

# Using Statistical Analysis in Mass Toxic Tort Cases: Are the Courts a Century Behind Science?

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United States District Judge Jack B. Weinstein, one of the mass torts deans of the judiciary, remarked in *The Role of Judges in a Government Of, By, and For the People*, that in mass tort cases, the “[f]ailure of the appellate courts to accept the law of large numbers and statistical analysis to prove cause, knowledge, and the like puts them more than a century behind science.”<sup>1</sup> In *Role of Judges*, Judge Weinstein also quoted Professor Margaret Berger’s observation that “[t]he courts’ handling of causation issues in toxic tort cases reveals a paradox,” such that “in toxic tort cases... courts not infrequently trample the evidentiary objective Justice Blackmun sought in *Daubert*—determinations on causation that are consistent with good science.”<sup>2</sup> As they relate to the realm of mass toxic torts, are these comments accurate and, perhaps more importantly, what is the appropriate intersection between law and science in such matters?

In this article, we explore the proposition that the courts fail to make proper use of statistical analysis and, thus, lag behind “good science” in the context of mass toxic torts actions. In particular, we discuss the use of epidemiological analysis as a facet of causation evidence and the use of statistical analysis to determine the propriety of medical monitoring relief.

Proof of causation in a mass toxic tort case will ordinarily involve reliance on epidemiological studies, which observe associations between exposure and disease to establish “general causation,”

i.e., that exposure to an agent may cause an increased incidence of disease. As observed in *Role of Judges*, the courts have tended to limit the use of epidemiological evidence, but such limitation should not automatically be viewed as a trampling on good science. Rather, as we discuss below, the National Academy of Sciences’ Institute of Medicine (IOM), in its recent report, *Improving the Presumptive Disability Decision-Making Process for Veterans*, recommends, among other things, that the approach to such decision-making be premised on “evidence-based decisions” and the proposition that “causation, not just association, [is] the target for decision-making.” Relevant to the treatment of epidemiological evidence in the *Daubert* arena, the IOM report articulates what the scientists consider to be “good science,” which is, of course, the measuring stick in a *Daubert* analysis.

Medical monitoring is another matter in toxic tort litigation that requires an intersection of statistical analysis, science, and medicine. A medical-monitoring claim is apt for statistical analysis as there must be a showing of, among other things, a quantifiable and significant increase in the risk of disease to the exposed population, a sufficiently sensitive medical test to detect the target disease, and “a demonstrable clinical value of medical monitoring (that is, as a result of early detection and diagnosis, treatment exists that improves either morbidity or mortality statistics).”<sup>3</sup> As illustrated in the article *A Quantitative Methodology for Determining the Need for Exposure-Prompted Medical Monitoring*, statistical analysis is particularly appropriate for a medical monitoring claim, rather than “simply relying on the opinions of retained medical experts.”<sup>4</sup> Such quantitative analysis will often reveal that medical monitoring is not warranted because medical testing is likely to do more harm than good on a

class-wide basis, contrary to the seeming logic that testing is appropriate so long as it leads to early diagnoses of the target disease.

## Epidemiological Analysis as Evidence of Causation

“When an agent’s effects are suspected to be harmful, we cannot knowingly expose people to the agent.”<sup>5</sup> Consequently, proof of causation in a non-pharmaceutical toxic tort will not be based on an experiment to show the effect of a given exposure to a substance in humans. Instead, in a toxic tort claim based on a chemical or other environmental exposure, evidence of causation ordinarily involves reliance on epidemiological studies of similar circumstances. “Epidemiology is the field of public health and medicine that studies the incidence, distribution, and etiology of disease in human populations.”<sup>6</sup> The “statistical analysis” generated by such studies infers “associations” between exposure and disease, but, fundamentally, “it should be emphasized that an association is not equivalent to causation.”<sup>7</sup> Indeed, with respect to causation, one court has recognized that “epidemiology, unlike many other scientific fields, includes an inherently subjective element: Epidemiology cannot objectively prove causation; rather, causation is a judgment for epidemiologists and others interpreting the epidemiologic data.”<sup>8</sup>

As Professor Berger’s observation denotes, the evidentiary crux of a toxic tort case is at what level do such epidemiological studies constitute sufficiently “good science” to satisfy the legal requirement for showing causation as an element of imposing liability. Judge Weinstein and Professor Berger appear to suggest that the courts have set the bar too high and that a broader intersection between the law and this type of statistical analysis should be permitted.

In cases involving chemical

exposures, the courts have often concluded that the plaintiffs' purported epidemiological evidence of causation is insufficient. In *Knight v. Kerby Inland Marine, Inc.*, for example, the two plaintiffs, who had worked as tankermen and on barges, alleged that they contracted lymphoma and bladder cancer due to exposure to benzene.<sup>9</sup> In support of these allegations, the plaintiffs proffered the expert testimony of an epidemiologist, Dr. Barry S. Levy.

After a *Daubert* hearing, the district court granted the defendants' motions in limine to exclude Dr. Levy's testimony, concluding that his proposed testimony lacked the necessary foundation, as:

[n]o peer-reviewed epidemiological study nor any published review of such studies has concluded that the specific chemicals at issue in this case are known—or even probable—causes of the plaintiffs' cancers. [Dr. Levy] relied on a variety of relatively dissimilar epidemiological studies to conclude that specific chemicals cause specific cancers. Most of the studies [Dr. Levy] cited failed to identify specific chemical exposures, involved potential exposure to chemicals not at issue in this matter, or inferred exposure by job category rather than by direct measurement.<sup>10</sup>

The district court noted that the studies relied upon were deficient as proof of causation, because rather than analyzing the particular exposure-disease scenario at issue, the studies "[lump[ed] numerous high-grade solvents together," identified "only a broad category of exposure" and failed to isolate "particular exposures," or failed to document "the level of exposure."<sup>11</sup>

Based on his remark in *Role of Judges*, Judge Weinstein might criticize this rationale as too restrictive and suggest that the law must accommodate the lack of or imprecision of the science, particularly in a mass tort context involving multiple chemicals and multiple diseases (i.e., the proverbial "toxic soup" case) where overall statistics indicate harm to some percentage of a population although no study is able to define a causal link between a given substance

and a given disease. Judge Weinstein suggests that "[i]f no one person can show by a preponderance of evidence that he was injured by a toxic substance . . . but demographics, epidemiology and statistics can demonstrate that some large number—say thirty percent—were injured by the substance and seventy percent by endogenous factors, the parties responsible should be ordered to pay thirty percent of the total damage they caused to be divided among the whole class."<sup>12</sup>

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### A finding of causation based on statistical analysis only showing association is not good science.

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Pre-*Daubert*, Judge Weinstein in fact addressed something of this very scenario in considering the claims of plaintiffs alleging a variety of injuries as a result of exposure to the chemical herbicide "Agent Orange." After engineering a class settlement in the case, a seminal mass toxic tort action, Judge Weinstein dismissed the claims of those class members who had opted-out of the class action on the grounds, among others, that the plaintiffs failed "to present credible evidence of a causal link between exposure to Agent Orange and the various diseases from which they are suffering."<sup>13</sup> Judge Weinstein noted that "[a] number of sound epidemiological studies have been conducted on the health effects of exposure to Agent Orange," but that "[n]o acceptable study to date . . . concludes that there is a causal connection between exposure to Agent Orange and the serious adverse health effects claimed by plaintiffs."<sup>14</sup>

Later in the Agent Orange history, Congress enacted the Agent Orange Act of 1991, which directed the National Academy of Sciences "to perform a comprehensive review of scientific and

medical information regarding the health effects of exposure to Agent Orange" and related herbicides and their components, and to update the review biennially.<sup>15</sup> The findings are utilized by the Veterans Administration (VA) to promulgate a benefits scheme for Vietnam veterans. The IOM's findings on the link between exposure and disease are divided into four categories: "sufficient evidence of association";<sup>16</sup> "limited or suggestive evidence of association";<sup>17</sup> "inadequate or insufficient evidence to determine association";<sup>18</sup> and limited or suggestive evidence of *no* association.<sup>19</sup> Generally, the VA awards benefits on the basis of the diseases falling into the first two categories of "sufficient" or "limited or suggestive" evidence of an association.

Setting aside Judge Weinstein's prior rejection of epidemiological evidence to prove causation of Agent Orange-related claims is an association-based approach akin to that utilized for Agent Orange benefits, an appropriate and just surrogate for the mass toxic tort arena that, in accordance with Judge Weinstein's admonition, would allow the courts to catch up with science and prevent the trampling of good science, against which Professor Berger warns.

A negative answer to these inquiries is found, interestingly, on the science side of the dialogue, with the scientists explaining that a finding of causation based on statistical analysis only showing an association is not good science. As the IOM's Vietnam Veterans Committee<sup>20</sup> acknowledged in its *2006 Update* report, "the target of evaluation is 'association,' not 'causality,' between exposure and health outcomes. As used technically, the criteria for causation are somewhat more stringent than those for association."<sup>21</sup> Notably, VA decisions to award benefits (healthcare coverage and disability compensation) to veterans are based on a lesser standard than legal causation, and, indeed, the VA will resort to certain "presumptions" where "scientific evidence is incomplete" and "gaps in the evidence related to causation" need to be bridged.<sup>22</sup> A separate IOM committee, the Committee on Evaluation of the Presumptive

Disability Decision-Making Process for Veterans (the Presumptive Disability Committee), was convened to review the past methods of making presumptions, such as the Agent Orange benefits scheme, and, “if needed, to make recommendations for an improved *scientific* framework that could be used in the future for determining if a presumption should be made.”<sup>23</sup>

As part of its work, the Presumptive Disability Committee “reviewed general methods by which scientists, as well as government and other organizations, evaluate scientific evidence in order to determine if a specific exposure causes a health condition.”<sup>24</sup> The committee recommended that, among other principles, presumptive disability determinations should be “evidence-based decisions” and that “causation, not just association, [should be] the target for decision-making.”<sup>25</sup> So, in short, according to the Presumptive Disability Committee, even for a determination where the standard is less than legal causation, mere association, as would be reflected in a purely statistical analysis, does not constitute good science.

Rather, good science requires that “[d]ecisions about presumptions *should* be grounded in a scientific evaluation of the *full range of evidence* that the exposure of interest *causes* the disease or disability.”<sup>26</sup> Contrary to a suggestion that statistical evidence might alone carry the day with respect to proving causation of a toxic tort, science requires much more. Statistical analysis is only a piece of the puzzle; determining causation of a mass toxic tort as a matter of science “involves review of statistical evidence from epidemiologic studies, evidence from experiments in other animals, and mechanistic evidence from basic biologic science.”<sup>27</sup> As the Presumptive Disability Committee explained the process in detail:

Because a statistical association between exposure and disease does not prove causation, plausible alternative hypotheses must be eliminated by careful statistical adjustment and/or consideration of all relevant scientific knowledge. Epidemiologic studies that show an association after

such adjustment, for example through multiple regression or instrumental variable estimation, and that are reasonably free of bias and further confounding, provide evidence but not proof of causation. Mechanistic knowledge about how particular agents might produce adverse health effects provides further evidence. For example, ionizing radiation is known to cause mutations in DNA that can result in cancer. Animal studies may provide further evidence by showing that an agent may induce in several different species the same effect observed in human studies, and by a mechanism that is conserved across species with key features of the mechanism observed.<sup>28</sup>

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**Deciding whether medical monitoring is necessary and appropriate involves a weighing of risks and benefits, something that is best accomplished by applying an objective and quantifiable method.**

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Strictly speaking, the IOM’s Presumptive Disability report does not provide a binding legal rule for the application of *Daubert* principles to epidemiological evidence in a toxic tort context, but it would nevertheless seem to be an important part of the dialogue and guidance on what “good science” is with respect to establishing a causal link between toxic exposure and disease that ought to be sought under *Daubert*.

### **Statistical Analysis as the Basis for Medical Monitoring**

In contrast to the shortcomings of utilizing statistical analysis as proof of causation in the mass toxic tort context, statistical analysis is appropriate, if not required, for determining a claim for medical monitoring, although such analysis will frequently be at odds with the adage that an ounce of prevention is worth a pound of cure. A determination

not to conduct medical monitoring on a class-wide or population-wide basis is a risk-utility determination based on whether false-positive and false-negative test results in the tested population will cause more overall harm than the benefit derived from the testing when it is accurate. As explained in the article *A Quantitative Methodology*, this analysis is quite susceptible to a quantitative result based on known factors, including the prevalence of the disease for which testing is being conducted and the “sensitivity” and “specificity” of the test being utilized. Based on these variables, a positive predictive value (PPV) can be calculated. Using the PPV model, courts can “more confidently and objectively decide whether medical monitoring is appropriate and necessary as a result of a specific chemical exposure,” as opposed to “simply relying on the opinions of retained medical experts.”<sup>29</sup>

The purpose of medical monitoring in the context of an allegedly toxic exposure is to detect disease for which an individual is at heightened risk due to the exposure before that patient becomes symptomatic so that treatment might be initiated as early as possible. Such monitoring is comprised of periodic diagnostic testing. (A single test would be referred to as medical screening.) Contrary to the “ounce of prevention” adage, it must be recognized that such testing (indeed, any medical testing) carries with it certain potential harms, including a false-negative test result, where the test erroneously fails to detect disease and the patient does not receive care; a false-positive test result, where the test erroneously concludes that a healthy patient has a disease, leading to unnecessary follow-up testing and care as well as mental anguish to the patient; harm to the “false-positive” patient from complications arising out of the unnecessary follow-up testing and care, including a series of tests, procedures, and complications, which is referred to as the “cascade effect”; and direct harm during the testing itself (e.g., a colon perforation during a colonoscopy).<sup>30</sup>

Given these potential harms, deciding whether medical monitoring is necessary



and appropriate on a class-wide basis involves a weighing of risks and benefits, something that is best accomplished by applying an objective and quantifiable method such as PPV. PPV utilizes three objective measures: the “sensitivity” of the test (i.e., the proportion of persons with the condition who are correctly identified as having the disease); the “specificity” of the test (i.e., the proportion of persons without the condition who correctly test negative; and the “prevalence” of the disease (i.e., the sum of background incidence of the disease and the additional incidence corresponding to the increased risk associated with the chemical exposure). These measures quantify how many persons with disease will be detected compared to the total number of all positives (i.e., true positives/all positives).<sup>31</sup> A low PPV means that an inordinately high number of healthy people will also test positive and “will be falsely alarmed” and unduly “subjected to unnecessary additional tests and procedures with all the attendant risks, including potential cascade effects.”<sup>32</sup>

As the PPV methodology shows, statistical analysis does in fact have a proper and needed place in mass toxic tort law as it relates to properly quantifying the benefits and risks of medical monitoring. The determination of medical monitoring claims via the PPV methodology is a matter in which the courts should catch up with science.

## Conclusion

The proposition that the law lags behind science in the mass toxic tort arena by failing to utilize the statistical analysis provided through the science of epidemiology to prove cause does not seem to be shared by the scientists themselves, who have indicated that good science requires proof of causation, not just the mere associations established via epidemiologic studies. On the other hand, with respect to determining medical monitoring claims, which frequently arise in mass toxic tort cases, statistical analysis is an appropriate and necessary tool that the courts should use.

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## Endnotes

1. Hon. Jack B. Weinstein, *The Role of Judges in a Government Of, By, and For the People*, Notes for the Fifty-Eighth Cardozo Lecture, sponsored by the Association of the Bar of the City of New York, delivered on November 28, 2007.

2. *Id.* at 22–23 (quoting Margaret A. Berger, *On Daubert* (2007) (forthcoming)).

3. Christopher P. Guzelian, Bruce E. Hillner, and Philip S. Guzelian, *A Quantitative Methodology for Determining the Need for Exposure-Prompted Medical Monitoring*, 79 IND. L. J. 57 (2004).

4. *Id.* at 57.

5. Michael D. Green, D. Michael Freedman & Leon Gordis, *Reference Guide on Epidemiology*, in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 339 (Fed. Jud. Ctr. 2d ed. 2000). “Experimental studies in which human beings are exposed to agents known or thought to be toxic are ethically proscribed. . . . Experimental studies can be used where the agent under investigation is believed to be beneficial, as is the case in the development and testing of new pharmaceutical drugs.” *Id.* at 339 n.14 (citations omitted).

6. *Id.* at 335. Toxicology is a related discipline that is relied upon in proving causation of a toxic tort claim. “Toxicology classically is known as the science of poisons,” or put another way, “the study of the adverse effects of chemicals on living organisms.” Bernard D. Goldstein and Mary Sue Henifin, *Reference Guide on Toxicology*, in REFERENCE MANUAL, *supra* notes at 403.

7. *Reference Guide on Epidemiology*, *supra* note 5 at 336.

8. *Knight v. Kerby Inland Marine, Inc.*, 363 F. Supp. 2d 859, 864 (S.D. Miss. 2005) (quoting *Reference Guide on Epidemiology*, *supra* note 5 at 374), *aff'd*, 482 F.3d 347 (5th Cir. 2007).

9. *Id.* at 860–61.

10. *Id.* at 866 (citing *Allen v. Pennsylvania Engineering Corp.*, 102 F.3d 194,

198 (5th Cir. 1996)) (“Scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs’ burden in a toxic tort case”).

11. *Id.* at 864–66.

12. *Role of Judges*, *supra* note 1, at 238.

13. *In re Agent Orange Prod. Liab. Litig.*, 611 F. Supp. 1223, 1229 (E.D.N.Y. 1985), *aff'd*, 818 F.2d 187 (2d Cir. 1987).

14. *Id.* at 1231.

15. VETERANS AND AGENT ORANGE: UPDATE 2006 1–2 (Institute of Medicine 2007).

16. “Sufficient evidence of association” exists where “evidence is sufficient to conclude that there is a positive association. That is, a positive association has been observed between exposure to herbicides and the outcome in studies in which chance, bias, and confounding could be ruled out with reasonable confidence. For example, if several small studies that are free of bias and confounding show an association that is consistent in magnitude and direction, there could be sufficient evidence of an association.” *Id.* at 11.

17. “Limited or suggestive evidence of association” is evidence that “suggests an association between exposure to herbicides and the outcome, but a firm conclusion is limited because chance, bias, and confounding could not be ruled out with confidence. For example, a well-conducted study with strong findings in accord with less compelling results from studies of populations with similar exposures would constitute such evidence.” *Id.*

18. “Inadequate or insufficient evidence to determine association” occurs when “[t]he available studies are of insufficient quality, consistency, or statistical power to permit a conclusion regarding the presence or absence of an association.” Examples would be studies that “fail to control for confounding, have inadequate exposure assessment, or fail to address latency.” *Id.* Exclusion of epidemiological studies for failing to document the level of exposure, a

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removal as a mass action under CAFA and is subsequently severed into individual cases by defendant.

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### Endnotes

1. Cooper v. R.J. Reynolds Tobacco Co., No. 3:08-cv-153-J-32HTS, 2008 WL 4093715 (M.D. Fla. Aug. 29, 2008), rev. denied, No. 08-90021 (11th Cir. Oct. 28, 2008).

2. Engle v. Liggett Group, Inc., 945 So. 2d 1246 (Fla. 2006).

3. Cooper, *supra* note 1.

4. Bullard v. Burlington N. Santa Fe Ry. Co., 535 F.3d 759, 762 (7th Cir. 2008).

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## New Legislation Creates Enhanced Duties for Manufacturers of Children's Products

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13. Section 101 (a)(2)(D).

14. Section 101 (d).

15. Section 101 (a)(2)(E).

16. Section 101(b)(2)(A).

17. Section 101 (b)(1)(A)–(B).

18. Section 101(b)(2)(A).

19. Section 102 (a)(1)(A)–(B).

20. Section 102 (a)(2).

21. Section 102 (g)(1).

22. Section 103 (a)(5).

23. Section 217 (a) (1).

24. Section 217(c)(1).

25. Section 217(c)(2).

26. Section 218.

27. Section 212.

28. Section 214.

29. Section 219.

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ground on which the court in *Knight* found certain studies to be insufficiently reliable, is consistent with this criteria for inadequate or insufficient evidence.

19. Such a “no association” finding may be made where “[s]everal adequate studies, which cover the full range of human exposure, are consistent in not showing a positive association between any magnitude of exposure to the herbicides of interest and the outcome. A conclusion of “no association is inevitably limited to the conditions, exposures, and length of observation covered by

the available studies. *In addition, the possibility of a very small increase in risk at the exposure studies can never be excluded.”* *Id.* at 12.

20. Committee to Review the Health Effects in Vietnam Veterans of Exposure to Herbicides.

21. VETERANS AND AGENT ORANGE, *supra* note 15, at 2.

22. Improving the Presumptive Disability Decision-Making Process for Veterans 7–8 (Institute of Medicine 2008).

23. *Id.* at 1.

24. *Id.* at 2.

25. *Id.* at 5.

26. *Id.* at 136 (emphasis added, final emphasis in original).

27. *Id.* at 172–73.

28. *Id.* at 173.

29. *A Quantitative Methodology*, *supra* note 3, at 57.

30. *Id.* at 66–73.

31. *Id.* at 81, 88–92.

32. *Id.* at 89.