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Articles

*479 GENERAL CAUSATION AT A CROSSROADS IN TOXIC TORT CASES

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I. Introduction

The traditional tort elements in a negligence cause of action are duty, breach of duty, proximate causation, and injury. [FN1] Each of these terms is infused with legal meaning, and policy considerations typically govern their construction. The emergence of a toxic torts jurisprudence, from out of the larger domain of the law of civil wrongs, has helped accentuate the policy-sensitive nature of the conventional tort elements.

A sensitivity to policy breeds controversy. In the toxic tort cases and legal scholarship, most of the buzz has been on the concept of proximate causation. This Article focuses on the issue of actual causation, the factual aspect of the larger proximate causation inquiry. More specifically, the normative question is what standard ought to govern actual causation determinations in toxic tort litigations. To illustrate the reformist tide, one influential view has been that the causation element should be abolished altogether in actions alleging exposure and harm coinciding with the corporate distribution of toxic products. [FN2] Another has been that a general statistical showing of *480 enhanced risk that appears to satisfy the more-likely-than-not legal standard should alone suffice to establish actual cause. [FN3]

This Article rejects the first proposal, critiques the second, and ultimately sides with a variant of the new statistical approach to causation in toxic tort cases. Toward these ends, Part II briefly discusses the factors that motivate the paradigm set forth by those who would abolish causation (the "abolitionists") in this context. The abolitionists both presuppose the very general causation finding that their proposal inveighs against, and depreciate the current system's ability to pin liability on distributors of toxic products. In the final analysis, their proposed solution would be unstable.

Part III discusses the alternative articulation by some jurists of an epidemiological substitute for specific causation proofs. The primary focus will be on judicial treatment of the notion that proof of a two-fold, or 2.0, epidemiological increase in relative risk may satisfy the civil more-likely-than-not burden of persuasion. This Part also suggests that differential diagnoses--which eliminate competing causal explanations *481 in a particular case--should emerge as the primary mechanism by which bonus probabilities may be assigned to those epidemiological proofs that indicate less than a two-fold relative risk ratio.

Part IV then evaluates the practical efficacy of a bright-line legal assessment of epidemiological evidence. When judges create or refine legal standards, the pull is naturally in the direction of bright lines that both secure the present decision and supply guidance for future decision making. The two-fold relative risk finding in epidemiological studies provides such a bright line, but the probativeness of such an outcome in vacuo may be illusory. For example, validity threats may plague epidemiological findings that otherwise point to the powerful likelihood of a causal connection; by the same token, ideal studies indicating even a minor relative risk increase may be significantly probative in conjunction with the case-specific elimination of confounding factors.

Finally, Part V addresses the theoretical issue of whether epidemiological evidence--even assuming an ideal study free of validity threats--should be admitted in legal cases at all, either as the sole evidence of causation when they establish a greater than 2.0 relative risk factor, or in conjunction with case-specific proof when they do not. This Article takes the position that

(Cite as: 108 Penn St. L. Rev. 479)

the epidemiological evidence should be deemed admissible, both to address the abolitionists' concerns, and because law and society would likely consider the resulting evidentiary mechanism to be legitimate.

II. Beyond the Abolitionist Program

The "abolitionists"--this Article's term for those who propose to abolish the causation element of proof in toxic tort lawsuits--lament that, in toxic tort litigations, unlike traditional tort cases, causality is profoundly elusive both because biology does not afford clear and distinct explanations of the causal mechanisms by which toxic exposures produce birth defects, cancers, and other diseases, and because such medical problems are not usually traceable back to any one particular source. [\[FN4\]](#) A conventional demonstration of "but for" causation is therefore impossible. [\[FN5\]](#)

Under the noncausal model proposed by Margaret Berger, a professor at the Brooklyn Law School--upon which model the abolitionist program is based--courts would impose liability in negligence for failure to provide adequate information relating to product risks, and proof that the failure had caused plaintiff's injury would not be *482 required. [\[FN6\]](#) Berger's later qualifications, however, suggest that her model ultimately remains a causal one, and that her model's corrective work is accomplished merely through a burden-shifting mechanism. [\[FN7\]](#) Consequently, says Berger, defendants should be given the opportunity to prove that exposure to their product could not plausibly have resulted in certain adverse health reactions, or should be entitled to a reduction in damages if they are able to establish that some other causal factor, such as smoking, contributed to the particular plaintiff's injury. [\[FN8\]](#)

In other respects, however, the causal presuppositions that course through Berger's model are less obvious. She proposes, for instance, that "[i]f a corporation fails to exercise the appropriate level of due care, it should be held liable to those put at risk by its action, without regard to injuries that eventually ensue." [\[FN9\]](#) By this language, Berger presupposes that a corporation's liability under her model will be predicated on some minimal finding that there are risks associated with its product. The corporation, moreover, will be held liable to those "put at risk." [\[FN10\]](#)

Requisite to any such finding of risk, however, is some showing of a product's general capability of causing harm. [\[FN11\]](#) Accordingly, when examined closely, it becomes clear that Berger's model presupposes that the finding of a general causal capability is a precondition to the imposition of liability. Thus, by assuming the existence of the very factual element that the abolitionist paradigm is committed to eliminating, that paradigm is rendered incoherent.

Berger's proposal is also deficient in a couple of other important respects. First, Berger claims a moral impetus for her model, which stems from the imperative of providing injured plaintiffs with "equitable compensation." [\[FN12\]](#) She criticizes the requirement that plaintiffs prove actual causation on this ground, citing the "enormous transaction costs" that this requirement may entail. [\[FN13\]](#) But in the final analysis, Berger's model admittedly also fails to provide plaintiffs with "a full measure of *483 traditional damages." [\[FN14\]](#) Berger replaces transaction costs with trade-off costs, conceding that "[i]n exchange for relieving plaintiffs of having to prove general causation, a possible fair trade-off might be to release defendants from having to pay for plaintiffs' pain and suffering," as well as punitive damages, and to provide them with a defendant-favorable method of damage scheduling. [\[FN15\]](#) It is not immediately apparent that transaction costs would ordinarily exceed the value of such trade-off concessions, and it seems reasonable to suppose that, indeed, the latter would often far surpass the former.

As a further difficulty, Berger proposes that, under the noncausal scenario, a corporation that distributed toxic substances would not be liable if it could show that it had not violated its duty to inform itself and others about the risks created by its product. [\[FN16\]](#) Yet here Berger curiously brackets the ample skepticism about corporate motivations that otherwise impels her project. For what reason is there to assume that a defendant that would recklessly imperil masses of workers and consumers by burying its head in the sand when it comes to product dangers, would sponsor and interpret research in an unbiased manner under the new noncausal regime?

The abolitionists' impulse is well-intentioned. They believe that traditional tort doctrine gives corporations a substantial incentive to bury their heads in the sand when it comes to the health and safety risks posed by their products "because the future likelihood that a causal connection can be proved between the corporation's conduct and plaintiff's injury appears minimal compared to the cost of present compliance." [\[FN17\]](#) This view, too, may be an overreaction. For one thing, the problem historically has been not so much a failure to suspect a causal link between the toxic substance and health problems,

(Cite as: 108 Penn St. L. Rev. 479)

but rather a negligent, reckless, or willful refusal to disclose this possibility to workers, consumers, or the public. [FN18] In the current causal system, however, if a *484 product's risk or hazard is scientifically discoverable, the court will impute this knowledge constructively to the manufacturer. [FN19]

In the resulting regulatory regime, if any entity investigated the causal risks associated with a substance or product, the corporate manufacturer or seller is deemed to have been privy to the investigator's findings. Moreover, the defendant is under a continuing duty to investigate and warn, and any discoveries that could have been made during the period of distribution or exposure are also attributed to that defendant. [FN20] If neither the scientific, technological, nor industrial communities research the risks associated with a substance, and no risk of a causal nexus is ever framed, it is difficult to comprehend the legal mechanism by which liability could acceptably be imposed. The link between a negligent actor and some harm afflicting a party who happens to have been exposed to that actor's conduct (or product) is literally nothing if not causal. [FN21]

It should be relatively uncontroversial that the state itself may justifiably fine or otherwise punish the sort of negligence the abolitionists identify. But a discrete acausal compensatory subsystem within the larger tort regime would be vulnerable to destabilization by the corporate sector's perennial clamor against the perceived illegitimacy of a new species of strict liability resulting in multimillion, and perhaps *485 multibillion, dollar outcomes. [FN22]

Thus, while some theory-minded legal scholars are understandably engaged by an abolitionist platform that seems both politically progressive and informed by an ethic of care, [FN23] it seems reasonable to assume that the abolitionists' position will not withstand the test of time. The tort system's machinery is likely adaptable enough to incorporate sufficiently pliant standards of causality to address complex and nonobvious causal mechanisms. A more resilient response to the large-scale toxic tort phenomenon preserves the causation element while adjusting in the area of the case-specific evidence that must be adduced. [FN24]

Moreover, even in typical tort cases, proofs of actual causation are not ordinarily rooted in scientifically precise demonstrations of causal mechanisms. In other words, when it comes to the causal element, the divide between the typical tort and the toxic tort scenario may not be as distinct as the abolitionists presuppose. Nor does the cognitive maneuver in a toxic tort case involving little-understood biological mechanisms necessarily depart radically from that in the garden variety tort action. Under either scenario, the fact finder hears of exposure and subsequent harm, and may or may not infer a causal connection. In either event, the precise causal mechanism is unobserved, perhaps unobservable, and the trier infers to the best explanation. [FN25] The philosopher David Hume taught that we cannot induce necessary causal connections from *486 observing mere regularities, [FN26] and the regularities of constant conjunction are all we can observe, even when the Mack truck strikes.

A response to the toxic tort dilemma that should better harmonize with the current flow of tort jurisprudence, and that will likely be more enduring than outright abolition of the causal element of proof, involves a turn toward the sort of statistical approach that is increasingly countenanced in American courts. Indeed, jurists have been moving closer to acceptance of a statistical alternative to traditional causal proofs. The most notable development concerns the use of epidemiological studies to establish specific, and not solely general, causation. The remainder of this Article examines the prospects and problems associated with this development.

III. The Epidemiological Alternative to Specific Causation in Caselaw

A. The Limits of Specific Causation

Plaintiffs in personal injury cases establish the element of specific causation when they show that a defendant's misconduct--or plaintiffs' exposure to the defendant's product--actually caused the plaintiffs' specific injuries. [FN27] In toxic tort litigations, plaintiffs also discretely proffer evidence of general causation, which establishes that the substance they were exposed to is capable of causing the injury or disease with which they are afflicted. [FN28] General causation is automatically established when an actual causal connection has been shown, because something that has caused an effect is necessarily capable of causing that effect. [FN29] But toxic tort litigations ply the two causal levels apart. [FN30]

*487 Although the idea of a second level of causal proof in toxic tort jurisprudence may at first glance suggest a heightened

(Cite as: 108 Penn St. L. Rev. 479)

evidentiary burden for plaintiffs, [\[FN31\]](#) the general causation element functions to justify relaxing the level of specific causation evidence that would otherwise be required. Establishing specific causation under traditional standards would be "oppressively problematic" in the toxic tort area [\[FN32\]](#) because these cases typically involve long latency periods between exposure and illness, as well as disease types that may be associated with multiple causal factors. [\[FN33\]](#) At the same time, the specific causation requirement usually endures in some form, [\[FN34\]](#) and plaintiffs who establish a substance's general harmful propensity may still fail to demonstrate that it probably caused their injury. [\[FN35\]](#)

In some cases, researchers can design and administer controlled scientific experiments to gauge whether a substance is capable of causing a particular condition or injury, and such an experiment could yield objective criteria with which to underwrite the inference of specific causation in a particular case. [\[FN36\]](#) In an increasing variety of toxic tort *488 cases, however, it is not feasible to experiment directly, and there will be little apparent case-specific basis on which to infer specific causation. [\[FN37\]](#)

B. Judicial Development of an Epidemiological Alternative

1. The Early Views

The question arises whether causation may be inferred from general causal evidence that is not case specific. As stated, judges in toxic tort cases have been somewhat open to the idea of a statistical alternative to traditional causal proofs. [\[FN38\]](#) The most notable issue before the courts has been whether epidemiological studies--relevant in determining general causal capabilities--may be used as the sole or predominant evidence of specific causation. [\[FN39\]](#)

An epidemiological study compares the incidence rates of disease among otherwise similarly-situated exposed and unexposed groups. [\[FN40\]](#) In the epidemiologist's discipline, the significant factor is not absolute risk, but relative risk. [\[FN41\]](#) A disease rate across populations, regardless of *489 exposure to any particular substance, yields an absolute risk of contracting that disease. [\[FN42\]](#) The determination of a relative risk, on the other hand, yields the risk among the exposed group relative to the general unexposed population. [\[FN43\]](#)

Perhaps the earliest judicial decision addressing the appropriateness of using epidemiological relative risk findings for determining actual causation was *Cook v. United States*. [\[FN44\]](#) The plaintiffs in *Cook* claimed that their rare neurological disorder, Guillain-Barre Syndrome ("GBS"), had been caused by their federally-sponsored swine flu vaccinations. [\[FN45\]](#) By holding that the plaintiffs' epidemiological evidence was legally insufficient to prove causation because it failed to show a relative risk greater than twice the upper limit of the baseline risk, [\[FN46\]](#) the court implicitly sanctioned the significance of such a two-fold finding.

The *Cook* court reasoned that, whenever the relative risk to vaccinated persons is greater than two times the risk to unvaccinated persons, it is more likely than not--because there is a greater than 50 percent probability--that a given case of GBS afflicting a vaccinee has resulted from that vaccination. [\[FN47\]](#) The court reasoned hypothetically as follows: Suppose the relative risk for vaccines nine weeks after vaccination is two- i.e., that they are twice as likely to experience onset of GBS after that interval as are persons in the unvaccinated population during the calendar week. If fifty GBS cases occur among a million unvaccinated persons that week, then a hundred cases would be expected among a million nine-week vaccinees. Of that hundred, fifty would have been expected without vaccination, while the other fifty are explained only by the event of vaccination. Thus, the likelihood that a given nine-week vaccinated case of GBS is attributable to vaccination is 50%. Similarly, if the relative risk of GBS to nine-week vaccinees is four, then 75% of all nine-week vaccinees are vaccine-linked. Once the relative risk rises above two, it becomes more probable than not that a given case was caused by the vaccine. [\[FN48\]](#)

Other courts followed *Cook* in deeming an epidemiological showing of a relative risk greater than two to be probative on the causation issue. [\[FN49\]](#) *490 Courts facing the issue thus appeared willing to accept an epidemiological substitute for specific causal evidence if that statistical proof established a greater than two-fold relative risk, and thereby satisfied, in the judicial reckoning, the more-likely-than not standard.

Judicial reliance on bright-line standards offers guidance, creates predictability, and engenders a certain comfort level in dealing with technical issues. [\[FN50\]](#) The risk, however, is that a bright-line standard will foster rigid decision making that

(Cite as: 108 Penn St. L. Rev. 479)

may tend to provide false, unfair, unreliable, or otherwise unintended outcomes. [FN51] In the present context, problems arise when the judiciary institutionalizes a two-fold relative risk standard. Most obviously, the legal order then discounts circumstances in which the particular plaintiff has actually been harmed by a substance or in which the epidemiological studies indicate a relative risk of 2.0 or less. [FN52] In such a situation, the bright-line standard would engender what is known as a Type II error, a false negative outcome that denies recovery to a meritorious claimant. [FN53] Strict adherence to the 2.0 standard would also, however, risk Type I error, involving false *491 positives. [FN54] Factually, alternative factors may have caused a particular plaintiff's disease; methodologically, an epidemiological study finding a greater than two-fold relative risk may be flawed. [FN55]

False negative outcomes are especially likely in jurisdictions that view the 2.0 mark as an admissibility standard. This judicial move is understandable, however. Rules of evidence prescribe that only relevant evidence is admissible, [FN56] and that evidence is only relevant if it tends to make the existence of a material fact more probable or less probable than it would be absent the evidence. [FN57] It would appear that a statistical study that supports the conclusion that a causal relation was more likely than not is, for that reason, relevant under the evidentiary standard. Thinking along these lines, a number of courts have deemed the 2.0 relative risk finding to constitute an admissibility threshold. [FN58]

During the early 1990s, courts around the New York area were attempting to hash out the epidemiological issue when non-"signature" diseases arose in the context of the asbestos litigation. [FN59] In the Joint *492 Eastern & Southern Districts Asbestos Litigation, [FN60] for example, the U.S. District Court considered the case of John Maiorana, a former sheet-metal worker who died of colon cancer at the age of forty after having been occupationally exposed to asbestos-containing products. [FN61] As would be expected in such a case, the plaintiff's causal evidence was epidemiological. [FN62] The court concluded, however, that studies showing a merely "elevated" risk of colon cancer for asbestos-exposed individuals were not "admissible" to establish a causal connection because, absent any direct evidence of causation, a plaintiff can meet the "preponderance of the evidence" standard only by showing that asbestos exposure has a relative risk greater than 2.0 for colon cancer. [FN63]

In *Landrigan v. Celotex Corporation*, a New Jersey State appellate court similarly concluded that, where the occupational history of 17,800 insulators exposed to asbestos revealed fifty-nine deaths from colon cancer, in a study population for which thirty-eight deaths were "expected," the relative risk of 1.55 did not sufficiently demonstrate causation. [FN64] The court disconcertedly took the view that an epidemiological study must show a relative risk in excess of two in order to be statistically significant. [FN65]

*493 The Supreme Court of New Jersey overturned the appellate court's reasoning in *Landrigan*. [FN66] The court distinguished the judicial gatekeeping role in determining admissibility from the scientist's role in designing, implementing, and drawing conclusions from epidemiological studies. [FN67] Thus hinting at a contextualist paradigm that accounts for the different epistemic circumstances of the scientific expert and the judiciary, [FN68] the court recognized that lay decision makers could not be expected to know technical and scientific matters in the way experts in *494 their respective disciplines do. [FN69] More specifically, the *Landrigan* panel stated that the court's role is not to describe "how to structure an epidemiological study, analyze the data, draw conclusions about the study population, and, if possible, extrapolate from statistical results inferences about specific individual subjects." [FN70] The court instead assumes the higher order task of evaluating the expert's own explanations of the factual bases and methodologies used in the studies to determine whether the expert's opinion "will assist the trier of fact to understand the evidence or determine a fact in issue." [FN71]

This epistemological posture is incompatible with strict adherence to a judicially-imposed relative risk standard. Instead, any such bright line would appear sensible only if the epidemiological community itself abided by such a marker. Yet the New Jersey high court did not go quite so far as to defer to the scientific or epidemiological community's own judgment about whether the strength, consistency, and plausibility of a statistical association warranted a causal inference in any particular legal case. [FN72] It did not, that is, flatly rule out judicial reliance on a strict 2.0 *495 threshold in the absence of other causal proof, but suggested instead that there will ordinarily be some sort of case-specific evidence, even if simply a record of asbestos exposure or, even better, the clinical presence of asbestos at the tumor site. [FN73]

Under such circumstances, a study showing a relative risk less than 2.0 could support a finding of specific causation, and would be deemed "one piece of evidence, among others, for the court to consider in determining whether the expert has

(Cite as: 108 Penn St. L. Rev. 479)

employed a sound methodology in reaching his or her conclusion." [\[FN74\]](#) The court, in other words, remains focused on its task of determining whether the expert herself possesses the knowledge requisite to arriving at the conclusion; this is a threshold judicial decision that is a function of whether the expert's scientific or other evidence fulfills the court's admissibility criteria. If the expert opines that a causal relationship exists with respect to the particular plaintiff, but relies solely on an epidemiological finding of a less than two-fold relative risk, then perhaps, as a legal matter, the expert's testimony is not "based upon sufficient facts or data" and should be excluded. [\[FN75\]](#)

This articulation followed another oft-cited New Jersey asbestos opinion, *Grassis v. Johns-Manville Corporation*, [\[FN76\]](#) in which the intermediate appellate court rebounded from its clumsy ruling in *Landrigan*. The *Grassis* reasoning merits quoting in some detail:

Defendants argue that there should be a threshold of a 2.0 correlation before an expert should be permitted to rely upon an epidemiological study. They urge that only when this figure is exceeded can it be said that the particular factor is more likely than not to have produced the particular injury. This assertion proves too much. Assuming a large group of potential plaintiffs, a causative factor of 1.99 and significant evidence eliminating other known causes, defendants' proposition would still exclude the epidemiological proof. Even though the physical problems of just under one-half of the plaintiffs (without reference to the additional causative proof) would have been statistically "caused" by the factor being studied, none could recover. Yet, if a new study raised the risk factor to 2.01, all of the plaintiffs could use the study to collect damages, although for nearly one-half of the group, the risk factor was not an actual cause of the condition. *496 This makes little sense, scientifically or legally. [\[FN77\]](#)

Accordingly, the emerging jurisprudential view is that epidemiological studies, which by their nature pertain to general causal capabilities, are good evidence of specific causation, especially when proffered in conjunction with some relevant non-statistical proof. Given such a further evidentiary showing--the sort that is feasible in toxic tort contexts, it should become less pressing upon the judiciary's collective consciousness that epidemiological proofs be governed by a bright line admissibility standard.

2. The Dutch Book Dilemma

It is useful to try to better understand the intuitive draw of the 2.0 relative risk standard for evidentiary gatekeepers in legal cases. On the one hand, any finding greater than 1.0 suggests that exclusion of the causal evidence, or a finding of insufficiency on that element of the case, chances Type II error involving a false negative legal outcome. [\[FN78\]](#) At least some individual, in other words, whose disease did result from the defendant's toxic product, would be denied her day in court.

On the other hand, policy considerations may deem this risk acceptable, and implicitly deem true the null hypothesis stating that a relative risk ratio less than 2.0 demonstrates the lack of any causal association. [\[FN79\]](#) The dilemma resides in the fact that, although some members of the afflicted population--being greater than expected but not greater than twice that number--will have a valid causal claim against a defendant, it is statistically less likely than not that any particular group member will have such a claim. [\[FN80\]](#)

Under such a circumstance, to argue that the causal explanation nevertheless merits submission to the jury is to encounter what is known as a "Dutch book" situation. [\[FN81\]](#) In a Dutch book event, a set of wagers is such that under any circumstance the total pay-off is negative. [\[FN82\]](#) The difficult claim implicitly offered by proponents of the epidemiological evidence in the legal context might be that, even though the statistical probability of causation in any particular case is less than (or equal to) 50 percent, the causal explanation itself imbues the cause of action with *497 bonus probability, permitting a finding that a causal connection is more probable than not.

The philosopher Bas van Fraassen argues against the abductive inferential reasoning process known as "inference to the best explanation" ("IBE") by attempting to show that, in Dutch book style, IBE makes us incoherent. [\[FN83\]](#) A brief review and critique of van Fraassen's approach may engender insights useful in developing a fresh approach to the problem of epidemiological proofs in legal cases.

Van Fraassen begins with an orthodox Bayesian actor, [\[FN84\]](#) personified as "Bayesian Peter," who "uses no ampliative rule,

(Cite as: 108 Penn St. L. Rev. 479)

but only logic." [FN85] Peter and we are given a statistical model for the probabilities that a die is biased based on tossing results. [FN86] The model introduces a factor X of bias, which can come in N different degrees: $X(1), \dots, X(N)$. [FN87] "If the die has bias $X(I)$, then the probability of ace on any one toss equals I/N ." [FN88] Of course, this is not exactly right, because, for one thing, this formula discounts the possibility of ace with an unbiased die. So van Fraassen notes that the model as a whole has a certain bias. [FN89]

In all events, the focus is on the perfect bias, $X(N)$, which gives ace the probability $(N/N) = 1$; the graph van Fraassen provides indicates, for instance, that the posterior probability of perfect bias ($N = 10$) after the first four tosses coming up ace is 39.5 percent, and after the first ten tosses 67 percent. [FN90] These posterior probabilities are updated after further tosses by a "conditionalization" rule. [FN91] Ultimately, such a rule gives rise to the predictive engine so important in testing scientific hypotheses. Thus, under a perfect bias model, the probability of tossing a fifth ace after the first four, once we have correctly weighted the posterior probabilities to arrive at an average, is 87 percent, and of *498 drawing an eleventh, is 95.5 percent. [FN92]

This is all quite abbreviated, and does not do justice to van Fraassen's more extended explication, which is itself sometimes difficult to follow. But let us trust that the field has been prepared for the attack. Peter meets a "Preacher"--preaching his praise of IBE--who convinces him "that the hypotheses of bias are like little or specialized laws, which also have explanatory power," [FN93] and that, in light of this explanatory success, bonus probabilities should be awarded to certain hypotheses. [FN94] Accordingly, after a run of four aces, Peter redistributes his probabilities, now assigning the probability of a fifth ace at 90 percent. [FN95]

Van Fraassen's experiment takes on new complexities, positing a series of bets by which, if Peter relies on the 90 percent estimate, he will be sure to lose come what may, and will even be able antecedently to compute this inevitably on his own. [FN96] "Thus, by adopting the Preacher's rule, Peter has become incoherent--for even by his own lights, he is sabotaging himself." [FN97] If we commit to a rule for the revision of opinion, it should probably entail a Bayesian extension of non-ampliative canons of logic "to all forms of opinion and opinion change." [FN98] Accepting the Preacher's rule that the explanatory aspect of a theory makes its hypotheses more probable makes us incoherent.

It is difficult to grapple with Van Fraassen's Dutch book claim. It probably reacts to such views as Gilbert Harman's (van Fraassen's colleague in the Philosophy Department at Princeton), who said, "The best explanation is more than just a highly probable explanation. It must also make what is to be explained considerably more probable than would the denial of that explanation." [FN99] More recently, Stathis Psillos, a philosopher of science at the University of Athens, said that, by virtue of its confirmational value, the explanatory aspect of a theory raises the theory's probability. [FN100]

Psillos, however, is assuming a context different from the one in which van Fraassen's Preacher operates. Psillos considers a case in which there are, let's say, ten theories, T_1, \dots, T_{10} , each of which explains a single phenomenon e_i ($i = 1, \dots, 10$). [FN101] Assume that we *499 arrive at theory T^* , which unifies $T_1 ? T_{10}$ and is thus more informative than any one of them, or even than their mere conjunction. On strictly probabilistic grounds, the probability of T^* will be less than or equal to the any of the individual probabilities of $T_1 ? T_{10}$. [FN102] But this "does not show that the probability of T^* cannot be high enough to warrant belief Rather, it is some of the features of the potential explanation which, having confirmational value, increase the theory's probability." [FN103]

So perhaps van Fraassen and proponents of IBE are speaking past one another. If so, the character of the Preacher does not fulfill its function of indicting IBE, but merely represents a misapplication of the principle that, in the right circumstances, a theory's likelihood is enhanced by virtue of its explanatory power. Intuitively, anyhow, this would seem to be the case with respect to Psillos's T^* . One example may illustrate the cogency of that model.

Assume that T_1 explains that Stoplight B consistently switches from red to green x t-units after Stoplight A does because the two are linked to one another. T_2 explains that Stoplight C is similarly linked to Stoplight B, and so on through Stoplight Z. Each theory thus explains a single phenomenon and entails a certain singular connection. After a time we posit T^* , which hypothesizes that all of the stoplights are hooked into a grid for the entire city, and are thereby coordinated overall. T^* entails more, and is more informative, than any T_1, \dots, T_n , or even than their conjunction, $T_1 \& \dots \& T_n$. The strict probability of T^* may be constrained by the probabilities of the individual theories, but its capability of uniting previously unrelated

(Cite as: 108 Penn St. L. Rev. 479)

phenomena (e.g., the Stoplight B-Stoplight A phenomenon and perhaps a Stoplight R-Stoplight Q phenomenon) and of yielding novel predictions (e.g., Stoplight Q will change $x + y$ t-units after Stoplight A) carries significant confirmational value. [\[FN104\]](#)

In van Fraassen's experiment, the Preacher touts the view that explanatory power should translate into increased credence in the more explanatory hypotheses. [\[FN105\]](#) Bayesian Peter is led into incoherence when he adjusts his probabilities accordingly. [\[FN106\]](#) But this is because, unlike the situation involving Psillos's T*, the explanatory aspect of the hypotheses of bias does not have an independent confirmational value. Nor should a proponent of IBE argue otherwise. So the Preacher does not accurately reflect the abductive position.

***500** From the perspective of this philosophical digression, it might seem more understandable both that courts are drawn toward a strict 2.0 relative risk standard when it comes to admissibility decisions concerning epidemiological studies, and that findings that do not exceed the 2.0 threshold may nevertheless be deemed admissible when offered in conjunction with some other evidentiary factors. In the former circumstance, when the epidemiological study is the only evidence grounding the plaintiff's causal claim, there seems to be no basis on which to afford bonus probabilities to the causation proof. When, however, there is some other case specific factor-- perhaps the plaintiff's high exposure, perhaps asbestos fibers near the tumor site, perhaps a differential diagnosis eliminating certain alternative causal possibilities in the plaintiff's life--then the causal hypothesis may take on enhanced confirmational value.

3. The Confirmational Value of Differential Diagnoses

The principal barrier to establishing specific causation by conventional methods in toxic tort actions concerns the causal mechanism linking the substance to the disease. The problem, in other words, concerns chiefly the causal capabilities of the toxic substance at issue. What proof is there, for instance, that silicone exposure increases the presence of injurious antinuclear antibodies? [\[FN107\]](#) How can a plaintiff establish that Bendectin ingestion causes birth defects? [\[FN108\]](#) More than 100,000 substances or their derivatives are registered for use in commercial applications, but researchers have studied the health implications of only a small portion of these. [\[FN109\]](#) This is the difficulty to which the abolitionists respond when they suggest that traditional causal evidentiary requirements discourage corporations from researching potential health hazards that may be associated with their products. [\[FN110\]](#)

Of less concern is the ability to identify and eliminate competing causal explanations for diseases and disease-types. Whereas levels of ***501** research about commercial products and their components may be directly sensitive to the manufacturer's particularized corporate interests, research into medical conditions and causal factors ordinarily flows from a different and more diffuse political dynamic that exists within the medical and scientific communities. [\[FN111\]](#) Corporate and scientific interests ordinarily combine to promote, rather than suppress, the investigation of medical afflictions, although deciding how to allocate resources toward one disease type rather than another may be politically charged. [\[FN112\]](#)

Thus, competing causal explanations for particular disease types will generally tend to be accessible to litigants in a toxic tort proceeding. Indeed, the problem for the toxic tort plaintiff often arises precisely because alternative causes have been identified for her type of disease. [\[FN113\]](#) For this reason, defendant corporations do have an incentive to marshal available scientific knowledge to discover possible alternative etiologies, thereby reducing the estimated probability that their products have caused the harm. [\[FN114\]](#)

The understanding of competing sources of a disease or medical condition has a double resonance for the defendant manufacturer; this information decreases the likelihood of a causation inference linking its ***502** product to the plaintiff's malady, but it also equips the plaintiff with a means of proffering a case-specific causal analysis that eliminates some of the competing possibilities. Any such case-specific showing adds confirmational value to the plaintiff's causal hypothesis, and may well shore up the probative significance of an epidemiological proof that comes up shy of the 2.0 threshold.

Accordingly, an alternative to the abolitionist idea for toxic tort litigations--one that would overcome the jurisprudential and institutional obstacles facing the all-out abolitionist program--combines general causal epidemiological outcomes with differential diagnosis testimony. A differential diagnosis tests a falsifiable hypothesis--e.g., that Mr. Maiorana's asbestos exposure caused his colon cancer--by determining whether the plaintiff's case-specific circumstances may rule out

(Cite as: 108 Penn St. L. Rev. 479)

alternative causes. [\[FN115\]](#) Nor should the admissibility of differential diagnosis evidence be controversial. The medical community has widely embraced this methodology, subjected it to peer review, and affirmed that it infrequently leads to incorrect results. [\[FN116\]](#)

The cases reflect the medical community's view. In *Cavallo v. Star Enterprise*, [\[FN117\]](#) for instance, the court emphasized that the process of differential diagnosis may significantly affect the determination about specific causation because the preponderance of the evidence standard may require ruling out other possible causes of an injury. [\[FN118\]](#) The differential diagnosis, of course, will itself be subject to the court's gatekeeping scrutiny, and may not be deemed admissible in a particular case if the court finds it insufficiently reliable. [\[FN119\]](#)

***503** Moreover, differential diagnosis testimony is even more likely than an epidemiological study to be accepted as the sole evidence of causation in a case. [\[FN120\]](#) Importantly, that testimony will be case-specific, and will therefore align with the traditional method of establishing specific causation. A plaintiff who proffers both epidemiological studies and a differential diagnosis should therefore be in a fairly strong position.

Differential diagnoses will often, however, not eliminate all other causal factors that may have contributed to a plaintiff's disease. A physician might testify that the individual plaintiff, afflicted with cancer and exposed to defendant's radiation, has never smoked, has a low-fat diet, has no significant genetic susceptibility, and has no other discernible conditions or circumstances that might create the predicate for the onset of the disease. [\[FN121\]](#) But a total elimination of competing causes may frequently be unlikely.

It is especially under these circumstances that epidemiology and differential diagnosis will be mutually reinforcing. Each will tend to confirm the probative characteristic of the other, and the offer of each will add confirmational value to the causal hypothesis overall. Accordingly, for instance, one court stated that an expert need not eliminate "each and every possible alternative cause," [\[FN122\]](#) especially when epidemiology demonstrates the product's general injurious capability. [\[FN123\]](#) As the District of Columbia Circuit Court stressed in *Ambrosini v. *504 Labarraque*, [\[FN124\]](#) "[t]he fact that several possible causes might remain 'uneliminated' . . . only goes to the accuracy of the conclusion, not the soundness of the methodology." [\[FN125\]](#)

By the same token, this Article proposes that reservations about the admissibility or probativeness of epidemiological studies finding relative risk ratios greater than 1.0 but not greater than 2.0 should be overcome--especially as the ratio approaches the 2.0 threshold--when the plaintiff has been able to supplement her causal presentation with valid differential diagnosis testimony.

IV. Evaluating the Validity of Epidemiological Studies

A. Introduction

Litigants proffering epidemiological studies, those challenging either their admissibility or the weight that the trier of fact should afford them, and courts seeking to rule on admissibility or simply to comprehend the science, should be aware of possible study problems. Whether the epidemiologist has undertaken a cohort study (prospectively or retrospectively comparing the incidence of disease in the exposed group to that within a group representing the general population) or a case-control study (retrospectively comparing people diagnosed with the disease to those not having the disease), the central issue is always whether a credible and valid causal claim has been established. [\[FN126\]](#)

Certain questions immediately arise about the validity of an epidemiological study. These include the following: have the subjects been accurately tallied; have the disease cases been reported and accounted for; are there detection problems resulting from the long latency periods that characterize some etiologies; [\[FN127\]](#) have the case and control populations remained constant or shifted over the course of the study; were the subjects exposed to other hidden toxins; and more generally, are there extraneous or confounding factors at play, such that plaintiffs would have developed their diseases regardless of the exposures being studied? [\[FN128\]](#)

These issues and others are systematically treated in the literature exploring various threats to the validity of epidemiological research. [\[FN129\]](#) ***505** An initial caveat is that, understandably, the foci of the medical and legal factual analyses are not

(Cite as: 108 Penn St. L. Rev. 479)

precisely the same. The scientific method assesses the probability that the harm is associated with the exposure, as well as the probability that the observed relationship may be an artifact of the experimental process. [\[FN130\]](#) The scientific approach, in other words, concerns the mathematics of the relationship, and such factors as the consistency and strength of that relationship. [\[FN131\]](#)

The legal fact finder, in some contrast, wants to know about the actual event; the real-world physical connection between the plaintiff's exposure to the defendant's product and the horrible ailment the plaintiff brings into court. This distinction should not be overblown, however, because in either event the recipient of the statistical information--whether a scientist or a lay fact finder--will decide whether that information is explanatory, and whether to draw a causal inference.

The question is thus always whether there are any problems that prevent observers from drawing a valid causal inference from the epidemiological data. The following subsections suggest some of the problems that may arise in the scientific analytic process. Threats to study validity have been widely discussed in the literature, [\[FN132\]](#) and so subparts (B) through (D) merely highlight the most notorious offenders: sampling error; measurement error; and design error and related interpretive problems. The difficulties discussed are meant to be illustrative, not exhaustive.

B. Sampling Error

One problem that may threaten the validity of a social scientific study arises when the researcher has erred in selecting a sample population. [\[FN133\]](#) A general rule guiding social science research is that the *506 size and quality of the sample selected must sufficiently represent the characteristics of the population studied. [\[FN134\]](#) The usefulness of even a perfectly designed study may be limited if sampling errors have occurred.

In the epidemiological context, the sample of cigarette smokers or children of Bendectin users, for example, may be too small to support an inference about the diverse population group studied. The study has to be large enough to reflect that diversity, but not so large that a similarity of non-exposure background variables becomes impracticable. [\[FN135\]](#) Also at times, the control population may, by chance, contain a large number of individuals who are naturally immune to the disease at issue, out of proportion to immunities occurring in the larger population. [\[FN136\]](#) Alternatively, from a toxic tort plaintiff's point of view, the control population may contain a randomly excessive number of people susceptible to the disease.

As a somewhat related point, characteristics peculiar to the study group will threaten the "external" validity of the study--i.e., the validity of generalizing the study outcomes. [\[FN137\]](#) It is possible that the causal inference will not easily apply to other population groups, or to groups exposed in different settings. In one study of the relationship between oral hygiene and periodontal disease, [\[FN138\]](#) for example, the fact that the participants, unlike the rest of us, cleaned their teeth under supervision threatened external validity. [\[FN139\]](#)

C. Design Error and Interpretive Problems

Because epidemiological research seeks to convey causal information, the studies must be designed to be sufficiently sensitive to causal influences. At the design stage, the epidemiologist has to *507 calculate the sample size most likely to meet the study's goals. [\[FN140\]](#) She has to perform a number of operations, such as adjusting sample size and establishing the frequency with which to collect data, and settle on the length of the study, in order to reckon how much statistical power the study should afford to detect an effect of a given magnitude. [\[FN141\]](#) A lack of statistical power will tend to produce false negatives, hence overlooking causal connections that do exist. [\[FN142\]](#)

At the post-study interpretive stage, the researcher relies on the known variances and sample sizes to compute the magnitude of the effect that could have been reasonably detected with a certain level of confidence, conventionally set at 95 percent. [\[FN143\]](#) When this established interpretive approach leads to provisional acceptance of the null hypothesis, the clinician will not be able to draw an epidemiologically-supported causal connection in any particular case, even when there is one. [\[FN144\]](#) As a general rule, in their large population studies, researchers--and the regulators who rely on them--will not deem very small elevations in the risk of disease accompanying exposure to a toxic substance to be statistically meaningful. [\[FN145\]](#)

Moreover, as a practical matter, the fixed periods of time devoted to particular studies may not be long enough to capture diseases manifesting after long latency periods. [\[FN146\]](#) Cancer, for instance, is characterized by latency periods varying

(Cite as: 108 Penn St. L. Rev. 479)

from five to perhaps forty years, *508 and to thoroughly detect causal factors the epidemiologists would have to monitor exposed and non-exposed groups over such extended periods. [\[FN147\]](#)

At the opposite end of the spectrum from the very long-term studies required to track diseases characterized by long latency periods, "cross-sectional" epidemiological studies examine exposures and diseases at particular points in time. [\[FN148\]](#) The temporal connection between the exposure and the condition is uncertain, and so the ability to draw any causal inference is limited. [\[FN149\]](#) Nonetheless, courts presiding over mass tort litigations have sometimes concluded that "cross-sectional studies are valid and generally accepted study designs in epidemiology" if the temporal relationship between the exposures and the outcomes is considered and controlled for. [\[FN150\]](#)

Additionally, any single epidemiological study may be vulnerable to biases and the influence of extraneous details that would tend to factor out if more studies were conducted. [\[FN151\]](#) Along these lines, Thomas Cook, a Faculty Fellow at the Institute for Policy Research at Northwestern University, and Donald Campbell, during his lifetime an eminent social psychologist and Schweitzer Professor in the Maxwell School at Syracuse University, jointly commented that "[s]ince single operations both underrepresent constructs and contain irrelevancies, construct validity will be lower in single exemplar research than in research where each construct is multiply operationalized in order to triangulate on the referent." [\[FN152\]](#)

Further, a study may be of limited usefulness, and the translatability of causal inferences may be impaired, if the exposure histories of the study's subjects tend toward one extreme or the other. If, for instance, an epidemiological study examines asbestos miners exposed to extraordinarily high dosages of toxic fibers, its findings may not readily *509 translate to workers more moderately exposed. [\[FN153\]](#)

D. Measurement Error

A study's descriptive accuracy will depend significantly (albeit not wholly) on the accuracy of its measurements. Epidemiological studies test causal theories that connect variables to the data, and because the quality of the data depends on measurement, inaccuracies can throw the study off and result in problematic conclusions. [\[FN154\]](#)

Measuring is often a matter of classifying, and so misclassifications count as measurement errors. [\[FN155\]](#) Consider, for instance, a study of the exposure to asbestos among insulators constructing battleships at a particular naval facility. If the researcher counts the dust emanating from nearby cutting operations as generating a certain level of airborne asbestos fibers, when in reality the cut materials happen to be asbestos-free--or inversely should she deem true asbestos sources to be innocuous--then the classification errors will entail measurement inaccuracy. Exposure misclassifications naturally engender biases in the risk estimates. [\[FN156\]](#)

Apart from classifying inaccurately, measurements of the same phenomenon may be so varied as to be unreliable. [\[FN157\]](#) Unreliability prevents the observer from drawing any causal inference with a requisite degree of confidence. As one scholar suggests, a particular laboratory might attempt to assess the presence of benzene in an air sample, but find randomly varying concentrations of benzene in parts per million, within 1 percent of the mean value, with each attempt. [\[FN158\]](#) Thus, a researcher using the laboratory's findings may not be able to link the dose associated with that air sample to the subject's response, and any causal *510 assessment would be problematic.

Testing for distributions of error is usually not too difficult in the physical sciences because the conditions can be altered in a controlled manner. [\[FN159\]](#) When studying human populations, however, testing for error is problematic for a number of reasons. People and their circumstances change over time, and individuals may alter their behavior, subconsciously or intentionally, when under examination. [\[FN160\]](#) Moreover, the population of factory workers, semiconductor "clean room" employees, or shipyard crew members assessed one month may be differently composed the next.

Apart from problems of assessing measurement reliability, researchers must account for measurement validity. [\[FN161\]](#) Reliable measurement does not ensure validity, as "a measurement process is valid only when it measures precisely what the researcher intends it to measure." [\[FN162\]](#) The researcher first has to conceptualize her terms and categories in a way that coheres with an approach taken in the relevant scientific community. One industrial hygienist may use the term asbestos worker to define any employee working in spaces containing more than a predefined minimum threshold level of asbestos concentration; another may reserve the term for those who mined or otherwise handled raw asbestos fibers. Thus, it is

(Cite as: 108 Penn St. L. Rev. 479)

important to clarify research terms with sufficient precision to avoid ambiguous readings on the part of a given study's targeted audience.

The researcher then refines the definitions by making them operational. [\[FN163\]](#) A study of a population exposed to asbestos may examine those exposed in shipyards, or in the asbestos mines, or in insulation manufacturing plants. The operational definitions provide study parameters, but conceptual narrowing has to fit the study's ultimate aims; measurement validity is placed at risk if operational definitions are so tight as to block out important data, or so broad as to permit contamination by extraneous factors. [\[FN164\]](#) An unrepresentative sample *511 group, resulting from an ill-conceived operational definition, will taint the validity of a study's measurement procedure. [\[FN165\]](#) In general, invalid measurement will impair the researcher's ability to interpret the data in a meaningful way. [\[FN166\]](#) Concerns about measurement validity thus intersect with some of the design and construct validity issues previously discussed. [\[FN167\]](#)

E. Conclusion

Validity threats may impair the usefulness of epidemiological studies in toxic tort litigations, regardless of whether the studies are proffered in satisfaction of a bright-line statistical assessment of causation or as the complement to case-specific proof such as differential diagnosis. The probativeness of the two-fold relative risk finding may prove illusory if the study or studies generating that outcome are themselves seriously flawed.

On the other hand, properly-constructed studies using sound measurement procedures and well-planned sampling techniques should prove significantly probative--even for minor increases in relative risk--when offered in conjunction with the case-specific elimination of confounding factors. Moreover, plaintiffs able to demonstrate flaws in epidemiological studies proffered by toxic tort defendants will thereby indirectly bolster the persuasiveness of their own causal proofs, even if these are otherwise weak. [\[FN168\]](#) Thus, a finding that plaintiff's causation evidence is admissible will usually thwart a defendant's motion for *512 summary judgment.

V. The Acceptability of Epidemiological Statistics in Legal Cases

A. Introduction--Admissibility

So far this article has argued that the abolitionists' concern about the difficulties of establishing causation in toxic tort cases is best addressed, not by constructing toxic tort litigation as a discrete subspecies of tort law in which the causation element is erased wholesale, but by relaxing the plaintiff's evidentiary burden through expanded reliance upon epidemiological statistical evidence. The idea finding some acceptance in the caselaw has been that proof of a two-fold epidemiological increase in relative risk can satisfy the civil more-likely-than-not burden of persuasion, and that some additional case-specific evidence, most likely differential diagnoses, may allow for the assignment of bonus probabilities confirming the probative value of those epidemiological proofs that indicate less than a two-fold relative risk ratio. [\[FN169\]](#)

But should epidemiological evidence--even assuming an ideal study free of validity threats--be admitted at all in legal cases on the issue of specific causation, either as the sole evidence of causation when they establish a greater than 2.0 relative risk factor, or in conjunction with case-specific proof when they do not? After all, epidemiology addresses only the issue of general causation, and even a slight increase in relative risk above 1.0-- factoring in a stipulated confidence level--indicates that the substance under study is capable of causing the injurious outcome. In *Allison v. McGhan Medical Corp.*, [\[FN170\]](#) for example, the Eleventh Circuit acknowledged that the epidemiological finding of a 1.24 relative risk showed a "significant statistical correlation" between silicone and increased antinuclear antibodies, [\[FN171\]](#) yet concluded that this level of "association is far removed from proving [specific] causation." [\[FN172\]](#)

However, if statistically significant relative risk ratios less than 2.0 but greater than 1.0 demonstrate a capability to cause the outcome, then this evidence is probative and should not be deemed inadmissible on relevancy grounds. [\[FN173\]](#) If such a study survives the court's *513 methodological scrutiny under *Daubert*, it should be admitted, although it remains possible that an expert's conclusion about specific causation based solely on the study would itself fail to meet Evidence Rule 702's requirement that the expert's testimony be "based upon sufficient facts or data." [\[FN174\]](#)

B. The Statistical Turn

(Cite as: 108 Penn St. L. Rev. 479)

The traditional trend in legal theorizing about statistical evidence has been to explain, in various ways, why naked statistical proofs are not acceptable, and will not be admitted for the jury's consideration. For example, the paradigmatic case standing for the exclusion of statistical testimony had been *People v. Collins*, [FN175] in which a couple robbed the complainant in the San Pedro area of Los Angeles. [FN176] One of the robbers was an African American male having a mustache and beard, and driving a yellow car; the other was a white woman, just over five feet tall, with a blonde ponytail. [FN177] The defendants fit the descriptions of the robbers, and at the time of the robbery were in the San Pedro area. [FN178]

At the trial the prosecutor proffered the testimony of a mathematics instructor to establish that, given the robbers' joint characteristics, there was an overwhelming probability that any couple matching their description had been the assailants. [FN179] The prosecutor assigned individual probabilities to a number of the characteristics common to the robbers and the defendants--a 1/1000 probability, for example, that an interracial couple would be in a car together in Los Angeles; a 1/10 probability that a "Negro" man would have a beard, and so forth--and the witness applied the statistical "product rule" to conclude that there was no more than a one in twelve million chance that any couple possessed the defendants' notable characteristics. [FN180] The jury convicted the defendants. [FN181]

On appeal, the *Collins* court held that the admission of the statistical evidence was prejudicial error. [FN182] First, the testimony lacked an adequate foundation, because there was no evidence underwriting the individual *514 probabilities, and because the individual factors were not shown to have been statistically independent. [FN183] Second, said the court, the entire statistical evidentiary exercise "was gravely misguided. At best, it might yield an estimate as to how infrequently bearded Negroes drive yellow cars in the company of blonde females with ponytails." [FN184] The crucial question, however, not addressed by the statistical surmise was: which of the few couples, if any, that might fit the defendants' description had committed the robbery? [FN185]

A second paradigmatic and oft-mentioned case is *Smith v. Rapid Transit, Inc.* [FN186] A bus, negligently driven, forced Smith's car off the road, but Smith could not identify the bus line. [FN187] The court accepted that, mathematically, it was more likely than not that the defendant owned the bus. [FN188] But this was not enough. [FN189] For example, said the court, quoting from its earlier opinion in *Sargent v. Massachusetts Accident Company*, [FN190] "the fact that colored automobiles made in the current year outnumber black ones would not warrant a finding that an undescribed automobile of the current year is colored and not black, nor would the fact that only a minority of men die of cancer warrant a finding that a particular man did not die of cancer." [FN191]

These days, however, courts recognize that, in a way, all evidence is probabilistic, if only because no evidence is certain. [FN192] As Judge Posner explained:

What powers the intuition that the plaintiff should lose the bus case is not the explicitly probabilistic nature of the evidence, but the evidentiary significance of missing evidence. If the 51/49 statistic is the plaintiff's only evidence, the inference to be drawn is not that there is a 51 percent probability that it was a bus owned by A that hit *515 the plaintiff. It is that the plaintiff either investigated and discovered that the bus was actually owned by B (and B might not have been . . . judgment-proof and so not worth suing), or that he simply has not bothered to conduct an investigation. If the first alternative is true, he should of course lose; and since it may be true, the probability that the plaintiff was hit by a bus owned by A is less than 51 percent and the plaintiff has failed to carry his burden of proof. If the second alternative is true--the plaintiff just hasn't conducted an investigation--he still should lose. [FN193]

Litigants increasingly rely on scientific evidence, and its use in legal cases has grown exponentially since *Collins*, *Smith*, and *Sargent* were decided. [FN194] The general tendency is to permit evidence having a statistical component under the standards that govern other scientific proofs. [FN195] As construed by the courts, beginning with *Cook*, epidemiological studies showing a relative risk greater than 2.0 say that there is greater than a 50 percent probability that any individual who both was exposed to toxic substance <<THETA>> and contracted disease <<xi>> would not have contracted << xi>> absent the exposure to <<THETA>>. [FN196] In the final analysis, this is not too different from the sort of proof rejected in *Smith*.

The statistical turn in evidentiary practice occurred in the late 1980s in the context of molecular genetics, and dealt with deoxyribonucleic acid, or DNA, which is the double-stranded molecule that carries an individual's genetic code. In the rape and murder case of *Spencer v. Commonwealth*, *516 [FN197] for instance, the Supreme Court of Virginia explained the testing process comparing the defendant's DNA to that taken from semen stains at the crime scene. [FN198] Based on the

(Cite as: 108 Penn St. L. Rev. 479)

initial database that had been available at the trial, the court reported that "the chance that anyone other than Spencer produced the semen stains was one in 135 million." [\[FN199\]](#) The updated database, available later in the trial, decreased the probability of a random match to one in 705 million. [\[FN200\]](#) Upholding Spencer's conviction based in part on this evidence, the court concluded that DNA testing was a reliable scientific technique, and that the tests had been properly conducted in the case. [\[FN201\]](#)

Courts now overwhelmingly accept DNA evidence. [\[FN202\]](#) Because the theory underlying the science is well established, legal challenges tend to be case specific, concerning such matters as the technical handling of samples, possible contamination, laboratory techniques, interpretive ambiguities, and possible matches between crime scene samples and innocent members of the suspect population. [\[FN203\]](#) William Thompson, a professor in the Department of Criminology, Law, and Society at the University of California, notes the view of some experts that having valid estimates of laboratory error rates is as important as having valid frequency estimates. [\[FN204\]](#) Statistics concerning DNA frequencies permit the scientist to estimate the probability of a coincidental match that might implicate an innocent person; on top of that, however, collection, handling, processing, and typing problems can result in false positives, and legal opponents challenging the use of DNA evidence may focus on these vulnerabilities. [\[FN205\]](#)

Concerning the possible misinterpretations of DNA proofs by courtroom players, statistician David Balding describes both the *517 "prosecutor's fallacy" and the "defendant's fallacy." [\[FN206\]](#) The prosecutor may tend to conflate the notion of "A implies B" with "B implies A," thus taking $P(A/B)$ -- or the probability of A given B--to imply $P(B/A)$. [\[FN207\]](#) From the calculation demonstrating that the probability of the DNA evidence given a suspect's innocence is very small, one cannot infer that the probability that the suspect is innocent, given the DNA evidence, is comparably small. [\[FN208\]](#)

Defense counsel, on the other hand, may construe a DNA profile frequency of one in one million to imply that, in a population pool of one hundred million individuals, the probability that the defendant is guilty is only one in one hundred, or $p = .01$. This, says Balding, ignores background information-- temporal, spatial, relational, and so forth--that almost always exists to some extent, and that will make some individuals more likely suspects than others. [\[FN209\]](#)

It is obvious that epidemiological evidence will not pinpoint the cause of a harm with the precision of a DNA proof. In some ways, however, the trier of fact may be less likely to misestimate the probative quality of the epidemiological showing--less likely, that is, to be overwhelmed by the numbers. Even if made aware of fallacious courtroom interpretations, and the mishandling of biological samples, a figure representing an outcome as one in 705 million not only will carry more probative force, but will hold more potentially prejudicial sway, than will a relative risk ratio of approximately 2.0.

In all events, the question is whether the statistical turn that has allowed for the introduction of DNA evidence will be wide enough to sweep in epidemiological statistical outcomes. A brief look at some of the theoretical work that has debated the use of statistical proofs should help address this issue.

C. A Theoretical Justification

The final issue is thus whether law and society will deem legal outcomes that hinge upon epidemiological proofs to be legitimate. The normative engine driving the legal system has been variously described as a search for truth, [\[FN210\]](#) a ritual by which to realize the peaceful settlements of social conflicts, [\[FN211\]](#) a mechanism for promoting fairness, *518 justice, and efficiency, [\[FN212\]](#) a means for projecting desirable norms and legal rules to society, [\[FN213\]](#) and a vehicle for producing stable outcomes. [\[FN214\]](#)

Scholars have traditionally objected to statistical proofs because they appear to conflict with one or more of these normative goals. Tribe's famous paper expresses special skepticism about the ability of lay fact finders to assign Bayesian prior probabilities to a set of initial facts. [\[FN215\]](#) Tribe rails against the "dehumanization of justice" that would result from forcing jurors to quantify the acceptable risk they are willing to run of sentencing an innocent defendant to prison. [\[FN216\]](#) Harvard Law Professor Charles Nesson, advancing the thesis that law projects normative legal rules, objects to statistical proofs that may transform the message from one of morality to one of "crude risk calculation." [\[FN217\]](#)

The standing objections to the use of statistical proofs were primarily descriptive, being explanations of why courts were not, in fact, permitting the introduction of such proofs. [\[FN218\]](#) The description no longer applies to DNA evidence, and it is

(Cite as: 108 Penn St. L. Rev. 479)

useful to explore some of the ways in which this statistical approach overcomes the traditional objections. If epidemiology's statistical findings are increasingly accepted, then the emerging shift applies to this area of the scientific evidence as well.

The most salient factor counseling the introduction of both DNA and epidemiological proof may be need. Need sometimes drives *519 evidentiary policy. [FN219] Illustratively, DNA findings have resulted in the exoneration of a significant number of individuals wrongly convicted. [FN220] Used for inculpatory purposes, the DNA analysis may be the most probative way of linking the defendant to the semen found at the crime scene, [FN221] the assailant's blood shed in the struggle, [FN222] the clump of hair taken from the victim's fist. [FN223] With respect to epidemiological proof, the abolitionists do a good job of showing why, at a minimum, the traditional principles for establishing specific causation need to be relaxed in toxic tort litigations. [FN224] Accordingly, the need factor characterizing the judicial acceptance of DNA proof and of epidemiological outcomes is comparable.

A second important factor, which must accompany any showing of need, is reliability. It is now beyond dispute that the genetic science underlying DNA research and epidemiological methodologies are both *520 well accepted in the respective scientific communities. [FN225]

This is not to say, of course, that the computation of the percentage of blue buses operating in a particular city, or of the proportion of colored to black cars sold, might be unreliable. But it is to say that such computations should not be systematically needed in types of litigation that occur repeatedly. Even in the isolated case, such as Smith, when naked statistics may be the only evidence linking a party to an event, need is questionable. As Judge Posner has stated, the diligence of the party proffering the proof is most immediately implicated. [FN226]

It will seldom be the case, however, that parties will offer DNA or epidemiological evidence as a smokescreen to cover a lax investigation. The effort they must make to obtain these sorts of scientific data will alone not be insignificant. Moreover, the proponents of this evidence must already have worked hard to create a suitable litigation context receptive to the information. They must be in a position to assert, for instance, that the epidemiological findings fit their clients' exposure histories, occupational environments, and so forth. But, at the same time, the burdens should ordinarily not be so extensive or prohibitive as to justify an outright abolition of the causal dimension, pace Berger.

Nor are legal outcomes predicated on DNA and epidemiological forms of statistical proof vulnerable to destabilization, either because new evidence may readily emerge to discredit the judgment, or because the community may be able to engage in the statistical computation on its own, hence impugning the need for discrete juries and evidentiary presentations. Under the Collins scenario, for instance, it is possible that another couple matching the assailants' descriptions might be spotted *521 somewhere near the San Pedro area. [FN227] Under the Smith scenario, the ritual performance of a jury trial would simply seem gratuitous were the outcome merely to hinge on an assessment of the proportion of vehicles owned and operated by a defendant in a given city. [FN228]

If one accepts Nesson's Durkheimian view of verdicts--that they project norms because they are about past events, not the evidence [FN229]--it is again doubtful that the admission of DNA and epidemiological proofs will rub one the wrong way. Perceiving that legal outcomes have rested on the practically irrefutable statistical connection discovered between a defendant's blood type and the assailant's sperm (when tests on each reveal statistically matching DNA), or on the showing of relative risk that demonstrates the causal capabilities of a manufacturer's product, otherwise conscientious citizens are not likely, on this basis, to take their chances in some malevolent or hazardous scheme. [FN230]

It is also significant that the presentation of DNA or epidemiological evidence does not threaten to transmogrify the triers of fact into amateur statisticians. In his landmark evidentiary piece, Tribe reacted against a proposal jointly authored by Michael Finkelstein, Professor of Law at the Columbia University School of Law, and William Fairley, a Harvard-trained statistician, that expert witnesses be brought into legal cases to help jurors engage in an explicitly Bayesian assessment of their probability functions. [FN231] The DNA or epidemiological proffer, by contrast, leaves intact the juror's everyday computational apparatus. [FN232] If the ordinary juror organizes the information revealed at trial into a narrative story "in which causal and intentional relations between events are central," [FN233] this sort of statistical *522 presentation should not throw her off course.

(Cite as: 108 Penn St. L. Rev. 479)

All of this being said, it is also reasonable to expect that the new acceptance of statistical forms of proof will spur a shift in the legal system's underlying normative commitments. If adjudications have tended in the past to privilege the evidentiary mechanism's dispute resolution function over its truth-seeking (and perhaps fairness) goals, more precise technologies now threaten to destabilize those resolutions. Recently, for instance, Manhattan District Attorney Robert M. Morgenthau sought the dismissal of the judgments of conviction entered against five men implicated in the notorious "Central Park jogger" attack, asserting that new DNA evidence and a confession from an alternative assailant had convinced him of their innocence. [\[FN234\]](#)

Epidemiology is not precise relative to molecular genetics. [\[FN235\]](#) But if the studies are demonstrably valid, their statistically significant findings should be delivered to the trier of fact in the interests of fairness and truth-seeking. Although a dispute resolution paradigm may counsel a conservative approach to evidentiary standards in some cases, courts should resist incentives to adhere to traditional notions of the burden of persuasion on the issue of specific causation.

VI. Conclusion

Traditional approaches to specific causation are not feasible in toxic tort litigations. Scholars and jurists advocating an end to the causal element in such cases lament that causality is profoundly elusive in the toxic tort context both because biology does not explain the causal mechanisms by which toxic exposures engender injuries, and because it is difficult to trace any such injuries to any one particular source.

But it is not necessary to abolish the causal element altogether. On the one hand, the abolitionist proposal likely faces insurmountable institutional and jurisprudential obstacles. On the other hand, this proposal is likely incoherent because it must presuppose some finding of the general causal nexus that it seeks to bypass.

The middle way relies on the new judicial acceptance of certain forms of statistical proof. Epidemiological findings, especially when conjoined with case-specific differential diagnosis testimony, support ***523** causal inferences and should be attainable in a wide variety of toxic tort cases. Although validity threats may impair the usefulness of epidemiological studies in toxic tort litigations, properly-constructed studies using sound measurement procedures and well-planned sampling techniques should prove significantly probative when offered in conjunction with the case-specific elimination of confounding factors.

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[\[FN1\]](#). [Weinberg v. Whatcom County](#), 241 F.3d 746, 751 (9th Cir. 2001); [Jobe v. ATR Mktg.](#), 87 F.3d 751, 753 (5th Cir. 1996).

[\[FN2\]](#). See, e.g., Margaret A. Berger, [Eliminating General Causation: Notes Towards a New Theory of Justice and Toxic Torts](#), 97 *Colum. L. Rev.* 2117, 2140 (1997) (arguing that a new legal standard of corporate product liability should be formulated that eliminates the plaintiffs' burden of establishing causation); Carl F. Cranor & David A. Eastmond, [Scientific Ignorance and Reliable Patterns of Evidence in Toxic Tort Causation: Is There a Need for Liability Reform?](#), 64 *Law & Contemp. Probs.* 5, 46 (2001) (contending that tort law may have to be modified to deal with the lack of understanding of the causal properties of toxic substances); Glenn Shafer, Causality: [Causality and Responsibility](#), 22 *Cardozo L. Rev.* 1811, 1834 (2001) (relying on Berger, advancing a probability claim that a defendant should pay under certain circumstances regardless of the harm caused); Wendy E. Wagner, [Choosing Ignorance in the Manufacture of Toxic Products](#), 82 *Cornell L. Rev.* 773, 852 (1997) (arguing that causation requirements should be reformed because they provide manufacturers with an incentive to remain ignorant about the hazards of their products); see also [Blue Cross & Blue Shield of N.J., Inc. v. Philip Morris, Inc.](#), 133 F. Supp. 2d 162, 174 (E.D.N.Y. 2001) (Hon. Jack B. Weinstein) (asserting that "[t]here is considerable merit in Professor [sic] Margaret A. Berger's suggestion that traditional general causation proof is so difficult in toxic tort cases that it should

(Cite as: 108 Penn St. L. Rev. 479)

not be required, but that alternative elements of the cause of action should suffice"). Regarding Blue Cross & Blue Shield of N.J., Inc., it should be noted that Berger's article articulating the noncausal model appeared as part of a Columbia Law Review symposium issue in tribute to Judge Weinstein. Moreover, Weinstein and Berger have collaborated. See Jack B. Weinstein & Margaret A. Berger, Weinstein's Federal Evidence § 404.12[3] (Joseph M. McLaughlin ed., Matthew Bender 2d ed. 1997).

[FN3]. See, e.g., *In re Hanford Nuclear Reservation Litig. v. E. I. Dupont*, 292 F.3d 1124, 1137 (9th Cir. 2002) (discussing "how epidemiological proof can be adapted to meet the 'more likely than not' burden of proof by requiring statistics to reflect a relative risk factor of 2.0 before a plaintiff can recover"); *Allison v. McGhan Med. Corp.*, 184 F.3d 1300, 1315 n.16 (11th Cir. 1999) (stating that the threshold for concluding that a toxic substance more likely than not caused a disease is 2.0, because a relative risk greater than 2.0 implies a greater than 50 percent likelihood of causation); *Magistrini v. One Hour Martinizing Dry Cleaning*, 180 F. Supp. 2d 584, 591 (D.N.J. 2002) (asserting that causality may be inferred from an epidemiological assessment of relative risk, and therefore that "the threshold for concluding that an agent was more likely than not the cause of an individual's disease is a relative risk greater than 2.0") (quoting Michael D. Green et al., Reference Guide on Epidemiology, in Federal Judicial Center, Reference Manual on Scientific Evidence 384 (2d ed. 2000)).

[FN4]. Berger, *supra* note 2, at 2120-21.

[FN5]. *Id.* at 2122.

[FN6]. *Id.* at 2143.

[FN7]. *Id.* at 2144-45.

[FN8]. *Id.*

[FN9]. *Id.* at 2134.

[FN10]. *Id.*

[FN11]. See also *id.* at 2144 ("According to this model, once plaintiffs proved the manufacturers' negligence in failing to reveal substantial information highly relevant to assessing the potential risks of asbestos exposure, a prima facie case of liability would be made out for those able to substantiate exposure and ill health."); see generally *Env'tl. Def. Fund. Inc. v. EPA*, 548 F.2d 998, 1008 (D.C. Cir. 1976) (noting that, to conclude that certain pesticides "pose a carcinogenic risk to humans..., the Administrator must show a causal connection between the uses of the pesticides challenged and resultant exposure of humans to those pesticides").

[FN12]. Berger, *supra* note 2, at 2150.

[FN13]. *Id.*

[FN14]. *Id.* at 2145.

[FN15]. *Id.*

[FN16]. *Id.* at 2148.

[FN17]. *Id.* at 2134.

[FN18]. See, e.g., Affidavit of Charles J. Roemer, former Chairman, Paterson Industrial Commission, *In re Johns-Manville Corp.*, Debtors Civ. No. 465-83C/1- 84C/688-83C (Bankr. S.D.N.Y., Sept. 29, 1982), reprinted in Barry I. Castleman, *Asbestos: Medical and Legal Aspects* 152-54 (2d ed. 1986) (describing statements of Vandiver Brown, General Attorney and President of Johns-Manville Corporation, that, although "Johns-Manville's physical examination program had, indeed, also produced findings of X-ray evidence of asbestos disease among workers exposed to asbestos,... it was Johns-Manville's

(Cite as: 108 Penn St. L. Rev. 479)

policy not to do anything nor to tell the employees of the X-ray findings" because such a company approach would be "foolish" because "if Johns-Manville's workers were told, they would stop working and file claims against Johns-Manville, and that it was Johns-Manville's policy to let them work until they quit work because of asbestosis or died as a result of asbestos-related diseases").

[FN19]. [George v. Celotex Corp.](#), 914 F.2d 26, 28 (2d Cir. 1990) (agreeing that a manufacturer of a toxic product "is held to the knowledge of an expert in its field"); [Borel Fibreboard Paper Products Corp.](#), 493 F.2d 1076, 1089- 90 (5th Cir. 1973) (concluding that, as a constructive expert, the manufacturer has a duty to "keep abreast of scientific knowledge, discoveries, and advances and is presumed to know what is imparted thereby," and also to test fully and inspect its products to uncover all dangers that are scientifically discoverable); see also [Dartez v. Fibreboard Corp.](#), 765 F.2d 456, 461 (5th Cir. 1985); [Wright v. Carter Products, Inc.](#), 244 F.2d 53, 59 (2d Cir. 1957); 1 Louis R. Frumer & Melvin I. Friedman, *Products Liability* § 2.22[1], at 2- 1062-64 (1990) (collecting cases, and reporting that the "manufacturer must keep abreast of scientific advances and is under a duty to make tests to ascertain the nature of its product. In this scientific age the manufacturer undoubtedly has or should have superior knowledge of his product").

[FN20]. See, e.g., [Liriano v. Hobart Corp.](#), 94 Civ. 5279 (SAS), 1996 U.S. Dist. LEXIS 7727, at *13 (S.D.N.Y. June 6, 1996) (affirming that the manufacturer's duty to warn of all potential dangers which it knew, or in the exercise of reasonable care should have known, to exist, "does not cease at the time a product is manufactured or sold; rather, a manufacturer has a continuing duty to warn of dangers it discovers thereafter"); [Cover v. Cohen](#), 473 N.Y.S.2d 378, 385 (1984) (holding that a manufacturer may "incur liability for failing to warn concerning dangers in the use of a product which come to his attention after manufacture or sale, through advancements in the state of the art, with which he is expected to stay abreast, or through being made aware of later accidents involving dangers in the product of which warning should be given to users").

[FN21]. See [Palsgraf v. Long Island R.R. Co.](#), 162 N.E. 99, 99 (N.Y. 1928) (reiterating that "[p]roof of negligence in the air, so to speak, will not do") (citation omitted); see also [Black v. Consol. Freightways Corp.](#), 219 F. Supp.2d 243, 251 (E.D.N.Y. 2002).

[FN22]. See Ellen Wertheimer, [Unknowable Dangers and the Death of Strict Products Liability: The Empire Strikes Back](#), 60 *U. Cin. L. Rev.* 1183, 1184 (1992) (documenting the erosion of strict tort liability and commenting that "[f]aced with the choice of holding manufacturers liable for dangers unknowable at the time of manufacture or abolishing strict products liability altogether, courts and legislatures have elected the latter course"); see also Robert L. Rabin & Marc A. Franklin, *Tort Law and Alternatives: Cases and Materials* 442- 600 (5th ed. 1992) (reporting that "strict liability" has been on the wane in products liability caselaw since the late 1980s); see generally Charles Nesson, [The Evidence or the Event? On Judicial Proof and the Acceptability of Verdicts](#), 98 *Harv. L. Rev.* 1357, 1373 (1985) (discussing evidentiary rules that arguably safeguard "the legal system's concern for continuing acceptance of the verdict" and "the stability of judgments"); cf. [Bowers v. Hardwick](#), 478 U.S. 186, 194 (1986) (indicating that legal institutions are self-conscious not to create regimes that leave them "vulnerable" to perceived "illegitimacy").

[FN23]. See Virginia Held, *Reason, Gender and Moral Theory*, in *Phil. & Phenomenological Research* 321-44 (supplement, Fall 1990), reprinted in *Ethics: History, Theory, and Contemporary Issues* 677, 683 (Steven M. Cahn & Peter Markie eds., 2d ed. 2002) (noting that proponents of a new "'ethic of care'... think it should supersede 'the ethic of justice' of traditional or standard moral theory").

[FN24]. See supra note 3 and accompanying text.

[FN25]. See generally Michael Devitt, *Realism and Truth* 44-46 (2d ed. 1991); Gilbert Harman, *Knowledge, Inference, and Explanation*, 5 *Am. Phil. Q.* 164, 168 (1968).

[FN26]. David Hume, *An Enquiry Concerning Human Understanding* 60 (Prometheus Books 1988) (1748) (concluding that "we are never able, in a single instance, to discover any power or necessary connexion; any quality, which binds the effect to the cause, and renders the one an infallible consequence of the other. We only find, that the one does actual, in fact, follow the other").

[FN27]. [Amorgianos v. AMTRAK](#), 303 F.3d 256, 268 (2d Cir. 2002); [Kelley v. American Heyer-Schulte Corp.](#), 957 F. Supp.

(Cite as: 108 Penn St. L. Rev. 479)

[873, 881 n.9 \(W.D. Tex. 1997\)](#).

[FN28]. [Amorgianos, 303 F.3d at 268; In re Paoli R.R. Yard PCB Litig., 35 F.3d 717, 751 \(3d Cir. 1994\); Merrell Dow Pharm., Inc. v. Havner, 953 S.W.2d 706, 714-15 \(Tex. 1997\)](#); see Susan Epstein, Comment: [Tort Reform To Ensure the Inclusion of Fertile Women in Early Phases of Commercial Drug Research, 3 U. Chi. L. Sch. Roundtable 355, 377 \(1996\)](#) (explaining that "[t]here are two levels of proof for causation in toxic tort suits: general causation and specific causation").

[FN29]. Cf. [Tyra v. State, 897 S.W.2d 796, 798 \(Tex. Crim. App. 1995\)](#) (analogously emphasizing that "a thing which actually causes death is, by definition, 'capable of causing death'") (citing [Texas Penal Code § 1.07\(a\)\(17\)\(B\)](#)).

[FN30]. See Jean Macchiaroli Eggen, [Toxic Torts, Causation, and Scientific Evidence After Daubert, 55 U. Pitt. L. Rev. 889, 896-97 \(1994\)](#) (explaining that "[n]owhere has the bifurcation of the standard of causal proof between general causation, based on probabilities, and specific causation, based on individualized factual connections, been as pronounced as it is in the toxic tort case").

[FN31]. See, e.g., Danielle Conway-Jones, [Factual Causation in Toxic Tort Litigation: A Philosophical View of Proof and Certainty in Uncertain Disciplines, 35 U. Rich. L. Rev. 875, 894 \(2002\)](#) (gleaning that "[t]he current trend in toxic tort cases, however, is to erect substantive and procedural obstacles, for example, a dual factual causation test").

[FN32]. Jean Macchiaroli Eggen, [Clinical Medical Evidence of Causation in Toxic Tort Cases: Into the Crucible of Daubert, 38 Hous. L. Rev. 369, 372 \(2001\)](#).

[FN33]. Id.

[FN34]. See, e.g., [Borel v. Fibreboard Paper Products Corp., 493 F.2d 1076, 1094 \(5th Cir. 1973\)](#) (concluding that, although "it is impossible, as a practical matter, to determine with absolute certainty which particular exposure to asbestos dust resulted in injury to Borel... the jury could find that each defendant was the cause in fact of some injury to Borel," who was generally exposed to all defendants' products); [Rutherford v. Owens-Illinois, Inc., 941 P.2d 1203, 1219 \(Cal. 1997\)](#) (products liability case brought by estate of worker exposed to asbestos-containing products and died of lung cancer, holding that "plaintiffs may prove causation in asbestos-related cancer cases by demonstrating that the plaintiff's exposure to defendant's asbestos-containing product in reasonable medical probability was a substantial factor in contributing to the aggregate dose of asbestos the plaintiff or decedent inhaled or ingested, and hence to the risk of developing asbestos-related cancer, without the need to demonstrate that fibers from the defendant's particular product were the ones, or among the ones, that actually produced the malignant growth").

[FN35]. See Joseph Sanders, Complex Litigation at the Millennium: [Kumho and How We Know, 64 Law & Contemp. Prob. 373, 385 n.116 \(2001\)](#).

[FN36]. See [Hollander v. Sandoz Pharms. Corp., 289 F.3d 1193, 1212 \(10th Cir. 2002\)](#) (describing procedures of "rechallenge" (exposing a patient to the same suspected causal substance) and "dechallenge" (removing that substance) that "resemble controlled experiments" and that may effectively inform the specific causation question); [Adams v. Indiana Bell Tel. Co., 2 F. Supp. 2d 1077, 1095 \(S.D. Ind. 1998\)](#) (stressing that, "[w]hen an association is identified among variables, a controlled experiment will help identify which independent variable caused the effect"); see also [Adams v. Ameritech Servs., Inc., 231 F.3d 414 \(7th Cir. 2000\)](#); [In re TMI Litig. Cases Consol. II, 911 F. Supp. 775, 803 \(M.D. Pa. 1996\)](#) (reiterating that the causal inferences that can be drawn from observational studies "rest on a less secure foundation than that provided by a controlled randomized experiment") (quoting Federal Judicial Center, Reference Manual on Scientific Evidence 351 (1994)); see generally Joelle Anne Moreno, [Beyond the Polemic Against Junk Science: Navigating the Oceans that Divide Science and Law with Justice Breyer at the Helm, 81 B.U. L. Rev. 1033, 1077-78 \(2001\)](#) (stating that, of three procedures for collecting data, anecdotal evidence, observational studies, and controlled experimentation, "controlled experiments are widely considered the best method for testing theories of causation").

[FN37]. See [Merrell Dow Pharm. v. Havner, 953 S.W.2d 706, 715 \(Tex. 1997\)](#).

[FN38]. See, e.g., In re Joint E. & S. Dist. Asbestos Litig.: [Maiorana v. United States Mineral Prods. Co., 52 F.3d 1124, 1128](#)

(Cite as: 108 Penn St. L. Rev. 479)

[\(2d Cir. 1995\)](#) (agreeing that statistical "epidemiological evidence is indispensable in toxic and carcinogenic tort actions where direct proof of causation is lacking").

[\[FN39\]](#). See generally *Schneck v. Int'l Bus. Mach. Corp.*, Civ. No. 92- 4370 (GEB), 1996 U.S. Dist. LEXIS 17486, at *100 (D. N.J. June 25, 1996) (noting the controverted issue in the litigation concerning the purported "limitations on epidemiology's ability to prove individual causation").

[\[FN40\]](#). See generally Michael R. Alderson, *An Introduction to Epidemiology* (1976); Abraham M. Lilienfeld & David E. Lilienfeld, *Foundations of Epidemiology* 3 (2d ed. 1980) (noting that "[e]pidemiology is concerned with the patterns of disease occurrence in human populations and of the factors that influence these patterns"); Kenneth J. Rothman, *Modern Epidemiology* 23 (1986) ("The fundamental task in epidemiologic research is thus to quantify the occurrence of illness. The goal is to evaluate hypotheses about the causation of illness and its sequelae and to relate disease occurrence to characteristics of people and their environment."); Linda A. Bailey et al., *Reference Guide on Epidemiology*, in *Federal Judicial Center, Reference Manual on Scientific Evidence* 121 (1994).

[\[FN41\]](#). See generally Alderson, *supra* note 40.

[\[FN42\]](#). See Rothman, *supra* note 40, at 23.

[\[FN43\]](#). See *id.*; Bailey et al., *supra* note 40, at 168.

[\[FN44\]](#). [545 F. Supp. 306 \(N.D.Cal. 1982\)](#).

[\[FN45\]](#). [Id. at 307](#).

[\[FN46\]](#). [Id. at 316](#).

[\[FN47\]](#). [Id. at 308](#).

[\[FN48\]](#). [Id. at 308 n.1](#) (emphasis added).

[\[FN49\]](#). See [Manko v. United States](#), [636 F. Supp. 1419, 1434 \(W.D.Mo. 1986\)](#), *aff'd*, [830 F.2d 831 \(8th Cir. 1987\)](#) (concluding that a relative risk of two or less means that exposure is not the probable cause of the disease at issue); [Marder v. G.D. Searle & Co.](#), [630 F. Supp. 1087, 1092 \(D.Md.1986\)](#) (deeming risk ratios of 1.3 and 1.9 to be insufficient to permit a finding of probable causation of harm by defendant's intra-uterine device, saying that "a two-fold increased risk is an important showing for plaintiffs to make because it is the equivalent of the required legal burden of proof--a showing of causation by the preponderance of the evidence or, in other words, a probability of greater than 50%") (emphasis added); see also [Wheelahan v. G.D. Searle & Co.](#), [814 F.2d 655 \(4th Cir. 1987\)](#); [In re Agent Orange Product Liab. Litig.](#), [597 F. Supp. 740, 785 \(E.D.N.Y. 1984\)](#), *aff'd*, [818 F.2d 145 \(2d Cir.1987\)](#) (asserting that plaintiffs must prove at least a two-fold increase in the incidence of the disease allegedly caused by exposure to the suspected toxic substance); cf. [In re Agent Orange Prod. Liab. Litig.](#), [611 F. Supp. 1223, 1261 \(E.D.N.Y. 1985\)](#) (discussing the "strong" and "weak" versions of the preponderance rule with respect to epidemiological evidence); see generally Michael D. Green, [Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of Agent Orange and Bendectin Litigation](#), [86 Nw. U. L. Rev. 643, 647 \(1992\)](#).

[\[FN50\]](#). See [People v. P. J. Video, Inc.](#), [501 N.E.2d 556, 565 n.1 \(1986\)](#) (Hancock, Jr., J., dissenting) (indicating that bright line rules create predictability, precision, and guidance).

[\[FN51\]](#). [Festo Corp. v. Shoketsu Kinzoku Kogyo Kabushiki Co.](#), [234 F.3d 558, 592 \(Fed. Cir. 2000\)](#) (Plager, C.J., concurring) (lamenting that the majority's "attempt [in patent law cases] to limit indeterminacy" with a set of bright-line rules adhering to the doctrine of equivalence "trade[d] off areas of uncertainty for a degree of rigidity" and the "potential for unintended... consequences that may do nothing but exacerbate the problem").

[\[FN52\]](#). See Sana Loue, *Epidemiological Causation in the Legal Context: Substance and Procedures*, in *Statistics for Social*

(Cite as: 108 Penn St. L. Rev. 479)

Science and Public Policy: Statistical Science in the Courtroom 263, 275 (Joseph L. Gastwirth ed., 2000) (commenting that strictly applying a 2.0 risk ration "is ill-advised because it would a priori preclude recovery by individuals actually harmed by a specific exposure where less than a relative risk of 2.0 is demonstrated") [hereinafter Statistics for Social Science and Public Policy].

[FN53]. See *id.* at 275-76.

[FN54]. David L. Faigman, [Mapping the Labyrinth of Scientific Evidence](#), 46 *Hastings L.J.* 555, 568 (1995) (discussing and defining Type I and Type II errors).

[FN55]. Part III, *infra*, discusses a number of the potential flaws that may threaten the validity of an epidemiological study.

[FN56]. See [Fed. R. Evid. 402](#).

[FN57]. See [Fed. R. Evid. 401](#).

[FN58]. E.g., [Allison v. McGhan Med. Corp.](#), 184 F.3d 1300, 1315 n.16 (11th Cir. 1999) (upholding the district court's ruling precluding the admission of study showing a 1.24 relative risk linking silicone exposure in breast implants to increased antinuclear antibodies characteristic of plaintiff's thyroid disease); [Daubert v. Merrell Dow Pharm.](#), 43 F.3d 1311, 1321 (9th Cir. 1995) (concluding that relative risk less than two offered to show a causal link between Bendectin ingestion and birth defects "would not be helpful, and indeed would only serve to confuse the jury, if offered to prove rather than refute causation. A relative risk of less than two may suggest teratogenicity, but it actually tends to disprove legal causation as it shows that Bendectin does not double the likelihood of birth defects"); [DeLuca v. Merrell Dow Pharm., Inc.](#), 911 F.2d 941, 958-59 (3d Cir. 1990) (concluding in Bendectin case that plaintiffs would have to establish a relative risk of greater than two to survive summary judgment); [Sanderson v. Int'l Flavors & Fragrances](#), 950 F. Supp. 981, 999-1000 (C.D. Cal. 1996) (in suit for personal injuries arising from plaintiff's exposure to fragrances emanating from Boss, Drakkar Noir, Stetson, Joop! Homme, Calvin Klein's Obsession, Davidoff's Cool Water, and Freesia perfumes, court relies on Ninth Circuit's conclusion that findings of a relative risk less than two, linking such exposures to plaintiff's injuries, would "not be helpful, and indeed would only serve to confuse the jury, if offered to prove rather than refute causation"); [Hall v. Baxter Healthcare Corp.](#), 947 F. Supp. 1387, 1403 (D. Or. 1996) (holding that, "[t]o support admissible expert opinions, epidemiological evidence must fit the legal as well as the substantive issues of the case," and that, because the relevant legal standard in proving proximate causation is whether the silicone breast implants at issue were more likely than not a substantial causal factor in producing the plaintiffs' atypical connective tissue disease, "plaintiffs must be able to show a relative risk of greater than 2.0").

[FN59]. The issue of linking various diseases to workers' asbestos exposures was pronounced when these workers were afflicted with such non-signature diseases as colon cancer or lung cancer. Peter H. Schuck, Commentary: [Judicial Avoidance of Juries in Mass Tort Litigation](#), 48 *DePaul L. Rev.* 479, 498 (1998). Asbestosis is characterized by the presence of asbestos fibers in lung tissue. [Cimino v. Raymark Indus., Inc.](#), 751 F. Supp. 649, 655 (E.D. Tex. 1990). Malignant mesothelioma is a particular type of lung cancer the only known cause of which is exposure to asbestos, and which ordinarily involves a latency period of twenty to thirty-five years. See Leon S. Gottlieb, *The Range of Medical Abnormalities Resulting from Asbestos Exposure*, reprinted in *Sourcebook on Asbestos Diseases: Medical, Legal and Engineering Aspects--Asbestos Disease Update 1-35* (Special Supp. Mar. 1989); see also Karen Hamm Antman, *Malignant Mesothelioma*, 303 *N. Eng. J. Med.* 200-02 (1980).

[FN60]. [758 F. Supp. 199 \(S.D.N.Y. 1991\)](#).

[FN61]. See [In re Joint E. & S. Dist. Asbestos Litig.](#), 964 F.2d 92, 93 (2d Cir. 1992).

[FN62]. See [In re Joint E. & S. Dist. Asbestos Litig.](#), 758 F. Supp. 199, 202 (S.D.N.Y. 1991).

[FN63]. *Id.* at 202-03 (acknowledging a "strong" epidemiological result finding that for certain individuals with significant levels of asbestos exposure the relative risk of colon cancer was 1.68, with a 95 percent confidence range of between 1.34 and 2.09, but stating that Maiorana's asbestos exposure level did not appear high enough to place him in this high risk group).

(Cite as: 108 Penn St. L. Rev. 479)

[FN64]. [Landrigan v. Celotex Corp., 579 A.2d 1268, 1272 \(App. Div. 1990\)](#).

[FN65]. By way of explanation, a test of statistical significance gauges whether the null hypothesis can be rejected. See Janice Nadler & Mary R. Rose, Victims and the Death Penalty: Inside and Outside the Courtroom: [Victim Impact Testimony and the Psychology of Punishment](#), 88 *Cornell L. Rev.* 419, 434 n.91 (2003). If the null hypothesis is rejected, then the effect found in a sample is said to be statistically significant. Id. Before conducting a statistical analysis, the experimenter chooses a significance level, traditionally called 'alpha', which although subjective is traditionally set at 5 percent. See David W. Barnes & John M. Conley, *Statistical Evidence in Litigation* 34 (1986). The lower the significance level, the more the data must diverge from the null hypothesis to be deemed significant; the significance level chosen determines the probability of a Type I error. Id. at 33-34. Importantly, a statistically significant effect is not necessarily practically significant. David H. Kaye, [DNA Evidence: Probability, Population Genetics and the Court](#), 7 *Harv. J. Law & Tech.* 101, 126-27 (1993). In the epidemiological context, with higher numbers of study subjects, a lower relative risk will be considered statistically significant. See generally Gerald W. Boston, [A Mass-Exposure Model of Toxic Causation: The Content of Scientific Proof and Regulatory Experience](#), 18 *Colum. J. Envtl. L.* 181, 261 (1993); James J. Schlesselman, Sample Size Requirements in Cohort and Case Control Studies of Disease, 90 *Am. J. Epidemiol.* 365 (1969). When the numbers of cases are very low, relative risk must approach 2.0 to be deemed significant; with larger numbers of cases, relative risk may hover about 1.5 and be considered significant on the issue of causal capability. See generally David Hildebrand, *Statistical Thinking for Behavioral Scientists* (1986).

[FN66]. [Landrigan v. Celotex Corp., 605 A.2d 1079 \(N.J. 1992\)](#).

[FN67]. Id. at 1086; see [Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 596-97 \(1993\)](#) (recognizing that "there are important differences between the quest for truth in the courtroom and the quest for truth in the laboratory. Scientific conclusions are subject to perpetual revision. Law, on the other hand, must resolve disputes finally and quickly").

[FN68]. Contextualism is a theory of knowledge that asserts that an individual's knowledge claim is relative to his epistemic context. See Robert J. Fogelin, *Pyrrhonian Reflections on Knowledge and Justification* 208 (Oxford Univ. Press 1994) (emphasizing that "[u]nderstanding what a whole <<phi>> is involves understanding what will count as a part--and this will vary with context"); David B. Annis, A Contextualist Theory of Epistemic Justification, 15 *Am. Phil. Q.* 213 (July 1978); Keith DeRose, Contextualism and Knowledge Attributions, 52 *Phil. & Phenomenological Res.* 913 (1992); Robert Hambourger, Justified Assertion and the Relativity of Knowledge, 51 *Phil. Studies* 241, 262 (1987) (explaining that "we should say that there really is only one sense of 'know' involved here, but that the amount of evidence it requires for us to know something varies with indefinitely many standards of caution"); David Lewis, Elusive Knowledge, 74 *Australasian J. Phil.* 549, 550-51 (Dec. 1996) (saying that ascriptions of knowledge, like much of what we say, are context-dependent, and that infallibilist epistemology (or even epistemology generally) is maybe "a context that makes them go false. Then epistemology would be an investigation that destroys its own subject matter.... In the strict context of epistemology we know nothing, yet in laxer contexts we know a lot"); see also Ludwig Wittgenstein, *Philosophical Investigations* 22e (2nd ed., G. Elizabeth M. Anscombe trans., Blackwell Publishers 1997) ("If I tell someone without any further explanation: 'What I see before me now is composite,' he will have the right to ask: 'What do you mean by "composite" ? For there are all sorts of things that that can mean!--The question 'Is what you see composite?' makes good sense if it is already established what kind of complexity--that is, which particular use of the word--is in question.").

[FN69]. [Landrigan, 605 A.2d at 1086](#).

[FN70]. Id.

[FN71]. Id. (quoting N.J. Evid. R. 56(2)). In a similar vein, in the later context of the United States Supreme Court's decision in [Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579 \(1993\)](#), one commentator interpreted Karl Popper's falsifiability criterion of evaluation to entail a conception of the scientific method that people standing outside of science could apply to determine whether purported scientists are in fact doing science. @ Adina Schwartz, A Dogma of ["Empiricism" Revisited: Daubert v. Merrell Dow Pharmaceuticals, Inc. and the Need To Resurrect the Philosophical Insight of Frye v. United States](#), 10 *Harv. J. Law & Tech.* 149, 164 n.47 (1997). Consistently, Popper took it to be the first task of the logic of knowledge to put forward a concept of empirical science, in order to make linguistic usage, now somewhat uncertain, as definite as

(Cite as: 108 Penn St. L. Rev. 479)

possible, and in order to draw a clear line of demarcation between science and metaphysical ideas.@ Karl R. Popper, *The Logic of Scientific Discovery* 38-39 (Routledge 1992) (1959). But see Wendy E. Wagner, *Judicial Review of Statistical Analysis in Environmental Rulemakings*, in *Statistics for Social Science and Public Policy*, supra note 52, at 282-83 (explaining that courts tend to be overly deferential to definitive quantitative analyses, which in turn tends to discourage proffers of statistical proof that more realistically express scientific uncertainties).

[FN72]. Strength of association correlates to the relative risk, whereas consistency of association is gauged by comparing the cause and effect relation emerging "in one study with the results of other studies and with other relevant scientific knowledge." See [Maiorana v. United States Mineral Prods. Co.](#), 52 F.3d 1124, 1128 (2d Cir. N.Y. 1995); Gary D. Friedman, *Primer of Epidemiology* 183-84 (3d ed. 1987). Friedman further explains that an association attains plausibility if it makes sense in relation to known biological mechanisms or other epidemiological background knowledge. *Id.* at 184. He explains, for instance, that "[p]art of the attractiveness of the hypothesis that a high-saturated fat, high-cholesterol diet predisposes to atherosclerosis is the fact that a biologic mechanism can be invoked. Such a diet increases blood lipids, which may in turn be deposited in arterial walls. A correlation between the number of telephone poles in a country and its coronary heart disease mortality rate lacks plausibility as a cause-and-effect relationship partly because it is difficult to imagine a biologic mechanism whereby telephone poles result in atherosclerosis." *Id.*

[FN73]. Landrigan, 605 A.2d at 1078.

[FN74]. *Id.*

[FN75]. [Fed. R. Evid. 702 \(2000\)](#) (regarding the portion of [Rule 702](#) added by the 2000 amendments in response to the United States Supreme Court ruling in [Daubert](#), 509 U.S. 579 (1993)).

[FN76]. [591 A.2d 671 \(N.J. Super. Ct. App. Div. 1991\)](#).

[FN77]. *Id.* at 676.

[FN78]. See supra notes 52-53 and accompanying text (explaining the notions of Type I and Type II error).

[FN79]. See Loue, supra note 52, at 275-76 (explaining that acceptance of the null hypothesis when the inconsistent hypothesis is true constitutes a Type II error).

[FN80]. See supra note 58.

[FN81]. See Bas C. van Fraassen, *Laws and Symmetry* 158 (1989).

[FN82]. *Id.*

[FN83]. *Id.* at 169.

[FN84]. "Thomas Bayes was an eighteenth-century minister (and mathematician) who wanted to prove God existed based on the sample data that's the world." Alani Golanski, [Kahn's Reign and Its Metaphors for Law--A Critique in the Philosophy of Legal Culture](#), 27 *S.U. L. Rev.* 89, 146 (2000). A Bayesian probability exercise is formulaic, and begins with a prior probability that one adjusts in the light of new evidence, pursuant to a likelihood ratio, to deliver a posterior probability. See generally Morris Hamburg, *Statistical Analysis for Decision Making* § 2.3, at 77-80 (4th ed. 1987); Laurence H. Tribe, [Trial by Mathematics: Precision and Ritual in the Legal Process](#), 84 *Harv. L. Rev.* 1329, 1353 (1971) (discussing Bayes's Theorem).

[FN85]. van Fraassen, supra note 81, at 161.

[FN86]. *Id.*

[FN87]. *Id.* at 163.

[\[FN88\]](#). Id.

[\[FN89\]](#). Id.

[\[FN90\]](#). Id. at 164.

[\[FN91\]](#). Id. at 162 (stating that, to say that the orthodox Bayesian updates by simple conditionalization is also to say that he "updates by Bayes's Theorem").

[\[FN92\]](#). Id. at 163-65.

[\[FN93\]](#). Id. at 166.

[\[FN94\]](#). Id.

[\[FN95\]](#). Id.

[\[FN96\]](#). Id. at 169.

[\[FN97\]](#). Id. (emphasis omitted).

[\[FN98\]](#). Id. at 175.

[\[FN99\]](#). Gilbert Harman, Knowledge, Inference, and Explanation, 5 Am. Phil. Q. 164, 168 (1968).

[\[FN100\]](#). Stathis Psillos, Scientific Realism: How Science Tracks Truth 224 (1999).

[\[FN101\]](#). Id.

[\[FN102\]](#). Id.

[\[FN103\]](#). Id.

[\[FN104\]](#). This hypothetical can be framed in other terms to steer clear of constructed systems, our likely prior knowledge or intuitions about city planning, and so on.

[\[FN105\]](#). van Fraassen, *supra* note 81, at 169.

[\[FN106\]](#). Id.

[\[FN107\]](#). See [Allison v. McGhan Med. Corp.](#), 184 F.3d 1300 (11th Cir. 1999)

[\[FN108\]](#). See, e.g., [Daubert v. Merrell Dow Pharm.](#), 43 F.3d 1311 (9th Cir. 1995); [DeLuca v. Merrell Dow Pharm., Inc.](#), 911 F.2d 941 (3d Cir. 1990).

[\[FN109\]](#). See Carl F. Cranor & David A. Eastmond, [Scientific Ignorance and Reliable Patterns of Evidence in Toxic Tort Causation: Is There a Need for Liability Reform?](#), 64 *Law & Contemp. Probs.* 5, 11 (2001); James Huff & David Hoel, Perspective and Overview of the Concepts and Value of Hazard Identification as the Initial Phase of Risk Assessment for Cancer and Human Health, 18 *Scand. J. Work Env't & Health* 83, 85 (1992) (estimating that there are as many as 100,000 chemicals in the marketplace); see also Office of Tech. Assessment, U.S. Congress, *Screening and Testing Chemicals in Commerce* 1 (1995) (estimating approximately seventy thousand chemicals in commerce).

[\[FN110\]](#). See Berger, *supra* note 2, at 2134.

[\[FN111\]](#). See, e.g., Univ. of Copenhagen, Inst. of Pub. Health, Dep't of Health Services Research, at

(Cite as: 108 Penn St. L. Rev. 479)

<http://www.pubhealth.ku.dk/stf/index-e.html> (last visited May 5, 2003) (noting that the Institute was established in 1997 to examine "the political control of the health care sector, and its historical development").

[FN112]. See Robert C. L. Moffat, Legal Perspectives on Cloning: [Cloning Freedom: Criminalization or Empowerment in Reproductive Policy?](#), 32 Val. U. L. Rev. 583, 599 (1998) (cautioning about "the risks and dangers of permitting political interference in the conduct of scientific research"); Steven R. Salbu, [AIDS and Drug Pricing: In Search of a Policy](#), 71 Wash. U. L.Q. 691, 722 (1993) (discussing political implications of allocating tax dollars to AIDS research and care).

[FN113]. See, e.g., [In re Paoli R.R. Yard PCB Litig.](#), 35 F.3d 717, 755 (3d Cir. 1994) (noting district court's exclusion of testimony of expert who had "not even consider[ed] alternative causes for various diseases"); [Magistrini v. One Hour Martinizing Dry Cleaning](#), 180 F. Supp. 2d 584, 609 (D.N.J. 2002) (reiterating that, "'where a defendant points to a plausible alternative cause and the doctor offers no reasonable explanation' for why he still concludes that the chemical was a substantial factor in bringing about the plaintiff's disease, 'that doctor's methodology is unreliable'") (quoting [Paoli](#), 35 F.3d 717, 760); [In re Diet Drugs Prods. Liab. Litig.](#), MDL Docket No. 1203, 2001 U.S. Dist. LEXIS 1174, at *41 (E.D. Pa. Feb. 1, 2001) (stressing that "even when association between exposure and disease exists, it must be determined 'whether the exposure causes the disease or whether the exposure and disease are caused by some other confounding factor'") (quoting Reference Manual on Scientific Evidence 369 (2d ed. 2000)).

[FN114]. See [Watkins v. Fibreboard Corp.](#), 994 F.2d 253, 255 (5th Cir. 1993) (noting that the defendant's experts had discussed the probability that factors other than asbestos exposure had caused the plaintiff's pleural lung disease); [Haines v. Liggett Group, Inc.](#), 140 F.R.D. 681, 683 (D.N.J. 1992) (noting plaintiff's allegation that the defendants had prodded the Council for Tobacco Research to sponsor research showing that factors other than smoking caused the illnesses attributed to tobacco use), vacated by [975 F.2d 81](#) (3d Cir. 1992).

[FN115]. See generally Diagnostic and Statistical Manual of Mental Disorders (Michael B. First ed., 4th ed. 1994).

[FN116]. See [Berg v. E.I. DuPont de Nemours & Co.](#), 293 F.3d 1127, 1130 (9th Cir. 2002); [In re Paoli](#), 35 F.3d at 758; [Glaser v. Thompson Med. Co.](#), 32 F.3d 969, 978 (6th Cir. 1994) (recognizing that differential diagnosis is "a standard diagnostic tool used by medical professionals to diagnose the most likely cause or causes of illness, injury and disease").

[FN117]. [892 F. Supp. 756](#) (E.D. Va. 1995), aff'd in part, rev'd in part on other grounds, [100 F.3d 1150](#) (4th Cir. 1996).

[FN118]. [Cavello](#), 892 F. Supp. at 771; accord [Raynor v. Merrell Pharm.](#), 104 F.3d 1371, 1376 (D.C. Cir. 1997) (suggesting the importance, in the court's assessment, of an explanation of the etiology of the disease, and of a showing of the implausibility of most alternative causes).

[FN119]. See [Kennedy v. Collagen Corp.](#), 161 F.3d 1226, 1229-30 (9th Cir. 1998) (concluding that the an expert's differential diagnosis should satisfy the admissibility criteria suggested in Daubert when that analysis is based on verifiable evidence and scientific methodology that is well-accepted in the field); [Moore v. Ashland Chem. Inc.](#), 151 F.3d 269, 278-79 (5th Cir. 1998) (en banc), cert. denied, [526 U.S. 1064](#) (1999) (noting that the trial court has the discretion to conclude that a differential diagnosis is not sufficiently reliable for the jury to consider); [Cavallo](#), [100 F.3d at 1159](#) (commenting that a speculative differential diagnosis, not supported by the available scientific studies, is thus inadmissible).

[FN120]. See [Turner v. Iowa Fire Equip. Co.](#), 229 F.3d 1202, 1209 (8th Cir. 2000) ("If a properly qualified medical expert performs a reliable differential diagnosis through which, to a reasonable degree of medical certainty, all other possible causes of the victims' condition can be eliminated, leaving only the toxic substance as the cause, a causation opinion based on that differential diagnosis should be admitted."); [Westberry v. Gislaved Gummi AB](#), 178 F.3d 257, 262 (4th Cir. 1999) (holding that a reliable differential diagnosis alone may provide a valid foundation for a causation opinion, even when no epidemiological studies, peer-reviewed published studies, animal studies, or laboratory data are offered in support of the opinion).

[FN121]. See [In re Hanford Nuclear Reservation Litig.](#), Master Case File No. CY-91-3015-AAM, 1998 U.S. Dist. LEXIS 15028, at *588-89 (E.D. Wash. Aug. 21, 1998); see also [Grassis v. Johns-Manville Corp.](#), 591 A.2d 671, 675 (N.J. Super. Ct. App. Div. 1991) (holding that an epidemiological study finding relative risk of less than 2.0 was admissible to prove that

(Cite as: 108 Penn St. L. Rev. 479)

plaintiff's asbestos exposure had caused his colon cancer where testimony ruled out other known risk factors, such as family history, diet, alcohol consumption, and smoking).

[FN122]. [Lakie v. Smith-Kline Beecham, 965 F. Supp. 49, 57 \(D.D.C. 1997\)](#).

[FN123]. *Id.* (concluding that it was not "particularly significant that the doctors could not affirmatively eliminate spontaneous chromosomal mutation as a cause of plaintiff's MDS 5 q-minus.... This burden would be particularly onerous in this case given that spontaneous chromosomal mutation is a difficult phenomenon for scientists to understand.... This plaintiff has established that there is a clear link between benzene contamination and MDS. As a result, it is not fatal to the admissibility of plaintiff's experts' testimony regarding specific causation that her experts cannot eliminate every possible cause of her condition.").

[FN124]. [101 F.3d 129 \(D.C. Cir. 1996\)](#).

[FN125]. *Id.* at 140 (quoting [Mendes-Silva v. United States, 980 F.2d 1482, 1487 \(D.C. Cir. 1993\)](#)).

[FN126]. See Carl F. Cranor, *Regulating Toxic Substances: A Philosophy of Science and the Law* 29 (1993).

[FN127]. See *supra* text accompanying note 33, and notes 44-46.

[FN128]. See generally Cranor, *supra* note 126, at 29.

[FN129]. See Thomas C. Cook & Donald T. Campbell, *Quasi-Experimentation: Design and Analysis Issues in Field Settings* 39-40 (1979); Frank Hagan, *Research Methods in Criminal Justice and Criminology* 129-41 (4th ed. 1997); Sander Greenland & James M. Robins, *Confounding and Misclassification*, 122 *Am. J. Epidemiology* 495, 495-506 (1985).

[FN130]. See David Barnes, [Too Many Probabilities: Statistical Evidence of Tort Causation](#), 64 *Law & Contemp. Prob.* 191, 191 (2001).

[FN131]. *Id.* at 197; see also Nesson, *supra* note 22, at 1358 (distinguishing statements about the evidence presented at trial from statements about past acts and occurrences at issue in the litigation).

[FN132]. See generally Donald T. Campbell & Julian Stanley, *Experimental and Quasi-Experimental Designs for Research* (1963).

[FN133]. See Clay Calvert, [Toxic Television, Editorial Discretion, & The Public Interest: A Rocky Mountain Low](#), 21 *Hastings Comm. & Ent. L.J.* 163, 196-97 (1998) (discussing the FCC's rejection of a challenge filed by the Rocky Mountain Media Watch to the license renewal of four local television stations-- allegedly because the violence content of their news broadcasts did not serve the public interest--on the ground that the sampling size of news broadcasts used in Rocky Mountain's study was too small to determine whether the sample was representative of the overall effort of the stations during their license terms).

[FN134]. Earl Babbie, *The Practice of Social Research* 226 (7th ed. 1995).

[FN135]. See [Bloomquist v. Wapello County, 500 N.W.2d 1, 5 \(Iowa 1993\)](#) (reflecting that a difficulty inhering in the use of epidemiological studies is that these "require large numbers of patients whose backgrounds are similar in every respect except their exposure to the suspected substance").

[FN136]. See Barnes, *supra* note 130, at 198 (emphasizing that such sampling error is "an unavoidable property of inferential statistics"); see also David E. Bernstein, [The Admissibility of Scientific Evidence After Daubert v. Merrell Dow Pharmaceuticals, Inc.](#), 15 *Cardozo L. Rev.* 2139, 2167 (1994) (saying that "[e]pidemiology is an inexact science, and is prone to a range of methodological and sampling errors").

[FN137]. Cook & Campbell, *supra* note 129, at 37.

(Cite as: 108 Penn St. L. Rev. 479)

[FN138]. Niklaus P. Lang et al., Toothbrushing Frequency as It Relates to Plaque Development and Gingival Health, 44 J. Periodontal 396 (1973).

[FN139]. See Jan Lindhe & Gary Koch, The Effect of Supervised Oral Hygiene on the Gingivae of Children, 2 J. Periodontal Res. 215 (1967).

[FN140]. See Abigail C. Saguy, [Employment Discrimination or Sexual Violence? Defining Sexual Harassment in American and French Law](#), 34 *Law & Soc'y Rev.* 1091, 1093 (2000) (discussing the author's own research design, and the limited two-country sample size used).

[FN141]. See Cook & Campbell, supra note 129, at 39-40; David H. Kaye, Is [Proof of Statistical Significance Relevant?](#), 61 *Wash. L. Rev.* 1333, 1357-62 (1986) (explaining the statistical concept of power).

[FN142]. See Carl F. Cranor et al., Judicial Boundry Drawing and the Need for Context-Sensitive Science in [Toxic Torts After Daubert v. Merrell-Dow Pharmaceuticals, Inc.](#), 16 *Va. Env'tl. L.J.* 1, 41 (1966) (explaining that "[i]f researchers use a sample which is quite small in a study to detect relatively rare diseases, such as those typical of many cancers, and either the researcher or judge insists on less than a .05 chance of false positives, there is a risk of high false negatives or low statistical power").

[FN143]. Cranor, supra note 126, at 32.

[FN144]. See id. at 34.

[FN145]. E.g., [National Ambient Air Quality Standards for Particulate Matter](#), 62 *Fed. Reg.* 38652, 38656 n.7 (July 18, 1997) (reporting that, "[o]ver the course of a year, the few peak 24-hour concentrations of [particulate matter, or 'PM'] appeare[d] to contribute a relatively small amount to the total health risk posed by the entire air quality distribution as compared to the aggregated risks associated with the low to mid-range concentrations," and that there was "greater uncertainty about both the existence and the magnitude of estimated excess mortality and other effects associated with PM exposures as one considers increasingly lower concentrations approaching background levels").

[FN146]. See Heidi Li Feldman, [Science and Uncertainty in Mass Exposure Litigation](#), 74 *Tex. L. Rev.* 1, 25 (1995).

[FN147]. See Australian Radiation Protection and Nuclear Safety Agency, The Controversy over Electromagnetic Fields and Possible Adverse Health Effects, at http://www.arpansa.gov.au/is_emf.htm (last visited May 5, 2003).

[FN148]. See Loue, supra note 52, at 268.

[FN149]. See id.

[FN150]. *Allen v. IBM*, Civil Action No. 94-264-LON, 1997 U.S. Dist. LEXIS 8016, at *79 (D. Del. May 19, 1997).

[FN151]. See, e.g., [Blum v. Merrell Dow Pharm., Inc.](#), 33 *Phila. Co. Rptr.* 193, 222 (Pa. Com. Pl. 1996) (reporting in Bendectin case that defendant Merrell Dow had performed only one epidemiological evaluation, which appeared to contain "design and supervision problems, as well as other errors and irregularities").

[FN152]. Cook & Campbell, supra note 129, at 65; cf. Douglas Crawford- Brown, [Scientific Models of Human Health Risk Analysis in Legal and Policy Decisions](#), 64 *Law & Contemp. Probs.* 63, 65-66 (2001) (opining that risk evaluation entails analysis of the risk agent, the potentially affected population group, and the conditions under which exposures may occur, and that judgments of risk must therefore involve the community of scientists, not single assessments).

[FN153]. See, e.g., [United States v. Reserve Mining Co.](#), 380 *F. Supp.* 11, 52-53 (D. Minn. 1974) (questioning whether asbestos exposures in a community setting are comparable to exposures among asbestos workers).

[FN154]. See Vern R. Walker, The Structure of Factual Inference in Judicial Settings: [Theories of Uncertainty: Explaining](#)

(Cite as: 108 Penn St. L. Rev. 479)

[the Possible Sources of Error in Inferences, 22 Cardozo L. Rev. 1523, 1547 \(2001\)](#) (explaining that "[m]easurement is the process of classifying individual objects or events into the categories of a variable, and it generates the data for a scientific study").

[FN155]. See Paul Gustafson, *Measurement Error and Misclassification in Statistics and Epidemiology: Impacts and Bayesian Adjustments* (2003).

[FN156]. See Rebecca A. Johnson & Elizabeth V. Wattenberg, *Risk Assessment of Phenoxy Herbicides: An Overview of the Epidemiology and Toxicology Data*, at <http://www.24d.org/chapter3.pdf> (last visited May 5, 2003) (explaining that "differential misclassification" occurs when the measurement error is sensitive to disease status, whereas "nondifferential misclassification" is independent of disease status).

[FN157]. Edward G. Carmines & Richard A. Zeller, *Reliability and Validity Assessment* 12 (1979).

[FN158]. Walker, *supra* note 154, at 1547-48.

[FN159]. *Id.* at 1548 (noting that "[f]or many measurement processes, especially in the physical sciences, we can conduct reliability studies to determine distributions of error under different sets of circumstances"); cf. David Friedman, [More Justice For Less Money, 39 J. L. & Econ. 211, 231 \(1996\)](#) (suggesting that even in the soft science of economics, procedural modifications may help control for error distributions).

[FN160]. Walker, *supra* note 154, at 1548.

[FN161]. See David P. Farrington, [Methodological Quality Standards for Evaluation Research, 587 Annals 49, 59 \(2003\)](#).

[FN162]. Walker, *supra* note 154, at 1548-49 (citing Edwin E. Ghiselli et al., *Measurement Theory for the Behavioral Sciences* 266 (1981)).

[FN163]. See Am. Psychiatric Ass'n, *Diagnostic and Statistical Manual of Mental Disorders XXI* (4th ed. 1994).

[FN164]. See Kitty Calavita, [The Paradoxes of Race, Class, Identity, and "Passing": Enforcing the Chinese Exclusion Acts, 1882-1910, 25 Law & Soc. Inquiry 1, 33 \(2000\)](#) (noting that "[d]ifficulties in crafting acceptable operational definitions may expose inconsistencies or internal contradictions in the theory itself"); see also Am. Psychiatric Ass'n, *supra* note 163 (reporting that "[t]he concept of mental disorder, like many other concepts in medicine and science, lacks a consistent operational definition that covers all situations").

[FN165]. Fridah W. Mugo, *Sampling in Research*, at <http://trochim.human.cornell.edu/tutorial/mugo/tutorial.htm> (last visited Aug. 9, 2003).

[FN166]. See generally Fatimah L.C. Jackson, [Anthropological Measurement: The Mismeasure of African Americans, 568 Annals 154 \(2000\)](#); David B. Wilson, [Meta-Analytic Methods for Criminology, 578 Annals 71, 77 \(2001\)](#).

[FN167]. See *supra* notes 140-53 and accompanying text; see generally Barnes, *supra* note 130, at 201-04 (also discussing extrapolation error (e.g., in inferring a "but for" causal relationship between the defendant's misconduct and the plaintiff's harm), hypothesis testing error (e.g., in assumptions made about the credibility of the evidence), and statistical modeling error (e.g., in assumptions about the interrelationships among variables and how the variables may affect outcomes)).

[FN168]. Cf. [Pick v. American Med. Sys., Inc., 958 F. Supp. 1151, 1160 \(E.D. La. 1997\)](#) (ironically finding that the epidemiological evidence proffered by the plaintiffs did not support their claim that silicone can induce autoimmune disease, but also that, "from the Daubert standpoint, the methodology is arguably flawed due to the nature of the alleged disease and the design restrictions in the studies," and therefore that "the epidemiological evidence does not definitively rule out such a connection").

[FN169]. See *supra* notes 44-77.

(Cite as: 108 Penn St. L. Rev. 479)

[FN170]. [184 F.3d 1300 \(11th Cir. 1999\)](#).

[FN171]. [Id. at 1315](#).

[FN172]. [Id. at 1315 n.16](#).

[FN173]. Courts opining otherwise, as in Mr. Maiorana's case in *In re Eastern and Southern Districts Asbestos Litigation*, or in the intermediate appellate ruling in *Landrigan*, have been confused. See *supra* notes 59-65 and accompanying text.

[FN174]. [Fed. R. Evid. 702](#); see *supra* note 75.

[FN175]. [People v. Collins, 438 P.2d 33 \(Cal. 1968\)](#).

[FN176]. [Id. at 34](#).

[FN177]. *Id.*

[FN178]. *Id.*

[FN179]. *Id.* at 37.

[FN180]. *Id.* at 38. To illustrate the product rule, the mathematician explained that the probability of rolling a "2" on any one roll of a die is 1/6, and that the probability of rolling two "2's" in succession is 1/6 x 1/6, or 1/36. *Id.* at 37 n.8.

[FN181]. *Id.* at 33.

[FN182]. *Id.* at 38-39.

[FN183]. *Id.* By contrast, we derive the 1/6 probability in the exemplar from the known fact that the die has six faces. Moreover, were the die biased in such a way that the probability of rolling a "2" following any prior roll of a "2" was greater or less than 1/6, then the product rule would fail.

[FN184]. *Id.* at 40.

[FN185]. *Id.* An issue is whether the real problem in *Collins*-like situations is that the police are likely to stop their investigation once they have found a single match. The correct statistical question should be: how likely is it, when police continue to select couples out of the suspect population pool, that they would get more than one match?

[FN186]. [58 N.E.2d 754 \(Mass. 1945\)](#).

[FN187]. [Id. at 754-55](#).

[FN188]. [Id. at 755](#).

[FN189]. *Id.*

[FN190]. [29 N.E.2d 825 \(1940\)](#).

[FN191]. [Smith, 58 N.E.2d at 755 \(quoting Sargent, 29 N.E.2d at 827\)](#).

[FN192]. See [Howard v. Wal-Mart Stores, 160 F.3d 358, 360 \(7th Cir. 1998\)](#). More formally, however, "statistical" evidence involves observations that are interpreted under a probability model. See Beverly G. Mellon, *A Likelihood Approach to DNA Evidence*, in *Statistics for Social Science and Public Policy*, *supra* note 52, at 125, 128.

[FN193]. [Howard, 160 F.3d at 360](#).

(Cite as: 108 Penn St. L. Rev. 479)

[FN194]. See Federal Cts. Study Comm., Report of the Federal Courts Study Committee 97 (1990) (noting that "[e]conomic, statistical, technological, and natural and social scientific data are becoming increasingly important in both routine and complex litigation"); see generally Gerald W. Boston, [A Mass- Exposure Model of Toxic Causation: The Content of Scientific Proof and the Regulatory Experience](#), 18 Colum. J. Envtl. L. 181, 382 (1993) (emphasizing that courts are increasingly relying upon scientific evidence in toxic tort cases); Joe S. Cecil & Thomas E. Willging, [Accepting Daubert's Invitation: Defining a Role for Court-Appointed Experts in Assessing Scientific Validity](#), 43 Emory L.J. 995, 996 (1994) (arguing that the idea of court-appointed experts is likely to receive increasing attention as the Daubert admissibility criteria, governing [Federal Rules of Evidence 702](#) and [703](#), are applied to the growing amount of scientific and technical evidence used in litigations); Brenda Inman Rowe, Note, [A Possible Solution for the Problem of Juries Slighting Nonscientific Evidence: A Bayesian-Like Judicial Instruction](#), 24 Am. J. Crim. L. 541, 542 (1997) (discussing the speculation about whether juries are capable of evaluating "the sophisticated scientific evidence that is increasingly used in criminal trials").

[FN195]. See Federal Cts. Study Comm., supra note 194, at 97.

[FN196]. See [DeLuca v. Merrell Dow Pharm., Inc.](#), 911 F.2d 941, 958-59 (3d Cir. 1990); [Sanderson v. International Flavors & Fragrances](#), 950 F. Supp. 981, 999-1000 (C.D. Cal. 1996); [Hall v. Baxter Healthcare Corp.](#), 947 F. Supp. 1387, 1403 (D. Or. 1996); [In re Agent Orange Prod. Liab. Litig.](#), 597 F. Supp. 740, 785 (E.D.N.Y. 1984) (asserting that plaintiffs must prove at least a two-fold increase in the incidence of the disease allegedly caused by exposure to the suspected toxic substance).

[FN197]. [384 S.E.2d 775 \(1989\)](#).

[FN198]. [Id.](#) at 781-82.

[FN199]. [Id.](#) at 783.

[FN200]. [Id.](#) at 791.

[FN201]. [Id.](#) at 783.

[FN202]. See generally Paul C. Giannelli, *The DNA Story: An Alternative View*, 88 J. Crim. L. & Crimin. 380 (1997) (critiquing the overwhelming judicial acceptance).

[FN203]. See 3 David L. Faigman et al., *Modern Scientific Evidence: The Law and Science of Expert Testimony* § 25-1.2, at 211 (2002).

[FN204]. William C. Thompson, *Forensic DNA Evidence*, in *Expert Evidence: A Practitioner's Guide* (Patrick Lee & Bert Black eds., 1997), quoted in Richard O. Lempert et al., *A Modern Approach to Evidence: Text, Problems, Transcripts and Cases* 1117, 1134 (3d ed. 2000).

[FN205]. [Id.](#); see also Seymour Geisser, *Statistics, Litigation, and Conduct Unbecoming*, in *Statistics for Social Science and Public Policy*, supra note 52, at 73 (noting that the variable numbers of tandem repeats probed during DNA enzyme cutting are subject to measurement error using the restricted fragment length polymorphisms method, also discussed in the Spencer case).

[FN206]. David J. Balding, *Interpreting DNA Evidence: Can Probability Theory Help?*, in *Statistics for Social Science and Public Policy*, supra note 52, at 65-66.

[FN207]. [Id.](#) at 65.

[FN208]. [Id.](#)

[FN209]. [Id.](#) at 66.

[FN210]. See [Fed. R. Evid. 102](#) (prescribing that the evidentiary rules shall be construed, inter alia, toward the end of

(Cite as: 108 Penn St. L. Rev. 479)

ascertaining the truth).

[FN211]. Tribe, *supra* note 84, at 1376.

[FN212]. See Gerald Torres, Law, Knowledge, and the Academy: [Translation and Stories](#), 115 *Harv. L. Rev.* 1362, 1363 (2002).

[FN213]. Nesson, *supra* note 22, at 1357; see also Emile Durkheim, *The Division of Labor in Society* 108 (G. Simpson trans. 1933) (arguing that legal outcomes and punishments are designed to convey norms to upright citizens).

[FN214]. See Aharon Barak, [Foreword: A Judge on Judging: The Role of a Supreme Court in a Democracy](#) 116 *Harv. L. Rev.* 16, 29 (2002) (stressing that "[t]he judge must ensure stability with change, and change with stability").

[FN215]. Tribe, *supra* note 84, at 1358.

[FN216]. *Id.* at 1372-76.

[FN217]. Nesson, *supra* note 22, at 1362. But cf. Robert L. Birmingham & Nancy J. Dunham, *An Evidentiary Value Reading of Naked Statistical Proofs*, 31 *St. Louis U. L.J.* 797, 817 (1987) (critiquing Nesson's argument objecting to the use of statistical proofs and proposing a new model rooted in the sensitivity of the evidentiary mechanism to principal facts).

[FN218]. See [In re Agent Orange Prod. Liab. Litig.](#), 611 *F. Supp.* 1223, 1240 (D.C.N.Y. 1985); see also David L. Bazelon, *Science and Uncertainty: A Jurist's View*, 5 *Harv. Envtl. L. Rev.* 209 (1981); Michael Dore, *A Commentary on the Use of Epidemiological Evidence in Demonstrating Cause-in-Fact*, 7 *Harv. Envtl. L. Rev.* 429, 431 (1983); Richard J. Heafey, *Trial by Lottery: The Misuse of Epidemiology and Statistics To Prove Causation in Drug and Chemical Litigation*, 12 *Am. J. Trial Advoc.* 19 (1988); Donald W. Large & Preston Michie, *Proving that the Strength of the British Navy Depends on the Number of Old Maids in England: A Comparison of Scientific Proof with Legal Proof*, 11 *Envtl. L.* 555, 563-68 (1981); Richard W. Wright, [Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof: Pruning the Bramble Bush by Clarifying the Concepts](#), 73 *Iowa L. Rev.* 1001, 1061-65 (1988).

[FN219]. See, for example, the advisory committee note accompanying [Fed. R. Evid. 804\(b\)\(2\)](#), which explains that the dying declaration exception to the hearsay rule is rooted traditionally in "the exceptional need for the evidence in homicide cases."

[FN220]. See Barry Scheck et al., *Actual Innocence* 246 (2000) (discussing sixty-seven DNA exonerations obtained by Cardozo Law School's Innocence Project); see also Edward Connors et al., *Convicted by Juries, Exonerated by Science: Case Studies in the Use of DNA Evidence To Establish Innocence After Trial* (1996) (discussing twenty-eight cases); Jennifer Boemer, [Granting Post-Conviction Deoxyribonucleic Acid \(DNA\) Testing to Inmates](#), 27 *Wm. Mitchell L. Rev.* 1971, 1997-2000 (2001) (noting that most of the Innocence Project's exonerations involved a situation in which the DNA technology responsible had not been available at the time of trial); Naftali Bendavid, *For Innocent, DNA Proving Sturdy Ally in Five Years--The Innocence Project has Freed 32 Convicts Through DNA Testing*, *Chi. Trib.*, Oct. 27, 1997, at A4; see also Mark Donald, *Lethal Rejection: Jeanette Popp Still Mourns Her Murdered Daughter. Now She's Become a Compelling Voice in the Debate over the Death Penalty. Guess Which Side*, *Dallas Observer*, Dec. 12, 2002 (reporting that, "[a]ccording to the Innocence Project, false confessions or admissions were involved in 27 of the first 111 post-conviction DNA exonerations in this country. Last week in Manhattan, prosecutors would ask a judge to throw out the conviction of five men in the infamous 'Central Park jogger' case, after DNA evidence proved that the confessions given by the men--imprisoned for 13 years--were false.").

[FN221]. E.g., [Spencer v. Commonwealth](#), 384 *S.E.2d* 775 (1989).

[FN222]. E.g., *State v. Thompson*, WD 57595, 2001 Mo. App. LEXIS 903, at * 12 (June 5, 2001) (discussing testimony that "it is common to find a trail of the assailant's blood leading from the victim's body to the bathroom, where assailants go to clean up and tend to their wounds"), *rev'd on other grounds*, 68 *S.W.3d* 393 (Mo. 2002).

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[FN223]. E.g., *State v. Brooks*, C1-01-1253, 2002 Minn. App. LEXIS 651, at *9-10 (June 11, 2002) (noting that defense counsel had elected not to obtain DNA results on the hair found in the victim's hand because they suspected it might have been the defendant's hair).

[FN224]. But see Todd D. Brown, Comment, *The Power Line Plaintiff and the Inverse Condemnation Alternative*, 19 *B.C. Envtl. Aff. L. Rev.* 655, 666 (1992) (reasoning from traditional law school principles, asserting that "[b]asing liability on the results of epidemiological data not only would be improper, but would violate the well-established legal precedent that a plaintiff must prove actual causation to recover in tort").

[FN225]. See *DeLuca v. Merrell Dow Pharm., Inc.*, 911 F.2d 941, 954 (3d Cir. 1990) (noting that "the reliability of expert testimony founded on reasoning from epidemiological data is generally a fit subject for judicial notice; epidemiology is a well-established branch of science and medicine, and epidemiological evidence has been accepted in numerous cases"); James S. Liebman, *Desegregating Politics: "All-Out" School Desegregation Explained*, 90 *Colum. L. Rev.* 1463, 1521-22 (1990) (recognizing that "litigants and judges in toxic tort cases can utilize the conclusions of a well-established and scientifically accepted medical field--epidemiology--to determine whether and with what frequency certain chemical agents cause certain kinds of cancer"); William C. Thompson & Simon Ford, *DNA Typing: Acceptance and Weight of the New Genetic Identification Tests*, 75 *Va. L. Rev.* 45, 79 (1989) (reporting that DNA sequencing has become well accepted in the scientific community since it was developed in the early 1970s, and has "certainly become a key tool in the field of molecular biology"); see also Laurel Beeler & William R. Wiebe, Comment, *DNA Identification Tests and the Courts*, 63 *Wash. L. Rev.* 903, 954 (1988) (noting that the ability to link DNA outcomes to individuals is well established in the scientific community, and that "non-forensic DNA tests are used extensively in other scientific disciplines, demonstrating their reliability and acceptance by the general scientific community").

[FN226]. See supra note 192.

[FN227]. See supra notes 175-85.

[FN228]. See supra notes 186-91.

[FN229]. Nesson, supra note 22, at 1362 n.17.

[FN230]. See also Birmingham & Dunham, supra note 217, at 804 (asking "[p]ractically, would a citizen, upon observing in the prisoner case that the state prevailed not on an ordinary eyewitness proof, but on a .96 probability obtained by a naked statistical proof, for that reason resolve, 'I will murder if it is expedient that I do so?'). This critique applies, of course, even against traditionally condemned statistical proofs.

[FN231]. Tribe, supra note 84, at 1354; Michael O. Finkelstein & William B. Fairley, *A Bayesian Approach to Identification Evidence*, 83 *Harv. L. Rev.* 489 (1970) (suggesting that Bayesian probability analysis be incorporated in certain trial settings); see also Richard Lempert, *The New Evidence Scholarship: Analyzing the Processes of Proof*, 66 *B.U. L. Rev.* 439, 476 (1986) (arguing that the use of Bayes's Theorem would augment "justice in the sense of accurate verdicts").

[FN232]. See Alan M. Turing, *Computing, Machinery and Intelligence*, 49 *Mind* 433 (1950) (asking "can machines think?" and devising his "imitation game" substituting a machine for a man); see also Ray C. Paton et al., *An Examination of Some Metaphorical Contexts for Biologically Motivated Computing*, 45 *British J. Phil. Science* 505 (1994).

[FN233]. Nancy Pennington & Reid Hastie, *Evidence Evaluation in Complex Decision Making*, 51 *J. Personality & Soc. Psychol.* 242, 243 (1986); see generally W. Lance Bennett & Martha Feldman, *Reconstructing Reality in the Courtroom: Justice and Judgment in American Culture* 276-81 (1981) (advancing the story model of case presentation).

[FN234]. See supra note 220; see also Tom Perrotta & Daniel Wise, *Morgenthau Calls for Dismissal of All Jogger Case Convictions: District Attorney Persuaded by New DNA Evidence and Confession*, *N.Y. L.J.*, Dec. 6, 2002, at 1.

[FN235]. See generally Sean B. Carroll et al., *From DNA to Diversity: Molecular Genetics and the Evolution of Animal Design* (2001).

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