americans have never really been comfortable with multiple causation¹⁷⁶ We’re still stuck in an either/or ontological regime . . . [as the] atomism of our causal habits tends to the presumption that events are disconnected,”¹⁷⁷ a mindset that resonates in preference for the quantitative analyses of epidemiology.

Thus, the complexity of biological processes is a contributor to the poetic melee between law and science in general, and statistically-based epidemiology and clinical medicine in particular. To exemplify, a recent study examining what causes the shape of a bird’s egg was performed by a team of six experts from different fields.¹⁷⁸ If it took tools and expertise “from computer science, comparative biology, mathematics, and biophysics” to understand egg shapes, reducing human disease causation to single-cause epidemiological models¹⁷⁹ makes a mockery of both science and law.¹⁸⁰

Courts then confront diseases in toxic tort cases involving multiple causes or multi-system breakdowns that confound the expected linear sequence. Determining which “straw” broke the camel’s back is problematic, especially where disease mechanisms involve non-sequential steps¹⁸¹ which may be impossible to reconcile with both traditional legal and biological imperatives.

In sum, causal analysis in biology is confounded and compounded by biological themes which have little resonance in traditional law think; the body does not work in a direct linear fashion. Thus, one assault or environmental insult can be repaired by a bodily defense mechanism, while a second might overwhelm that system. And the

175. See *Schultz v. Akzo Nobel Paints, LLC*, 721 F.3d 426 (7th Cir. 2013).

176. Courts are not too happy with it either. See Barbara P. Billauer, *Admissibility of Scientific Evidence Under Daubert: The Fatal Flaws of ‘Falsifiability’ and ‘Falsification,’* 22 B.U. J. SCI. & TECH. L. 21 (2016). A critical flaw of Daubert’s reliance on Popper is that Popper requires an either-or disposition of a scientific hypothesis. Popper’s paradigm cannot accept the notion of multiple causation. See *id.*

177. Nelken, *supra* note 124, at 270-71.

178. See Judy Siegel, *Cracking the Mystery of the Avian Egg Shape*, JERUSALEM POST (June 27, 2017, 21:59), <http://www.jpost.com/Business-and-Innovation/Health-and-Science/Cracking-the-mystery-of-the-avian-egg-shape-498109>.

179. Rarely do today’s experts believe biostatistical means can account for multi-causal mechanisms. See, e.g., W. KEITH MORGAN & ANTONY SEATON, OCCUPATIONAL LUNG DISEASES 10 (1984). Some statistical programs can control for certain “confounding variables” but only in large population cohorts. *Id.* at 102. Controlling for unknown co-causal factors, genetic susceptibility or multi-step causation is outside the scope of current biostatistical practice. See DONA SCHNEIDER & DAVID E. LILIENFELD, LILIENFELD’S FOUNDATIONS OF EPIDEMIOLOGY 63-68 (4th ed. 2015).

180. See Scales, *supra* note 138 (“Indeed it is the very complexity of the models that has exacerbated the danger [of Popper’s] positivism in causation decisions.”).

181. See, e.g., N. Kochhar & D. Kaul, *Initiator-Promoter Coupling of Phospholipases D and A₂ in Platelets upon Cholesterol Incorporation* 314 FED’N EUR. BIOCHEMICAL SOCIETIES 1, 17-19 (1992).

same insult might provoke one response in one person and a different one in another. How can the law address causation, if disease, by its very nature, is idiosyncratic and plaintiff-specific, and *Daubert*-approved science, which for the most part turns on but/for causation, is unworkable and biologically repugnant?¹⁸²

E. TEMPORAL DECOHESION

If that is not enough, difficulties in conveying causal connections arise also from temporal lapses (even if linear) between a negligent act (or omission) and its manifestation, such as in latent diseases. Traditional legal doctrine provides that superseding and intervening causes occurring during these temporal interstices (between act and event) break the causal chain and short-circuit liability;¹⁸³ the longer the time lapse, the more people expect events other than the defendant's negligence to wreck their nuisance, and the more important evidentiary exclusion of other potential causal factors becomes.¹⁸⁴ As early as 1866, the court in *Ryan v. New York Central Railroad Co.*¹⁸⁵ noted "[i]t is a general principle that every person is liable for the consequences of his own acts, but not for remote damages. It is not easy at all times to determine what are proximate and what are remote damages."¹⁸⁶

The cultural prelude dates to biblical times, signaling a hard-wired expectation that these direct sequences will be provable as a clear nexus between act and harm, envisioned as a fluid, uninterrupted sequence.¹⁸⁷ Exodus Chapter 21 provides a casebook-worthy example:

182. Biomarker studies, however, present observable verifiable individual proof and was important in proving liability against W.R. Grace in the Woburn TCE cases, as well as in the *DuPont C-8 cases*. See STEPHANIE J. FRISBEE ET AL., *THE C8 HEALTH PROJECT: DESIGN, METHODS, AND PARTICIPANTS* (2009); see also *Leach v. E.I. DuPont de Nemours and Company*, No. 01C-608 (W. Va. Cir. Ct. Apr. 10, 2002); *Studies Finding Links Between PFOA Exposure and Adverse Human Health Effects*, HILL, PETERSON, CARPER, BEE & DEITZLER, PLLC <http://www.hpcbd.com/Personal-Injury/DuPont-C8/C8-Links-Between-Exposure-and-Health-Effects.shtml> (last visited Jan. 11, 2018).

183. See *White v. Schnoebelen*, 18 A.2d 185 (N.H. 1941) ("While a time lapse between events raise concerns, the time lapse itself is not necessarily dispositive. The terrorist's time bomb set to detonate after long delay set still accrues liability for its maker—but it makes causal proof that much more difficult: Through lack of care a person may set in motion forces which touch the person (or property) of another only after a long period and then only through new, fortuitous conduct A lapse of time may make it difficult or even impossible to prove that the bridge of causation is unbroken but if it appears on the balance of probabilities to be intact, it will bear the necessary weight of conveying.")

184. Wright, *supra* note 164.

185. 36 N.Y. 207 (1866).

186. *Ryan v. N.Y. Cent. R.R. Co.*, 36 N.Y. 207, 210 (1866).

187. *Cone v. Inter Cty. Tel. & Tel. Co.*, 40 So. 2d 148 (Fla. 1949). See generally Richard A. Epstein, *Causation—In Context: An Afterword*, 63 CHI.-KENT L. REV. 653 (1987).

כ וְכִי-יִכֶּה אִישׁ אֶת-עַבְדּוֹ אוֹ אֶת-אִמָּתוֹ, בְּשֵׁבֶט, וּמַת, תַּחַת יָדוֹ—נָקָם, יִנָּקֵם. 20 And if a man smite his bondman, or his bondwoman, with a rod, and he die under his hand, he shall surely be punished.

כא אִךְ אִם-יוֹם אוֹ יוֹמִים, תַּעֲמֹד--לֹא יִנָּקֵם, כִּי בְּכֶסֶף הוּא. {ס} 21 Notwithstanding if he continue a day or two, he shall not be punished; for he is his money (property).¹⁸⁸

The intervention of a day or two between action and harm constituted a temporal break sufficient for the Torah to consider the causal chain broken, vitiating liability. The medieval commentator, Rashi, raises the intervention of another person as an operative force that truncates the causal linkage, noting that even if some harm was caused by the initial blow, someone else might have struck the bondsman during the interim between the first blow and death.¹⁸⁹

Determining what constitutes an adequate nexus between act and damage is not clear cut, may be dictated by desired outcome,¹⁹⁰ and consequentially now is relegated to policy determination. “Legal or proximate cause is not logic. It is practical politics.”¹⁹¹ This is well-illustrated by the extra-judicial *Kishon* resolution affording compensation to the navy divers.

F. THE POETICS OF SCIENCE

Some among experts claim “that the culture of law should strive as far as possible to assimilate itself to the culture of science when dealing with scientific issues”¹⁹² But *how* could the culture of law assimilate itself to the culture of science when it does not even know what it is? *Why* should legal culture assimilate itself to science, and not vice versa, at least in the courtroom? And: *which* science are we talking about—clinical medicine, or epidemiology? To address this, a

188. *Exodus* 21:20-21. Rashi interprets “he shall not be punished, because he is his property,” as “[b]ut if someone else struck him, even if he lingered for twenty-four hours before he died, he [the other person] is liable [to incur the death penalty].” See Chabad, http://www.chabad.org/library/bible_cdo/aid/9882#showrashi=true.

189. Rashi interprets “he shall not be punished, because he is his property,” as “[b]ut if someone else struck him, even if he lingered for twenty-four hours before he died, he [the other person] is liable [to incur the death penalty].” See Chabad.org, http://www.chabad.org/library/bible_cdo/aid/9882#showrashi=true.

190. Richard W. Wright, *Causation in Tort Law*, 73 CAL. L. REV. 1735, 1774 (1985).

191. See Mario J. Rizzo, *Foreword—Fundamentals of Causation*, 63 CHI.-KENT. L. REV. 397 (1987). (“[C]ausation-in-fact is the only truly causal issue.”).

192. *Id.*

preliminary discussion of the culture (and poetics) of science in general and the relevant sciences in particular is in order.

Science, too, has a poetics,¹⁹³ a history, a culture, and a philosophy intrinsic to its conduct and contribution to furthering knowledge.¹⁹⁴ Like all poetics, it is culture-specific,¹⁹⁵ meaning each field of science adds its own idiosyncratic contribution and is influenced by its own history, linguistics, and culture.

I begin by focusing on an accomplished poet and scientist, whose scientific interests were all-encompassing and who, for good measure, was also a lawyer: Johann Wolfgang von Goethe (1749-1832),¹⁹⁶ also credited with coining the term “biology.”¹⁹⁷ Better known for his literary and dramatic works (even called the German Shakespeare), Goethe’s contributions to science and philosophy of science are of no less significance.¹⁹⁸ These include *Metamorphosis of Plants*¹⁹⁹ and the popularization of the Goethe barometer.²⁰⁰ To gain a comprehensive view of geology, Goethe collected 17,800 rock samples—the largest private collection of minerals in Europe.²⁰¹ His most significant works influenced both Schopenhauer and Wittingstein (who in turn influenced Popper), involved color theory, and resulted in his most important work,²⁰² *Theory of Colours*.²⁰³

Goethe’s art informed his legal practice,²⁰⁴ as he argued that laws could not be created by pure rationalism since geography and history shaped habits and patterns, just as his view of life informed his sci-

193. See generally CLAIRE PRESTON, *THE POETICS OF SCIENTIFIC INVESTIGATION IN SEVENTEENTH-CENTURY ENGLAND* (1st ed. 2015).

194. MARIO BIAGIOLO & JESSICA RISKIN, *NATURE ENGAGED: SCIENCE IN PRACTICE FROM THE RENAISSANCE TO THE PRESENT 1-12* (2012).

195. See STEVE FULLER, *SOCIAL EPISTEMOLOGY, SCIENCE, TECHNOLOGY, AND SOCIETY* 121 (2d ed. 2002) (“Since virtually no one in one culture can think beyond the core sets of beliefs because they appear self-evident . . . [,] the historian must read the culture’s texts ‘deep’ in order to reenact the thought processes of their authors.”).

196. DAVID SEAMON ET AL., *GOETHE’S WAY OF SCIENCE 1* (David Seamon & Author Zajoc eds., 1998).

197. DENNIS L. SEPPER, *GOETHE AND THE POETICS OF SCIENCE* 219-20 (2005) (“Goethe believed that all . . . works of science . . . were guided by certain basic intuitions . . . ,” a statement that would have appalled Popper).

198. Robert Paris, *Goethe and Studies About Nature*, *MATIER ET EVOLUTION* (2014), <http://matierevolution.fr/spip.php?article3502>.

199. JOHANN WOLFGANG VON GOETHE, *THE METAMORPHOSIS OF PLANTS* (2009). It was published in German in 1790. Goethe’s *Nature* was written in 1781.

200. See also GEORG WILHELM FRIEDRICH HEGEL, *HEGEL’S PHILOSOPHY OF NATURE: ENCYCLOPAEDIA OF THE PHILOSOPHICAL SCIENCES* (1830).

201. Paris, *supra* note 198.

202. See JOHANN PETER ECKERMANN, *CONVERSATIONS OF GOETHE* (J.K. Moorhead ed., John Oxenford trans., 1998).

203. JOHANN WOLFGANG VON GOETHE, *THEORY OF COLOURS* (1840) (originally published in German in 1810).

204. SEPPER, *supra* note 197.

ence.²⁰⁵ His scientific approach was phenomenological,²⁰⁶ and he believed intuition²⁰⁷ was the instrument by which one grasps the biological archetype.²⁰⁸

Goethe's understanding of science was far more sophisticated historically than anything that preceded it. He knew, long before twentieth-century philosophers and historians, that the most diverse forces and influences go into the making of science: social movements, intellectual fashions, religious convictions, personal predilections, and a host of other things. More basically, however, he thought that sciences were constructed on the basis of the ways of conceiving and presenting things, *Vorstellungsarten*.²⁰⁹

Goethe noted that "proving a hypothesis is a more limited, rhetorical goal that should be attempted only after the poetical structure has been laid down."²¹⁰ Foreshadowing modern science (and at distinct odds with Popper), Goethe stressed the importance of a multidisciplinary approach.²¹¹ His view of how science must be constructed was thus comprehensive, synthetic, and holistic,²¹² and he believed scien-

205. SEAMON ET AL., *supra* note 196.

206. *Phenomenology*, STANFORD ENCYCLOPEDIA OF PHILOSOPHY (2013), <https://plato.stanford.edu/entries/phenomenology>, (Phenomenology is the study of structures of consciousness as experienced from the first-person point of view). *See also* CLAUDE BERNARD, AN INTRODUCTION TO THE STUDY OF EXPERIMENTAL MEDICINE 50 (1865) (regarding the importance of a "phenomenological" view of science).

207. Per Goethe:

Each person has certain characteristic contexts for seeing things and certain characteristic ways of trying to put things together . . . *Vorstellungsarten* . . . For example, atomism is not just a theory but also a recurrent manner in which a certain type of intelligence tries to comprehend the basic phenomena of nature.

SEPPER, *supra* note 197, at 221. *See also* RUDOLF STEINER, THE THEORY OF KNOWLEDGE IMPLICIT IN GOETHE'S WORLD-CONCEPTION (2002) ("In inorganic nature, we see causes and effects as separate from each other In the organic world, we perceive species and genera of organisms and try to determine their mutual relationships."); *infra* note 209 and accompanying text.

208. *See* Paris, *supra* note 198.

209. SEPPER, *supra* note 197, at 221, 223.

210. *Id.* at 218.

211. *See* Johann Wolfgang von Goethe, INTERNET ENCYCLOPEDIA OF PHILOSOPHY, <http://www.iep.utm.edu/goethe/>ep.utm.edu/goethe/#H3 (last visited Nov. 2017) ("[Newton] commits the error of taking as his premise a single phenomenon, artificial at that, building a hypothesis on it, and attempting to explain with it the most numerous and unlimited phenomena."); *see also* SEPPER, *supra* note 197, at 218 ("Newton . . . presented experiments only to drive forward a narrative that intends to discredit competitor theories and to prove his own By being very selective of the phenomena, Newton produced an appearance of comprehensiveness that was as much artifice as reality.").

212. SEPPER, *supra* note 197, at 222.

tific representations should be as close an analogy to how nature is constructed as possible.²¹³

Goethe's views, which reject atomism or scientism, have been embraced by modern scientists.²¹⁴ Philosopher Tom Sorell offers a definition of scientism that justifies its rejection by modern scientists: "Scientism is a matter of putting too high a value on natural science in comparison with other branches of learning or culture."²¹⁵ Even Sir Richard Doll notes "that it seems therefore that experimental toxicological data on carcinogenesis can forewarn of potential hazard to man, but that our final decision must rest on a full assessment of our total knowledge, including that derived from human observations and the economic, sociological, and ethical features of society."²¹⁶

Goethe also pointed out that every language has different capacities for expressing phenomenality,²¹⁷ thereby acknowledging the confounding aspects of language in conveying scientific messages. Per Goethe, in science, words affect not just the meaning, but the outcome. Hence, he demanded "that the language used to describe each domain be derived from that domain."²¹⁸ Moreover, "Goethe's scientific work strove not to weave a web of words but rather to use words to give us access to things."²¹⁹ Indeed, Goethe probably would have been horrified to learn that a raw statistical and purely mathematically-based "science" of epidemiology²²⁰—uninformed by biology or experience—is driving the causal analysis in law today.²²¹

Professor Sepper's analysis is telling:

When a modern scientist applies mathematics to nature, he ordinarily abstracts from the natural situation, often by us-

213. J.J. BONO, MAKING KNOWLEDGE: HISTORY, LITERATURE, AND THE POETICS OF SCIENCE (2010).

214. Scales, *supra* note 138, at 274. Scales writes that "the scientism, what we might call the quasi-empiricism of the toxic tort regime is the worst case of positivism," an implicit attack on Popper. *Id.*

215. See Thomas Burnett, *What is Scientism*, AM. ASS'N ADVANCEMENT SCI., <https://www.aaas.org/page/what-scientism> (last visited Jan. 11, 2018) (discounting the views of atomism (or scientism)).

216. WORLD HEALTH ORG., INTERPRETATION OF NEGATIVE EPIDEMIOLOGICAL EVIDENCE FOR CARCINOGENICITY 6 (N.J. Wald & R. Doll eds., 1985). Doll's work is discussed further in notes 327-335 *infra* and accompanying text.

217. SEPPER, *supra* note 197, at 225.

218. Peter David Luborsky, *Goethe's Scientific Language in Prose and Poetry* (1993) (unpublished Ph.D. dissertation, University of Massachusetts Amherst; on file with author).

219. SEPPER, *supra* note 197, at 225.

220. See Thomas Kuhn, *Mathematical Versus Experimental Traditions in the Development of Physical Science*, 7 J. INTERDISC. HIST. 1 (1976); BERNARD, *supra* note 206 (Claude Bernard, the father of experimental physiology, castigated the use of statistics and averages in biological experimentation, writing an entire subchapter on the matter).

221. SEPPER *supra* note 197, at 217-18, 225.

ing a simplified model that serves as an extended analog
 [But to Goethe] the object is less to have a mathematical
 formula . . . than to gain a deepened and more unified experi-
 ence of the phenomena²²²

As Goethe said:

[T]here is a fairly sharp contrast with the practice of the more
 mathematized natural sciences. In hardly any sense do the
 mathematized natural sciences aim to help one observe the
 phenomena in an orderly fashion. Instead, they highlight an
 observable aspect or even abstract entirely from the observa-
 ble so that the observable is treated as merely an index of
 what is invisible.²²³

Far removed from Popper in his view of science, Goethe's view, I
 submit, is closer to the reality of today's biology and toxic tort causal
 proof.

Goethe's views also align with chemist George Gore, who, in 1878,
 wrote a universally acknowledged book on science, scientists, scien-
 tific discovery, and its culture.²²⁴ Without discussing poetics as such,
 Gore remarks on the importance of using precise and clear language
 in science,²²⁵ a view expressed by other scientists as well. The impor-
 tance of correct use of language lies not only in being able to report
 research well; it is with language that we do most of our thinking.²²⁶
 Minimizing the importance of the mathematical aspects of science and
 distinguishing between qualitative truth (what we might call *validity*)
 and quantitative truth or accuracy (what we might call *reliability*),²²⁷
 Gore notes that qualitative truth is far more important than quantita-
 tive truth.²²⁸

222. *Id.* at 215.

223. *Id.* at 218. *See also* Johann Wolfgang von Goethe, *The Experiment as Mediator Between Object and Subject*, IN CONTEXT 19-23 (Fall 2010). This essay, written in 1793, is perhaps a forerunner of Bohr's view of quantum physics, where it is believed that there is no barrier between observer and observed, each influencing the other. *Id.* *See also* SEPPER, *supra* note 197.

224. *See generally* GEORGE GORE, THE ART OF SCIENTIFIC DISCOVERY (1878).

225. GORE, *supra* note 224, at 76-78.

226. 1 CITIZEN SCIENTISTS LEAGUE, SCIENTIFIC WORK AND CREATIVITY: ADVICE FROM THE MASTERS 41 (2012). *See also* W.I.B. BEVERIDGE, THE ART OF SCIENTIFIC INVESTIGATION (Reginald Smith ed., 2012).

227. GORE, *supra* note 224, at 148-49. *See* Barbara Pfeffer Billauer, *Daubert Debunked: A History of Legal Retrogression and the Need to Reassess "Scientific Admissibility,"* 21 SUFFOLK J. TRIAL & APP. ADVOC. 4-5 (2015-2016) (providing the legal importance of discerning the difference of the two terms and concepts).

228. GORE, *supra* note 224, at 150 ("Qualitative truths of simple existence are the very foundation of science and the nearest approaching to absolute as any truths we know.").

In fact, in an echo of Goethe, many theorists view reliance on statistics as persuasive evidence with a jaundiced eye.²²⁹ “Statistics help us in drawing conclusions from our data by ensuring that our conclusions have a certain reliability, but even statistical conclusions are strictly valid only for events which have already occurred.”²³⁰

G. RHETORIC

In science, the credit goes to the man who convinces the world, not the man to whom the idea first occurs—Sir Francis Darwin, Eugenics Review, April 1914.

Another element of legal poetics law falls under the heading of rhetoric.²³¹ American courts and commentators typically view the causal link between conduct and injury as a matter of factual inquiry.²³² When there is not enough “objective” science to prove a causal connection, the plaintiff’s claim is doomed, “no matter how serious the harm or how egregious the defendant’s misdeeds”²³³ This has not stopped intrepid advocates from pursuing the matter using unconventional means of persuasion such as the media and advocacy—referred to by Aristotle and others as rhetoric.²³⁴ In other words, the “ignoble” art of persuasion.²³⁵

This view illustrates why we instinctively reach for tools of persuasion (including probabilities and statistics) in tort cases when the “science” of causation²³⁶ is ambiguous or too imprecise to satisfy legal standards of proof. It must be noted that “persuasion” can lead to obfuscation, and here statistics shines. As Ben Goldacre tweeted:

229. See generally JAMES CHANDLER ET AL., QUESTIONS OF EVIDENCE: PROOF, PRACTICE AND PERSUASION ACROSS THE DISCIPLINES 401-28 (1st ed. 1994) (*nota bene* Mary Poovey’s comment at page 420: “According to various accounts, statistics limits itself to recording observable facts . . . [;] it limits itself to a static description of the present, scorning historical and comparative narratives”).

230. CITIZEN SCIENTISTS LEAGUE, *supra* note 226, at 147.

231. See WHITE, *supra* note 117.

232. Nelken, *supra* note 124, at 253.

233. David M Engel, *Discourse of Causation in Injury Cases, Exploring Thai and American Legal Cultures*, in FAULT LINES: TORT LAW AS CULTURAL PRACTICE 252 (Engel & McCann eds., 2009).

234. WHITE, *supra* note 117, at 31 (“Rhetoric, . . . fills in the gap when science is absent. The main aim of science is that it contributes to knowledge by informing us of what is knowable in the sense that can be demonstrated Rhetoric is . . . what we do when science doesn’t work. Instead of dealing with what is “known” it deals with what is probably the case. Rhetoric is the art of establishing the probably by arguing from our sense of the probable.”).

235. *Id.*

236. SHEILA JASANOFF, SCIENCE AT THE BAR: LAW, SCIENCE, AND TECHNOLOGY IN AMERICA (1995). Jasanoff describes causation as the seminal issue that characterizes toxic tort cases. *Id.*

“statistics will confess to anything, if you torture them hard enough.”²³⁷

Consequently, we face a paradox: at the very time when hazards and catastrophes appear to become most nefarious, they simultaneously slip through the nets of proof, laws of liability, and systems of compensation with which the legal and political systems attempt to capture and remedy them Moreover, as such consequences become incalculable, statistics turn into a form of obfuscation . . . “the possibility of determining causality becomes hopelessly complicated”²³⁸

And of course, “there are lies, damned lies and statistics.”²³⁹ The cultural view of statistics is, in a word, disdain. Even some aspects of classical “probability theory” has been attacked “as marking a huge complexifying project.”²⁴⁰ This has not stopped the legal community from lapping it up in the guise of epidemiological studies, notwithstanding inherent difficulties in translating its use into legalistic terminology.

Statistics has also come under attack by statisticians. Biostatistician Richard Royall notes:

Standard statistical methods regularly lead to the misinterpretations of results of scientific studies. The errors are usually quantitative. But sometimes they are qualitative—sometimes the evidence is judged to support one hypothesis over the other, when the opposite is true. These misinterpretations are not a consequence of scientists misusing statistics. They reflect instead a critical defect in current theories of statistics.²⁴¹

Royall explains that his entire book is an indictment of statistical theory, which has produced “a seriously defective methodology, revealed in the inability of that theory to answer fundamental questions

237. *Medical Library Association Meets and Tweets*, NLM IN FOCUS (June 7, 2016), <https://infocus.nlm.nih.gov/2016/06/07/medical-library-association-meets-and-tweets/>. See also DARRELL HUFF, HOW TO LIE WITH STATISTICS (1954), <http://www.horace.org/blog/wp-content/uploads/2012/05/How-to-Lie-With-Statistics-1954-Huff.pdf> (quote originally attributed to Ronald H. Coase, 1991 Nobel Prize Winner in Economics, as “if you torture the data hard enough, it will confess”).

238. See FRANK FISHER, CITIZENS, EXPERTS, AND THE ENVIRONMENT: THE POLITICS OF LOCAL KNOWLEDGE 50, 54, 147 (2000).

239. Popularized by Mark Twain, who attributed it to Benjamin Disraeli.

240. MIOARA MUGUR-SCHÄCHTER & ALWYN VAN DER MERWE, QUANTUM MECHANICS, MATHEMATICS, COGNITION AND ACTION: PROPOSALS FOR A FORMALIZED EPISTEMOLOGY, 257 (2006).

241. RICHARD ROYALL, STATISTICAL EVIDENCE: A LIKELIHOOD PARADIGM xii (1997).

about what the results of routine statistical analyses means.”²⁴² (!) No, as Royal said, “All is not well”²⁴³ in the world of statistics.

In fact, the mathematicalization of disease and its transmission is notoriously inaccurate without consideration of biological parameters.²⁴⁴ Further, “[m]any philosophers and social scientists have succeeded in showing that statistical analysis of risks . . . function[s] as a form of moralizing in the name of mathematical objectivity.”²⁴⁵

“Honest” persuasive scientific efforts, however, which fall short of objective scientific experiment should not be rejected out of hand. Persuasion played a huge role in launching modern science.²⁴⁶ For example, before “all the evidence was in,” proponents of two alternative theories of bodily defense mechanisms argued viciously and championed their positions publicly—based on clues supplied by nature, pattern recognition, comparisons—and insults and innuendo.²⁴⁷ In the debate between the role of various cells in immunological response, Elie Metchnikoff and Paul Ehrlich’s attacks on each other were legendary.²⁴⁸ Both theories would have failed a Popperian analysis (because both could be falsified),²⁴⁹ and hence would have been rejected under *Daubert* as unreliable and unscientific. Nevertheless, they both turned out to be correct; ultimately the two shared the 1908 Nobel Prize in Medicine.²⁵⁰

H. BRIDGING THE GAPS: SOCIAL EPISTEMOLOGY AND POETICS OF LEGAL CAUSATION

Difficulties in reconciling epidemiology and legal causation have been addressed under the rubric of social epistemology, “a multi-fac-

242. ROYALL, *supra* note 241.

243. *Id.*

244. Barbara Pfeffer Billauer, *Weapons of Mass Hysteria, Faulty Biothreat Predictions, and Their Impact on National (In) Security: A Case-Study of Smallpox or: The Misuse of Mathematical Modeling to Project Biothreats, Terrorism Attacks, and Epidemics*, 27 HEALTH MATRIX 347 (2017).

245. *Id.* See also FISHER, *supra* note 238, at 89.

246. See Mary Poovey, *Figures of Arithmetic, Figures of Speech: The Discourse of Statistics in the 1830s*, in QUESTIONS OF EVIDENCE, PROOF, PRACTICE AND PERSUASION ACROSS THE DISCIPLINES, 401-05 (1991).

247. See generally LUBA VIKHANSKI, IMMUNITY: HOW ELIE METCHNIKOFF CHANGED THE COURSE OF MODERN MEDICINE 106 (2016) see also OLGA METCHNIKOFF, LIFE OF ELIE METCHNIKOFF 1845-1916 (1921). Metchnikoff did not so much prove phagocytes were a key component of the immune system; he argued it—passionately and persuasively, selecting evidence to support his case, all the while Koch and Behring were arguing other cells were more important. *Id.*

248. PAUL DE KRUIF, MICROBE HUNTERS 190-224 (1926).

249. See Barbara P. Billauer, *supra* note 176 (indicating neither could disprove (falsify) the theory of the other—which stands to reason, since they were both right). See also BERNARD, *supra* note 206.

250. *Nobel Prize in Physiology or Medicine 1908*, NOBEL PRIZE, https://www.nobelprize.org/nobel_prizes/medicine/laureates/1908 (last visited Jan. 13, 2018).

eted discipline which seeks to overcome traditional decision-making . . . [which is] performed in a dichotomous yes-no format.”²⁵¹ Akin to poetics in focusing on the cultural dimensions of decision-making,²⁵² social epistemology evaluates the multiple influences society is heir to in making decisions in the absence of scientific consensus. Sadly, it appears that neither Goethe nor Gore’s views, (nor the poetically-oriented approach of other scientists)²⁵³ have made their way into social epistemology.²⁵⁴

I. SOCIAL EPISTEMOLOGY IN THE COURTROOM

The aforementioned *Kishon Cases*²⁵⁵ were subjected to intense social epistemological inquiry, suggesting the disparate outcomes arose not from scientific conflicts but the different cultural milieu²⁵⁶ surrounding the two plaintiff groups, the commando-divers and the fishermen.²⁵⁷ According to Smadar Ben-Asher²⁵⁸ and Tal Golan,²⁵⁹ the results can be traced, at least in part, to the higher social regard in which the elite Naval Commandos were held,²⁶⁰ manifestations of cul-

251. See FISHER, *supra* note 238.

252. See FULLER, *supra* note 195.

253. See DONALD C. GOELLNIGHT, THE POET-PHYSICIAN: KEATS AND MEDICAL SCIENCE 8-12 (1984) (regarding the contributions to the poetics of science by William Harvey, of blood circulation fame, and chemist Sir Humphry Davy, a friend of Coleridge’s, who discovered six elements and pioneered the field of electrochemistry).

254. JASANOFF, *supra* note 236, at 7.

255. Tal Golan, *The Kishon Affair: Science, Law, and the Politics of Causation in the Israeli Context*, 23 SCI. IN CONTEXT 535-69 (2010).

256. See generally Smadar Ben-Asher & Nurit Goren Tzivoni, *Protective Identification as a Defense Mechanism When Facing the Threat of an Ecological Hazard*, 18 PSYCHOANALYSIS, CULTURE & SOC’Y 11, 17-35 (2006). In the *Kishon Cases*, the epidemiologists (and court) opined that low level exposures were not carcinogenic; in the TCE/Woburn case, the experts hypothesized that low level exposures to a pollutant weakened the immune system, making the exposed more susceptible to cancer. A scientific investigation measured indicia of such effects and found changes in T and immune cell (NK cell) ratios, establishing the hypothesis was correct. For some reason, cellular changes and biomarker testing do not seem to have been done in the *Kishon Cases*.

257. See generally Golan, *supra* note 255.

258. Smadar Ben-Asher, *Hegemonic, Emancipated and Polemic Social Representations: Parental Dialogue Regarding Naval Commandos Training in Polluted Water*, 12 PAPERS ON SOC. REPRESENTATIONS 6.1.-6.12 (2003).

259. See Golan, *supra* note 255. See also Ben-Asher, *supra* note 258.

260. Smadar Ben-Asher & Ella Ben-Atar, *The Attitude of the Local Press to Marginal Groups: Between Solidarity and Alienation*, 22 ISR. AFF. 528-48 (2016).

ture²⁶¹ and media coverage,²⁶² a force exemplified in the *DuPont C-8 cases*.²⁶³

J. BRIDGING THE LAW-SCIENCE SCHISM

According to Ben-Asher, the clash between law and science is impacted by the way issues are “framed” by the media, which shapes the public’s thinking,²⁶⁴ much the way a good lawyer influences a legal resolution. This phenomenon, or “interpretive package,” Ben Asher argues, is what happened in the Kishon Affair.²⁶⁵ Nevertheless, in that case, the framing of issues had little (no) impact on the outcome. The courts rejected the claims of both the divers and the fishermen. Even the scientists convened by the military panel (a biochemist and epidemiologist) rejected causal associations. Only the jurist on the military panel found a connection, using an arcane legal loophole,²⁶⁶ clearly swayed by cultural imperatives.

K. FRAMING AND INTERDISCIPLINARY BORROWING

Framing does play an important role in interdisciplinary borrowing, however, which includes the law’s use of epidemiology. Thus, “historians frequently focus on the fact, say, that the mathematical technique contributed significantly to the solution of the physicist’s problem, rather than on the fact that the physicist had to refashion the problem in order to take advantage of the mathematical technique.”²⁶⁷ In the legal setting, framing requires the law to recast its problem in epidemiological—rather than legal or even clinical—terms, i.e., posing the causation question in statistical fashion, which allows for only one answer: the causal claim is either all wrong or all right, akin to proving but/for causation. An alternative, more medically correct approach, and which tracks Goethe’s views, would consider multiple or concurrent causation, i.e., that more than one outcome is possible. In other words, the defendant’s product caused the harm *and* other conditions did as well (say, by contributing, exacerbating, or predisposing the plaintiff to disease).

261. JONATHAN HARR, *A CIVIL ACTION* (1995).

262. See generally ROBERT BARTLETT, *THE RESERVE MINING CONTROVERSY: SCIENCE, TECHNOLOGY AND ENVIRONMENTAL QUALITY* (1980); see also FRANK D. SCHAUMBURG, *JUDGMENT RESERVED: A LANDMARK ENVIRONMENTAL CASE* (1976).

263. See, e.g., National Rich, *DuPont’s Worst Nightmare*, N.Y. TIMES (Jan. 6, 2016) <https://www.nytimes.com/2016/01/10/magazine/the-lawyer-who-became-duponts-worst-nightmare.html> (last visited Nov. 20, 2017).

264. Smadar Ben-Asher & Ella Ben-Atar, *supra* note 260.

265. *Id.*

266. Golan, *supra* note 255.

267. FULLER, *supra* note 195, at 19.

Another example of flawed interdisciplinary borrowing is the failure to address methodological limitations of the borrowed field. In science, and especially in the practice of epidemiology, hidden, undetectable, or even unpreventable methodological weaknesses can render the results unreliable and the conclusions flimsy or difficult to interpret,²⁶⁸ such as where the disease is rare or the test population small.²⁶⁹ For example, the epidemiological study convened by the Kishon military panel was intense—but the population studied was decidedly small by epidemiological standards—5,000 soldiers were identified (only 3,000 showed up for testing), compared to the tested population of about 70,000 in the *DuPont C-8 cases*.

The social epistemological bridge in the *Kishon Cases* sought by Ben Asher and Golan failed, in large measure, because the parties did not appreciate the limits of epidemiology, an approach probably doomed from the outset in this case. It is difficult to understand the same failing by the military panel epidemiologist, who eventually disregarded the study's negative results and bowed to the jurist on the panel, the eminent Meir Shamgar, and recommended the military accept responsibility, although perhaps on a lower level because science "could not exclude the possibility of a causal connection between . . . exposure and disease,"²⁷⁰ thereby at least tepidly acknowledging the limitations of the field. The resolution was not well-met. The result was neither scientifically valid nor legally consistent—the scientists merely acquiesced to Shamgar's sense of justice.²⁷¹

This conflict between science-produced-by-experts and its legal reception is summed up by Jasanoff,²⁷² who states that "the representation of law and science as fundamentally different enterprises has given rise to . . . strikingly recurrent themes . . . that of 'culture clash between lawyers . . . and scientists.'"²⁷³ Because she lumps all "science" and its cultural and historical underpinnings into one category,²⁷⁴ whether her assertions apply equally to the practice of medicine and epidemiology, which often consists of starkly raw statistics, is unknown.

268. JASANOFF, *supra* note 236, at 120.

269. Golan, *supra* note 255 (citing David Rosenberg, *The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System*, 97 HARV. L. REV. 849, 851-929 (1984)). Golan cites Rosenberg who uses the term "multivariate analysis," but it is not clear Rosenberg really understands the term. Golan's "perpetuating" the concept shows how statistical errors creep into social discourse and legal opinions. *Id. See, e.g., Manko v. Unites States*, 830 F.2d. 831 (8th Cir. 1986).

270. *See* Golan, *supra* note 255, at 608.

271. *Id.*

272. JASANOFF, *supra* note 236, at 69-92.

273. *Id.* at 7.

274. *Id.*

Perhaps the main obstacles to “interdisciplinary borrowing,” however, are difficulties in communication which generate confusion and conflicting legal decisions. As Fuller states:

It becomes more likely that several teams of researchers will assent to the same set of sentences but apply them in ways that suggest those sentences have quite different meanings Because of the ease with which it can conceal epistemic differences, the communicative process itself is the main source of cognitive change.²⁷⁵

L. SCIENTIFIC EPISTEMOLOGY: THE EXPERT-DETERMINATIVENESS OF TOXIC TORT CASES

“Expert testimony is one of the prominent areas in which science and law collide.”²⁷⁶ As the aforementioned cases illustrate, the type of expert affects the courts’ determinations. In fact, perhaps it can be said that toxic tort cases are “expert-determinative.”²⁷⁷

An expert is “defined as someone with mastery over a body of knowledge and its relevant techniques.”²⁷⁸ In a sense, the testimony regarding causal proof are struggles over “the authority of knowledge”²⁷⁹ before the court’s determination which reflects the field it believes has the “superior knowledge.” Jasanoff, however, recognizes subjective elements experts bring to the courtroom, recommending deconstructing expert testimony and “exposing . . . underlying subjective preconceptions”²⁸⁰ Then she pointedly asks “[w]hose knowledge should count as valid science, according to what criteria and applied by whom?”²⁸¹ Physics education specialist Carl Wenning²⁸² concurs, discussing different ways different fields come to know things²⁸³ and noting the “way of knowing” differs whether one’s opinions are based on concepts or numbers, philosophy or biology.²⁸⁴ This,

275. FULLER, *supra* note 195.

276. 1 OXFORD UNIV. PRESS, *LAW AND SCIENCE: CURRENT LEGAL ISSUES* xi, 259 (Michael D. A. Freeman, Andrew D. E. Lewis & Helen Reece eds., 1998).

277. To the best of my knowledge, this is the first work to use this term.

278. See FISHER, *supra* note 238.

279. Kenneth A. Bruffee, *Social Construction, Language, and the Authority of Knowledge: A Bibliographical Essay*, 48 *COLL. ENG.* 773-90 (1986). Statements attributed to Kuhn claim “scientific knowledge is a social construct.” *Id.* at 774, 779. See ANNE RUGGLES GERE, *WRITING GROUPS: HISTORY, THEORY, AND IMPLICATIONS* 72 (1987).

280. JASANOFF, *supra* note 236, at 19.

281. *Id.*

282. See Carl J. Wenning, *Scientific Epistemology: How Scientists Know What They Know*, 5 *J. PHYSICS TCHR. EDUC.* 3 (2009) (“Scientific inquiry is only one epistemological approach to knowledge.”).

283. See GORE, *supra* note 224.

284. Wenning, *supra* note 282.

of course, influences the way one conceptualizes (mentally frames) an issue, thereby coloring the research and opinion.

The view that scientists are best suited to opine on scientific precepts also is increasingly being challenged.²⁸⁵ Fisher goes so far as championing lay advocacy, voiding the validity of medical judgment earned from years of clinical experience. It appears he would have us believe we might be better off visiting the neighborhood shaman than being seen by a licensed physician, although he is reserving his ire for those viewing the world in mathematical terms, such as statisticians and physicists. To support the illegitimacy of modern science, Fisher conjures the world of chaos theory and quantum physics, where cats are simultaneously alive and dead,²⁸⁶ eviscerating traditional notions of causation, and concluding that “we learn that what one observes in the physical world depends in important ways on where one stands”²⁸⁷

Even challenges between accredited traditional experts are intense in toxic tort causation. Thus “[c]onventional’ scientists, on the one hand, [have] argued that there was ‘absolutely no proof’ a given pollutant was harmful and derided dissenting scientists as tainted by nonscientific and emotional tendencies.”²⁸⁸ “‘Frontier’ scientists, on the other hand, argued that those who demanded high levels of proof had their own unscientific commitments, whether in their predispositions or in their loyalties to the industries for which they worked or from which they received financial support.”²⁸⁹

Into this conflict wades *Daubert*, theoretically, at least, allowing the “frontier” scientist to have her or his say. But, as we wind our way down the slippery slope of scientific epistemology, we see that perhaps *Daubert* cannot save us after all, and we are adhering to *Frye*,²⁹⁰ only accepting testimony (at least in epidemiology) in accord with the scientific mainstream.²⁹¹ Fisher notes that “what we have generally taken as ‘knowledge’ . . . is, rather than the product of objective measures per se . . . the outcome of consensus.”²⁹² In short, in one fell swoop, Fisher does away with *Daubert*, revitalizes *Frye*, and explains

285. *Id.*

286. See MURRAY GELL-MAN, *THE QUARK AND THE JAGUAR: ADVENTURES IN THE SIMPLE AND COMPLEX* 152-53 (1994) (providing a description of Schrodinger’s Cat).

287. See FISHER, *supra* note 238, at 72.

288. *Id.* at 100.

289. *Id.* at 100-01.

290. 293 F. 1013 (D.C. Cir. 1923).

291. New York still holds by the *Frye* standard. *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923), admits only scientific testimony accepted by the general consensus of the scientific community. The *Frye* standard is far more restrictive than *Daubert*, which allows novel scientific evidence, as long as the data is derived from research employing a reliable methodology. See Billauer, *supra* note 227, at 21.

292. See FISHER, *supra* note 238, at 105.

why epidemiologists and clinicians are barred from giving testimony outside the scientific mainstream.

It is not surprising, then, that *Milward*²⁹³ and the *Kishon Cases* rejected the testimony of the treating physician and “frontier epidemiologist.” Notwithstanding *Daubert*,²⁹⁴ “new” views are rejected and choice-of-expert is based on the “going trend” or the general consensus. And so, courts celebrate epidemiological evidence and permit epidemiologists to interpret data as they will—but only if the views reflect the scientific consensus. The code *ipse dixit*, then, actually means that experts can furnish whatever opinions they see emanating from the data—but only if mainstream scientists agree. Since epidemiologic testimony requires data extrapolation, who knows just how far along the extrapolative chain courts will go²⁹⁵ before crying *ipse dixit!*?²⁹⁶ When it comes to frontier science, this can be a subjective determination. But which scientific community’s views will influence the court? What is *ipse dixit* in one specialty may be a bona fide judgment call in another, and outright quackery in a third.

Further, the decision regarding what is *ipse dixit* in epidemiology and who are acceptable experts have one thing in common: they rapidly change with time, and causal conclusions change in tandem, illustrating Kuhn’s conclusion that not until enough scientists sign on to the prevailing paradigm could science change.²⁹⁷ To illustrate, prior to 1955, smoking would not have caused cancer because most experts said it did not; prior to 1960, Thalidomide would not have caused phocomelia because most experts said it did not;²⁹⁸ and prior to 1987, Bendectin caused birth defects because many scientists said it did.²⁹⁹ The science did not change, but the scientific perception had.

The continuum of changing expert opinion parallels the change in courts’ evaluation of who is an expert (or what field is considered scientifically legitimate). Over time, potential experts become more revered in their fields; they themselves have not changed, their

293. 664 F. Supp. 2d 137, 148 (D. Mass. 2009).

294. Billauer, *supra* note 227.

295. “Risk assessment” is based on the precautionary principle and used for regulatory standard setting and has an accepted (and reliable) methodology, but it is a pure exercise in *ipse dixit* (i.e., scientifically unsubstantiated opinion). NAT’L RESEARCH COUNCIL, RISK ASSESSMENT IN THE FEDERAL GOVERNMENT: MANAGING THE PROCESS 1 (1996) [the RED BOOK]. *See also* RODERICKS, *supra* note 106.

296. *See* Gen. Elec. Co. v. Joiner, 522 U.S. 136 (1997) (discussing how *ipse dixit* means opinion testimony untethered to data).

297. *See* THOMAS KUHN, THE STRUCTURE OF SCIENTIFIC REVOLUTIONS (1982).

298. Francis Kelsey of the FDA did not think this. *See* N.Y. Times, *The Shadow of the Thalidomide Tragedy*, YouTube (Sept. 23, 2013), <https://www.youtube.com/watch?v=41n3mDoVbvk>.

299. JOSEPH SANDERS, BENDECTIN ON TRIAL: STUDY OF MASS TORT LITIGATION (1998).

education has not changed, their opinions have not changed. The world's perception of them, however, has.

To illustrate, assuming Sir Austin Bradford Hill, the famed formulator of the nine Hill Principles³⁰⁰ (the “Ten Commandments” of interpreting epidemiological data), were offered as an expert today, that proffer might be rejected. Sir Austin was not a medical doctor and had no formal training in health sciences or epidemiology. His degree was in economics. His integration of statistics into epidemiology was novel and pioneering, posing a problem for jurisdictions adhering to *Frye* which reject novel science even with sound methodologies. Only his work with Professor Doll later enabled him to be called an epidemiologist. At what point along his career would courts have allowed Hill to testify highlights the problems in expert selection, especially in epidemiology, which does not require medical credentials.

In sum, the principles emerging from our poetics inquiry advises that poetic failure derives from competing history, language, and culture. It is confounded by the lack of clear temporal connection, elusive when complex causal mechanisms generate diseases with long latency periods arising at the microscopic level. The process of generating and acquiring the knowledge that qualifies one to be an expert differs by field and commentator. Goethe, a bona fide scientist, relies on the most “unscientific” of skills—intuition. He and others embrace a “holistic” view demanding that “science” resemble the messy world of nature, while Popper (and hence *Daubert*) demands yes-no dichotomous experimentation that all but eliminates the notion of multiple causation. The perspective of the arbiter and the manner of framing the question affects the approach and opinion—leading to clashes between laity and scientific expert, and within the scientific community generates clashes between pioneers of “frontier science” and the old guards of “prevailing knowledge.”

V. THE POETICS OF EPIDEMIOLOGY AND MEDICINE

A. INTRODUCTION

That Popper's views have been appropriated into law (via *Daubert*) as opposed to those of Bernard, Gore,³⁰¹ and Goethe, for example, without the *Daubert* court even giving a tacit acknowledgment of competing views, indicates the poverty of scientific literacy in the legal community. Popper's sinkholes alone would drown a legal

300. See A.B. Bradford-Hill, *The Environment and Disease: Association or Causation*, 58 PROC. ROYAL SOC'Y MED. 295 (1965). But see KENNETH L. ROTHMAN & SANDER GREENLAND, *MODERN EPIDEMIOLOGY*, 17-20 n.74 (2d ed. 1998).

301. See Billauer, *supra* note 176, at 22.

arbiter trying to appreciate the nuanced physiology of causal consequences in toxic tort causation.³⁰² But when epidemiologists differ in understanding their own field's uses and purposes, (i.e., the ultimate intra-cultural conflict), the law must re-evaluate its reliance on a field where we cannot be sure what the experts are conveying—or if they are trying to refashion their culture to fit the law's culture—even as they misinterpret legal doctrine.

I introduce the topic by noting that Jessica Ogden of the London School of Hygiene and Tropical Medicine recognizes the importance of poetics in public health, indicating its value in evaluating not just the effects of disease on people, but the effects history, language, and culture have on the ways people affect diseases which “have impacted changes in public health perspectives and the locus of blame for public health “failures.”³⁰³

B. OVERVIEW

Causal proof in toxic tort cases generally³⁰⁴ relies on epidemiologic evidence.³⁰⁵ Thus, *Brock v. Merrell Dow Pharmaceuticals, Inc.*³⁰⁶ noted the superiority of epidemiologic studies to establish causal proof³⁰⁷ and held “insufficient” any expert's opinion not grounded in a statistically significant epidemiologic study.³⁰⁸ Michael Green stated that “[t]he most desirable evidence is epidemiologic, be-

302. Bernard Black & David Lilienfeld, *Epidemiologic Proof in Toxic Tort Litigation*, 52 *FORDHAM L. REV.* 5 (1984) (“This Article loosely defines toxic tort cases as those in which the plaintiff seeks compensation for harm allegedly caused by exposure to a substance that increases the risk of contracting a serious disease, but does not cause an immediately apparent response.”).

303. Jessica Ogden, *Compliance Versus Adherence: Just a Matter of Language? The Politics and Poetics of Public Health*, in *MANAGING TUBERCULOSIS: AN INTERDISCIPLINARY APPROACH* 215 (1999).

304. *But see In re Meridia Prods. Liab. Litig.*, 328 F. Supp. 2d 791, 800 (N.D. Ohio 2004) (“No requirement exists that a party *must* offer epidemiological evidence to establish causation.”) (emphasis in original). *See also* *Wells v. Ortho Pharm. Corp.*, 788 F.2d 741, 745 (11th Cir. 1986).

305. *See* *Lynch v. Merrell-Nat'l Labs.*, 830 F.2d 1190, 1194 (1st Cir. 1987) (holding that “[s]tudies of this sort [of animal toxicology], singly or in combination, do not have the capability of proving causation in human beings in the absence of confirmatory epidemiological data”).

306. 874 F.2d 307 (5th Cir. 1989).

307. *See In re Meridia*, 328 F. Supp. 2d at 800 (quoting *Soldo v. Sandoz Pharm.*, 244 F. Supp. 2d 434, 536 (W.D. Pa. 2003)) (“This does not mean that conclusive published epidemiologic studies are required in every case alleging cause and effect. In this case, however, other types of evidence upon which plaintiff might reasonably rely are equally absent.”).

308. *Nota bene* the *Brock* court's understanding of the statistical aspects of epidemiology is seriously flawed regarding statistical significance and confidence intervals. Michael 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 (1992).

cause it can best be generalized to support inferences about the effect of an agent in causing disease in humans.”³⁰⁹

One cannot fault courts for their reliance on epidemiology.³¹⁰ After all, it has been used in toxic tort cases for well-nigh thirty years, popularized by Irving Selikoff to sustain funding for his asbestos work,³¹¹ and embraced by Judge Weinstein as the only simulacra of science that supported a connection between Agent Orange and the diseased claimants before him.³¹² During the 1990s, as the legal system continued to struggle with complexities of proving toxic tort causation, epidemiology moved from being a useful adjunct to being almost indispensable.³¹³

It must be noted, however, that there are various subspecialties in epidemiology.³¹⁴ Legal opinions generally reflect the focus and mindset of “public health epidemiology,”³¹⁵ which I define as the use of epidemiology in the practice of public health.³¹⁶ In that the primary goal of public health is disease-prevention or improvement of population health,³¹⁷ a prospective or future-oriented focus,³¹⁸ its use

309. *Id.* at 646 (“Epidemiologic studies are the primary generally accepted methodology for demonstrating a causal relation between the chemical compound and a set of symptoms or a disease.”).

310. *Renaud v. Martin Marietta Corp.*, 749 F. Supp. 1545, 1553 (D. Colo. 1990), *aff'd*, 972 F.2d 304 (10th Cir. 1992) (“[I]n the absence of direct evidence, plaintiffs in toxic tort cases can present circumstantial evidence in order to show that it is probable that they were exposed to contaminants. The two primary forms of circumstantial evidence in a toxic tort case are etiological evidence and epidemiological evidence.”).

311. *See, e.g.*, I.J. Selikoff, et al., *The Occurrence of Asbestosis Among Insulation Workers in the United States*, 132 ANNALS N.Y. ACAD. SCI. 139-55 (1965).

312. *In re Agent Orange Prod. Liab. Litig.*, 597 F. Supp. 749 (E.D.N.Y. 1984).

313. By the early 1990s, U.S. courts were regularly dismissing toxic suits that could not support their claims with solid epidemiological studies. *See SANDERS, supra* note 299.

314. For example, the Johns Hopkins Department of Epidemiology is organized into five epidemiological areas: Chronic Disease, Clinical, Human Genetics/Genetic, Infectious Disease, and Occupational and Environmental. *History of the Department*, JOHNS HOPKINS BLOOMBERG SCH. PUB. HEALTH, <https://www.jhsph.edu/departments/epidemiology/about/history.html> (last visited Jan. 13, 2018). *See also*, NAT'L RESEARCH COUNCIL, ENVIRONMENTAL EPIDEMIOLOGY: THE USE OF THE GRAY LITERATURE AND OTHER DATA 3 (1997).

315. *See* DAVID L. STREINER ET AL., PDQ (PRETTY DARNED QUICK) PUBLIC HEALTH 6 (2011) (noting that epidemiology and public health differ). An example of intraspecialty disconnect surfaces early. *See* E. DOUGLAS SCHUTCHFIELD & C. WILLIAM KECK, PRINCIPLES OF PUBLIC HEALTH PRACTICE 197 (1997) (stating that epidemiology is not only the study of population health determinants but the “application of this study” to these problems).

316. *See* SCHUTCHFIELD & KECK, *supra* note 315, at 3 (“Public health applies the knowledge gained from science to the improvement of the health status of groups of people . . .”).

317. *See* MOYSES SZKLO & F. JAVIER NIETO, EPIDEMIOLOGY: BEYOND THE BASICS 207 (2000) (discussing the two goals of epidemiologic studies, primary prevention and secondary prevention).

of the tool of epidemiology³¹⁹ to quantitatively assess the health of the population reflects that perspective.³²⁰ When courts talk about epidemiology,³²¹ i.e., randomized clinical studies, cohort studies, and case-control studies,³²² they are referring to this model.³²³

Notwithstanding courts' veneration of the field to prove general causation,³²⁴ current practice eschews its use to inform specific causation, "except in those relatively rare cases in which illness occurs in more than fifty percent of the population."³²⁵ Epidemiologists might be surprised to learn that their own luminaries are, in large measure, responsible for the confusing state of affairs.³²⁶

318. See STREINER ET AL., *supra* note 315 ("The basis of public health is trying to prevent or reduce future risk of harm."). *But see id.* at 138 ("[E]pidemiology tells us whether *past* exposures has increased the risk of some outcome for a group of people . . . [.] risk-assessment is used to estimate whether *current* or *future* exposure will affect a city or community.") (emphasis in original). Streiner conflates their scientific validity and is incorrect. The prospective cohort method is exactly as the name implies, examining a population going forward without defining notions of past exposures. See LEON GORDIS, *EPIDEMIOLOGY* 1 (1996); ROTHMAN & GREENLAND, *supra* note 300, at 4; SZKLO & NIETO, *supra* note 317. As to equating risk assessment with epidemiology in terms of scientific validity, most case law and commentators vehemently disagree. See, e.g., *Parker v. Mobil Oil Corp.*, 857 N.E.2d 1114 (N.Y. 2006).

319. SCHUTCHFIELD & KECK, *supra* note 315, at 4.

320. *Id.* at 22.

321. While discussing tools designed to measure population health, the Federal Manual describes epidemiology as "the branch of science that studies the etiology, or cause, of disease." FED. JUDICIAL CTR., *supra* note 24, at 335. See *Glastetter v. Novartis Pharm. Corp.*, 252 F.3d 986 (8th Cir. 2001) (providing yet another example of poetic failure); see also GORDIS, *supra* note 318, at 3.

322. FED. JUDICIAL CTR., *supra* note 24, at 345-47. See also MICHAEL D. GREEN, *BENDECTIN AND BIRTH DEFECTS: THE CHALLENGES OF MASS SUBSTANCE LITIGATION* 27 (1996).

323. An excellent exposé of the nuances generated by misconception of public health epidemiology was written by Nancy Kreiger. Nancy Kreiger, *Questioning Epidemiology, Objectivity, Advocacy, and Socially Responsible Science*, 89 AM. J. PUB. HEALTH 1151-53 (1999) (underscoring how epidemiology is but one of the basic sciences of public health). See also *In re Joint E. & S. Dist. Asbestos Litig.*, 52 F.3d 1124, 1128 (2d Cir. 1995) ("Epidemiology is the study of disease patterns in human populations."). The court in the *Kishon Cases* went on at great length discussing these studies—although irrelevant to the opinion.

324. See *In re Asbestos Litig.*, 52 F.3d at 1128 ("Epidemiological evidence is indispensable in toxic tort carcinogenic tort actions where direct proof of causation is lacking."). This case involved asbestos-caused colon cancer.

325. See JASANOFF, *supra* note 236, at 121.

326. See *In re Fibreboard Corp.*, 893 F.2d 706, 712 (5th Cir. 1990) (determining that population risk could not substitute for findings of individual causation). The court stated:

It is evident that these statistical estimates deal only with general causation, for "population-based probability estimates do *not* speak to a probability of causation in any one case; the estimate of relative risk is a property of the studied population, not of an individual's case." This procedure does prove a particular defendant's asbestos "really" caused a particular plaintiff's disease; the only "fact" proven is that *in most cases* the defendant's asbestos *would have been* the cause.

Fibreboard, 893 F.2d at 712 (emphasis in original).

Sir Richard Doll opined that “[e]pidemiologic data cannot determine the probability of causation in any meaningful way because of individual differences.”³²⁷ Responding to Irving Selikoff’s propagation of asbestos-related lawsuits (some alleging asbestos caused ubiquitous diseases, such as lung cancer),³²⁸ Professor Doll³²⁹ claimed:

[A]sbestos is a cause of lung cancer in this practical sense is incontrovertible, but we can never say that asbestos was responsible for the production of the disease in a particular patient, as there are many other etiologically significant agents to which the individual may have been exposed, and we can speak only of the extent to which the risk of the disease was increased by the extent of his or her exposure.³³⁰

In isolating epidemiology for population-use only³³¹ and in denouncing its applicability for individuals,³³² Doll paved the way for the two-step process of general and specific causation.³³³ Professor Leon Gordis, a contributor to the *Federal Manual on Scientific Evidence*,³³⁴ concurred, informing us that “epidemiology answers questions about *groups*, whereas the court often requires information about *individuals*.”³³⁵

327. Professor Doll’s comments came at the apex of the asbestos legal controversy in the 1980s—and it may be that he was trying to ratchet down the litigation hysteria. See RICHARD DOLL & RICHARD PETO, *THE CAUSES OF CANCER* 1249, 1307 (1981). The author had two personal meetings with Professor Doll in the winter and spring of 1986 where these sentiments were displayed.

328. See generally IRVING J. SELIKOFF & E. CUYLER HAMMOND, *HEALTH HAZARDS OF ASBESTOS EXPOSURE* (1979); see E. CUYLER HAMMOND et al., *Asbestos Exposure, Cigarette Smoking and Death Rates*, in *ANNALS N.Y. ACAD. SCI.* 473 (1979). But see DOLL & PETO, *supra* note 327, at 1249 (“It is again not plausible that any material percentage of the total number of cancers in the whole United States derives from asbestos . . .”).

329. Professor Doll asserted that, except for cigarette smoke, environmental causes of disease are but a miniscule fragment of attributable cancer causes. See DOLL & PETO, *supra* note 327, at 1256 (stating that such causes “account[] for no more than 7% of cancers in the non-occupationally exposed”).

330. Richard Doll, *Proof of Causality: Deduction from Epidemiological Observation*, 45 *PERSP. IN BIOLOGY & MED.* 499, 500 (2002). Accord Nathan Schachtman, *Substituting Risk for Specific Causation*, SCHACTMAN L. <http://schachtmanlaw.com/substituting-risk-for-specific-causation/> (last visited Jan. 13, 2018).

331. See also DAVID A. FREEDMAN, *STATISTICAL MODELS AND CAUSAL INFERENCE* 151-57 (2010) (“Epidemiologic methods were developed to prove general causation There is a considerable gap between relative risks and proof of specific causation.”).

332. See, e.g., *Casey v. Ohio Med. Prods.*, 877 F. Supp. 1380 (N.D. Cal. 1995); see generally Andrew See, *Use of Human Epidemiology Studies in Proving Causation*, 67 *DEF. COUNS. J.* 478, 478 (2000) (“Epidemiology studies can be used to establish only general causation.”).

333. Rosenberg, *supra* note 269, at 856-57. See also *In re Agent Orange Prod. Liab. Litig.*, 597 F. Supp. at 749.

334. MICHAEL D. GREEN, D. MICHAEL FREEDMAN & LEON GORDIS, *REFERENCE GUIDE ON EPIDEMIOLOGY OF THE FEDERAL MANUAL ON SCIENTIFIC EVIDENCE* (3d ed. 2011). Green’s scientific confusion juxtaposed with Gordis’s legal illiteracy contribute to the problem.

335. GORDIS, *supra* note 318, at 1.

The multi-credentialed Sana Loue³³⁶ illustrates how this mantra became mainstream and misunderstood, thereby misinforming the legal culture:

Additionally, epidemiology is concerned with the quest for truth on a population level and seeks to explain disease occurrence and prevention on a population level . . . [;] law is concerned with truth on an individual level, and seeks a determination as to whether the disease or condition at issue in a specific individual is attributable to a specific cause or factor. The dissonance in both purpose and perspective creates a tension between the two disciplines that is perhaps most apparent in their approach to evaluation and a determination of causation.³³⁷

Loue makes a telling point. Sadly, she bases it on erroneous facts. First, Loue's characterization of epidemiology, so prevalent in legal opinions, discounts the rich and larger culture, history, and utility of the field, discussed below. Second, clinical medicine, too, seeks truth on an individual level. It, too, is informed by epidemiology—and vice-versa. Ignoring this relationship exemplifies a cultural xenophobia epidemiologists bring to the courtroom that has infected the law.

C. HISTORY

Perhaps it can be said that modern epidemiology derived from the activities of Dr. John Snow (personal physician to Queen Victoria), which determined the *cause* of the 1854 London cholera epidemic.³³⁸

Snow's epidemiology would have been determinative in a lawsuit against the Lambeth Company,³³⁹ as it convincingly determined the Broad Street well that they owned and maintained³⁴⁰ was the "cause" of the cholera epidemic. By comparison, the medical cause, *vibrio cholerae*, was discovered by Robert Koch (originator of Koch's postu-

336. She has a JD, PhD in Medical Anthropology, PhD in Epidemiology, MPH, MSSA in Social Work, and MA in Education. *Staff*, CASE W. RES. U. SCH. MED., <https://case.edu/medicine/faculty-development-diversity/about/staff/loue.html> (last visited Jan. 13, 2018).

337. Sana Loue, *Epidemiological Causation in the Legal Context: Substance and Procedures*, in STATISTICAL SCIENCE IN THE COURTROOM 264 (Joseph L. Gastwirth ed., 2000).

338. *But see* Stephen Thacker & Ruth Berkelman, *Public Health Surveillance in the United States*, 10 EPIDEMIOLOGICAL REVIEWS 164-65 (1988) (discussing early practices).

339. ABRAHAM LILIENFELD, FOUNDATIONS OF EPIDEMIOLOGY 1 (1976).

340. Not all residents in proximity to the well became ill, discrediting, at first instance, the hypothesis that the well was the causal source. Upon further inquiry, workmen employed by the New Street Brewery, who drank the Brewery's malt and did not drink the well water, did not become ill! No exposure—no disease. *See* EDWARD R. TUFTTE, VISUAL AND STATISTICAL THINKING: DISPLAYS OF EVIDENCE FOR MAKING DECISIONS 5-8 (1997).

lates—sometimes confused with Hill’s principles by various courts),³⁴¹ in a laboratory twelve years later. Snow tested the well water in a laboratory—and found nothing amiss. Only the population study allowed him to “prove” the well at Broad and Cambridge “caused” the epidemic.³⁴²



Figure 1: The Broad Street Wall³⁴³

341. See *Miller v. Pfizer, Inc.*, 196 F. Supp. 2d 1062 (D. Kan. 2002), *aff'd* 356 F.3d 1326 (10th Cir. 2004). The court repeatedly referred to the Hill Principles as Koch’s Postulates. None of the four sets of law firms involved—nor their experts, the judge, or the law clerks—caught the error, attesting to the scientific illiteracy of the legal profession.

342. JOHN SNOW, ON THE MODE OF COMMUNICATION OF CHOLERA 42 (1855).

343. *John Snow Memorial and Pub*, Wikimedia Commons, <https://commons.wikimedia.org/w/index.php?curid=357998> (last visited Jan. 13, 2018).

Frank Fisher's work further demonstrates the misconceptions about the field, its history, and its capabilities:

Because traditional epidemiology tends to limit itself to the broad and generalizable trends related to . . . health problems, it overlooks the disparate concentrations of such disorders As such, it neglects disproportionate risks in occupation and workplace exposures assumed by low-income and working-class persons of color, especially women.³⁴⁴

While Fisher may have a bona fide complaint regarding today's epidemiology, the advent of the field was far different. Foundational principles derived from the study of occupational diseases in the poor or under-privileged.³⁴⁵ In the late 1800s, Dr. Alice Hamilton's³⁴⁶ meticulous exposure-data collection of impoverished laborers³⁴⁷ revealed hitherto elusive causal connections between occupational exposures and diseases.³⁴⁸ In 1908, Louis Brandeis, with the help of his sister-in-law, Josephine Goldmark, submitted the first scientific brief to the United States Supreme Court (which came to be called "The Brandeis Brief"), and which addressed occupational hazards faced by women,³⁴⁹ containing almost one hundred pages of epidemiological and sociological data.³⁵⁰

Fisher³⁵¹ also relies on Abraham Lilienfeld's³⁵² definition of epidemiology to support his claims,³⁵³ echoed in a legal seminal paper by Black and David E. Lilienfeld (Abraham's son):

The elucidation of the relationship between a disease and a factor (e.g., a toxic substance) suspected of causing it lies within the domain of epidemiology. The epidemiologist examines this relationship in the context of populations, comparing the disease experiences of people exposed to the factor with those not so exposed.³⁵⁴

344. See FISHER, *supra* note 238.

345. Percival Pott investigated scrotal cancer in chimney sweepers in 1775. See DAVID L. STREINER ET AL., *supra* note 315, at 6, 163.

346. See ALICE HAMILTON, *EXPLORING THE DANGEROUS TRADES* 128-62 (1943). Her work led to enactment of the Occupational Safety and Health Act of 1970, Pub. L. No. 91-596, 84 Stat. 1590 (1970), the year she died.

347. The integration of epidemiology and toxicology was key to epidemiology, as practiced a century ago. HAMILTON, *supra* note 346, at 163. Today, such integrated use is posed as novel. See Douglas L Weed, *Environmental Epidemiology Basics and Proof of Cause/Effect*, 181-82 *TOXICOLOGY* 399-403 (2002).

348. HAMILTON, *supra* note 346.

349. *Muller v. Oregon*, 208 U.S. 412 (1908). See also Billauer, *supra* note 227.

350. *Id.*

351. FISHER, *supra* note 238, at 147.

352. LILIENFELD, *supra* note 339.

353. Black & Lilienfeld, *supra* note 302.

354. *Id.*

With this foundational background, it is no wonder courts try to limit the use of epidemiology to general causation.³⁵⁵ But, Fisher and Professor Lilienfeld's son would have done well to revisit Abraham Lilienfeld's original description, discussed below, which details a far broader blueprint for the field.

D. LEGAL HISTORY

The legal history of epidemiology is recounted in Tal Golan's multiple works³⁵⁶ and need not be detailed here, other than to say that in the 1960s and 1970s concerns for the environment took center stage in policymaking and relied on experimental toxicology for standard setting.³⁵⁷ Eventually, both industry and civil action groups challenged the science behind the standards, which, *inter alia*, required interspecies³⁵⁸ and high-dose-low-dose extrapolation.³⁵⁹ Regulators admitted the fragility of the method,³⁶⁰ but justified it on the basis of necessity. Some suggested better scientific tools might be found in the arsenal of epidemiology.

Initially, objections came from both sides:

Criticism of the newly-fangled epidemiology was by no means limited to die-hard experimentalists [or lawyers]. Scientists committed to genetic views of disease faulted epidemiology for focusing attention on environmental effects, while those committed to social explanations of disease faulted it for focusing attention on individual factors abstracted of social context. Perhaps the most sophisticated critiques came from biostatisticians anxious to protect the integrity of their science

355. See *See, supra* note 332.

356. Tal Golan, *Epidemiology, Tort and the Relations Between Science and Law in the Twentieth Century Courtroom*, NATURE ENGAGED: SCIENCE IN PRACTICE FROM THE RENAISSANCE TO THE PRESENT 163-83 (2012) [hereinafter *Science and Law*]. See also Tal Golan, *The History of Epidemiological Evidence in the Twentieth Century American Courtroom*, in NATURE ENGAGED: SCIENCE IN PRACTICE FROM THE RENAISSANCE TO THE PRESENT (2012) [hereinafter *History of Epidemiological Evidence*]; see generally TAL GOLAN, LAWS OF MAN AND LAWS OF NATURE: A HISTORY OF SCIENTIFIC EXPERT TESTIMONY (2004) [hereinafter GOLAN, LAWS OF MAN]. But see Nathan Schachtman, *Tal Golan's Preliminary History of Epidemiologic Evidence in U.S. Courts*, SCHATMAN L., <http://schachtmanlaw.com/tal-golans-preliminary-history-of-epidemiologic-evidence-in-u-s-courts/> (last visited Jan. 13, 2018) (a brilliant essay on Golan's opinions).

357. See also David Schorr, *Historical Analysis in Environmental Law*, in OXFORD HANDBOOK OF HISTORICAL LEGAL RESEARCH, (Markus Dubber & Christopher Tomlins eds., 2017).

358. See JASANOFF, *supra* note 236, at 69-92.

359. Hertz-Picciotto, *Epidemiology and Quantitative Risk Assessment: A Bridge from Science to Policy*, 85 AM J PUB. HEALTH 484-91 (1995) ("The emphasis [of risk-assessment] is not on establishing rigid rules, but rather on ensuring a consistent, reliable process that makes optimum use of available data.")

360. See *Reserve Mining Co. v. EPA*, 514 F.2d 492 (8th Cir. 1975); *Ethyl Corp. v. EPA*, 541 F.2d 1 (D.C. Cir. 1976).

from political pressures, and from epidemiologists who were concerned that too much would be claimed for their fledgling science, which was just starting to make inroads into medicine. These critics were able to point out various methodological difficulties inherent to epidemiological research, from sampling and selection biases to confounding variables, which further undermined epidemiology's capacity to establish authoritative causal claims.³⁶¹

To compensate for deficiencies in epidemiology,³⁶² scientists involved in policymaking³⁶³ introduced "quantitative risk assessment,"³⁶⁴ and social scientists introduced "the precautionary principle".³⁶⁵

Disdained by scientific purists, this pragmatic program of epidemiology was warmly embraced by the expanding regulatory regimes of the late twentieth century. [This] so-called "black-box epidemiology"—a technical, policy-driven epidemiology . . . shunned biological hypotheses and concentrated on computing the risks facing taxpayers³⁶⁶

As Tal Golan writes:

The epidemiologists traded up their mechanical rulers first for punch cards and then for software programs, and got comfortable with the new tools of multivariate correlation and regression, and exotic tests of statistical significance. By the end of the 20th century, the reduction of causes to a distributed network of risk factors had become prevalent and increasingly informed medical research, as well as regulatory and legal action. In theory, some continued to insist, this was not a science of causation. *In practice, however, it was exactly this—a hunt for causes; if not for science then certainly for administrative and legal action.*³⁶⁷

361. GOLAN, LAWS OF MAN, *supra* note 356, at 23.

362. *See id.* at 166 ("Epidemiology, they pointed out, was not an experimental science. It could neither sufficiently control its data nor test the veracity of its conclusions. Thus, while epidemiology remained useful in generating causal hypotheses, only experimental science could reliably validate them.")

363. RODERICKS, *supra* note 106, at 57. *See also* THOMAS A. BURKE, ET AL., REGULATING RISK, THE SCIENCE AND POLITICS OF RISK (1993).

364. AM. CHEM. SOC'Y, UNDERSTANDING RISK ANALYSIS, A SHORT GUIDE FOR HEALTH, SAFETY AND ENVIRONMENTAL POLICY MAKING (1998), http://raperkins.net/ENVE_652/Module08/Submodule8D_%20RiskCom/risk_book.pdf. But see John D. Graham & Jonathan Baert Wiener, Risk vs. Risk: Tradeoffs in Protecting Health and the Environment (1995).

365. GOLAN, LAWS OF MAN, *supra* note 356, at 23.

366. Neil Pearce, *Traditional Epidemiology, Modern Epidemiology, and Public Health*, 86 AM. J. PUB. HEALTH 678-83 (1996) ("The key issue has been the shift in the level of analysis [of epidemiology] from the population to the individual."). *See also* Milton Terris, *Epidemiology and the Public Health Movement*, 3 J. PUB. HEALTH POL'Y 315-29 (1987); Hertz-Picciotto, *supra* note 359.

367. GOLAN, LAWS OF MAN, *supra* note 356, at 27 (emphasis added).

Policymakers (many of whom are lawyers) and epidemiologists, however, differed on what constitutes “epidemiological best evidence.”³⁶⁸ “Policy-makers preferred systematic reviews and qualitative research, focusing on consistency and strength of evidence, the quality of data, bias in the evidence, and relevancy . . . compared to epidemiologists who favored quantitative studies and . . . focused on methodology.”³⁶⁹

These difficulties should have sounded a warning bell.³⁷⁰ Nevertheless, faced with similar problems, judges, too, turned to epidemiology.³⁷¹ By the late 1980s, epidemiologic proof of toxic tort causation had become a growth industry.³⁷² Early (and erroneous) parameters were institutionalized in *Manko v. United States*³⁷³ in 1986.³⁷⁴ Thereafter, “junk epidemiology” bankrupted the Silicone Breast Industry (via loose testimony of unscrupulous “experts”) and dismantled Dalkon Shield production.³⁷⁵ Junk science³⁷⁶ led to the *Daubert trilogy*,³⁷⁷ which sought to prevent more bad science from polluting precedent. Even earlier, demands for more precise proof generated the general and specific causation requirement, which *Daubert* then sentenced to the pre-trial stages for determination before a jury could hear the relevant evidence.³⁷⁸

E. EPIDEMIOLOGY: WHAT IT DOES DEPENDS ON WHOM YOU ASK

With this overview in mind, I revisit Abraham Lilienfeld’s perspective on epidemiology, generated prior to the toxic-tort industry boom and hence unscathed by the legal climate. Lilienfeld’s view was that the field has *three* basic purposes, including: (1) determining etiological factors of diseases, such as investigating what food *caused* an outbreak of food poisoning or to determine what chemicals and what

368. O’Lily Donoghue Jenkins, et al., *Evaluating and Using Observational Evidence: The Contrasting Views of Policy Makers and Epidemiologists*, FRONT PUB. HEALTH, Dec. 6, 2016, at 1; P Shabecoff, *Radon Alert: The EPA Goes After the Carcinogen that Nature Made*, N.Y. TIMES, Sep. 18, 1988, at 1, 5. See also L.A. Cole, *Radon Scare—Where’s the Proof?*, N.Y. TIMES, Oct. 6, 1988, at A31.

369. O’Lily Donoghue Jenkins, et al., *supra* note 368, at 1.

370. *Id.*

371. Billauer, *supra* note 227.

372. JACK C. FISHER, SILICONE ON TRIAL: BREAST IMPLANTS AND THE POLITICS OF RISK (2015).

373. 636 F. Supp. 1419 (W.D. Mo. 1987).

374. *Manko v. United States*, 636 F. Supp. 1419 (W.D. Mo. 1987).

375. FISHER, *supra* note 372.

376. PETER W. HUBER, GALILEO’S REVENGE: JUNK SCIENCE IN THE COURTROOM 206-09 (1991).

377. *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993).

378. *Science and Law*, *supra* note 356. See also *History of Epidemiological Evidence*, *supra* note 356; see generally GOLAN, LAWS OF MAN, *supra* note 356.

degree of exposure *caused* the disease;³⁷⁹ (2) determining if a *causal* hypothesis developed by a clinician or experimenter is consistent with the distribution of disease in the population; and (3) acting as a basis for preventive and public health services.³⁸⁰

Practitioners in these areas have different mindsets and perspectives. The first group determines what happened in the past—much like tort litigation. The second investigates what is happening in the present, much like standard-setting by policymakers. The third tries to affect the future, much like social activists. One therefore may ask whether public health epidemiology, with its focus on the future, is relevant to proof of past causation in tort law?

Victor Schoenbach of the Gilling School of Public Health disagrees with Gordis regarding epidemiology's role in causation, noting "epidemiologic research, whether descriptive or analytic, etiologic or evaluative, generally seeks to make causal interpretations."³⁸¹ Abraham Lilienfeld would concur³⁸² as he looked at causation from a more pragmatic approach, both in medicine and epidemiology.³⁸³ Proponents of "clinical epidemiology"³⁸⁴ and etiologic epidemiology also concur, leav-

379. The CDC Epidemiologic Intelligence Service ("EIS") practices etiologic epidemiology. "EIS officers . . . [are] ready responders, identifying causes of disease outbreaks . . ." *Epidemic Intelligence Survey*, CDC (Apr. 19, 2017), <https://www.cdc.gov/eis/diseasedetectives.html>. Among EIS trainees are not just medical doctors, but experts in nutrition, scientists, and veterinarians, which would have been useful for the *Kishon* court to have noticed. While they use population studies to generate their answers, their focus is on solving individual causes of diseases. *See generally*, BERTON ROUCHCE, *THE MEDICAL DETECTIVES* (1947).

380. LILIENTELD, *supra* note 339, at 3-9.

381. VICTOR SCHOENBACH & WAYNE D. ROSAMOND, *UNDERSTANDING THE FUNDAMENTALS OF EPIDEMIOLOGY: AN EVOLVING TEXT* 355 (2002). In a 2002 article, Douglas Weed and Pamela Mink provide a succinct and thoughtful discussion of this twenty-year long "remarkable disciplinary rift," concluding that an epidemiologist can be a "full-fledged epidemiologist" whether he or she does etiologic research alone, combines public health practice and policymaking with research, or spends most of her or his time "making the public health system work." Weed & Mink, *Roles and Responsibilities of Epidemiologists*, 12 *ANNALS EPIDEMIOLOGY* 67, 70 (2002). *See also* SCHOENBACH & ROSAMOND, *supra* note 381, at 91, 223, 272 ("[D]eriving an etiologic analysis is a critical function of epidemiology.").

382. As would Dona Schneider and David Lilienfeld. SCHNEIDER & LILIENTELD, *supra* note 179, at 4.

383. LILIENTELD, *supra* note 339, at 252. "The essentials of the epidemiological approach in determining the specific etiologic agent of a disease are demonstrated in their simplest form by the investigation of a food poisoning outbreak." *Id.* at 15. "The demonstration of a statistical relationship between a disease and biological or social characteristics is the first step in the epidemiologic analysis of the etiology of the disease." *Id.* at 247.

384. *See UCSF Program on Epidemiology and Biostatistics*, CLINICAL EPIDEMIOLOGY & HEALTH CARE RES. INST. HEALTH, <https://epibiostat.ucsf.edu/clinical-epidemiology> (last visited Jan. 13, 2018) ("Clinical Epidemiology is the application of principles of epidemiology to clinical medicine. While classical epidemiology is the study of the distribution and determinants of diseases in populations, clinical epidemiology is the application of the principles and methods of epidemiology to conduct, appraise or apply

ing one to wonder why courts numbly embraced the views of Doll and Gordis (whose views are enshrined in the *Federal Manual on Scientific Evidence*), without batting a collective eyelash.

Professor Haroutune Armenian³⁸⁵ also claims epidemiologists investigate causation in individual cases, a practice he calls “case-investigations,”³⁸⁶ which are performed by hospital and clinical epidemiologists as well as clinicians.³⁸⁷ “Clinical investigations try to explain the reason(s) why this particular person developed the disease [T]he epidemiologist-clinician uses prior knowledge about the risk-factors and etiology of the disease to explain the occurrence of the incident event,”³⁸⁸ a practice similar to Dr. Butler’s in *Milward*,³⁸⁹ discussed above.³⁹⁰ In Armenian’s approach, the primary concern is the individual patient,³⁹¹ and the investigation provides “as complete an explanation as possible of the *etiology* of the specific problems affecting the case”³⁹²

This disconnect between clinical epidemiologists and public-health researchers is explained by Silverman, who notes that “basically the fields do not talk to each other and do not trust one another.”³⁹³ With this in mind, it is worthwhile to compare Abraham Lilienfeld’s and Armenian’s goals of epidemiology with Gordis’, the legal go-to expert, whose own textbook lists five objectives, all public health driven.³⁹⁴ By comparison, in addition to Armenian and Lilienfeld, David Katz, in his *Review of Epidemiologic Approaches*, defines nine “Contributions of Epidemiologists to the Medical Sciences,” four of which are individual and clinician-relevant. The ninth is “providing expert testimony in court.”³⁹⁵

clinical research studies focusing on prevention, diagnosis, prognosis, and treatment of disease. Clinical Epidemiology is the basic science of Evidence-based Medicine.”).

385. Abraham Lilienfeld preceeded Gordis as Chair of the Epidemiology Department at Johns Hopkins School of Public Health; Armenian succeeded Gordis.

386. HAROUTUNE K. ARMENIAN & SAM SHAPIRO, EPIDEMIOLOGY AND HEALTH SERVICES 4 (1998).

387. *Id.*

388. *Id.* at 109-11.

389. 664 F. Supp. 2d 137 (D. Mass. 2009).

390. ARMENIAN & SHAPIRO, *supra* note 386, at 109-11.

391. *Id.* at 4.

392. *Id.* at 15.

393. WILLIAM A. SILVERMAN, WHERE’S THE EVIDENCE?: DEBATES IN MODERN MEDICINE 91-92 (1998).

394. ARMENIAN & SHAPIRO, *supra* note 386.

395. David L. Katz, *Epidemiology*, BIostatistics & PREVENTIVE MED. REV. 3-5 (1997).

F. MULTIPLE CAUSATION AND CAUSAL THINKING

Breaking with current law-think, Professor Lilienfeld emphasizes differentiating between one-cause disease models (but/for causation) and multi-factor diseases, which he illustrates with tuberculosis (“TB”).³⁹⁶ While undoubtedly TB is caused by *Mycobacterium tuberculosis*, the bacillus is not found in every TB patient, nor does everyone exposed to the agent become sick—as additional susceptibility and lifestyle factors are required.³⁹⁷ These multicausal situations, Lilienfeld claims, violates the first of Koch’s postulates of causation and inevitably generates the circular reasoning³⁹⁸ the *Milward*³⁹⁹ court so violently castigated. Contrary to what legal experts would like us to believe, then, sometimes, even when we know the cause and have the ability to prove it, scientific proof does not always work.⁴⁰⁰

Lilienfeld attributes these dichotomous perspectives found within epidemiology—to culture clash. Most “poetically,” he notes that:

[D]ifferences in causal thinking [especially where multiple causal agents are involved]—depend on the frame of reference within which the investigator operates [W]hen multiple etiological factors . . . each act[ing] independently . . . produce a change . . . at a cellular level . . . which becomes the necessary pre-condition of the disease process,⁴⁰¹ this should be regarded as part of the disease process.

G. SIGNIFICANT STATISTICAL CONTORTIONS

Progressive epidemiologists claim a “newer epidemiology” exists that has, in fact, adopted a multi-causal approach. Its proponents assert that “the notion of a single cause of disease [is] . . . firmly rejected in favor of multiple causation.”⁴⁰² In the courtroom, however, statisticians revert to uni-causal models, perhaps because statistics has not evolved sufficiently to address multi-causal situations with confidence.

While law is supposed to be predicated on comparative or qualitative assessments,⁴⁰³ epidemiologists speak in the statistical language of risks and probabilities, setting the stage for the frisson in transla-

396. LILIENFELD, *supra* note 339, at 249-51.

397. *Id.* at 250.

398. *Id.* at 251.

399. 664 F. Supp. 2d 137 (D. Mass. 2009).

400. Presumably this could occur as a result of false negative test results. MELVIN SHIFFMAN, *ETHICS IN FORENSIC SCIENCE AND MEDICINE* 97 (1999).

401. LILIENFELD, *supra* note 339, at 251.

402. SCHUTCHFIELD & KECK, *supra* note 315, at 22.

403. See LAWRENCE COHEN, *THE PROVEABLE AND THE PROBABLE* (1977).

tion.⁴⁰⁴ Generally, case law requires data to be statistically significant,⁴⁰⁵ but epidemiologists Rothman and Greenland decry this requirement, noting that while “the notion of statistical significance has come to pervade epidemiologic [and legal] thinking . . . [,] [it] offers less insight into epidemiological data than alternative methods that emphasize estimation of interpretable measures.”⁴⁰⁶ Most courts disagree, however.⁴⁰⁷

The hypocrisy of relying on epidemiological statistical significance becomes apparent in the “Viagra vignette.” In law, statistical significance all but guarantees admissibility. Conversely, it appears that in science, its presence means nothing—unless scientists say so. Two recent statistically significant works “proved” Viagra “causes” melanoma, at least by legal standards, i.e., the relative risk was almost doubled, a finding many courts equate with proof of *specific* causation.⁴⁰⁸ At least one article ratified a biologically plausible mechanism.⁴⁰⁹ Nevertheless, the scientific consensus is that it is not Viagra, *per se*, that causes melanoma, but the lifestyle associated with it. This *ipse dixit* exculpation is made by scientists⁴¹⁰ who seem fearful of maligning the male wonder-drug (or perhaps take the drug them-

404. The bright line of a “relative risk greater than 2.0 means that d was more likely than not caused by c,” is perhaps the key perversion of law-epi translations. *Id.* (citing *Manko v. United States*, 636 F. Supp. 1419 (W.D. Mo. 1987)). Consideration of relative risks—which pertains to the *average* risk of a population, without consideration of confidence intervals, is wholly non-determinative. See M. KHOURY, ET AL., *FUNDAMENTALS OF GENETIC EPIDEMIOLOGY* 77 (1993).

405. See BRADFORD-HILL, *supra* note 300. *But see* STEVEN A. KOEHLER & PEGGY A. BROWN, *FORENSIC EPIDEMIOLOGY* 6 (2009) (arguing against statistical significance). However, in the legal arena, we are mandated to assure reliability of results and statistical significance is the only guarantee courts have that the results were not merely an accident of chance.

406. ROTHMAN & GREENLAND, *supra* note 300, at 6.

407. The practice described by Rothman and Greenland sounds remarkably akin to the “qualitative epidemiology” used by Professor Linn and dismissed by Judge Shapiro in the *Kishon Cases*.

408. JASANOFF, *supra* note 236, at 69-92.

409. Marie Ellis, *Does Viagra Encourage Melanoma Cells to Proliferate?*, MED. NEWS TODAY (Mar. 11, 2016), <https://www.medicalnewstoday.com/articles/307729.php>.

410. Wen-Qing Li, et al., *Sildenafil Use and Increased Risk of Incident Melanoma in US Men: A Prospective Cohort Study*, 174 *INTERNAL MED.* 964-70 (2014). Finding a statistically significant, nearly doubled risk, the authors suggest biological plausibility via an increase in intracellular cGMP levels. A second study confirmed the findings (first time users experiencing a 21% increased risk). See Stacy Loeb et al., *Use of Phosphodiesterase Type 5 Inhibitors for Erectile Dysfunction and Risk of Malignant Melanoma*, 313 *INTERNAL MED.* 2449, 2449-55 (2015). The eleven authors of both articles discount the significance or health risk of their statistically significant data. One (Loeb) went so far as to do an auxiliary data check to *disprove* her results, which appears to be unpublished. See Arlene Weintraub, *Viagra Doesn't Cause Melanoma but The Viagra Lifestyle Might*, FORBES (June 23, 2015, 11:00 AM), <https://www.forbes.com/sites/ardeneweintraub/2015/06/23/report-viagra-doesnt-cause-melanoma-but-the-viagra-lifestyle-might/#10eca7fb25a9>.

selves). The hypocrisy goes farther. In a third attempt to exculpate the Viagra-melanoma causal connection, a meta-analysis examined three case-control studies and two cohorts. That attempt, too, found a statistically significant excess risk. These results were disavowed⁴¹¹ because they did not demonstrate a dose-response curve, i.e., the statistically excess disease was found in those taking low doses of the drug, directly contradicting the reasons for rejecting the initial study's findings. In the initial studies, the *ipse dixit* was that Viagra confounds for lifestyle factors (i.e., excess sun exposure, and presumed that men who take Viagra are more likely to spend time in the sun). Applying this logic to the meta-analysis, only men who take low doses of Viagra live the good life in the sun or have a lifestyle that confounds for the "real cause" of melanoma.⁴¹² The inconsistencies and logical flaws of this reasoning seem to elude the scientists.⁴¹³

This vignette is illustrative of the fact that epidemiology's purpose depends on whom you ask, who is doing the interpreting,⁴¹⁴ or reflects a self-consciousness of the variable validity of the field within public health.⁴¹⁵ For many epidemiologists, "causal laws are essentially working assumptions or tools of the scientist rather than verifiable

411. Susan Mayor, *Phosphodiesterase Inhibitors such as Viagra do not Increase Melanoma Risk, Review Finds*, 357 *BRIT. MED. J.* 2468 (2017). See also *Erectile Dysfunction Medicines do not Cause Melanoma, Analysis of Large Studies Finds*, *CLINICAL PHARMACIST* (May 19, 2017) ("A meta-analysis reveals a significant association between the use of Viagra and low-stage melanoma but the criteria for causation were not met.").

412. BRETT SALISBURY, *PROOF THE SUN DOES NOT CAUSE SKIN CANCER* (2016).

413. One wonders if physicians should be prescribing high doses of the drug—as it appears to confer some protective effect.

414. Compare GUNTHER F. CRAUN & REBECCA L. CALDERON, *HOW TO INTERPRET EPIDEMIOLOGICAL ASSOCIATIONS* (2005) (attributing excess cardiac mortality and morbidity in Japan to the hardness of the drinking water), with RICHARD W. WILKINSON, *UNHEALTHY SOCIETIES: THE AFFLICTION OF INEQUALITY* 18, 130-34 (1996) (attributing it to perceived national inequality).

415. See Hannes G. Pauli & Kerr L. White, *Scientific Thinking, Medical Thinking and Medical Education: Questions derived from their Evolution in the 20th Century*, *ROUND TABLE DISCUSSION*, http://www.who.int/hrh/en/HRDJ_2_3_02.pdf (last visited Jan. 13, 2018) ("[D]octrine of specific etiology is biased in favor of mechanistic phenomena, laying the ground for the subsequent tremendous development of technology in medicine and its dissociation from the psychic and social domains, considered to be a matter of intuition. The patient, then, scientifically speaking, becomes a machine The term diagnosis represents not statements of facts but of medical concepts. As such it is an instrument of thinking, in the sense that its use, non-use or alternative use will lead to different actions and effects. For example, to use the diagnostic term either of angina pectoris or coronary occlusion in an identical situation will set the minds of those making diagnostic or therapeutic decisions in different directions. In one case the orientation will be more towards a patient's subjective perception, towards the person as a whole; in the other more towards an underlying substratum or mechanism, involving the vessels of the heart.").

statements about reality”⁴¹⁶ Susser complains about the “mindless abuse of complex statistical packages” (such as multivariate analysis)⁴¹⁷ to infer some sort of flawed “reality.”⁴¹⁸ Professor Scales bitterly notes that “epidemiology is not the be all and end all of causal inquiry, and the courts—by and large—have turned their ‘gate-keeping function’ into more of a ‘search and destroy’ mission. Most real epidemiologists think it is nonsense.”⁴¹⁹ Indeed, if epidemiologists themselves decry the lack of reliability of their field, how can courts accept it? After all, Sir Richard Doll, the eminent epidemiologist upon whose rhetoric much of the legal framework is based, even raised “the image of epidemiology as informed common sense.”⁴²⁰

Perhaps, then, it can be said that problems using epidemiology in the legal context derive from:

confusing two rather different enterprises[;] on the one hand, there is the issue of legitimating knowledge claims, which is decided by the conventions of a culture and will depend on the interests that the culture has in acquiring knowledge [in epidemiology] on the other hand there is the issue of explaining knowledge which involves studying their causal origins [medicine].⁴²¹

H. LANGUAGE

If lawyers do not understand science, that is understandable, and perhaps curable—but the problem becomes multiplied when scientists do not understand each other. Even simple, non-scientific words produce confusion in scientists of different persuasions, reflecting the cognitive (and cultural) milieu of each. Matthew Cobb recounts a story where Nobel Prize winner Joshua Lederberg began a correspondence with physicist and Fermi Prize winner John von Neumann in 1955:

The two men soon realized that each of them did not understand what the other meant by [by the simple word] ‘information’, and that Lederberg eventually concluded that this was because they were thinking at very different levels. For biologists, he argued, the propagation and evolutionary elaboration

416. A comment taken from from Hubert Blalock. See FREDERIC J. FLERON, *RUSSIAN STUDIES AND COMPARATIVE POLITICS: VIEWS FROM METATHEORY AND MIDDLE-RANGE THEORY* 72 (2016).

417. See also ROTHMAN & GREENLAND, *supra* note 300, at 6 (on multi-variate analysis).

418. JOSSEY-BASS, *METHODS IN SOCIAL EPIDEMIOLOGY* 41-42 (J. Michael Oakes & Jay S. Kaufman eds., 2006). Susser’s comments regarding misuse of multivariate analysis and substituting “risk factor” identification for causal determinations did, however, reawaken multiple cause considerations into epidemiology.

419. See Scales, *supra* note 138, at 273.

420. OXFORD UNIV. PRESS, *supra* note 276, at 259.

421. FULLER, *supra* note 195, at 18.

tion, of complexity is ‘self-evident’—they were interested in the detail of how such a system could work. The logician von Neumann, however, was looking for the foundations of an axiomatic theory of reproduction—something much more abstract and not linked to biology at all.⁴²²

The linguistics of epidemiology are even more problematic. In discussing a paper published in 1975, the beginning of the legal epidemiology boom, Rothman and Greenland noted that although “[n]o new concept or definition was proposed . . . [,] the paper was useful because so many readers did not know the distinctions among the basic measures used in epidemiology.”⁴²³ As late as 1998, they write that understanding the fundamental underpinnings of epidemiology such as “causation” and “induction periods” have not yet been fully integrated into the conceptual bedrock of the discipline.⁴²⁴

[This] disagreement about basic conceptual and methodologic points has led in some instances to profound differences in the interpretation of data [This does] not necessarily attest to the thick-headedness of epidemiologists; a more charitable interpretation would be that the basic ideas fundamental to the new science have not yet displaced traditional thinking.⁴²⁵

A detailed analysis of misused terminology such as confusing “attributable risk”⁴²⁶ with “relative risk,”⁴²⁷ affecting multi-causal analysis,⁴²⁸ or scientific “validity” with “reliability,” affecting evidentiary admissibility⁴²⁹ is outside the scope of this article. Suffice it to say that that these basic terms are used differently in law, medicine, and even within epidemiology. To illustrate, we take a brief look at the

422. MATTHEW COBB, *LIFE’S GREATEST SECRET: THE RACE TO CRACK THE GENETIC CODE* 146 (2015). *See also* KUHN, *supra* note 297 (providing a similar example. Here a chemist and physicist were asked if a single atom of helium was a molecule; the chemist said yes; the physicist said no).

423. ROTHMAN & GREENLAND, *supra* note 300, at 4.

424. *Id.*

425. *Id.*

426. GORDIS, *supra* note 318, at 155 (“Attributable risk . . . is defined as the amount or proportion of disease incidence (or disease risk) that can be attributed to a specific exposure.”). Thus, attributable risk measures how many cases can be prevented if a single substance or exposure is eliminated. *Id.* at 122. *See also* Katz, *supra* note 395, at 122 (noting it is “[t]he proportion of the total risk for a particular outcome attributable to a particular exposure”); KHOURY ET AL., *supra* note 404. While the concept is critical to understand, “there are still some conceptual problems in its definition and interpretation measures. . . .” Its use is only applicable to a sufficient-cause model (i.e., but/for causation). *Id.*

427. GORDIS, *supra* note 318, at 142. Relative risk measures the *average* risk comparing exposed and unexposed groups. *Id.*

428. SEYMOUR GARTE, *GENETIC SUSCEPTIBILITY TO CANCER* 75-88 (1980).

429. *See* Billauer, *supra* note 227, at 4-5.

words “cause,” “etiology,”⁴³⁰ and “association.”⁴³¹ In the next section, I evaluate the derivative terms “differential diagnosis” and “differential etiology.”

The Federal Judicial Manual on Scientific Evidence takes pains to differentiate “cause” and “etiology,”⁴³² reserving the former to the legal profession and limiting the latter to the medical profession. However, contrary to the legal consensus, Armenian reports that doctors do use the word “causation” and do so “to refer to the reason a specific patient succumbed to a particular illness.”⁴³³ Dorland’s Medical Dictionary defines “a cause” as “that which brings about any condition or produces any effect.”⁴³⁴ Thus, in medicine, “cause” is used the same way it is in law. Qualifying terms, such as “exciting cause” (that leads directly to a specific condition) or “immediate cause”—or even “proximate cause”—are also available in medicine.⁴³⁵ The term “etiology,” Armenian states, is something the medical profession uses when it talks about the process in a general population.⁴³⁶ *Dorland’s Medical Dictionary* defines “etiology” as “the study or theory of the factors that cause disease and the method of their introduction into the host,”⁴³⁷ an epistemological concept that has no bearing on causal proof or medical diagnosis, although, perhaps, it may be relevant in epidemiology.

Fisher, the social scientist, wades into this linguistic menagerie,⁴³⁸ erroneously noting that for the purposes of establishing cause or etiology, epidemiology “is generally the initial step in a health-related environmental risk assessment and favored to establish legal causation.”⁴³⁹

In sum, Professors Doll and Gordis on one hand, and Professors Lilienfeld, Armenian, Shapiro, Katz, and Shai Linn, on the other, clearly see epidemiology through different lenses. The latter group suggest that epidemiology might be suited to inform specific causation

430. GREEN, FREEDMAN & GORDIS, *supra* note 334, at 443.

431. *See generally* Fraser v. 310-52 Townhouse Corp., 57 A.D.3d 416 (N.Y. App. Div. 2008) (differentiating association from causation).

432. GREEN, FREEDMAN & GORDIS, *supra* note 334, at 443-44.

433. ARMENIAN & SHAPIRO, *supra* note 386, at 111.

434. *Cause*, DORLAND’S MEDICAL DICTIONARY 285 (1988).

435. *Id.*

436. *Id.*

437. *Etiology*, DORLAND’S MEDICAL DICTIONARY 587 (1988).

438. *See* KOEHLER & BROWN, *supra* note 405, at 6 (“Epidemiological studies can never prove causation; that is, it cannot prove that a specific risk factor actually causes the disease being studied. Epidemiological evidence can only show that this risk factor is associated (correlated) with a higher incidence of disease in the population exposed to that risk factor. The higher the correlation the more certain the association, but it cannot prove the causation.”)

439. FISHER, *supra* note 238, at 147. *But see* Fraser, 57 A.D.3d at 416 (holding, in a mold case, that an “association” is not equivalent to “causation”).

under certain conditions. The former approve it to only establish some loose general “association,” legally translated into general causation. As to requiring conventional biostatistical references of reliability, i.e., confidence intervals,⁴⁴⁰ p-values, and statistical significance, that, too, depends on whom you ask.

I. THE POETICS OF MEDICINE

Although scant attention is paid to poetics in public-health epidemiology,⁴⁴¹ poetics has generously informed the culture of medicine (and vice-versa).⁴⁴² Contrasting early references to the practice of public health (such as quarantining leprosy patients mentioned in the Bible), the formalized practice of epidemiological research does not really begin until 1700 with Ramazzini’s *De Morbis Artificum Diatriba* (*Diseases of Workers*).⁴⁴³ By contrast, medical research can be dated at least as early as Galen, born in 129 C.E. The 1,700-year head start medicine has over epidemiology endows it with a sort of staying power.⁴⁴⁴ While it has been said that at the end of the day medicine is an “empirical” process, i.e., one does what works,⁴⁴⁵ it endures as a field—precisely because it does work. By contrast, at least some in authority believe that epidemiology does not.⁴⁴⁶

The long history of medicine generates a rich culture,⁴⁴⁷ poignantly and poetically depicted in great works of literature, such as Sinclair Lewis’s *Arrowsmith*, which explored the conflicts between the

440. T Christoffel & S P Teret, *Epidemiology and the Law: Courts and Confidence Intervals*, 81 AM. J. PUB. HEALTH 1661-66 (1991).

441. Notable exceptions are Paul deKruif’s *Microbe Hunters*, a work of fictionalized non-fiction, and Upton Sinclair’s, *The Jungle*, which memorably depicts the intersection of law and public health.

442. See, e.g., GOELLNIGHT, *supra* note 253 (“The reason that the influence of medical science on Keat’s poetry and letters has not been fully documented is quite simply that the literary critics, with a few notable exceptions, lack the necessary medical knowledge to appreciate such influence.”).

443. W. KEITH MORGAN & ANTONY SEATON, OCCUPATIONAL LUNG DISEASES 2-3 (1984). Ramazzini, too, studied disease in “the poor trades.” *Id.* at 2. See Barbara Pfeffer Billauer, *Benjamin Franklin: State Statesman-Scientist and Father of Scientific Statecraft*, PROC. POL’Y STUD. ORG. (2009) (discussing how Benjamin Franklin did the first epidemiological study in America, regarding effectiveness of variolation in “Negroes and Caucasians”).

444. S.C. PANDA, MEDICINE: SCIENCE OR ART? 127-38 (2006) (“Medicine is sometimes considered a science, and sometimes an art; the object of medical science is to study disease.”). See *id.* (concluding that it is both).

445. R.C. LEWONTIN, BIOLOGY AS IDEOLOGY 5 (1991).

446. Zafrir Rinat & Noa Shpigel, *Israel Drops Controversial Study on Links Between Pollution and Cancer in Haifa Area*, HAARETZ (Jun 25, 2017), <https://www.haaretz.com/israel-news/premium-1.797814>.

447. See generally John Saunders, *The Practice of Clinical Medicine as an Art and as a Science*, 174 WESTERN J. MED. 137-41 (2001) (noting medicine is an applied science that is defined as pure science applied to a particular set of problems, a distinction first made in 1620).

medical practitioner and researcher, and A.J. Cronin's *The Citadel*. The poetry of Keats,⁴⁴⁸ who was trained as a doctor, adds to the treasury of medical-poetics literature and ratifies the importance of the narrative in the practice of medicine: "Through the study of narrative, the physician can better understand patients' stories of sickness and his or her own personal stake in medical practice."⁴⁴⁹ The medical history, a manner eliciting the medical narrative, informs the diagnosis, even providing good exposure evidence if carefully taken, although perhaps not appreciated in the legal arena.

The dichotomy of culture, approach, and mindset between epidemiologist-researcher and medical practitioner can be summed up as follows:

[T]he academic investigator and the bedside practitioner arrive at their callings through different routes of socialization and indoctrination. For both groups, a principle problem is uncertainty, but . . . medical scientists turn to formal tests to control for confounding variables in efforts to distinguish between rival hypotheses. On the other hand, clinicians generally proceed inductively: they are preoccupied with the puzzles of individual cases, and with generating *experience-based post-hoc hypotheses*. The practitioner's calling demands closure [like the law] and uncertainty is resolved through action, diagnosis and treatment. Science is abstract and open-ended . . . [The medical] practice is concrete and forces closure,⁴⁵⁰ [much like law].

J. LANGUAGE: DIFFERENTIAL DIAGNOSIS AND DIFFERENTIAL ETIOLOGY

The disconnects between clinical medicine and law balloons in the terms "differential diagnosis"—the traditional reasoning doctors use to identify possible medical causes of a patient's condition—and "differential etiology,"⁴⁵¹ a non-medical term that courts (not doctors) use to characterize medical causation, reflecting the linguistic discord and cultural cacophony. The *Federal Manual* authors admit to their linguistic sleight of hand:

Courts have come to use certain medical terms such as *differential diagnosis* and *differential etiology* in ways that differ

448. GOELLNIGHT, *supra* note 253.

449. R. Charon et al, *Literature and Medicine: Contributions to Clinical Practice*, in 122 ANNALS INTERNAL MED. 599-606 (1995).

450. SILVERMAN, *supra* note 393, at 91 (emphasis in original).

451. See *Westberry v. Gummi*, 178 F.3d 257 (4th Cir. 1999). To validate this malapropism, the *Federal Manual* authors leapfrog to the conclusion that "differential etiology' more closely suggests determination of [legal] cause." GREEN, FREEDMAN & GORDIS, *supra* note 334, at 443-44.

from their common usage in the medical profession. For example, although environmental and occupational health physicians may use the term “differential diagnosis” to include the process of determining whether an environmental or occupational exposure caused the patient’s disease, most physicians use the term to describe the process of determining which of several diseases caused the patient’s symptoms.⁴⁵²

“Differential etiology,” then, is legal jargon concocted to refer to extrinsic causes of disease, something the *Federal Manual* authors incorrectly assume is of no concern to physicians. In fact, doctors are indeed interested in extrinsic causes, although only those pertaining to the patient (specific causation, anyone?). As Armenian says, “[w]ithin the medicolegal environment of liability cases, we are interested in assessing causation at the individual level rather than demonstrating a general etiologic relation between disease and exposure.”⁴⁵³

Armenian illustrates the importance of ascertaining the “cause” in a medical context. In one case, the patient presented with allergic rhinitis and diagnosed with angioedema (gross swelling) of her eyelids, lips, and tongue. She was treated symptomatically to reduce the life-threatening condition—and then the search began as to the cause: was it a seasonal allergy? A genetic condition? Or something more insidious? Further medical investigation revealed the “cause” to be a severe allergy to pine nuts, elicited from the history that revealed the temporal onset immediately after consuming hors d’oeuvres at a Christmas party.⁴⁵⁴

Although extrinsic factors can be vital to making a diagnosis⁴⁵⁵ and certainly influence treatment, the *Federal Manual* takes it upon themselves to tell treating physician-experts how to practice medicine: “An expert’s opinion on diagnosis and his or her opinion on external causation should generally be assessed separately, since the basis for such opinions are often quite different.”⁴⁵⁶

452. TERRENCE F. KIELY, SCIENCE AND LITIGATION: PRODUCTS LIABILITY IN THEORY AND PRACTICE 146 (2d ed. 2002) (citing FEDERAL REFERENCE MANUAL ON SCIENTIFIC EVIDENCE, MARY SUE HENIFER ET AL., *Reference Guide on Medical Testimony*, at 443-44 (2d ed.)). The entire paragraph as written in the *Federal Manual* is medically imprecise: “Symptoms” refers to only subjective complaints made by a patient; they are unverifiable and often not repeatable. Presumably, the authors meant to include “signs” that are objective manifestations of illness, e.g., pulse, blood pressure and temperature, and laboratory test results.

453. ARMENIAN & SHAPIRO, *supra* note 386, at 111.

454. *Id.* at 122-23.

455. Revised U.S. surveillance case definitions (diagnostic criteria) often include a history of exposure to extrinsic factors.

456. GREEN, FREEDMAN & GORDIS, *supra* note 334, at 443 n.160.

VI. AN ANALYSIS OF THE CAUSAL CONUNDRUM

In sum, the conundra presented at the outset can be grouped into three categories:

- Expert-determinativeness and failure to recognize the different cultures and practices of different fields and sub-field;⁴⁵⁷
- Misunderstanding the pathology of disease and ‘etiology’; and
- The tortured linguistics of general and specific causation

Recall that in the *DuPont C-8 cases*, six medically unrelated conditions were determined to be linked to the same chemical. In the *Kishon Cases*, the courts ruled this was a medical impossibility; in *Milward*,⁴⁵⁸ the court found general causation *did* exist for benzene; in the *Kishon Cases*, it ruled that it did not. In the *DuPont C-8 cases*, the linguistic divide was between plaintiffs and defense lawyers; in the *Kishon Cases*, a cultural schism divided science and the court.

The cases illustrate that lawyers, judges, and experts all have different understandings of various words, pointedly “specific” and “general,” when used in concert with the word “causation.” It is this tangled web of words and notions, these linguistic acrobatics and clashing cultures, that generate the science-law disconnect and must be unraveled before progress can be made in providing a framework to resolve problems in proving legal causation.

A. THE CULTURE OF MEDICAL PRACTICE: DR. BUTLER’S APPROACH

Notwithstanding references to required quantitative proof,⁴⁵⁹ “the practice of medicine is one of judgment, not mathematics.”⁴⁶⁰ Yet the court in *Milward*⁴⁶¹ views Butler as an ersatz epidemiologist who did not comply with the norms of epidemiology—as practiced by epide-

457. See *In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 758 (3d Cir. 1994), *cert. denied*, 513 U.S. 1190 (1995) see also *Kannakeril v. Terminix Int’l, Inc.*, 128 F.3d 802, 807 (10th Cir. 1997) (holding “differential diagnosis” is a “a technique that involves assessing causation with respect to a particular individual”).

458. 664 F. Supp. 2d 137 (D. Mass. 2009).

459. See, e.g., the *Kishon Cases*.

460. See SANDERS, *supra* note 299, at 137-41. Sanders states:

Good physicians use their personal judgment to affirm what they think to be true in a particular situation. Their knowledge is not purely subjective, for they cannot believe just anything, and their judgment is made responsibly and with universal intent—that is, they take it that anyone in the same position should concur. It is practical wisdom. Medical practice demands such judgments on a daily basis. The good doctor is able to reflect on diverse evidence and to apply it in a particular context. No computer could replace him or her, for the judgment cannot be reached by logic alone. Here medical practice as art and science merge.

Id. See generally PANDA, *supra* note 444; see also KATHRYN MONTGOMERY, *HOW DOCTORS THINK: CLINICAL JUDGMENT AND THE PRACTICE OF MEDICINE* (2005).

461. 664 F. Supp. 2d 137 (D. Mass. 2009).

miologists,⁴⁶² rather than as a clinician⁴⁶³ who uses epidemiology selectively⁴⁶⁴ to inform diagnoses and treatment of her specific patient's illness in the customary fashion of clinicians, a view supported by Armenian.⁴⁶⁵

As to Butler's differential diagnosis, the court looked at this, too, in a quantitative light.⁴⁶⁶ In what can only be called legal *chutzpah*, the law crafts a medical nonsense term, "differential etiology," and then says clinicians cannot use this diagnostic technique because too many causes are unknown. But as Butler said, "[f]or an epidemiologist, if there is no known cause—the disease is idiopathic; for a clinician, [e]very case of leukemia has some cause . . . [,] and only [t]hose cases with unidentified causes get hit with the 'idiopathic' tag."⁴⁶⁷

Let's now look at this case in real-life terms. The incidence of APL⁴⁶⁸ is five to ten out of 25,000,000 people.⁴⁶⁹ We know an underlying genetic malformation is responsible for the condition.⁴⁷⁰ As to what triggers its expression, we only know three associated risk factors, two of which were ruled out, benzene exposure being the third. We obviously don't know the seven unknown causes. We also have a

462. See *Milward v. Acuity Specialty Prods. Grp., Inc.*, 639 F.3d 11, 17-19 (1st Cir. 2011) (citing *Daubert v. Merrill Dow Pharm., Inc.*, 509 U.S. 579, 590 (1993)) (discussing how the purpose of *Daubert* is to ensure an expert opinion maintains "the same level of intellectual rigor that characterizes the practice of an expert in the relevant field").

463. DAVID L. SACKETT ET AL., CLINICAL EPIDEMIOLOGY: A BASIC SCIENCE FOR CLINICAL MEDICINE 295, 300 (1985).

464. N.J. WALD & R. DOLL, INTERPRETING NEGATIVE EPIDEMIOLOGICAL EVIDENCE FOR CARCINOGENICITY 7 (1985) ("Negative human evidence may mean very little, unless it relates to prolonged and heavy exposure.").

465. ARMENIAN & SHAPIRO, *supra* note 386, at 117-18. The authors provide:

The [medical] case investigation does not aim at establishing all possible etiologies for the condition; our primary objective is to explain the outcome in the particular individual . . . and the inferences we make refer primarily to an explanation pertaining to the individual. We are not necessarily aiming at explaining all phenomena of a similar nature.

Id. Further, it should be noted that "skimming" medical literature is an acceptable practice in clinical medicine. See SILVERMAN, *supra* note 393, at 102 ("The glut of medical literature resulted in a methodology (or set of guidelines) advising doctors how to keep abreast of medical literature."). A selective pragmatic method advised to address the "hopeless problem of trying to read everything" was produced by clinical epidemiologists at McMaster University.

466. See *Ruggiero v. Warner Lampert*, 424 F.3d 249, 254 (2d Cir. 2005) (determining differential diagnosis cannot prove general causation); *Glastetter v. Novartis Pharm. Corp.*, 252 F.3d 986, 989 (8th Cir. 2001).

467. *Milward v. Rust-Oleum Corp.*, 820 F.3d 469, 478 (1st Cir. 2016).

468. C.C. Coombs, M. Tavakkoli, & M. S. Tallman, *Acute Promyelocytic Leukemia: Where Did We Start, Where are We Now, and the Future*, 5 BLOOD CANCER J. 1 (2015).

469. *Acute Promyelocytic Leukemia Facts, Cancer Stat Facts 2014-2015*, LEUKEMIA & LYMPHOMA SOC'Y, https://www.lls.org/sites/default/files/National/USA/Pdf/Publications/FS26_APL_FactSheet.pdf (last visited Jan. 13, 2018).

470. APL is caused by a specific chromosome translocation. *Acute Promyelocytic Leukemia (APL)*, RUSH U. MED. CTR., <https://www.rush.edu/services/conditions/acute-promyelocytic-leukemia-apl> (last visited Jan. 13, 2018).

patient sitting before us, who just happens to have had thirty years of high-level benzene exposure. I suggest a reasonable clinician would conclude that, more probably than not, this patient's very rare disease came from the benzene.

Let's further assume the patient presented with a rare disease associated with lead exposure, but also associated with other causes, some known, some not. In performing her differential diagnosis, the doctor can rule out known causes. As to unknown causes, these, of course, she can't. Assume further the patient had a thirty-year exposure to lead. The doctor can now treat the patient as if his substantial exposure to lead caused the problem and begin chelation therapy, or conclude that since we don't know seventy to eighty percent of the causes of this condition, the doctor should do nothing.⁴⁷¹ In that case, I suggest the doctor would be guilty of malpractice.

To be sure, there are serious scientific problems with Butler's position, discussed at length in Michael Hoenig's excellent article, "When Experts Cherry Pick Studies."⁴⁷² Among them, her subscription to the "there is no safe level of exposure" school of carcinogenesis (an unproven scientific "belief" that initially caused her to reject studies at odds with her position). Had the court based its rejection on this, it might have been a cogent and legally valid decision. But it did not. Instead, it faulted her because her techniques comported with standard epidemiological standards.

B. PUTTING THE PIECES TOGETHER: GENERAL AND SPECIAL CAUSATION

The definitions of general causation and specific causation⁴⁷³ take the guesswork out of figuring out the meaning—one would think. And yet, as noted earlier, DuPont's lawyers in the C-8 cases claimed they did not mean what the plain words suggest they meant. But on reflection, the phrase might well be ambiguous. Does it mean:

- Can the substance cause disease in theory, because of its biological makeup? Or is mathematical certainty (or statistical significance) required?
- Can the substance cause disease in animals that serve as acceptable human surrogates?
- Can the substance cause disease in small doses?

471. See RICHARD ROYALL, *STATISTICAL EVIDENCE: A LIKELIHOOD PARADIGM* 1-3 (1997).

472. Michael Hoenig, *When Experts 'Cherry-Pick' Among Competing Studies*, N.Y. L.J., (May 9, 2016, 3:00 AM), <https://www.law.com/newyorklawjournal/almID/1202757013908/>.

473. GREEN, FREEDMAN & GORDIS, *supra* note 334, at 444.

- Can the substance cause any cancer, or just the cancer complained by the plaintiff?
- Does general exposure include levels at which the plaintiff was exposed, as both the *DuPont* and the *Kishon* cases suggest?

One case defined both general and specific causation as bearing on whether *the type of injury at issue can be caused or exacerbated*⁴⁷⁴ by the substance. If Mr. Milward's cancer was triggered by one of the seven unknown causes, is it not a fair inference to say the benzene exposure at least exacerbated that cause? The customary practice in occupational medicine would so opine. The epidemiologist, however, cannot measure that proposed exacerbation—and so says it cannot. The issue is who decides? Is this to be a consensus opinion? Made by epidemiologists? Medical doctors? Toxicologists? Oncologists? International scientific agencies? Regulatory agencies? And specifically regarding epidemiology—what “sub-field” are we talking about? Public-health epidemiologists? Clinical epidemiologists? Hospital epidemiologists? Statisticians without medical degrees?⁴⁷⁵

One might argue that if an administrative agency is regulating a substance, that agency has concluded the substance *can*—at least under certain circumstances—cause disease. But the *Parker*⁴⁷⁶ court's rejection of agency determinations does not buy this. And neither does the *Kishon* courts' rejection of the IARC determination. Thus, rather than a uniform scientific conclusion that a substance *can* cause cancer (or some other disease), the case-by-case determination generates an untenable divide regarding scientific carcinogenicity.

I suggest that if a substance is characterized as probably carcinogenic by a reputable and neutral scientific organization, or regulated by a national environmental agency, general causation is established and the issue of sufficient exposure should be shunted to specific causation. After all, if there is not some basic level of evidence to warrant regulation, why would the substance be regulated in the first place?

But what if no reputable agency, regulatory or scientific, has so concluded, as in the case of a new substance or pharmaceutical? The cases seem to suggest that a “lone warrior's” view, not substantiated

474. *Wade-Greux v. Whitehall Labs., Inc.*, 874 F. Supp. 1441 (D.V.I. 1994), *aff'd*, 46 F.3d 1120 (3d Cir. 1994).

475. *See, e.g.*, DAVID A. FREEDMAN, *STATISTICAL MODELS: THEORY AND PRACTICE* (2005); *see also* DAVID A. FREEDMAN, *ON TYPES OF SCIENTIFIC ENQUIRY: THE ROLE OF QUALITATIVE REASONING* (2008). Freedman is a statistician who wrote at length about *Manko v. United States*, 636 F. Supp. 1419 (W.D. Mo. 1987).

476. 857 N.E.2d 1114 (N.Y. 2006).

by consensus, would not be admissible. Of course, that is not what *Daubert* says,⁴⁷⁷ but nonetheless, that is what courts do.

The solution to admissibility of “frontier-expert testimony” is simply to follow *Daubert*:⁴⁷⁸ if that expert can show a reliable and valid methodology, the testimony should be admitted.⁴⁷⁹ But, this claim begs the question, in pioneering “science” who makes the determination that the “novel” method is reliable?⁴⁸⁰ Other like-minded authorities?⁴⁸¹

Exactly what birthed the double requirement of specific and general causation is not clear. Golan asserts it was to accommodate the public health ideology of tort law (i.e., but/for causation).⁴⁸² But the practice may have outlived its usefulness. At the end of the day, the underlying pertinent *legal* question of general causation becomes a policy issue. How broadly do we want to construe the term to afford the plaintiff a chance to be heard? Not until courts resolve this policy issue can the remaining questions be answered.

Similar questions present themselves when determining what is encompassed under “specific causation”:

- If proof of general causation has already been proven, is it necessary to revisit the matter in every case alleging exposure to that chemical?
- What if the bulk of causes have not yet been identified—is there some quanta of unknown causes that must be ruled out before a differential diagnosis is accepted?

C. HOW DID IT ALL COME ABOUT AND WHAT IS IT ALL FOR?

Oliver Wendell Holmes noted:

[A] consideration of the earliest instances will show . . . that vengeance, not compensation . . . was the original object [of tort law] . . . ; that the various forms of liability known to

477. See *Amorgianos v. Nat'l R.R. Passenger Corp.*, 303 F.3d 256, 267 (2d Cir. 2002) (discussing how *Daubert* attempts to strike a balance between a liberal admissibility standard for relevant evidence on the one hand and the need to exclude misleading “junk science” on the other).

478. The *Daubert* court reminded us that “expert opinion based on a methodology that diverges ‘significantly from the procedures accepted by recognized authorities in the field . . . cannot be shown to be “generally accepted as a reliable technique.”” *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 584 (1993) (quoting *Daubert v. Merrell Dow Pharm., Inc.*, 951 F.2d 1128, 1129 (9th Cir. 1991)).

479. *Bayer ex rel. Petrucelli v. Dobbins*, 885 N.W.2d 173, 180 (Wis. Ct. App. 2016).

480. *Merrell Dow Pharm., Inc. v. Havner*, 953 S.W.2d 706, 719 (Tex. 1997).

481. See Billauer, *supra* note 176 (discussing a history of good science rejected by fellow scientists because of its “novelty”).

482. *Science and Law*, *supra* note 356, at 163-83. *History of Epidemiological Evidence*, *supra* note 356. GOLAN, LAWS OF MAN, *supra* note 356. *But see* Schactman, *supra* note 357 (a brilliant essay on Golan’s opinions).

modern law spring from the common ground of revenge
It shows that they have started from a moral basis, from the
thought that someone was to blame.⁴⁸³

Holmes is not content to rest on the past as arbiters of present
and future normative conduct, lamenting that:

[P]recedents survive in the law long after the use they once
served is at an end the reason for them has been forgotten.
The result of following them must often be failure and confu-
sion from the merely logical point of view Every impor-
tant principle which is developed by litigation is in fact and at
bottom the result of definitely understood views of public
policy.⁴⁸⁴

Indeed, public policy has drifted toward compensation, redress or
cure, often in a no-fault context. Especially in cases of indeterminate
liability, Professors Porat and Stein note the focus now rests “on two
principal theories of tort liability: that of deterrence and that of correc-
tive justice”⁴⁸⁵ Corrective justice, per Porat and Stein, highlights
doing justice to the victim, while retributive justice (or blame) empha-
sizes doing justice to the wrongdoer. In corrective justice, or what we
might call “curative justice,” “[t]he victim is compensated for the dam-
age that he or she wrongfully sustained, and the wrongdoer is forced
to make good the damage by compensating the victim.”⁴⁸⁶

Compensation can be seen as the closest thing the law has to “cur-
ing” the victim. Where the operative “question is seen as causation,
not blame . . . [,] this implicitly suggests that cure, rather than re-
venge, can be the aim.”⁴⁸⁷ Revenge, according to White, is the product
of blame, while determining how the injury can be healed (cure) is a
superior societal objective.⁴⁸⁸

Recall that the cultural imperative of public health epidemiology
is to identify (and remove) a specific causal agent to prevent future
harm. Nuanced, it is akin to “blaming” an agent for society’s illnesses.
By contrast, clinical medicine attends to a patient (or plaintiff) who
has already been injured; the doctor seeks to *cure* the patient—a far
more consonant approach with modern jurisprudence than current
case law would admit.⁴⁸⁹

483. HOLMES, *supra* note 124, at 22.

484. *Id.*

485. ARIEL PORAT & ALEX STEIN, TORT LIABILITY UNDER UNCERTAINTY 11 (2001).

486. *Id.* at 12.

487. WHITE, *supra* note 117, at 18 (“To stop the obsessive (if understandable) pro-
cess of blaming and excusing, free[s] the mind to think how the wound can be
healed”).

488. *Id.*

489. Oliphant, *supra* note 148, at 152. In Scandinavia, for example, tort law was
translated into a legal science that rejected the ideas of blame, interpersonal justice,

In that clinical medicine (whose focus is on individual causation) is inherently intertwined with epidemiology,⁴⁹⁰ clinical epidemiologists would certainly opine that epidemiology can inform specific causation.⁴⁹¹

VII. CONCLUSION

Perhaps it can be said that “public health” is concerned with “general causation” (more accurately causal associations), while clinical medicine is concerned with specific causation. While currently courts look to epidemiology as defined in the public-health context, “[s]olving problems in public health is fundamentally different from medical diagnosis and treatment,”⁴⁹² something courts have yet to fully realize.

Thus, pediatrician William A. Silverman suggests that medicine is far closer in its objectives to law than epidemiology could ever hope to be.⁴⁹³ In this context, an occupational medicine physician, familiar with the general literature of the field, although not versed in the epidemiological minutiae, may be the best suited to resolving specific causation.⁴⁹⁴ Armenian probably would concur.⁴⁹⁵

I also propose that epidemiology as used in clinical practice is far more ingrained and widespread than currently believed,⁴⁹⁶ a subject that requires further exploration. Nevertheless, should this hypothesis be sustained, courts may not be as skittish in their view of clinicians and clinical testimony—even if not utilized according to the standards of epidemiological practice—to prove specific causation.

However, forging answers to modern causation questions will require a multi-causal approach, something to which society is culturally averse, and statistics has yet to fully address. The legal impact of susceptibility genes⁴⁹⁷ also must be evaluated. Plaintiffs predisposed to disease because of a genetic malformation should not garner for the

and rights. Although the traditional tort language was retained, the decisive question became “who ought to bear the risk?” rather than who was at fault. *Id.*

490. See CDC use of epidemiology to establish diagnostic criteria, case-definitions, treatment and prognosis, an in-depth discussion, which is outside the scope of this Article.

491. Most authorities say at least two studies reaching statistical significance are required before causal connection is demonstrated. David Barnes, *Too Many Probabilities: Statistical Evidence of Tort Causation*, 64 L. & CONTEMP. PROBS. 236 (2001).

492. ARMENIAN & SHAPIRO, *supra* note 386, at 15.

493. SILVERMAN, *supra* note 393, at 91.

494. There are difficulties with this approach. For one, it could jeopardize the doctor-patient relationship if the doctor refuses to testify.

495. ARMENIAN & SHAPIRO, *supra* note 386, at 111 (citing A.S. Evans, *Causation and Disease: A Chronological Journey*, 5 AM. J. EPIDEMIOLOGY 249-58 (1994)).

496. CDC, PRINCIPLES OF EPIDEMIOLOGY IN PUBLIC HEALTH PRACTICE 1 (3d ed. 2006).

497. Richard Doll, *An Epidemiological Perspective of the Biology of Cancer*, 93 CANCER RES. 963 (2005).

defendant a “get out of jail free card.” In these cases, where the plaintiff has developed a disease associated with a known pathogen or carcinogen, if the plaintiff can establish certain exposure—even if below that required to cause disease in the general population, we can assume we are dealing with the modern-day “egg-shell plaintiff” and that person should recover. Such an approach would avoid the travesty of manipulating science or tort law to redress the wrong accruing “when the law has not caught up with science.”⁴⁹⁸

Perhaps the most pragmatic solution comes from Lilienfeld:

In medicine and public health, it would appear reasonable to adopt a pragmatic concept of causality. A causal relationship would be recognized to exist whenever evidence indicates that etiologic factors for a part of the complex of circumstances that increases the probability of the occurrence of disease and that a diminution of one or more of these factors decreases the frequency of that disease.⁴⁹⁹

In summary, this research presented an innovative methodology, that of poetics and social epistemology, to examine conflicts in causal proof. Based on this methodology, the research further suggests that the current requirement of proving both “specific causation” and general causation is unnecessary, duplicative, and unnecessarily onerous. Further, I suggest that, as the law recognizes its current limitations, relegating general causal proof to policy determinativeness is warranted. Finally, a new conceptual mode of assessment is necessary. As Professor Almog says:

Since science has begun to distrust general explanations and solutions . . . the grand challenge for literature [and law] is to be capable of weaving together the various branches of knowledge, the various “codes” into a manifold and multifaceted vision of the world.⁵⁰⁰

498. See *Collins v. Xytex Corp.*, No. A16A1139 (Ga. Ct. App. filed Mar. 4, 2016).

499. LILIENFELD, *supra* note 339, at 13, 252.

500. ITALIO CALVINO, SIX MEMOS FOR THE NEXT MILLENIUM 66 (1988), http://media.roportal.ro/forum/uploads/monthly_09_2011/post-133160-0-64778800-1316969107.ipb. See also Almog, *supra* note 120.

CODA:

*“So they were left in the middle of Australia, Old Man Kangaroo and Yellow-Dog Dingo, and each said, ‘That’s your fault.’”*⁵⁰¹

The purpose of tort law, of course, is to prove liability—including causation. In today’s toxic tort legal climate the plaintiff tells the defendant, “it is your fault” and the defendant answers, “you cannot prove it was not something else”—your genes or some risky lifestyle choice you made. In other words, “maybe it is *your* fault.”

Whose approach is better? Old-Man Kangaroo and Yellow-Dog Dingo could not have done a better job of pointing fingers than the clinician and the law’s embrace of public-health epidemiology.

501. KIPLING, *supra* note 2.

