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CAUSATION, STATISTICAL EVIDENCE, AND TOXIC TORTS

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In this paper, I present a puzzle about how courts react to statistical evidence. The basic puzzle is that while some types of statistical evidence are considered insufficient to establish causation, other types of statistical evidence are considered sufficient. While the types of statistical evidence that are cyonsidered insufficient to establish causation have received significant scholarly attention, much less attention has been paid to the types of statistical evidence that courts consider sufficient.[1] The latter types of statistical evidence are especially prominent in toxic tort cases, which makes such cases a natural springboard for exploring solutions to the puzzle.

This paper proceeds in three stages. First, I set out what I take to be the basic intuitive puzzle, as well as some prominent views on why establishing causation on the basis of certain types of statistical evidence is problematic. To follow, I discuss in some detail the nature of toxic tort cases and how statistical evidence is generally utilized to establish causation. To close, I show how prominent accounts are unable to address this puzzle about the reaction of courts to different types of statistical evidence, and I put forward a tentative solution that both aligns with bedrock legal principles and is supported by philosophical argument.

1. THE PUZZLE OF STATISTICAL EVIDENCE

Let us start with an example that is often used to illustrate the problem with statistical evidence:

> *Blue Bus*: There are two bus companies in town, the Blue Bus Company and the Grey Bus Company. One day, an out-of-control bus injures Sal.

1 For influential discussions of the insufficiency of statistical evidence (and related) questions, see Tribe, "Trial by Mathematics"; Cohen, *The Probable and the Provable*; Brook, "Inevitable Errors"; Thomson, "Remarks on Causation and Liability"; Posner, "An Economic Approach to the Law of Evidence"; and Redmayne, "Exploring the Proof Paradoxes." A notable exception to the lack of discussion of the sufficiency of statistical evidence is Hawthorne et. al, "Statistical Evidence and Incentives in the Law."

> Sal, who saw that it was a bus that caused his injuries, brings a claim for damages. To establish that it is more likely than not that the Blue Bus Company caused Sal's injuries, Sal points out that the Blue Bus Company owns and operates 80 percent of the buses on local bus routes. The Blue Bus Company concedes that the bus that hit Sal was being operated negligently; however, it contests that the evidence presented by Sal is sufficient to establish that it is more likely than not that the Blue Bus Company caused his injury. Since the Blue Bus Company concedes the question of negligence, the only issue that the judge must rule on to establish liability is whether the evidence is sufficient to establish that it is more likely than not that the Blue Bus Company caused Sal's injuries. The judge concludes that the evidence presented by Sal is sufficient to establish the likelihood of causation and thus that the Blue Bus Company is liable for damages.[2]

Most people have the intuition that holding the Blue Bus Company liable in this case is somehow inappropriate. This is *prima facie* puzzling since, to meet the preponderance of evidence burden with respect to causation, one needs only to establish that it is more likely than not that the defendant caused the plaintiff's injuries. As such, if the judge concludes that in light of the evidence presented, it is more likely than not that a bus from the Blue Bus Company hit Sal, then it is not clear what would stand in the way of a finding of liability.

Many scholars are tempted to say that a finding of liability in Blue Bus is inappropriate because the statistical evidence presented is insufficient to establish the likelihood of causation. They contrast such evidence with what they call *individualized* or *particular* evidence, such as eyewitness testimony. For instance, if an eyewitness testified that the bus that hit Sal was blue, most agree that this would be sufficient to establish the likelihood of causation, even if eyewitnesses sometimes make mistakes in identifying the color of buses. The intuitive difference between statistical and individualized evidence seems to be preserved even if, given the Blue Bus Company's market share and the rate of errors in eyewitness testimony, the likelihood that it caused injury in each case is the same.

Before putting forward some more specific proposals as to the supposed problem with the type of statistical evidence presented in Blue Bus, a nearby case is worth considering:

> *Blue Lung*: For over twenty years, the Nuclear Dump Company has been illegally emitting a specific type of toxic fume that is known to cause a

2 The first application of the Blue Bus scenario to the statistical evidence issue was in Thomson, "Liability and Individualized Evidence." It is based on the fact pattern of *Smith v. Rapid Transit Inc.*, 317 Mass 469 (Mass 1945).

> rare disease called blue lung. After contracting blue lung, nearby resident Maria sues the Nuclear Dump Company in civil court. Maria's doctor testifies that in her professional opinion, it is very likely that Maria contracted blue lung as a result of inhaling the specific type of toxic fume that the Nuclear Dump Company emits and incredibly unlikely that she contracted it in an unrelated manner. The Nuclear Dump Company concedes that it negligently emitted toxic fumes but contests the sufficiency of Maria's evidence to establish that toxic fumes caused Maria's illness. The judge in the case finds Maria's evidence sufficient to establish that it was more likely than not that emissions caused her illness and, since the Nuclear Dump Company already conceded its negligence, finds it liable for damages.

While finding the Blue Bus Company liable for damages seems inappropriate, the case against Nuclear Dump Company is in many respects stronger. At a minimum, as we will see below, courts certainly treat such cases differently. For now, however, it is simply worth noting that insofar as we think that there is a relevant difference between these cases, then we will have a puzzle on our hands. This is because, at least *prima facie*, the evidence used to establish the likelihood of causation in both Blue Bus and Blue Lung is purely statistical.

With these cases in mind, let us take a look at some more specific proposals as to the problem with purely statistical evidence. While this list is not exhaustive, it is meant to provide a basic idea of the diversity of proposals that have been put forward in this regard.[3] Regardless of which of these proposals we adopt, it will be the case that in the context of toxic torts, non-individualized, purely statistical evidence ought to be insufficient to establish that it is more likely than not that the defendant's product caused the plaintiff's injuries.

> *Causal Account*: Purely statistical evidence is problematic because it lacks the appropriate causal link to the proposition for which it is taken to be evidence.[4]

> *Sensitive Account*: Purely statistical evidence is problematic because a belief in liability based on such evidence will not track the truth of the matter. In particular, if someone other than the defendant were liable,

3 For discussion of a slightly expanded list, see Enoch and Fisher, "Sense and Sensitivity," 565–71.

4 This causal account is based on the view put forward in Thomson, "Remarks on Causation and Liability" and "Liability and Individualized Evidence." A similarly causal view is put forward by Wright, "Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof."

> the juror or judge (most probably) would still have formed the belief that the defendant was liable.[5]
>
> *Normal Account*: Purely statistical evidence is problematic because it does not provide normic support for the truth of the proposition for which it is taken to be evidence. In particular, the statistical evidence does not make it the case that the falsity of the proposition would require more explanation than its truth.[6]

The plan for the rest of the paper is as follows. First, in section 2, I discuss in some detail how courts handle the evidence used to establish the likelihood of causation in toxic tort cases. To follow, in section 3, I argue that none of the above accounts can satisfactorily account for such cases. To close, in section 4, I put forward my own tentative proposal as to why courts treat these cases differently that both aligns with fundamental legal principles and is on solid moral and epistemological ground.

2. TOXIC TORTS

In this section, I provide a basic discussion of toxic torts and a more detailed discussion of one way in which a plaintiff can establish that it is more likely than not that the defendant's product caused the plaintiff's injuries. While this is not the only way in which a plaintiff can establish the likelihood of causation, it is the simplest illustration of how statistical evidence is put to use in this area of the law.

One helpful way of thinking about toxic torts is put forward by Bert Black and David Lilienfield, who define toxic tort cases as follows:

> Toxic tort cases [are] those in which the plaintiff seeks compensation for harm allegedly caused by exposure to a substance that increases the risk of contracting a serious disease, but does not cause an immediately apparent response. These cases generally involve a period of latency or incubation prior to the onset of the disease. In most cases the increased risk of the disease does not diminish or dissipate, even with the cessation of exposure.[7]

5 This sensitive account is based on the view put forward by Enoch et al, "Statistical Evidence, Sensitivity, and the Legal Value of Knowledge"; Enoch and Fisher, "Sense and Sensitivity"; and Enoch and Spectre, "Sensitivity, Safety, and the Law."

6 This normal account is based on the view put forward by Smith, "When Does Evidence Suffice for Conviction?"

7 Black and Lilienfield, "Epidemiologic Proof in Toxic Tort Litigation," 732.

Well-known examples of toxic tort cases include claims of harms after exposure to asbestos, Agent Orange, insecticides, hazardous wastes, and lead paint.

In a toxic tort case, a plaintiff must establish both the defendant's negligence and her own damages.[8] Probably the most challenging, controversial, and contested element in any toxic tort case, however, is causation.[9] Even from our simple description of toxic torts above, some of these challenges should be apparent. In particular, exposure to harmful chemicals may result in harm and injuries decades later, and in almost all cases, exposure simply increases an individual's risk of suffering injuries, as opposed to ensuring that such harms will obtain. This is in addition to the fact that quite often, the chemicals being put into use are not fully understood by scientists in the field. Ora Fred Harris Jr. expounds on this problem quite eloquently:

> A common, generally accurate, evaluation of humankind's understanding of the behavior of hazardous or toxic wastes and the effect of exposure on humans points to a vast amount of scientific uncertainty. … Thus, a plaintiff attempting to establish that exposure to a particular substance has in fact caused his or her injury may face a dubious court or jury because of the lack of scientific certainty. Moreover, because this "new" tort injury can have a latency period of up to as many as twenty to thirty years, it may be, as a practical matter, virtually impossible to establish the requisite causal relationship between an exposure that may have taken place many decades ago and a recently manifested injury now claimed to be the consequence of that exposure. Not only does this long latency period stymie the toxic or hazardous exposure victim's ability to isolate the alleged substance that precipitated the injury, it also diminishes the chances of identifying the responsible parties.[10]

Faced with these complex issues, courts have developed a nuanced approach to causation in toxic tort cases. In order for a plaintiff's claim to be successful, she must establish both general and specific causation.[11] Conceptually speaking, in a case in which it is alleged at trial that an *F* caused a *G*, the question of general causation is "Can an *F* cause a *G*?"—which the plaintiff must prove is

8 For an excellent discussion of these elements of the plaintiff's case, see Roisman et. al, "Preserving Justice."

9 For more on causation in tort law, as well as issues that arise in difficult cases, see Wright, "Causation in Tort Law." For a discussion focused on causation in toxic tort litigation, see Conway-Jones, "Factual Causation in Toxic Tort Litigation."

10 Harris, "Toxic Tort Litigation and the Causation Element," 912.

11 For a helpful discussion of this distinction, see Gold, "The 'Reshapement' of the False Negative Asymmetry in Toxic Tort Causation," 1511.

more likely than not; and the question of specific causation is "Did an *F* cause this particular *G*?"—where the same burden of proof applies. To take an example outside of the toxic tort context, if it is alleged that a mosquito bite caused a seizure, the question of general causation is "Can a mosquito bite cause a seizure?" while the question of specific causation is "Did a mosquito bite cause this particular seizure?"

Returning to the context of toxic torts, in order to establish general causation, the plaintiff must establish that the chemical or substance in question can cause the sort of harm that the plaintiff suffered in the population at large—or at least in a subgroup of the population to which the plaintiff belongs.[12] In order to establish specific causation, the plaintiff must establish that the chemical or substance in question actually caused the harm that the plaintiff suffered. The burden of proof with respect to both the general and specific causation tests is a preponderance of evidence. This means that in order to prevail at trial, the plaintiff must establish that it is more likely than not that the defendant's product can cause the harm the plaintiff suffered in the general population and that it more likely than not caused the plaintiff's actual harm.

Notice that these questions are not independent. First, establishing general causation is necessary for establishing specific causation. In other words, unless it is more likely than not that an *F* can cause a *G*, then it will not be more likely than not that an *F* caused a particular *G*. Some courts have explicitly noted this, writing that testimony on specific causation "is unnecessary" if general causation cannot be established.[13] In addition, establishing specific causation is sufficient for establishing general causation. That is, if it is more likely than not that a particular *F* caused a particular *G*, then it is more likely than not than an *F* can cause a *G*. Some courts have explicitly noted this as well, writing that although the plaintiff must establish both general and specific causation, the court's "ultimate focus" is on specific causation.[14] With this in mind, if evidence presented establishes specific causation, then the court's relevant inquiry will be answered.

Let us take a look at the general and specific causation requirements in turn. One relatively straightforward manner in which a plaintiff can establish general causation is by showing that exposure to the defendant's product at

12 On the history of the distinction between specific and general evidence, see Gold, "The 'Reshapement' of the False Negative Asymmetry in Toxic Tort Causation," 1513. For arguments against this distinction, at least in determinations of standing, see "Causation in Environmental Law."

13 *Dunn v. Sandoz Pharmaceuticals Corp.*, 275 F. Supp. 2d 672, 676 (M.D.N.C. 2003).

14 *Henricksen v. ConocoPhillips Co.*, 605 F. Supp 2s 1123, 1176 (E.D. Wash 2009).

least doubles people's risk of harm.[15] This doubling of risk is often described as people exposed to the chemical having a "relative risk" of greater than 2. While not without controversy, some courts have explicitly connected their choice of a relative risk of greater than 2 to the fact that the applicable burden of proof is a preponderance of evidence.[16] Simply put, if being exposed to a chemical more than doubles people's risk of suffering a particular harm, and an individual suffers that harm after being exposed to that chemical, then it is "more likely than not" that individuals exposed to that chemical will be harmed. An example of a court explicitly embracing such reasoning is the Texas Supreme Court in *Merrell Dow v. Havner*:

> Assume that a condition naturally occurs in six out of 1,000 people even when they are not exposed to a certain drug. If studies of people who did take the drug show that nine out of 1,000 contracted the disease, it is still more likely than not that causes other than the drug were responsible for any given occurrence of the disease since it occurs in six out of 1,000 individuals anyway. . . . However, if more than twelve out of 1,000 who take the drug contract the disease, then it may be statistically more likely than not that a given individual's disease was caused by the drug.[17]

The court in *Havner* recognized that such reasoning is controversial. In particular, it recognized that drawing such a simple link between epidemiological findings and the legal burden of proof may be overly simplistic.[18] Even if the relationship between epidemiological findings and a burden of proof might not be one to one, however, the court concluded that "there is a rational basis for relating the requirement that there be more than a 'doubling of the risk' to our no evidence standard of review and to the more likely than not burden of proof."[19]

Even if the defendant concedes that the relative risk of its chemical to people is greater than 2, which would establish general causation, there remains the question of whether the chemical actually caused the plaintiff's injuries, which

15 For a thorough discussion of how different types of epidemiological studies can be utilized to prove both general and specific causation, including that people's relative risk is greater than 2, see Beecher-Monas, *Evaluating Scientific Evidence*, ch. 4.

16 Note that my claim is not that a relative risk greater than 2 is necessary to establish general causation, simply that it is sufficient.

17 *Merrell Dow Pharmaceuticals Inc. v. Havner*, 953 S.W. 2d 706, 717 (Tex. 1997). Reaffirmed in *Merck & Co., Inc. v. Garza*, 347 S.W. 3d 256 (Tex. 2011). See also *Cagle v. Cooper Companies (In re Silicone Gel Breast Implants Prod. Liab. Litig.)*, 318 F. Supp. 2d 879, 892 (C.D. Cal. 2004).

18 For an insightful critique along these lines, see Gold, "Causation in Toxic Torts"; and Kaye, "Apples and Oranges."

19 *Merrell Dow v. Havner*, 717.

is the question of specific causation.[20] In order to establish the claim that more likely than not, the chemical actually did harm the plaintiff, the plaintiff must rule out other plausible causes of her injuries. To take a classic example, while exposure to asbestos can increase a plaintiff's risk of lung cancer, recovery will be complicated if the plaintiff is also a heavy smoker.[21] Given the prolonged latency periods of many harmful chemicals, as well as the complex etiology of many diseases such as cancer, ruling out other plausible causes of a plaintiff's injuries is generally a central part of establishing specific causation.[22]

In order to address other plausible causes of her injuries, thus establishing specific causation, the plaintiff generally provides a "differential diagnosis" or, more accurately described, a "differential etiology."[23] In short, a differential etiology is "a patient-specific process of elimination that medical practitioners use to identify the 'most likely' cause of a set of signs and symptoms from a list of possible causes."[24] Differential etiology is often analogized to differential diagnosis because while the latter involves narrowing down to one likely ailment for purposes of providing care, the former involves narrowing down to one likely cause of the patient's ailment for purposes of establishing liability. As Ronald E. Gots puts it, "differential diagnosis is a quest for a diagnosis: what is wrong with the patient internally. It is not, inherently, a search for the ultimate cause (critical to liability) of that disease process or disorder."[25]

For our purposes, there are two important things to note about differential etiology. First, the goal of differential etiology is to examine and eliminate (or significantly decrease the likelihood) of other plausible causes of the plaintiff's injury, as opposed to putting forward any sort of story (causal or otherwise) that links the defendant's activity to the plaintiff's injuries. This is important because by eliminating other potential causes of the plaintiff's injury, the

20 There are of course a number of ways that the defendant might challenge the plaintiff's claim that the epidemiological studies presented demonstrate a relative risk of at least 2. I put these aside for the present discussion.

21 For more discussion of the difficulties associated with asbestos litigation, see White, "Asbestos and the Future of Mass Torts."

22 For a case in which a defendant was granted summary judgment in light of a failure to establish specific causation in this manner, see *Lennon v. Norfolk & W. Ry. Co.*, 123 F. Supp. 2d 1143, 1154 (N.D. Ind. 2000).

23 For an excellent discussion of how differential etiology is handled by the courts, see Sanders and Machal-Fulks, "The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases"; and Sloboda, "Differential Diagnosis or Distortion."

24 *Hall v. Baxter Healthcare Corp.*, 947 F. Supp. 1387, 1413 (D. Or. 1996).

25 Gots, "From Symptoms to Liability," 25. For further commentary on certain mistakes that courts make in handling epidemiological evidence in particular and scientific evidence more generally, see Bryant and Reinert, "The Legal System's Use of Epidemiology."

likelihood that the defendant caused the injury will increase, but this is only via indirect argument. As the Texas Supreme Court put this point:

> There can be many possible "causes," indeed, an infinite number of circumstances can cause an injury. But a possible cause only becomes "probable" when in the absence of other reasonable causal explanations it becomes more likely than not that the injury was a result of its action.[26]

The second, related point is that differential etiology is meant to examine other *plausible* causes of the plaintiff's injury.[27] As such, by its very nature, it will not examine unlikely sources of injury, thus leaving entirely open the possibility that the plaintiff's injuries were caused in a rare or extraordinary manner. The fact that such fanciful causal possibilities need not be explored further underscores my earlier point that at root, the goal of differential etiology is simply to make the defendant's liability for the plaintiff's injuries more likely.

Let us sum up. In order for a plaintiff to establish that a defendant's product caused her injuries, she must establish both general and specific causation. In order for a plaintiff to establish both general and specific causation, she can demonstrate both a relative risk to people of at least 2 and, via differential etiology, that other plausible causes of her injuries are unlikely. With this in mind, we can now revisit the accounts put forward in section 1 to see how they apply to the evidence that is commonly put forward to establish the likelihood of causation in toxic tort cases.

3. Specific Causation and Statistical Evidence

In this section, I discuss how the three accounts of the problematic nature of statistical evidence discussed in section 1 would categorize the evidence used to establish the likelihood of causation in toxic tort cases. What I will show is that regardless of which of these accounts we favor, the evidence that suffices to establish the likelihood of causation in toxic tort cases will be considered problematic. In particular, I argue that evidence that suffices to establish general causation is purely statistical, and, perhaps even more importantly, evidence that suffices to establish specific causation is purely statistical. The latter claim is even more important because, as discussed above, evidence used to establish specific causation is *ipso facto* evidence that is used to establish general

26 *Parker v. Employers Mutual Liability Insurance Co. of Wisconsin*, 440 W. 23 43, 47 (Tex. 1969).

27 For instance, in *Soldo v. Sandoz Pharms. Corp.*, 244 F. Supp. 2d 434, 551–52 (W.D. Pa. 2003), the court held that the plaintiff must rule out alternatives that the defendant shows to be plausible. But see *Wade-Greaux v. Whitehall Labs., Inc.*, 874 F. Supp. 1441, 1473 (D.V.I. 1994).

causation, so evidence used to establish general causation cannot have a property that evidence used to establish specific causation lacks.

As discussed above, plaintiffs can establish that it is more likely than not that the defendant's product caused their injuries in toxic tort cases by both showing that the defendant's product increased their relative risk by a factor of two (general causation) and that other plausible causes of the plaintiff's injuries were absent (specific causation). There should be little doubt that only statistical evidence is necessary to establish general causation. In order to show that the defendant increased the plaintiff's relative risk by a factor of two, the plaintiff must provide epidemiological studies showing that individuals who are exposed to the relevant chemicals or substances tend to suffer injuries similar to the plaintiff at double the rate of those who are not.[28] A showing that a chemical or substance increases the risk of a particular injury is patently statistical. As such, any scholar who finds statistical evidence problematic is likely to find relative risk findings problematic.

Even if evidence used to establish general causation were not statistical, if evidence used to establish specific causation were statistical, this would suffice to show that evidence used to establish causation is statistical. With this in mind. a question that is more important to consider is whether differential etiology testimony, or testimony with respect to specific causation, is purely statistical. If differential etiology testimony is in fact purely statistical, then a court's treatment of such evidence will present a *prima facie* challenge to those who believe that statistical evidence ought to be insufficient to establish the likelihood of causation.

To put my cards on the table, I think that differential etiology testimony is purely statistical. If I am right, then if we think that it is problematic to use purely statistical evidence to establish the likelihood of causation, we are likely to think it is similarly problematic to use differential etiology testimony in this manner. After all, as pointed out above, the doctor offering differential etiology testimony is not directly arguing that the defendant's activities caused the plaintiff's injury; instead, the doctor is testifying with respect to the likelihood of other plausible causes. As such, the doctor is not seeking to conclusively establish that the defendant's activities did in fact cause the plaintiff's injuries; instead, at least as differential etiology analyses are commonly thought of, the doctor is casting doubt on alternative explanations of the plaintiff's injuries.

28 As a reviewer has helpfully noted, additional analysis is necessary to move from epidemiological studies to causation. For further discussion, see Federal Judicial Center, *Reference Manual on Scientific Evidence*, 601. For our purposes, all that is important is that even with this additional analysis, only statistical evidence is necessary to establish both general and specific causation.

It is worth examining each of the accounts of the problem with statistical evidence put forward in section 1 to see why exactly each would consider differential etiology testimony to be as problematic as the statistical evidence presented in Blue Bus. Let us start with Judith Thomson's influential causal account.

> *Causal Account*: Purely statistical evidence is problematic because it lacks the appropriate causal link to the proposition for which it is taken to be evidence.

As Thomson spells out her view, in order for a piece of evidence to be individualized, or for it to be proper grounds for establishing the likelihood of causation, there must be some feature of the defendant's (putative) causing of harm that plays a causal role in the generation of that piece of evidence.[29] For Thomson, such individualized evidence "(putatively) guarantees the defendant's guilt," and, as such, the jury can be "sure beyond a reasonable doubt that there are facts available to it which guarantee that the defendant is guilty."[30]

There is one sense in which we might consider differential etiology testimony as "caused" by the defendant's actions, at least if the defendant actually is liable for the plaintiff's injuries. This is because the differential etiology testimony is caused by (in the sense of based on) the plaintiff's injuries, so if the defendant caused the plaintiff's injuries, then the defendant caused the differential etiology testimony. At the same time, if we dig a little deeper, we see that differential etiology testimony does not satisfy Thomson's requirements for individualized evidence. As stated above, according to Thomson, in order for us to possess individualized evidence that a particular defendant is guilty, there must be a feature that distinguishes the defendant from other possible causes of the plaintiff's injuries that can be assigned the appropriate causal role in producing the evidence. However, there is no unique or contrastive feature of the defendant's emissions in toxic torts cases that play a causal role in the differential etiology testimony. Even if the defendant had not caused the plaintiff's injuries, the differential testimony based on those injuries would be the same, namely, that the most likely cause was the defendant. As such, differential etiology evidence fails to be individualized in the manner Thomson favors.

29 For more discussion, see Thomson, "Liability and Individualized Evidence," 205.

30 Thomson, "Liability and Individualized Evidence, 214–15. A reviewer has questioned Thomson's account on the grounds that if no other buses were on the road, this would not be "caused" by the plaintiff but would surely not be simply statistical. On this point, I think Thomson has the right view. If there are no buses from the Grey Bus Company on the road, for instance, this increases the likelihood that a bus from the Blue Bus Company caused the accident. However, this evidence would be problematic in the same way that Thomson claims that market share evidence alone is.

Next, let us examine the view put forward by David Enoch and others:

> *Sensitive Account*: Purely statistical evidence is problematic because a belief in liability based on such evidence will not track the truth of the matter. In particular, if someone other than the defendant were liable, the juror or judge (most probably) would still have formed the belief that the defendant was.

Enoch et al. claim that if we establish the likelihood of causation based on purely statistical evidence, then it will be pure luck when our verdicts are correct. This is because statistical evidence will be available both when the defendant caused the plaintiff's injuries and when someone or something else did. In Blue Bus, for instance, evidence as to the Blue Bus Company's market share is available in cases in which it is responsible for the plaintiff's injuries as well as those in which the Grey Bus Company is responsible. As such, if a judge or juror reaches a conclusion about the likelihood of causation based on market share evidence, this conclusion will not track the truth of whether the defendant caused the plaintiff's injuries.[31] Enoch et. al contrast conclusions based on statistical evidence with those based on evidence such as eyewitness testimony. For instance, the testimony of an eyewitness who claims to have seen a bus from the Blue Bus Company hit the plaintiff would presumably not be available if the Grey Bus Company was responsible.[32] Thus, a conclusion with respect to the likelihood of causation based on eyewitness testimony would more successfully track the truth of whether the defendant actually caused the plaintiff's injuries.[33]

31 Note that this analysis applies only if we hold fixed the occurrence of the accident in our evaluation of the relevant counterfactual. If we do not hold fixed the accident's occurrence, then a belief in liability based on market share will be sensitive to the defendant's liability. This is because had the defendant not caused the plaintiff's injuries, most probably no one else would have, and if no one else caused the injuries, then the judge or juror would not have formed the belief in liability.

32 I say "presumably" because a witness may be mistaken.

33 Even this would be undermined if, for instance, the belief is formed on the basis of testimony from an eyewitness who, though telling the truth, would have lied in order to indemnify the defendant regardless. (For instance, see Smith, "When Does Evidence Suffice for Conviction?" 1202.) That said, we might think it particularly problematic if not only was it the case that our beliefs about liability did not track the truth of the matter, but we knew this to be the case ahead of time. Thomson ("Liability and Individualized Evidence") offers a similar line of reasoning on this point. In addition, it is worth noting that the Sensitivity Account does allow for certain types of evidence that we might think of as probabilistic. For instance, if traces of a fingerprint found at the crime scene are consistent with the defendant's, an expert might testify as to the likelihood of someone else having left those same traces. A judgment of guilt based on such evidence would be sensitive to

For reasons quite similar to those discussed above, beliefs formed on the basis of differential etiology testimony will not be sensitive to whether the defendant's product caused the plaintiff's injuries. Differential etiology testimony simply establishes that the defendant's activities were the most likely cause of the plaintiff's injuries; such testimony would still be offered, for instance, if one of the incredibly unlikely causes was actual. As such, if it is inappropriate to reach a conclusion about the likelihood of causation if such a conclusion would lack this sort of counterfactually sensitivity, we will be committed to the claim that the manner in which courts establish the likelihood of causation in toxic tort cases is problematic.[34]

To close this section, let us examine the view put forward by Martin Smith.

> *Normal Account*: Purely statistical evidence is problematic because it does not provide normic support for the truth of the proposition for which it is taken to be evidence. In particular, the statistical evidence does not make it the case that the falsity of the proposition would require more explanation than its truth.

Smith spells out the problematic nature of purely statistical evidence by pointing out that even with such evidence, we would not require any additional explanation if the proposition turned out to be false. To take a concrete example, if all but one ticket in a thousand-ticket lottery were green, then it would statistically be unlikely for a nongreen ticket to win. However, a green ticket winning would not need more explanation than a nongreen ticket winning; after all, some ticket was going to win, and the winning ticket would either be

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the defendant's guilt just as long as, had the defendant not committed the crime, traces of a fingerprint consistent with his would most probably not be found at the crime scene.

34 An anonymous reviewer wondered whether a variant of the sensitivity account might be more successful in this regard. They pointed out that differential etiology testimony might not have even been offered at trial if other causes (such as the plaintiff's own behavior or exposure to a product other than the defendant's) were clearly responsible for the plaintiff's illness. In this way, the very presence of difference etiology testimony at trial entails a certain likelihood that the defendant's product was liable. Even if we accept that the very presence of such testimony entails a certain likelihood that the defendant's product was liable for the plaintiff's injuries, it seems that such an approach does not address the Blue Bus problem. In fact, such an approach seems to fall prey to the same logic of the Blue Bus problem, which is that a certain likelihood of defendant liability is insufficient for a judgment. In response, one might point out that DNA evidence can suffice for a finding of liability, and a finding on this basis is surely better than a finding based on market share evidence. As I discuss in more detail below, however, those who have worries about statistical evidence can surely have worries about DNA evidence as well, and we would insist that, in some fundamental sense, such evidence fails to be an appropriate basis for conviction.

green or not.[35] Smith draws a direct analogy between such lottery cases and the use of statistical evidence in the courtroom. If a plaintiff simply establishes that it is highly likely that the defendant caused the plaintiff's injuries, this is insufficient because if the defendant did not cause the injuries, this would require no additional explanation. This is unlike a case in which, for instance, an eyewitness claims to have seen the defendant commit the crime: with such eyewitness testimony, not only is it unlikely that the defendant is innocent, it is also true that if the defendant actually is innocent, the eyewitness' testimony would need further explanation.

Regardless of what we think of the details of Smith's account, if we adopt it, we will consider a court's treatment of statistical evidence in toxic tort cases to be problematic. This is because the evidence presented in such trials simply tells us the frequency with which we can expect activities such as the defendant's to cause injuries such as the plaintiff's. Differential etiology testimony tells us nothing about which causal pathways would require more or less explanation. In fact, even if the plaintiff succeeds in establishing specific causation, the jury will have no reason to think that any particular cause of the patient's injuries is more or less normal than any other.

Given the above discussion, it seems hopeless to attempt to rule out the type of statistical evidence in Blue Bus on general epistemological grounds but allow for the type of statistical evidence presented in toxic tort cases.[36] Whatever our judgments are about toxic tort cases, they are relatively straightforward instances in which the court allows statistical evidence to establish the likely causal link between the defendant's product and the plaintiff's injuries. Instead of attempting to tease out some epistemological distinction with respect to the evidence presented, it is much more worthwhile to think about why it might be the case, both from a legal and philosophical perspective, that the courts handle the statistical evidence in Blue Bus and toxic tort cases so differently. It is to this task that I now turn.

35 In some sense of the word, drawing a green ticket in the lottery might be abnormal, in the sense of unexpected. However, this is not the sense that Smith is drawing on in developing his account, which focuses on what events would require more or less explanation.

36 Another option suggested by a reviewer is to focus on "case specificity," with the thought being that the evidence presented in differential etiology is specific to the plaintiff in a way that market share evidence is not. While I am open to this possibility, the primary difficulty is that at least at first glance, evidence in the Blue Bus case is case specific. For instance, we must know where the accident take place to establish that it is the bus from the Blue Bus Company that dominates the market in that location, and we must know that the accident was caused by a bus to get the inquiry off the ground.

4. BACK TO THE PUZZLE

At this point, I hope to have made the contours of the puzzle with statistical evidence and toxic torts clear. As stated at the outset of the paper, the basic puzzle is that while courts do not allow for the establishment of causation based on the type of statistical evidence presented in Blue Bus, they do allow for the establishment of causation based on the type of statistical evidence presented in toxic tort cases. In this section, I will first discuss why I think courts treat these types of statistical evidence differently and the legal justification for doing so. To follow, I will put forward some philosophical considerations in favor of the courts' approach.

In Blue Bus, the statistical evidence presented was that the Blue Bus Company operates 80 percent of local bus routes; as a general matter, courts consider this type of evidence insufficient to establish the likelihood of causation. In toxic tort cases, experts provide statistical evidence that the defendant's product is the likely cause of the plaintiff's injuries; as a general matter, this is considered sufficient to establish the likelihood of causation.[37] A legal distinction that we can put to use here is the distinction between direct and indirect (or circumstantial) evidence. As is commonly put in legal texts, direct evidence "proves a fact without an inference or presumption and which in itself, if true, establishes that fact."[38] Indirect evidence, on the other hand, is "evidence from which the fact-finder can infer whether the acts in dispute existed or did not exist."[39] Applying this distinction to the puzzle under discussion, the statistical evidence in toxic tort cases is direct, while the statistical evidence in Blue Bus is indirect.

Before saying more, it is worth being clear that the distinction between direct and indirect evidence is primarily a legal (as opposed to epistemological) one. While I think this legal distinction has important epistemological implications—at least in legal epistemology—it may be one that is most useful in thinking about particular court practices as opposed to, for instance, the proper justification of doxastic attitudes. Since the question I am interested in answering in this paper is why courts treat testimony in toxic tort cases differently than other types of statistical evidence, it is natural to root the discussion in the rules and traditions of the courts.

To see how the distinction between direct and indirect evidence relates to our puzzle, it is helpful to start with the broad distinction between what sort of

37 The language of specific causation is generally reserved for toxic torts; however, we might naturally say that the evidence presented in Blue Bus is insufficient to establish specific causation.

38 Bergman and Hollander, *Wharton's Criminal Evidence*, 1:8, quoting Montana Code Annotated, § 26-1-102(4) (1995).

39 Bergman and Hollander, *Wharton's Criminal Evidence*, 1:8.

determinations jurors are permitted to make "on their own"—by which I mean on the basis of their own judgment even if no evidence was formally admitted through trial procedures—and determinations that jurors are not permitted to make in this manner.[40] Two types of determinations that jurors are permitted to make on their own are as follows: (1) determinations of witness credibility and (2) determinations based on common knowledge of how the world works:

1. As a general matter, jurors can determine on their own whether a particular witness is being truthful. For instance, if a witness provides an alibi for the defendant, jurors can decide whether they think the witness is being honest or deceptive.
2. As a general matter, jurors can make certain inferences based on common knowledge of how the world works. For instance, expert testimony is not necessary to establish, as Mansfield puts it, "that the sun rises in the east or that a bullet fired into the brain will cause serious harm or death."[41] Of course, exactly what constitutes common knowledge of how the world works is contestable. At the same time, what is clear is that once the sort of information necessary for a finding of liability is far enough outside of common knowledge of how the world works, expert testimony or other formally introduced evidence is necessary.

For our purposes, one important example of 2 (i.e., a determination that jurors are not permitted to make on their own) is a determination that the exposure to the defendant's product caused (for instance) mesothelioma.[42] If no evidence was presented at trial that supported the claim that the defendant's product caused mesothelioma, jurors could not simply decide, based on their own personal understanding of the world, that the product caused this illness and the defendant was thus liable for medical expenses. This would remain true even if the defendant's product did cause mesothelioma and the juror—perhaps based on her own background research—knew this to be true.

The primary reason that we do not want jurors determining on their own that the defendant's product caused mesothelioma is that such a process does not allow the defendant appropriate opportunity for rebuttal. This is particularly troubling in criminal cases in which defendants have a constitutional right

40 For discussion, see Kirgis, "The Problem of the Expert Juror"; and Mansfield, "Jury Notice." See also McCormick, *Handbook on the Law of Evidence*; and Wigmore, *Evidence in Trials at Common Law*.

41 Mansfield, "Jury Notice," 395.

42 Another example of 2 is the judgment of whether an expert's methods are reliable; this gatekeeping function of keeping "junk science" out of the courtroom is played by judges. For discussion, see *Federal Rules of Evidence*, 702.

to be confronted by the evidence against them. Even in civil trials, however, allowing juries to make this sort of determination "on their own" is incredibly problematic, if for no other reason than juries' determinations on these matters cannot be vetted and challenged in the manner that expert testimony is.

With this distinction in mind, we can turn to the difference between direct and indirect statistical evidence. The term 'direct statistical evidence' is helpful in addressing our puzzle because such evidence speaks directly to the question that the court is attempting to answer, namely, whether it is more likely than not that the defendant's product caused the plaintiff's injuries. Otherwise put, on the basis of direct statistical evidence, jurors are permitted to decide whether it is more likely than not that the defendant's product caused the plaintiff's injuries. More specifically, jurors can reach this determination on the basis of 1 (e.g., the credibility of the individual providing expert testimony and perhaps of the plaintiff).[43] In contrast, I would argue that jurors ought not be permitted to determine the defendant's liability on the basis of indirect statistical evidence. Indirect statistical evidence about the Blue Bus Company's market share does not establish that it is more likely than not that the Blue Bus Company caused the plaintiff's injuries. In addition, to get from that statistic to a conclusion regarding the Blue Bus Company's liability, jurors would need to do more than draw on 2 (i.e., common knowledge of how the world works). Instead, they would need to take their own private epistemic journeys to arrive at that conclusion. For instance, to move from the premise that the Blue Bus Company runs 80 percent of the local bus routes to the conclusion that it is more likely than not that the Blue Bus Company caused the defendant's injuries, the jury must assume, *inter alia*, that the Blue Bus Company runs buses on the route where the accident was caused, that the accident was not caused by an out-of-town bus, and that the Blue Bus Company does not provide far superior training to its drivers than other bus companies.[44] While each of these claims may be true and may even be known, they ought not be left to jury members to determine on their own.

43 As a reviewer has helpfully noted, the jurors will have to find the plaintiff's testimony credible insofar as the plaintiff's testimony is necessary to establish the range of possible causes of their ailment. Additional determinations, including based on 2 (common knowledge of how the world works), will likely also be necessary in a civil trial. The point here stands insofar as this determination in a toxic tort trial, unlike the relevantly similar determination in a Blue Bus–type trial, will not stray from determinations that are permitted for jurors.

44 The possibility of an out-of-town bus worried the court in *Smith v. Rapid Transit* itself. Of course, if the Blue Bus Company provided far superior training, then the likelihood that it, as opposed to another bus company, caused the accident would be much lower.

As mentioned above, if the plaintiff does not formally present evidence to support the claims that are necessary for a juror's private epistemic journey from indirect statistical evidence to a conclusion about the likelihood of causation, allowing for such a conclusion would conflict with important legal principles. In particular, allowing for such private epistemic journeys conflicts with what Paul F. Kirgis calls the "fundamental principle of the Anglo-American adjudicative system . . . that cases must be decided based solely on evidence formally admitted through trial procedures."[45] If a juror takes a private epistemic journey from a piece of indirect statistical evidence to a conclusion about the probability of causation, this will not allow a defendant the appropriate opportunity to subject the assumptions of the journey to cross-examination or rebuttal.[46] (This is similar to the requirement, in the criminal context, that prosecutors present a "theory of the case" as to where, how, when, and why the defendant supposedly committed the crime of which she is accused.) Such a private epistemic journey is unnecessary when it comes to direct statistical evidence, however, as direct evidence answers a question posed by the court. More specifically, when an expert testifies that a defendant's product is the likely cause of the defendant's injuries, the defendant has the opportunity to challenge this testimony. In addition, the expert has been vetted according to the applicable rules of evidence. In this way, while the epistemic journey of jurors is inaccessible and likely lacking in expert judgment—the epistemic journey of experts has been vetted and is open to direct challenge.[47]

Not only does a court's disparate treatment of direct and indirect statistical evidence align with bedrock legal principle; it is also on solid epistemological and moral ground. This is because it is much more reasonable for a juror to reach a conclusion about the likelihood of causation on the basis of direct statistical evidence than it is to do so on the basis of indirect statistical evidence. In Blue Bus, even though the plaintiff presents uncontested evidence that the Blue Bus Company operates 80 percent of the buses in the area, there are at least two distinct reasons for which jurors should hesitate to reach a conclusion about the likelihood of causation on that basis. First, even if a juror knows that the Blue Bus Company operates 80 percent of buses in the area, she might only be

45 Kirgis, "The Problem of the Expert Juror," 493.

46 For discussion, see *Halverson v. Anderson*, 513 P. 2d 827, at 830 (Wash 1973).

47 This is also why it is acceptable for an expert to testify on the basis of statistical evidence even though it would be unacceptable for jurors to reach conclusions on their own based on that same evidence. In other words, experts can answer questions posed by the court on the basis of statistical evidence because (1) their methods are vetted and (2) they are subject to cross-examination and rebuttal. I thank an anonymous reviewer for pressing me to address this point.

.7 confident that a bus caused the plaintiff's injuries. This is not only a problem in this particular example—as the juror will not think it more likely than not that the Blue Bus Company is liable—but also a more general one, as courts may want to avoid forcing jurors to integrate a variety of different statistics in reaching verdicts.[48] Second, we should expect the plaintiff to select evidence that is most favorable to her case. As such, the fact that the plaintiff presents evidence that the Blue Bus Company owns 80 percent of the buses in the area strongly suggests that the likelihood of causation is much lower. One can safely assume that if the plaintiff gathered any stronger evidence, she would have presented it, and if she gathered any weaker evidence, she would have withheld it. With this in mind, when the plaintiff presents evidence regarding the Blue Bus Company's market share, a judge or juror should simply think of it as the best that the plaintiff could come with to establish the likelihood of causation.[49]

Direct statistical evidence differs from indirect statistical evidence in this regard because it answers a question posed by the court, namely, whether it is more likely than not that the defendant caused the plaintiff's injuries. Since this feature of the court is established and known ahead of time, there is no risk that statistics that speak to this question will be cherry-picked among amongst other possibilities. In addition, since no inferences are necessary between direct statistical evidence and the likelihood of causation, direct statistical evidence that is trustworthy provides a decisive answer to the question under consideration. With this in mind, although as a general matter, expert testimony can provide jurors with either indirect or direct statistical evidence, we can think of experts who offer direct statistical evidence as eyewitnesses to the likelihood that the defendant's product caused the plaintiff's injuries.

Another, perhaps more controversial way of stating this argument is in terms of what sort of evidence would enable jurors to know, conditional on admissible evidence, that it is more likely than not that the defendant caused the plaintiff's injuries. For this sort of account, we can look to Sarah Moss, who argues that "statistical evidence suffices to prove causation just in case the factfinder knows that causation is more than .5 likely."[50] My slight modification would be to work with the proposition that on the basis of admissible evidence, it is more likely than not that the defendant caused the plaintiff's injuries. For the reasons listed above, it is not easy for a factfinder to come to know that on

48 This concern is raised by Tribe, who thinks that a focus on this sort of math distracts jurists from more important questions about justice ("Trial by Mathematics").

49 For related discussion, see Posner, "An Economic Approach to the Law of Evidence," 1509. For a more expansive treatment of this issue with statistical evidence, see Allen and Pardo, "The Problematic Value of Mathematical Models of Evidence."

50 Moss, "Knowledge and Legal Proof," 26.

the basis of admissible evidence, it is more likely than not that the Blue Bus Company caused the plaintiff's injuries on the basis of its market share because the factfinder should leave open the possibility that other admissible evidence undermines the plaintiff's case. However, if a doctor provides testimony that in her professional opinion, it is more likely than not that the defendant's product caused the plaintiff's injuries, jurors would be in a much better position to have the knowledge necessary for a judgment of causation.

One question we might ask at this point is whether, based on proof of general causation, an expert might testify as to specific causation. I have three responses here. First, the simplest response is that to the extent that findings of liability are allowed without (nontestimonial) evidence of specific causation, they ought not be.[51] This is because evidence of general causation alone simply does not answer one of the two questions the court is attempting to answer, namely whether it is more likely than not that the defendant's product caused the plaintiff's injuries. This is problematic from a legal perspective because in order for a juror to reach a conclusion about specific causation based on evidence about general causation, she must take a private epistemic journey that includes assumptions that are unsupported by evidence formally presented by the plaintiff and thus difficult for the defendant to contest.

From a moral and epistemological perspective, it does not seem reasonable for a juror to reach a conclusion about the likelihood of specific causation without (nontestimonial) evidence of specific causation, just as it does not seem reasonable for a juror to reach a conclusion about the likelihood that a bus from the Blue Bus Company caused the plaintiff's injuries based on the Blue Bus Company's market share (even if an "expert" testified that, given that market share, it is likely that a bus from the Blue Bus Company caused the accident). More specifically, there is no reason to think that knowledge of 1 would bring with it knowledge of 2:

1. Given all reasonably available evidence, it is more likely than not that the defendant's product causes injuries such as those suffered by the plaintiff.
2. Given all reasonably available evidence, it is more likely than not that the defendant's product caused the plaintiff's injuries.

51 This is similar to the point that sometimes courts do allow for simply statistical evidence to ground judgments of liability. If this is true—and *Kaminsky v. Hertz*, 288 N.W. 2d 426 (Mich. Ct. App., 1980) is often cited in support—then those who oppose statistical evidence serving this role will also oppose what the court did in this particular case. A similar point can be made about convictions based solely on DNA evidence.

In contrast, when evidence is presented that speaks to specific causation, the defendant has appropriate opportunity for rebuttal, and it is reasonable for a juror to conclude that on the basis of admissible evidence, it is more likely than not that the defendant's product caused the plaintiff's injuries (and perhaps even easier for her to know this).

This is not my only response to the supposed practice by the courts, however. The second point I would make is that there is little consensus among courts when it comes to the weight to be given to testimony about general and specific causation. As Russelyn Carruth and Bernard Goldstein put it, "even courts enunciating similar rules using the same words do not always mean the same thing."[52] Given the confused manner of courts in speaking about this question, in particular their failure to clearly distinguish requirements for specific causation from requirements for general causation, it is difficult to reach any judgment with regards to whether testimony based on evidence about general causation actually suffices to establish specific causation.[53]

This leads to my third point, which is that the cases that are most often cited in support of the claim that testimony based on evidence about general causation can suffice to establish specific causation often to not employ such reasoning in reaching judgments. For instance, a footnote in *Allison v. McGhan Medical* states that a relative risk of greater than 2 "permit[s] an inference that the plaintiff's disease was more likely than not caused by the agent."[54] However, the expert testimony presented in that case did not meet this level of relative risk, so such testimony was not used to establish specific causation. A similar analysis applies to *Daubert v. Merrell Dow Pharmaceuticals*, in which the court stated that "for an epidemiological study to show causation under a preponderance standard, the relative risk … will, at a minimum, have to exceed 2."[55] Again, however, the expert testimony provided failed to meet this standard, so this case is not an instance of testimony based on general causation sufficing to establish specific causation.[56]

52 Carruth and Goldstein, "Relative Risk Greater Than Two in Proof of Causation in Toxic Tort Litigation," 203.

53 Carruth and Goldstein, "Relative Risk Greater than Two in Proof of Causation in Toxic Tort Litigation," 204.

54 *Allison v. McGhan Medical Corporation*, 184 F.3d 1300, 1315 (11th Cir. 1999).

55 *Daubert v. Merrell Dow Pharmaceuticals*, 43 F.3d 1311, 1321 (9th Cir. 1995).

56 Without belaboring the point, I would say the same about *Ambrosini v. Labarraque*, 101 F.3d 129 (DC Cir. 1996).

Perhaps the case that is most often put forward as showing that general causation suffices to establish specific causation is *Manko v. United States*.[57] In *Manko*, just as in *Allison* and *Daubert*, the court stated that "a relative risk greater than 2 means that the disease more likely than not was caused by the event."[58] However, the court in *Manko* then proceeded to perform an analysis of specific causation, though somewhat confusedly continuing to use the language of relative risk. In particular, the court wrote that our calculation of relative risk "must be adjusted to accommodate the possibility that the plaintiff's antecedent illness caused his [illness]."[59] In this way, even in *Manko*, it is simply not the case that testimony based on evidence about general causation sufficed, on its own, to establish that the defendant's product caused the plaintiff's disease.[60]

To be clear, the fact that many courts seem to suggest that testimony based on evidence about general causation can suffice to establish specific causation is troubling. At the same time, given how difficult it is to glean a clear message from these opinions, I am willing to wait until such reasoning is used in finding a defendant liable before concluding that courts in fact are willing to find defendants liable without any evidence of specific causation whatsoever.

One last possible solution to the above puzzle that is worth considering is whether courts are more likely to allow statistical evidence to prove causation when negligence has already been established. This certainly would align with a certain sentiment that if, for instance, a corporation has negligently emitted toxic waste, we are not that upset if they are found liable simply because it is more likely than not that its emissions have caused others harm. While this has intuitive plausibility, there are a number of reasons to think that it is not actual practice. First and perhaps most importantly, if the plaintiff cannot provide sufficient evidence of causation, then a court may never even reach the question of negligence. In fact, this is what happened in the original court case that inspired Blue Bus: the statistical evidence with respect to the defendant's bus ownership was insufficient to establish causation, so the question of negligence was never decided. Another reason to doubt that this is common practice is the range of cases in which the plaintiff needed only to establish causation in

57 For discussion, see Moss, "Knowledge and Legal Proof," 25; and Green and Powers, *Restatement (Third) of Torts*, § 28 c (4). The Restatement also has a list of other cases that are worth examining in this regard.

58 *Manko v. United States*, 636 F. Supp. 1419, 1434 (W. D. Mo. 1986).

59 *Manko v. United States*, 1437.

60 *Manko v. United States*, 1437: "Because a viral illness can cause [the plaintiff's injuries] and because plaintiff had a viral illness [before exposure to the defendant's product], this relative risk must be adjusted to accommodate the possibility that the plaintiff's antecedent illness caused [his injuries]."

order for the defendant to be liable for damages. This is most common in cases involving strict liability, such as with certain instances of the use of asbestos and the government's distribution of vaccines.[61]

According to my view, courts are more justified in considering direct statistical evidence sufficient to establish the likelihood of causation than indirect statistical evidence.[62] While I think this is the best explanation of court rulings in such cases, an alternate story we might tell here is that, given the unique factors present, toxic torts are simply anomalous. For instance, some might argue that because of the significant public interest in holding polluters liable for their emissions, courts relax their regular requirements for the establishment of causation. This "special exception" view is much less appealing, however, once we examine the wide range of cases in which direct statistical evidence is deemed sufficient for establishing the likelihood of causation.

Since *Smith v. Rapid Transit*—the case that inspired the original Blue Bus hypothetical—is most often cited as a paradigmatic instance in which statistical evidence was considered insufficient for establishing the likelihood of causation, it is instructive to look at contemporaneous case law for evidence in favor of my view.[63] As we will see, contemporaneous case law supports my view that while courts do not consider indirect statistical evidence sufficient to establish causation, they often consider direct statistical evidence to be sufficient.

As was the case with toxic torts, direct statistical evidence is most often offered by medical experts in order to establish the most likely cause of the plaintiff's injuries. Examples in which the direct statistical evidence provided by medical experts sufficed to establish the likelihood of causation include *Marlow*

61 See *In re Joint E. So Dist. Asbestos Lit.*, 827 F. Supp. 1014 (S.D.N.Y. 1993); and *Manko v. United States*, respectively, which both involve strict liability (liability without negligence).

62 For what it is worth, I take Richard Wright's argument in "Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof" as supportive of my second point (without sufficient attention to the first). In part, he writes:

> A judgment on what actually happened on a particular occasion is a judgment on which causal generalization and its underlying causal law was instantiated on the particular occasion. [Evidence on specific causation] connects a possibly applicable causal generalization to the particular occasion by instantiating the abstract elements in the causal generalization, thereby converting the abstract generalization into an instantiated generalization. Without such [evidence], there is no basis for applying the causal generalization to the particular occasion. (1051)

To be clear, I am not claiming that no amount of indirect evidence suffices for a judgment of civil liability. Instead, I am simply pointing to a distinction between expert testimony and market share statistics to explain their differential treatment by the courts.

63 For one example of *Smith v. Rapid Transit* being used in this manner, see Smith, "When Does Evidence Suffice for Conviction?"

v. Dike, in which liability was upheld on the basis of a doctor's testimony that the defendant's negligence was "the probable cause" of the plaintiff's injuries, and *Rash v. Albert*, in which the establishment of causation was upheld partly on the basis of medical testimony that, while other causes were also possible, the defendant's negligence "probably" caused the plaintiff's injuries.[64] A variety of Massachusetts workers' compensation claims from that time also support my view: in these cases, causation was established on the basis of expert testimony that it was more likely than not that the plaintiffs' injuries were ones that the defendants caused.[65] Lastly, in certain cases, the courts explicitly adopted the language of direct statistical reasoning to establish the likelihood of causation, such as when, in *O'Connor v. Griff*, the court held that, based on the evidence presented, an expert "might properly conclude" that "it was reasonably probable" that the defendant's negligence caused the plaintiff's injuries.[66]

Before closing, it is worth noting that although there is strong theoretical reason to prefer direct statistical evidence to indirect statistical evidence, there are at least three additional factors that will limit the extent to which direct statistical evidence is introduced at trial. First, experts cost money, so claims with little to no monetary damages are unlikely to involve competing experts. Second, in a variety of cases, individuals with the relevant expertise might not be widely available. For instance, while there might be a number of experts that can testify about fingerprint matches and cancer etiology, there may be none that can testify about more obscure matters such as whether Kantians are less likely to commit fraud than utilitarians. Third, regardless of whether an individual professes to be an expert on a particular matter, judges play a gatekeeping role in determining who can actually testify at trial. While the standards applied in particular courts will vary, commonly considered factors include the reliability of the expert's techniques, whether such techniques have

64 *Marlow v. Dike*, 168 N.E. 154 (Mass 1929); and *Rash v. Albert*, 271 Mass 247 (1930).

65 See for instance, *Blanchard's Case*, 277 Mass 413 (1956); *Geagan's Case*, 301 Mass 319 (1938); and *Cooper's Case*, 271 Mass 38 (1930).

66 *O'Connor v. Griff*, 307 Mass 120 (1940). Another precursor to toxic torts cases can be seen in *Sullivan v. Boston Elevated Railway*, 71 N.E. 90 (Mass 1904). See also *Comeau v. Beck*, 64 N.E. 2nd 436 (Mass. 1946), in which a doctor's testimony that the plaintiff's injury could have been caused by the defendant's negligence, alongside the plaintiff's good health prior to the accident, was sufficient for a finding of liability. In its basic structure, such a case mirrors the requirement of general causation, which is met by the doctor's testimony, as well as specific causation, which the jury is allowed to infer due to the simultaneity of the accident and the injury. I hope to explore this parallel in much more detail in future work.

been peer-reviewed and whether the technique or theory has general acceptance within the scientific or professional community.[67]

In light of these factors, we should expect to see expert testimony regarding direct statistical evidence much more often in toxic tort cases than in cases like Blue Bus. First, toxic torts cases often involve large sums of money, so experts will be worth investing in. Second, there are a range of scientists, doctors, and public health professionals with expertise regarding toxic substances and their impact on human health and well-being. Third, as a corollary to the second, there is widespread acceptance of certain methodologies and theories, as well as peer-reviewed journals publishing on such questions, that can assuage a judge's concerns that she may be admitting so-called junk science into the courtroom. Neither of these may be true with respect to experts who wish to testify on the likelihood that a particular bus company caused a particular sort of injury or property damage. While such testimony is certainly possible in any civil suit (subject to applicable rules of evidence regarding expert testimony), there are certain areas of the law, such as toxic torts, where we should expect it to be relatively commonplace.

5. Conclusion

In this paper, I have presented a puzzle about how courts react to purely statistical evidence and my own tentative approach to solving it. The basic puzzle is that while certain types of statistical evidence are not considered sufficient to establish the likelihood of causation, there are other types, such as those commonly put to use in toxic tort cases, that are considered sufficient. While a number of attempts have been made to explain why statistical evidence is insufficient to establish causation, few have attempted to square this claim with the range of cases in which this practice is common.

Through an examination of toxic torts, I have shown that it is untenable to claim that as a general matter, courts consider statistical evidence insufficient to establish causation. I have put forward a view according to which it is more justified to establish causation on the basis of direct statistical evidence than indirect statistical evidence. This is both because defendants have appropriate opportunity to rebut conclusions based on direct statistical evidence and because it is more reasonable to reach a conclusion about the likelihood of causation on the basis of direct statistical evidence. I have discussed case law that suggests that direct statistical evidence is sufficient to establish the

67 These factors are taken from *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993). For an accessible primer on the admissibility of expert testimony, see Cappellino, "*Daubert* vs. *Frye*."

likelihood of causation in a variety of contexts outside of toxic torts. This case law makes it even harder to sustain the sort of view defended by others according to which courts have a general aversion to using statistical evidence to establish the likelihood of causation. In place of such a simplistic view, I have argued, we should adopt one according to which the treatment of statistical evidence by courts is much more nuanced and multifaceted.

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