Note

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Hope for Hanford Downwinders?: The Ninth Circuit's Ruling in *In re Hanford Nuclear Reservation Litigation*

This Note explores the difficulties toxic tort plaintiffs face in attempting to establish that their exposures to known hazardous substances, rather than some independent factor, caused their diseases. Through an analysis of federal district court and Ninth Circuit Court of Appeals decisions, this Note examines how application of traditional tort burdens of proof to the realm of toxic tort litigation has imposed nearly insurmountable barriers for injured plaintiffs seeking relief. This Note examines in detail the Ninth Circuit's decision in *In re Hanford Nuclear Reservation Litigation*, a July 2002 case that only marginally relaxed the evidentiary burden toxic tort plaintiffs must meet in order to establish causation. Significantly, although the ruling may be useful to a limited number of *In re Hanford* plaintiffs, the effect of the court's opinion is largely illusory when viewed within the context of existing burdens to recovery. This Note concludes that federal legislation should be enacted to provide fair and just compensation to plaintiffs in mass toxic tort cases, such as *In re Hanford*, so long as those plaintiffs are able to meet prescribed minimum

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burdens relating to epidemiological and statistical proof of causation.

Part I of this Note examines the ongoing *In re Hanford* litigation, including the historical underpinnings of the case and the Ninth Circuit's recent opinion clarifying the parameters of relative risk and the use of statistical evidence in toxic tort litigation. Part II discusses the use of scientific evidence and expert testimony in toxic tort litigation as well as the development of evidentiary standards related to such evidence, starting with the Supreme Court's ruling in *Daubert v. Merrell Dow Pharmaceuticals*. Part III then examines the statistical basis of relative-risk calculations and the rationale for the "doubling of the risk" standard that nearly all courts employ. Part IV surveys the field of scholarly opinion concerning both the perceived overreliance and underreliance of courts on relative-risk calculations and the epidemiological data upon which such calculations are based. Finally, Part V analyzes a number of proposed theories for reformulating the evidentiary burden of proof for causation in toxic tort litigation.

I

THE HANFORD LITIGATION

A. History of the Hanford Nuclear Reservation

The Hanford Nuclear Reservation, located in southeastern Washington, is a 560-square-mile[1] plutonium production facility built in 1943 to meet the needs of the Manhattan Project.[2] The work of manufacturing weapons-grade plutonium began at the facility early in 1944, and reached its peak in the late 1940s and early 1950s during the nuclear proliferation of the Cold War.[3] Production continued until the last plant was deactivated in December 1988.[4] Unbeknownst to people living in the vicinity of Hanford, airborne emissions[5] of radioactive substances also

[1] Elouise Schumacher, *U.S. Confirms Health Risks Near Hanford in '40s, '50s*, THE SEATTLE TIMES, July 12, 1990, at A1 [hereinafter *Health Risks*].

[2] Gayle Greene, *In the Afterglow: Environmental Problems Caused by Nuclear Weapons Plant in Hanford, Washington*, THE NATION, Feb. 28, 1994, at 46.

[3] *See Health Risks*, *supra* note 1, at A1.

[4] Elouise Schumacher, *Fallout of Radiation Revelation: A Flurry of Questions*, THE SEATTLE TIMES, July 29, 1990, at A1 [hereinafter *Questions*].

[5] Smaller amounts of radioactive emissions were also discharged directly into the Columbia River from 1944 until the last of Hanford's eight original production reactors was shut down in 1971. *Id.*

peaked during the facility's first decade of operation (although new releases continued well into the 1970s), blanketing thousands of square miles of Washington and Oregon with radioactive isotopes such as iodine-131 and plutonium-239.[6]

As early as 1951, Hanford officials were aware that airborne emissions from the site posed a significant health risk to persons in the surrounding vicinity.[7] It wasn't until July 12, 1990, however, when the United States Department of Energy's Hanford Environmental Dose Reconstruction Project published a report entitled "Initial Hanford Radiation Dose Estimates," that the fears of thousands of Washington and Oregon "downwinders" were substantiated. The report focused on Hanford's releases of radioactive iodine[8] and concluded that over 685,000 curies[9] were released into the air from 1944 to 1947—with over 340,000 curies

[6] Official estimates of the level of iodine-131 released from Hanford between 1944 and 1956 are as follows: 1944—54,000 curies; 1945—340,000 curies; 1946—76,000 curies; 1947—24,000 curies; 1948—1,200 curies ; 1949—7,900 curies ; 1950—4,000 curies; 1951—18,800 curies; 1952—1,000 curies; 1953—700 curies ; 1954—500 curies; 1955—1,100 curies; 1956—400 curies. *Health Risks*, *supra* note 1 at A1. The airborne radiation emissions were detected as far south as Klamath Falls, Oregon, and as far north as Spokane, Washington. *Questions*, *supra* note 4, at A1.

[7] On January 12, 1951, a Hanford official acknowledged the health risks, stating: "[t]he particle problem still remains, in my opinion, a very serious health problem. This problem is present in other A.E.C. [Atomic Energy Commission] manufacturing plants and will be important in new installations, not only from the standpoint of real injury but because of the extreme difficulty of defense in cases of litigation." Plaintiffs' Consolidated Complaint at 26, para. 31, *In re* Hanford Nuclear Reservation Litig., 780 F. Supp. 1551 (E.D. Wash 1991) (No. CY-91-3015-AAM). *See also* Greene *supra* note 2, at 46 (detailing early challenges to the government's assurances about the harmlessness of radioactive emissions from Hanford including studies by Dr. Ernest Sternglass showing increased infant mortality around Hanford between 1943 and 1954, and the case of Dr. Thomas Mancuso, who was hired by the Atomic Energy Commission to study the health effects of radiation at Hanford only to be defunded when his study turned up high incidences of cancer).

[8] Researchers focused on radioactive iodine, which is an especially troublesome material because it is readily absorbed by the human thyroid gland. Once absorbed, radioactive iodine causes cancer and affects the gland's ability to properly regulate growth and metabolism. *See* Steve Green, *Thousands Exposed to Radiation in '40s, New Study Shows*, UNITED PRESS INT'L, July 13, 1990, *available at* LEXIS, News Library, UPI File.

[9] Dick Clever, *Study May Affect Hanford Lawsuit*, SEATTLE POST-INTELLIGENCER, April 20, 1994, at A1. Radioactive isotopes such as iodine 131 are unstable—and hence radioactive—due to an excess number of protons or neutrons in their nuclei. *See* David S. Gooden, *Radiation Injury and the Law*, 1989 BYU L. REV. 1155, 1160 (1989). Radioactive isotopes stabilize themselves by giving off energy in the form of radiation. *Id.* An element is radioactive for as long as it takes the nucleus of that element to shed enough protons or neutrons for it to become evenly balanced. *Id.* A curie is the traditional measure for radioactivity with one curie representing 3.7 times 10 disintegrations per second. *Id.* at 1162.

emitted by the facility in 1945 alone.[10] In comparison, the 1979 Three Mile Island nuclear accident in Pennsylvania released just fifteen curies of radioactive iodine,[11] while the 1986 Chernobyl nuclear accident in the former Soviet Union released an estimated 6,000,000 curies of radioactive iodine.[12] Based on data contained in the government's report, experts estimated that approximately 55,000 individuals could have been exposed to radiation levels high enough to cause adverse health effects.[13] The most disturbing revelations to surface from the government's report concerned details of the 1949 "Green Run," a large-scale, *deliberate* release of radioactive emissions from Hanford, undertaken at the behest of the U.S. military, in order to test equipment that had been set up to monitor the Soviet Union's emerging nuclear-weapons arsenal.[14]

B. In re Hanford *at the District Court Level*

Soon after the Department of Energy released its report documenting the long-term, large-scale release of radioactive substances from the Hanford Nuclear Reservation, thousands of individuals living within the affected regions of southeastern Washington and northern Oregon filed suit.[15] In February 1991, these separate actions were consolidated into a single lawsuit: *In re Hanford Nuclear Reservation Litigation*.[16]

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[10] *Health Risks*, *supra* note 1, at A1. Many other radioactive or toxic materials were also released from Hanford during that time, including cesium, strontium, plutonium, phosphorus, zinc, copper, neptunium, arsenic, chromium, manganese and krypton. *Questions*, *supra* note 4, at A1.

[11] *Health Risks*, *supra* note 1, at A1.

[12] *Questions*, *supra* note 4, at A1.

[13] *Id.*

[14] Trisha T. Pritkin, *Hanford: Where Traditional Common Law Fails*, 30 GONZ. L. REV. 523, 566 n.47 (1994); *Questions*, *supra* note 4, at A1; Greene, *supra* note 2, at 46.

[15] *In re* Hanford Nuclear Reservation Litig., CV-91-3015-AAM 1998 WL 775340, at *1 (E.D. Wash. Nov. 19, 1998).

[16] *Id.* The cases consolidated into *In re Hanford* are as follows: Evenson v. EPA, CY-90-3067-AAM; Hamilton v. E.I. DuPont de Nemours & Co., CY-90-3069-AAM; Wahpat v. General Electric Co., CY-90-3091-AAM; Criswell v. E.I. DuPont de Nemours & Co., CY-90-3106-AAM; Jaros v. E.I. DuPont de Nemours & Co., CY-90-3107. *Id.* at *1. Subsequent to the February 1991 consolidation, several additional cases were also consolidated into *In re Hanford*. *Id.* at *2. These later cases are as follows: Roseman v. General Elec. Co., CY-91-3045-AAM; Seaman v. E.I. DuPont de Nemours & Co., CY-91-3080-AAM; Miller v. E.I. DuPont de Nemours & Co., CY-92-3069-AAM; Durfey v. E.I. DuPont de Nemours & Co., CY-93-3087-AAM; Thomson v. E.I. DuPont de Nemours & Co., CY-94-3067-AAM. *Id.*

The consolidated action was refiled in federal court in the Eastern District of Washington as a class-action lawsuit consisting of plaintiffs who alleged that the operators of Hanford had acted intentionally and negligently in contaminating the region surrounding Hanford, thereby causing their illnesses.[17] Plaintiffs' consolidated complaint sought recovery "for redress for present and threatened future injuries resulting from Defendants' wrongdoing in the generation, storage, and use of vast quantities of radioactive . . . hazardous substances at [Hanford] and the release of those substances into the environment."[18] The *In re Hanford* plaintiffs alleged injuries including thyroid cancer, non-neoplastic thyroid diseases, and various non-thyroid cancers such as breast cancer, brain cancer, and lymphoma.[19] The five defendant companies named in the complaint—E.I. Du Pont de Nemours & Co., General Electric Co., UNC Nuclear Industries, Atlantic Richfield Co., and Rockwell International—all manufactured plutonium at the facility between 1943 and 1987.[20]

The district court divided the consolidated *In re Hanford* litigation into three phases.[21] The first phase permitted discovery by plaintiffs of Hanford's operating and emissions history while allowing the defendants to conduct discovery related to individual plaintiffs' exposures, medical histories, and relevant illnesses.[22] Although originally scheduled to last one year, this

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[17] The district court, commenting on the enormity of the litigation, stated:

> Plaintiffs, who conceivably could number into the hundreds of thousands, consist of all those persons who, at some time during the last 50 years, resided and/or had some property interest in an area which covers most of southeastern Washington, a portion of northeastern Oregon, and a small portion of western Idaho. . . . Given the scope of the plantiffs' claims, particularly with regard to the number and differing types of emissions and the differing harms alleged to have resulted from each, the potential enormity of this litigation, as well as the dollar amount of any recovery, is almost staggering.

In re Hanford Nuclear Reservation Litig., 292 F.3d 1124, 1128 (9th Cir. 2002).

[18] *In re* Hanford Nuclear Reservation Litig., 780 F. Supp. 1551, 1555 (E.D. Wash. 1991) (quoting Plaintiffs' Consolidated Complaint).

[19] *In re Hanford Nuclear Reservation Litig.*, 1998 WL 775340, at *2. On September 20, 1996, the claims of numerous *In re Hanford* plaintiffs were severed from that litigation and consolidated as *Berg v. E.I. DuPont de Nemours & Co.*, 293 F.3d 1127 (9th Cir. 2002). The *Berg* plaintiffs sought damages for emotional distress and medical monitoring due to their exposure to radiation; however, the *Berg* plaintiffs did not claim physical injuries based on their exposures. *Id.* at 1129.

[20] *In re Hanford Nuclear Reservation Litig.*, 1998 WL 775340, at *2.

[21] *Id.*

[22] *In re Hanford Nuclear Reservation Litig.*, 292 F.3d at 1129.

initial phase of the litigation was extended three times in three years and did not conclude until March 1995.[23] The second phase of the litigation was designed to focus on causation and included disclosure of all scientific expert witnesses and the filing of expert reports by both sides.[24] The third phase was designed to encompass general liability and any other pre-trial issues.[25]

Once the second phase of discovery began, the district court granted plaintiffs' request to bifurcate the causation phase into two separate stages.[26] The first stage would focus on "generic causation," while the second would focus on "individual causation."[27] Although the district court did not precisely define these two terms, generic causation is generally understood as a determination of whether a toxic/radioactive agent has the *capacity* to cause the diseases complained of by plaintiff,[28] while individual causation is a determination of whether the toxic/radioactive agent *actually did* cause a particular plaintiff's disease.[29]

During discovery on the issue of generic causation the *In re Hanford* plaintiffs and defendants developed "fundamentally dif-

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[23] *Id.*

[24] *Id.*

[25] *In re Hanford Nuclear Reservation Litig.*, 1998 WL 775340, at *2.

[26] *Id.*

[27] *Id.* at *4.

[28] In its hearing on defendant's summary judgment motion at the conclusion of the generic causation phase, the district court defined generic causation as asking the question of "whether an agent is capable of causing a particular disease." *In re Hanford Nuclear Reservation Litig.*, 1998 WL 775340, at *4. (citing Hilao v. Estate of Marcos, 103 F.3d 767, 788 (9th Cir. 1996) (Rymer, J., concurring in part and dissenting in part)). Similarly, on appeal, the Ninth Circuit defined generic causation as "the capacity of a toxic agent, such as radiation, to cause the illnesses complained of by plaintiffs." *In re Hanford Nuclear Reservation Litig.*, 292 F.3d at 1129.

[29] The district court did not specifically define what it meant by "individual causation" in either its order to bifurcate the causation phase or in its ruling on defendant's summary judgment motion. The Ninth Circuit, however, understood the term to mean that once generic causation was established "'individual causation' answers whether that toxic agent actually caused a particular plaintiff's illness." *In re Hanford Nuclear Reservation Litig.*, 292 F.3d at 1129 (citing Sterling v. Velsicol Chem. Corp., 855 F.2d 1188, 1200 (6th Cir. 1988)); *see also In re* "Agent Orange" Litig., 818 F.2d 145, 165 (2d Cir. 1987) ("[t]he relevant question . . . is not whether Agent Orange has the capacity to cause harm the generic causation issue, but whether it *did* cause harm and to whom. That determination is highly individualistic, and depends upon the characteristics of individual plaintiffs . . . and the nature of their exposure to Agent Orange."); Jones v. Allercare, Inc, 203 F.R.D. 290, 301 (N.D. Ohio 2001) ("the relevant question in this case will not be whether the products have the capacity to cause harm, but whether the products caused harm and to whom. Thus, the real causation issue in this case is individual, not general, in nature.").

ferent views on the nature of plaintiffs' burden."[30] Plaintiffs argued that, consistent with the generally recognized meaning of generic causation, they needed only to prove that emissions released from Hanford had the capacity to cause their claimed diseases.[31] Defendants, however, maintained that, under the Ninth Circuit's ruling in *Daubert v. Merrell Dow Pharmaceuticals*,[32] plaintiffs could not proceed to the individual causation stage unless they could offer proof that, for each claimed disease, the relevant plaintiff had been exposed to a dose of radiation that statistically doubled his or her risk of harm.[33] Based on their understanding of plaintiffs' burden of proof, the defendants filed a motion for summary judgment at the conclusion of discovery on generic causation. Defendants claimed that plaintiffs had failed to establish a genuine issue of material fact as to whether the reported emissions from Hanford had resulted in a statistical doubling of the risk of harm for any plaintiff.[34]

In July 1998, in a 762-page order, the district court in large part granted defendants' motion for summary judgment adopting defendants' understanding of the burden of proof needed for plaintiffs to advance past the generic causation phase. The court concluded that proof concerning whether radiation is capable of causing a given illness raises only a "possibility" that radiation is in fact the cause of that illness.[35] Relying heavily on its interpretation of *Daubert*, the court wrote: "Such evidence invites a jury to speculate whether radiation exposure is in fact a cause of the injury and, by itself, is of no assistance to a jury."[36] The court noted that because the *In re Hanford* plaintiffs lacked direct proof of causation, they instead had to use scientific evidence

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[30] *In re Hanford Nuclear Reservation Litig.*, 1998 WL 775340, at *3.

[31] *In re Hanford Nuclear Reservation Litig.*, 292 F.3d at 1130.

[32] 43 F.3d 1311 (9th Cir. 1995).

[33] *In re Hanford Nuclear Reservation Litig.*, 292 F.3d at 1130.

[34] *In re Hanford Nuclear Reservation Litig.*, 1998 WL 775340, at *3. In their motion for summary judgment, defendants conceded that radiation exposure is "capable of causing" several illnesses complained of by the plaintiffs including thyroid cancer and non-autoimmune hypothyroidism. *Id*. at *4. The defendants, however, did not agree with the plaintiffs concerning the exposure levels at which radiation was "capable of causing" these illnesses. *Id.* Defendants also argued that plaintiffs' evidence was insufficient to establish a genuine issue of material fact as to whether radiation was "capable of causing" the majority of other illnesses that plaintiffs alleged were caused by radioactive emissions from Hanford. *Id.*

[35] *Id.*

[36] *Id.* (citing Daubert v. Merrell Dow Pharm., Inc., 43 F.3d 1311, 1320-22 (9th Cir. 1995)).

and expert testimony based on epidemiological data.[37] The district court concluded that, regardless of whether such data is used at a generic or individual causation stage, if epidemiological data and statistical proof are relied upon to establish causation, such scientific evidence "meets the 'more-likely-than-not' sufficiency standard only if a 'doubling of risk' is shown."[38] Based on this conclusion, the court held that to survive summary judgment on the issue of generic causation, each plaintiff had to prove not only that radiation is capable of causing the injury in question, but that he or she had been exposed to a threshold dose of radiation that statistically doubled his or her risk of harm.[39]

The district court's conclusion that plaintiffs had to demonstrate a doubling of the risk to survive summary judgment on generic causation resulted in the exclusion of seventeen of the plaintiffs' expert witnesses who had not concluded that Hanford's radiation emissions resulted in a doubling of the risk to any particular plaintiff.[40] The court then established threshold levels of radiation exposure for each particular illness[41] and dismissed any claim where the plaintiff had not been exposed to the official doubling dose, regardless of any other particularized evidence.[42] As a result of the district court's requirement that plaintiffs establish a doubling-of-the-risk in order to advance past the generic causation stage, the vast majority of the Hanford downwinders' claims were dismissed.[43]

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[37] *Id.* at *5.

[38] *Id.*

[39] *Id.* at *10.

[40] *See In re* Hanford Nuclear Reservation Litig., 292 F.3d 1124, 1132 (9th Cir. 2002).

[41] For example, the court held that those plaintiffs asserting thyroid cancer claims had not presented a genuine issue of material fact and could not survive summary judgment unless their exposure to radioactive iodine was greater than five rads for plaintiffs aged zero to four when exposed; ten rads for plaintiffs aged five to nine when exposed; thirty-three rads for those aged ten to nineteen when exposed; and one hundred rads for those who were twenty and over when exposed. *Id.* While a "curie" measures the amount of radioactive material emitted during the decay of an unstable particle, a radiation absorbed dose, or "rad," is defined as the deposition of one hundred ergs of energy in one gram of material (in this case the human body) from ionization due to radiation emissions. *See* Thomas W. Lippman, *Nuclear Exchange Brewing at N.R.C.; Becquerel, Gray and Sievert May Obliterate Curie, Rad and Rem*, WASH. POST, July 19, 1989, at A21.

[42] *See In re Hanford Nuclear Reservation Litig.*, 292 F.3d at 1132.

[43] The only remaining claims after summary judgment were those meeting the district court's strict age, time, proximity, and dose requirements for thyroid cancer, non-autoimmune clinical and subclinical hypothyroidism, bone cancer, lung cancer,

C. The In re Hanford *Plaintiffs' Appeal to the Ninth Circuit*

The *In re Hanford* plaintiffs immediately appealed the district court's grant of summary judgement to the Ninth Circuit Court of Appeals. Plaintiffs raised three main arguments, all of which were based upon what they perceived as the district court's erroneous conclusion that *Daubert* mandated a threshold showing that a doubling-of-the-risk had occurred at the generic causation stage.[44] Plaintiffs argued that, by adopting the "doubling-of-the-risk" standard, the district court had prematurely decided issues of individual causation.[45] Further, plaintiffs contended that the district court's discovery order led them to believe that to survive summary judgment on generic causation they needed only to meet the "capable-of-causing" standard, not the doubling-of-risk standard; therefore, they argued, the district court had prejudiced plaintiffs' case because their understanding of the generic causation standard had shaped their production of expert reports.[46]

Nearly four years later, on June 18, 2002, the Ninth Circuit Court of Appeals reversed the district court's ruling in an opinion that significantly clarified and narrowed application of the "doubling of the risk" standard in toxic tort litigation in the Ninth Circuit.[47] The appellate court disagreed strongly with the district court's conclusion that a doubling-of-the-risk was required to advance past the generic causation phase and instead credited plaintiffs' argument that generic causation only addresses whether radiation exposure was "capable of causing" plaintiffs' claimed illnesses.[48] The Ninth Circuit concluded that the trial court had "in essence skipped the generic causation inquiry and decided issues of individual causation without the benefit of full discovery or particularized medical evidence."[49]

More importantly, the Ninth Circuit went on to hold that when considering the issue of individual causation in the *In re Hanford* litigation, it was erroneous to hold that a doubling-of-the-risk

salivary cancer, and breast cancer if the female plaintiff was lactating at the time of the exposure. *See id.*

[44] *See id.* at 1133.

[45] *Id.*

[46] *Id.*

[47] *Id.* at 1133-37, 1139.

[48] *Id.* at 1134.

[49] *Id.* at 1134-35.

was a strict requirement before causation could be established.[50] In so ruling, the Ninth Circuit held that both the defendants and the district court had improperly interpreted *Daubert* as uniformly mandating a doubling-of-the-risk standard when plaintiffs in toxic tort cases rely on epidemiological proof and statistical evidence to establish causation.[51] The Ninth Circuit concluded that *Daubert*'s holding was inapposite to *In re Hanford* because in *Daubert* the plaintiffs lacked scientific evidence showing that the drug in question (Bendectin) was even capable of causing plaintiff's birth defects.[52] In contrast to the complete lack of evidence supporting generic causation in *Daubert* (i.e., whether Bendectin was capable of causing birth defects), the Ninth Circuit noted that scientific and legal authority had long recognized that "[r]adiation is capable of causing a broad range of illnesses, even at the lowest doses."[53]

Because there was no dispute as to whether radiation was capable of causing the illnesses the *In re Hanford* plaintiffs alleged, the Ninth Circuit held that the district court had erred in requiring plaintiffs to prove exposure to a set threshold level of radiation that resulted in a doubling of their risk for contracting any particular illness.[54] Instead, the Ninth Circuit held that the "validity of a claim should not depend on whether a plaintiff was exposed to a fraction of a rem lower than the 'doubling dose'"[55] and concluded that "common sense alone mitigates against es-

[50] *Id.* at 1135.

[51] *Id.* at 1136-37.

[52] *Id.* The Ninth Circuit stated, "The only evidence plaintiffs had that Bendectin caused their own birth defects was (1) proof that their mothers took Bendectin during pregnancy, and (2) epidemiological evidence that mothers who used Bendectin during pregnancy bore more children with birth defects than mothers who did not use Bendectin." *Id.* at 1136.

[53] *Id.* at 1137. The Ninth Circuit also distinguished *Schudel v. General Electric Co*., 120 F.3d 991 (9th Cir. 1997), the only other Ninth Circuit case cited by the defendants and the district court as supporting their conclusions that the *In re Hanford* plaintiffs were required to meet the doubling of the risk standard during the generic causation stage. *Id.* at 1136. The Ninth Circuit noted that, in *Schudel*, the "sole causation evidence" put forth by plaintiffs to meet the more likely than not burden of proof under Washington state tort law was expert testimony that exposure to defendant's cleaning solvents "could possibly" have caused one of the plaintiff's neurological symptoms. *Id.* Because the *Schudel* plaintiffs lacked any other evidence of causation, their claims were required to meet the same substantiation requirements of *Daubert*. *Id.* Thus, the Ninth Circuit distinguished *Schudel* from *In re Hanford* on the same grounds as *Daubert*. *Id.* at 1133-37.

[54] *Id.* at 1137.

[55] *Id.*

tablishing a bright line threshold for safe irradiation."[56]

Based on its holding that the doubling of risk standard should not be employed as a strict, threshold requirement once the capacity of a given substance to cause a plaintiff's complained-of illness is established, the Ninth Circuit also concluded that the district court committed reversible error by excluding plaintiffs' experts simply because those experts had failed to conclude that a doubling-of-risk had occurred.[57]

II

Principles of Causation in Toxic Tort Litigation

A. Distinguishing Toxic Tort Causation from Traditional Tort Causation

The *In re Hanford* plaintiffs' difficulties with establishing that large-scale emissions of highly radioactive substances from the Hanford Nuclear Reservation, rather than some other independent factor, caused their documented medical conditions is commonly described as the "problem of the indeterminate plaintiff."[58] Numerous plaintiffs in other toxic tort cases have

[56] *Id.* (quoting *In re* TMI Litig., 193 F.3d 613, 727 n.179 (3d Cir. 1999)).

[57] *Id.* at 1138-39.

[58] The "problem of the indeterminate plaintiff," which is the focus of this Note, must be distinguished from what is commonly referred to as the "problem of the indeterminate defendant," which, in and of itself, stands as a substantial evidentiary burden for plaintiffs in toxic tort litigation. This problem arises when a plaintiff cannot ascertain which of the various defendants was actually responsible for producing the specific toxic substance to which plaintiff attributes his injuries. *See* Christopher L. Callahan, *Establishment of Causation in Toxic Tort Litigation*, 23 Ariz. St. L.J. 605, 612 (1991). Over the last several decades, certain courts considering the problem of the indeterminate defendant have refused to deny recovery to plaintiffs despite seemingly insurmountable proof problems. Instead, these courts have fashioned innovative liability schemes that stretch the boundaries of traditional tort causation principles. The most notable of these schemes are enterprise liability, adopted in *Hall v. E.I. DuPont de Nemours & Co*., 345 F. Supp. 353 (E.D.N.Y. 1972), and the market share liability schemes adopted by the California Supreme Court in *Sindell v. Abbott Laboratories*, 607 P.2d 924 (1980), and by the New York Court of Appeals in *Hymowitz v. Eli Lilly & Co.*, 539 N.E.2d 1069 (1989).

In *Hall*, plaintiffs were children who had been injured when blasting caps they were playing with exploded. 345 F. Supp. at 359. The explosion destroyed the blasting caps, thus making it impossible to identify the particular manufacturer of the blasting caps at issue. *Id.* at 372. Refusing to dismiss the case despite this lack of proof, the *Hall* court held that joint knowledge and action in failing to provide a warning label on the blasting caps was sufficient to state a claim for relief against all the manufacturers of such blasting caps. *Id.* at 373-74. The *Hall* court held that "joint control may be shown . . . [by] evidence that defendants, acting independently,

faced similar proof problems.[59] In fact, commentators have long argued that the peculiar nature of toxic tort injuries creates a virtually insurmountable hurdle for plaintiffs who attempt to establish that a causal link exists between a given toxic exposure and

adhered to an industry-wide standard or custom with regard to the safety features of blasting caps." *Id.* at 374.

Sindell involved a suit by children who had developed birth defects as a result of their mothers' ingestion of the morning sickness pill DES. 607 P.2d at 925. There had been numerous manufacturers of the drug and no plaintiff could identify any one of them as having manufactured the drug actually ingested by their mother. *Id.* Again, the court refused to dismiss the suit and instead stretched the bounds of traditional tort causation to hold defendants liable. Under the theory developed by the *Sindell* court, once the plaintiffs joined the manufacturers of a substantial share of the California market for the product that caused the injury, "each defendant [would be] held liable for the proportion of the judgment represented by its share of that market unless it demonstrates that it could not have made the product which caused plaintiff's injuries." *Id.* at 937.

Hymowitz involved a similar factual setting as *Sindell* but with the New York Court of Appeals taking a slightly different approach to market share liability. First, the court allowed plaintiffs to name defendants representing a substantial share of the *national* DES market, with liability being based upon a particular defendant's share of that market. 539 N.E.2d at 1085. Further, unlike *Sindell*, the defendants were not allowed to exculpate themselves from liability even if they could prove their particular drug was not ingested by any plaintiff's mother. *Id.* at 1089. Thus, the only way a defendant could avoid liability was for that defendant to prove that it either had never marketed DES or that it had not marketed DES for use during pregnancy. *Id.* at 1092. A defendant who marketed DES for use during pregnancy but whose product the plaintiff's mother could not have ingested because it was not marketed in the relevant geographical area could still be held liable. *See also*, Abel v. Eli Lilly & Co., 343 N.W.2d 164 (Mich. 1984) (adopting an alternative liability scheme under which all alleged tortfeasors must be joined as defendants); Martin v. Abbott Labs., 689 P.2d 368 (Wash. 1984) (adopting what the court termed "market-share alternate liability" under which all defendants who are unable to clear themselves from liability are presumed to have equal market shares totaling 100%).

Commentators have suggested that mass toxic tort litigation cases involving indeterminate defendants present an ideal context for bending the traditional tort recovery requirements, even in the absence of a clear causal link. *See*, *e.g.*, Callahan, *supra* note 58, at 614-16. These commentators argue that eliminating the need for a plaintiff to identify a particular defendant as having caused his injuries does not defeat the principle that a defendant should properly be held liable for only the injuries that it has caused. *Id*. at 616. This is so because (theoretically, at least) under a liability scheme such as market share liability, if every injured person were to file suit in a jurisdiction that had adopted market share liability, each defendant would only be held liable for that part of the damage it actually caused. *Id.* In this way, the traditional underpinnings of tort law are upheld, despite the reformulation of proof requirements.

[59] *See*, *e.g.*, Daubert v. Merrell Dow Pharm., Inc., 43 F.3d 1311 (9th Cir. 1995); *In re* Breast Implant Litig., 11 F. Supp. 2d 1217 (D. Colo. 1998); Hall v. Agent Orange Prod. Liab. Litig., 597 F. Supp. 740 (E.D.N.Y. 1984); *In re* "Agent Orange" Prod. Liab. Litig., 597 F. Supp. 740 (E.D.N.Y. 1984); Allen v. United States, 588 F. Supp. 247 (D. Utah 1984); Merrell Dow Pharm., Inc. v. Havner, 953 S.W.2d 706 (Tex. 1997); Landrigan v. Celotex Corp., 605 A.2d 1079 (N.J. 1992).

the later onset of their illness.[60] This distinct problem necessitates a different undertaking than the establishment of causation in more traditional tort cases. The difference is due primarily to the absence of any objectively verifiable causal chain in toxic tort cases and because a toxic tort plaintiff's injury is neither traumatic nor sudden.[61] Instead, injuries caused by toxic/radioactive exposures, such as those in *In re Hanford*, occur as unobservable, sub-cellular chemical disruptions after prolonged exposure to both the substance in question and a myriad of other substances which may or may not themselves be harmful to the individual.[62] The unobservable nature of such injuries are, in turn, followed by a long latency period before any signs of illness can be detected.[63]

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[60] *See*, *e.g.*, Callahan, *supra* note 58, at 605-06; Trisha T. Pritikin, *Hanford: Where Traditional Common Law Fails*, 30 GONZ. L. REV. 523 (1995-1996); David Rosenberg, *The Causal Connection in Mass Exposure Cases: A 'Public Law' Vision of the Tort System*, 97 HARV. L. REV. 849, 869 (1984); Melissa Moore Thompson, *Causal Inference in Epidemiology: Implications for Toxic Tort Litigation*, 71 N.C. L. REV. 247, 251 (1992).

[61] For a detailed analysis of the reasons why causation is more problematic in toxic tort litigation see *Allen v. United States*, 588 F. Supp. 247 (D. Utah 1984), *rev'd on other grounds*, 816 F.2d 1417 (10th Cir. 1987), *cert. denied*, 484 U.S. 1004 (1988). The *Allen* court observed three primary difficulties: 1) the long latency periods following exposure to toxic/radioactive substances allow for possible involvement of "intervening causes" that obscure the connection between plaintiff's exposure and his illness; 2) the "non-specific" nature of the toxic injury obscures the causal connection and differentiates it from traumatic injures which are much easier to trace; 3) the difficulty in distinguishing a disease caused by toxic exposure from diseases that are attributable to either natural or spontaneous causes. *Id.* at 405-06.

[62]

> Radiation injury occurs at the cellular level. The interaction between radiation and cellular water causes toxicity within the cell. This cell damage can affect an organism in four different ways: (1) cell damages may be repaired without injury to the organism; (2) cell damage may cause cell death; (3) the cell may continue to function but lose its reproductive capabilities; and (4) the cell damage may go unrepaired and may modify the cell's code for reproduction.

Craig A. Barr, *A Practical Guide to Proving and Disproving Causation in Radiation Exposure Cases: Hanford Nuclear Site and Radioactive Iodine*, 31 GONZ. L. REV. 1, 4 (1995-1996). Cancer develops in the fourth scenario. *Id.*

[63] Bert Black & David E. Lilienfeld, *Epidemiologic Proof in Toxic Tort Litigation*, 52 FORDHAM L. REV. 732, 738, 744 (1984). Such a scenario obviously differs from a more traditional tort scenario in a number of important ways. As an example, if a plaintiff is injured when he is hit by a negligent motorist who failed to yield at a pedestrian crosswalk, none of the issues inherent in toxic tort litigation need be addressed. There is a clear, observable causal chain because any witness to the accident will testify that they saw the car hit the plaintiff and moments later saw the plaintiff with a broken leg. There is no possible alternate explanation for how the

Despite the differences between injury in traditional and toxic tort cases, courts continue to require plaintiffs in toxic tort litigation to meet the same burden of proof on causation as traditional tort plaintiffs.[64] As a result, plaintiffs in toxic tort litigation have the burden of proving by a preponderance of the evidence, that "but for" the defendants' release of radioactive emissions, they would not have been injured. The disparity then between toxic tort litigation and more traditional tort litigation lies in the significant substantive impediments plaintiffs encounter in attempting to satisfy the preponderance-of-evidence burden.

Because of the practical limitations that result from the traceability issues and long latency periods, the goal of toxic tort plaintiffs in most cases is not to prove the existence of a specific, verifiable and direct link between their illness and exposure. Instead, they seek to prove that there is a sufficiently high *probability* that their exposure to a given substance caused their injuries to allow imposition of liability.[65] To accomplish this goal, toxic tort plaintiffs necessarily rely on scientific evidence, including the results of epidemiological studies[66] and expert testimony based upon those studies, to sufficiently satisfy causation.[67]

B. The Admissibility of Scientific Evidence and Expert Testimony

Probability of causation based on epidemiological studies and expert testimony typically plays a pivotal role in determining whether the plaintiff will prevail in a toxic tort case. Therefore, evidentiary challenges to the admissibility of such evidence, like the summary judgment motion filed by the defendants in *In re*

plaintiff's leg was broken and the plaintiff would not have to expend any resources to establish the obvious fact that the driver of the car caused the injury.

[64] Callahan, *supra* note 58, at 609.

[65] *See* Troyen A. Brennan, *Causal Chains and Statistical Links: The Role of Scientific Uncertainty in Hazardous-Substance Litigation*, 73 CORNELL L. REV. 469 (1998).

[66] *See* Black & Lilienfeld, *supra* note 63, at 738-39. Epidemiology is defined as the study of the incidence, determinants, distribution, and control of disease in human populations. *Id.* at 736. Accordingly, epidemiological studies are undertaken to explore and clarify a possible association between a toxin and a disease within a given population. *Id.*

[67] See Barr, *supra* note 62, at 19 ("Expert testimony for both epidemiological studies and experimental evidence is indispensable."). *See also* Black & Lilienfeld, *supra* note 63, at 738 (explaining that to establish causation in toxic tort cases plaintiffs usually must rely on expert witnesses and the results of epidemiological studies).

Hanford, are a critical part of nearly every toxic tort case.[68] The Supreme Court's 1993 decision in *Daubert v. Merrell Dow Pharmaceuticals, Inc.* governs the standard of admissibility for such scientific evidence.[69]

In *Daubert*, the Supreme Court held that the recently enacted Federal Rule of Evidence (FRE) 702[70] had displaced the traditional *Frye*[71] test of admissibility for scientific testimony. Under *Frye*, the determinative factor for admissibility of scientific evidence was the "general acceptance" of such evidence within the relevant scientific community.[72] Under *Frye*, data based on novel and/or cutting edge scientific research were often excluded because consensus within the scientific community as a whole, rather than the particular judge hearing the case, determined admissibility.[73] Under *Daubert*, however, the Supreme Court charged trial judges with the duty to act as "gatekeepers" of scientific testimony.[74]

This new gatekeeping function required trial courts to assess the admissibility of scientific evidence and testimony using a two-prong analysis. First, from FRE 702's phrase "scientific, technical, or other specialized knowledge" the Supreme Court derived a standard of reliability:[75] To be sufficiently reliable, scientific evidence must have a "grounding in the methods and procedures of science," that is, be "derived by the scientific method" and amount to more than "subjective belief or unsupported speculation."[76] Second, from FRE 702's phrase "assist the trier of fact," the Supreme Court set a standard of relevance:[77] The expert testimony must "fit" the case for which it is proffered, meaning that the testimony must be "relevant to the task at hand" in that it

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[68] *See*, *e.g.*, Michael V. Cires & Martha K. Wivell, *Protecting Your Evidence Against "Junk Science" Attacks*, TRIAL, Nov. 1991, at 35.

[69] Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579 (1993).

[70] Federal Rule of Evidence 702 states: "If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise."

[71] Frye v. United States, 293 F. 1013 (D.C. Cir. 1923).

[72] *Daubert*, 509 U.S. at 589.

[73] Kristina L. Needham, *Questioning the Admissibility of Nonscientific Testimony After* Daubert, 25 FORDHAM URB. L.J. 541, 544-45 (1998).

[74] *Id.*

[75] *See* Heidi Li Feldman, *Science and Uncertainty in Mass Exposure Litigation*, 74 TEX. L. REV. 1, 7 (1995).

[76] *Daubert*, 509 U.S. at 594.

[77] Feldman, *supra* note 75, at 8.

"logically advances a material aspect of the proposing party's case."[78] The Supreme Court articulated four factors to guide courts in evaluating whether proffered scientific evidence constituted scientific knowledge that would assist the trier of fact in understanding or determining a fact at issue: testability, peer review and publication, rate of error, and general acceptance.[79]

After announcing the new standards for admissibility of scientific evidence, the Supreme Court remanded *Daubert* back to the Ninth Circuit Court of Appeals for further proceedings.[80] The Ninth Circuit's ruling on remand (known as *Daubert II*) was the first decision to apply the Supreme Court's two-pronged, reliability/relevance test for the admissibility of scientific testimony and evidence.[81]

C. The Ninth Circuit's Daubert *Ruling*

In *Daubert II*, the Ninth Circuit reconsidered the dismissal of a toxic tort suit brought on behalf of two children against Merrell Dow Pharmaceuticals.[82] The children suffered from limb reduction birth defects alleged to have been caused by their mother's ingestion of the morning sickness drug Bendectin.[83] Like nearly all toxic tort cases, the plaintiffs had sought to establish causation through introduction of expert testimony that was based in large part on data from epidemiological studies.[84] The Ninth Circuit was therefore required to evaluate the reliability and relevance of plaintiffs' scientific evidence in light of *Daubert*'s new standard of admissibility.

Applying the reliability prong to the facts of the case, the Ninth Circuit noted that several factors weighed against admitting plaintiffs' expert testimony.[85] First, none of the experts had conducted any studies on Bendectin prior to being hired as experts.[86] Second, none of plaintiffs' experts had ever published or

[78] *Daubert*, 509 U.S. at 591, 597.

[79] *Id.* at 593-94.

[80] *Id.* at 598.

[81] Daubert v. Merrell Dow Pharm., Inc., 43 F.3d 1311 (9th Cir. 1995).

[82] The United State District Court for the Southern District of California had earlier granted defendant's motion for summary judgment. *See* Daubert v. Merrell Dow Pharm. Inc., 727 F. Supp. 570 (S.D. Cal. 1989).

[83] *Daubert*, 43 F.3d at 1313.

[84] *Id.* at 1320.

[85] *Id.* at 1317.

[86] *Id.*

solicited formal review for any of their studies.[87] The court held: "We've been presented with only the experts' qualifications, their conclusions and their assurances of reliability. Under *Daubert*, that's not enough."[88] Plaintiffs' failure to pass muster under the reliability prong led the Ninth Circuit to conclude that remand might be appropriate so that the plaintiffs could augment their experts' reports in light of *Daubert*'s new requirements. However, the court felt that remand was appropriate only if the plaintiffs could satisfy the relevance prong of admissibility.[89]

The Ninth Circuit construed the relevance or "fit" standard as requiring a "valid scientific connection to the pertinent inquiry as a precondition to admissibility."[90] The court held that "[h]ere, the pertinent inquiry is causation."[91] Plaintiffs had sought to establish causation using expert testimony. The court held that such proof was permitted, but that plaintiffs had to carry the traditional burden of proof by establishing that their injuries resulted from ingestion of Bendectin and not some other independent factor.[92] The court concluded: "[T]his means that plaintiffs must establish not just that their mothers' ingestion of Bendectin increased somewhat the likelihood of birth defects, but that it more than doubled it—only then can it be said that Bendectin is more likely than not the source of their injury."[93] The court also held that for an epidemiological study to demonstrate causation under a preponderance standard, the relative risk of limb reduction defects would have to exceed two.[94] None of plaintiffs' experts had so concluded.[95] The Ninth Circuit concluded that under *Daubert*'s relevance requirement, studies with a relative risk of less than two would not be helpful and would "only serve to confuse the jury."[96] Based on this holding, the court upheld summary judgment for defendant Merrell Dow.[97]

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[87] *Id.* at 1318.
[88] *Id.* at 1319.
[89] *Id.* at 1320.
[90] *Id.*
[91] *Id.*
[92] *Id.*
[93] *Id.*
[94] *Id.* at 1321. In *Daubert II*, because the background data of limb reduction defects was roughly one per thousand births, the court held that plaintiffs would have to show that the incidence of such defects was more than two per thousand among children of mothers who took Bendectin. *Id.* at 1320.
[95] *Id.* at 1321-22.
[96] *Id.*
[97] *Id.*

The breadth of the Ninth Circuit's application of *Daubert*'s relevance standard proved to be the primary point of contention between the district court and the Ninth Circuit Court of Appeals in *In re Hanford*. The district court interpreted the Ninth Circuit's relevance analysis in *Daubert II* as setting a threshold evidentiary requirement, under which scientific evidence and expert opinion that did not demonstrate a relative risk of greater than two was necessarily excluded as irrelevant. On its face that was the Ninth Circuit's conclusion in *Daubert II*. In *In re Hanford*, however, the Ninth Circuit was able to distinguish its own holding by concluding that although a relative risk of greater than two is generally a requirement for satisfying *Daubert*'s relevance prong, the relative risk calculation does not act as a threshold evidentiary requirement when the capability of a substance to cause the injuries plaintiffs allege has already been established.

III

RELATIVE RISK

As *In re Hanford* and *Daubert* illustrate, the ability of a plaintiff to establish causation in toxic tort litigation typically turns on whether plaintiff's scientific evidence and expert testimony are admissible to help establish the probability that exposure to a toxic/radioactive substance, rather than some independent factor, caused plaintiff's injury. The standard of relevance most courts use to determine the admissibility of such evidence examines whether the scientific evidence demonstrates a "doubling of the risk," or, as some courts formulate it, whether the data demonstrate a "relative risk" of greater than two. It is important to understand both the predicate rationale courts employ to justify their reliance on relative risk calculations as well as the basic methods employed by scientists and statisticians in calculating actual relative risk values.

A. Calculating Relative Risk and Probability of Causation

A relative risk value is derived from epidemiological studies of human populations exposed to a particular substance.[98] Relative

[98] *See* Roy Alan Cohen & Jodi F. Mindnich, *Expert Testimony and the Presentation of Scientific Evidence in Toxic Tort and Environmental Hazardous Substance Litigation*, 21 SETON HALL L. REV. 1009, 1035-36 (1991); Jon Todd Powell, *How to Tell the Truth With Statistics: A New Statistical Approach to Analyzing the Bendectin*

risk represents the degree of likelihood that an individual exposed to a particular toxic substance will contract a given disease compared to the degree of likelihood that an unexposed individual will contract the same disease.[99] Put another way, relative risk equals the "risk of disease in a population segment exposed to a particular substance, divided by the risk of disease in the rest of the population."[100] When the relative risk value of an epidemiological study equals one, there are identical proportions of persons with the disease in both the exposed group and the unexposed group, thus demonstrating the absence of any observable association between exposure to a substance and the onset of disease.[101] A relative risk figure of more than one, however, indicates that there are a greater number of people within the exposed population who have contracted a particular disease than in the unexposed population.[102] Thus, a relative risk of more than one demonstrates the existence of an observable association between exposure to a substance and the onset of disease.

Once a relative risk value is derived, it is often used to calculate a probability of causation ("PC") formula.[103] The PC formula is defined as the percentage of risk in the exposed population that is attributable to the substance under analysis, and it is calculated using the following formula: PC=1-1/x, where "x" is the relative risk.[104] The following hypothetical illustrates how a relative risk value is used to calculate a PC formula, which, in turn, produces a meaningful figure for use in analyzing causation

Epidemiological Data in the Aftermath of Daubert v. Merrell Dow Pharmaceuticals, 31 HOUS. L. REV. 1241, 1251 (1994).

99 Thompson, *supra* note 60, at 250-51.

100 *Id.*

101 Cohen & Mindnich, *supra* note 101, at 1035; Thompson, *supra* note 60, at 251. For example, a relative risk value would equal one if epidemiological studies indicated that a specific type of cancer developed in one out of every one thousand individuals within a population segment after widespread exposure to a particular toxic substance, but studies indicated that the normal background rate for that same type of cancer was also one out of every one thousand.

102 Thompson, *supra* note 60, at 251; *see also* Cohen & Mindnich, *supra* note 101, at 1035; Powell, *supra* note 98, at 1251-52. For example, a relative risk value of two would be derived when an epidemiological study indicated that a specific type of cancer developed in two out of every one thousand individuals within a population segment exposed to a particular toxic substance, where the background rate for that same type of cancer was only one out of every one thousand individuals.

103 *See* Cohen & Mindnich, *supra* note 101, at 1035; Mark Parascandola, *What Is Wrong with the Probability of Causation?*, 39 JURIMETRICS J. 29, 31 (1998); Powell, *supra* note 101, at 1251.

104 Powell, *supra* note 98, at 1254; Parascandola, *supra* note 103, at 31-32.

in toxic tort litigation.[105]

If there are 150 cases of a disease within a population segment of 1,000 people who were exposed to a particular toxic substance, and only 100 cases of the same disease in a similar population of 1,000 people who were not exposed to the toxic substance, the applicable relative risk value is 1.5.[106] Once a relative risk of 1.5 is derived, the PC formula discussed above is used to arrive at a probability of causation equal to .33, or 33%.[107] Therefore, when 150 exposed individuals within a population of 1,000 develop a disease where normally only 100 out of 1,000 individuals would develop the same disease, there is only a 33% probability that exposure to the toxic substance in question, rather than some other independent factor, caused any one individual's disease.

However, using this same methodology, if there had been 300 instances of a particular disease in an exposed population where only 100 individuals would normally have contracted the disease, the relative risk would be 3.0 and the probability of causation

[105] It is important to point out that the discussion herein concerning the use of epidemiological evidence is necessarily limited in its scope and not an exhaustive analysis of the scientific underpinnings of epidemiological evidence or the complex mathematical calculations that such studies routinely entail. Many commentators, however, have written extensively on such issues. *See*, *e.g.*, David W. Barnes, *Too Many Probabilities: Statistical Evidence of Tort Causation*, 64 LAW & CONTEMP. PROBS. 191, 198-205 (2001) (detailing statistical calculations used by epidemiologists to interpret data, including concepts of statistical sampling, experimental design and measurement, statistical modeling, hypothesis testing, and extrapolation); Andrew A. Marino & Lawrence E. Marino, *The Scientific Basis of Causality in Toxic Tort Cases*, 21 U. DAYTON. L. REV. 1, 6-22 (1995) (explaining a number of principles relating to the use of epidemiology in the courtroom including dependent variables, dosimetry, risk factors, systemic variation, internal versus external validity, and rate of error); Bruce R. Parker, *Understanding Epidemiology and its Use in Drug and Medical Device Litigation*, 65 DEF. COUNS. J. 35, 35-56 (1998) (explaining different types of epidemiological studies including prospective cohort studies, retrospective cohort studies, case-control studies, nested case-control studies; as well as factors that may contribute to unreliability within those studies such as selection bias, measurement bias, confounding factors, and random chance; as well as tools used to interpret statistical data arrived at in epidemiological studies such as standard deviation, confidence intervals, and rates of error); Thompson, *supra* note 60, at 268-74 (examining epidemiological principles including statistical association, temporality, biological plausibility and coherence, dose-response, consistency, analogy, experimental evidence, and specificity).

[106] A relative risk value of 1.5 is arrived at by dividing 150 (the number of cancers in the exposed population) by 100 (the number of cases in the unexposed population).

[107] A 33% probability of causation is arrived at when the relative risk value is 1.5 through the following calculation: probability of causation equals one minus one divided by 1.5; i.e., [1 – (1/1.5)], which equals [1 – .66] which equals .33, or 33% probability of causation.

would be 67%.[108] Thus, in this second hypothetical, there would be a 67% chance that a given individual within the exposed population had contracted their disease through exposure to the toxic substance in question, rather than some other independent factor.

B. Application of the Preponderance of Evidence Standard to the Relative Risk Value

In the second example above, the relative risk of 2.0 results in there being a 50% probability that plaintiffs contracted their disease through exposure to the toxic substance in question, rather than some independent variable. Applying the relative-risk standard to the legal requirement that a plaintiff prove causation by a preponderance of the evidence (i.e., prove it is more probable than not that "but for" exposure to the substance in question the illness would not have occurred) it is easy to see why many courts have concluded that a relative risk of greater than two is important evidence for making a determination regarding causation. When relative risk is more than two, and the corresponding probability of causation is greater than 50%, civil courts applying the preponderance-of-evidence standard can reasonably conclude that a given plaintiff's disease was more likely than not caused by exposure to the chemical in question.[109]

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[108] A 67% probability of causation is arrived at where the relative risk value is 3.0 through the following calculation*:* probability of causation equals one minus one divided by 3.0; i.e., [1 – (1/3.0], which equals [1 – .33] which equals .67, or 67% probability of causation.

[109] Before the rise of epidemiology as the central means for plaintiffs to establish probability of causation in the toxic tort context, commentators observed two distinct views concerning the scientific proof needed to satisfy the preponderance of evidence standard. *See* Rosenberg, *supra* note 60, at 867. The so called "strong" version of the preponderance rule maintained that a plaintiff could not prevail on his claim without producing both statistical correlations from epidemiological studies indicating that the relative risk exceeded two *and* particularistic evidence providing direct and specific proof of a causal relationship between the substance in question and an individual plaintiff's injuries. *Id.* at 869. In contrast, some courts used the "weak" version of the preponderance rule under which a plaintiff could prevail by merely coming forward with evidence regarding the statistical correlation between the chemical in question and his disease. *Id.* at 874-75. Over time, the "weak" version gained prominence, such that the distinction between the two views is no longer made. *See* Joseph Sanders & Julie Machal-Fulks, *The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law*, 64 LAW & CONTEMP. PROBS. 107, 119 n.124 (2001).

C. Hall v. Baxter Healthcare Corp.

Hall v. Baxter Healthcare Corp.[110] provides a typical example of a trial court exercising its "gatekeeping" role to exclude plaintiffs' scientific testimony under the preponderance rule. Holding that a relative risk of less than two fails to satisfy *Daubert*'s relevance prong,[111] *Hall* involved litigation over plaintiffs' "atypical connective tissue disease" (ACTD), a condition allegedly caused by the rupture and/or degradation of plaintiffs' silicone breast implants.[112] In December 1996, the United States District Court for the District of Oregon granted defendants' motions in limine to exclude plaintiffs' expert testimony concerning causation.[113] The court analyzed the issue by applying *Daubert*'s two-pronged admissibility test,[114] and held that for plaintiffs' proffered epidemiological evidence on causation to meet the reliability requirement, "the relative risk . . . arising from the epidemiological data . . . will, at a minimum, have to exceed [two]."[115] Plaintiffs' experts based their opinions upon sixteen published epidemiological studies assessing the relationship of silicone breast implants and connective tissue disease.[116] The court noted that the highest relative risk demonstrated by any of the plaintiffs' studies was only 1.24.[117] The court concluded: "Therefore, these studies cannot support expert testimony that silicone 'more likely than not' causes disease or signs and symptoms of disease in wo-

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

111 *See id.* at 1411-14.

112 *Id.* at 1391. The defendants in *Hall* included Baxter Healthcare Co., Bristol-Myers Squibb, Minnesota Mining and Manufacturing, and Dow Corning Corp. *Id.* at 1391 n.1.

113 *Id.* at 1394.

114 *Id.* at 1396. The court wrote "The task before this court . . . is two-pronged. First, the court must determine whether plaintiff's experts' testimony reflects 'scientific knowledge,' . . . and was 'derived by the scientific method.' Second, the court must ensure that the proposed testimony 'fits,' that is, that the testimony is 'relevant to the task at hand' in that it 'logically advances a material aspect of the proposing party's case.'" *Id.*

115 *Id.* at 1403 (citing Daubert v. Merrell Dow Pharm., Inc., 43 F.3d 1311, 1321 (9th Cir. 1995)).

116 *Id.* at 1404.

117 *Id.* at 1405. Plaintiffs offered one other study, referred to in the opinion as the "Liang-Schottenfeld abstract," that reported a relative risk of 2.27 for undifferentiated connective tissue diseases in breast implant patients. *Id.* at 1404. The court refused to consider this study, however, because it was unpublished, and because the study only included three women with breast implants, thus severely calling into question its epidemiological significance. *Id.* at 1405.

men."[118] As a result, the court excluded every scientific study put forth by the plaintiffs and ruled that not a single one of plaintiffs' experts would be allowed to testify.[119] The motion in limine was accordingly granted.[120]

IV

Critiquing Judicial Reliance on Relative Risk and Epidemiological Evidence

Since 1982, at least thirty-one court opinions, including *In re Hanford*, *Daubert*, and *Hall*, have addressed the question of relative risk in toxic tort litigation.[121] In twelve of those cases, courts held that a relative risk of greater than two is necessary to support a reasonable inference of causation.[122] However, in fourteen other cases, courts held that a relative risk of greater than two was not a strict requirement.[123] Similarly, of the twenty-one cases addressing whether expert opinion on causation must be based on data indicating a relative risk of more than two, ten courts held in the affirmative while eleven declined to adopt such a requirement.[124]

Given courts' divergent treatment of relative risk, it is not surprising that commentators are similarly divided. Some commentators criticize overreliance by courts on relative risk. These authors contend that the relative risk calculation, and the epidemiological data upon which such calculations are based, are not proper grounds for awarding recovery in toxic tort litigation.[125] On the other side of the argument are numerous other commentators who argue that relative risk calculations and epidemiological data actually fail to take into account many injuries that should be compensible under tort law. These authors conclude that any relative risk values demonstrating a positive correlation between exposure to toxic/radioactive substances and later onset of disease are relevant and should be considered at trial.[126]

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[118] *Id.* at 1405.
[119] *Id.* at 1414-15.
[120] *Id.* at 1414.
[121] Russellyn S. Carruth & Bernard D. Goldstein, *Relative Risk Greater Than Two in Proof of Causation in Toxic Tort Litigation*, 41 Jurimetrics J. 195, 197 (2001).
[122] *Id.* at 200.
[123] *Id.* at 201.
[124] *Id.* at 201-02.
[125] *See infra* notes 130-48 and accompanying text.
[126] *See infra* notes 149-60 and accompanying text.

A. Arguments That Courts Put Too Great an Emphasis on Epidemiological Evidence and Relative Risk Calculations

One major criticism of judicial reliance on relative risk calculations comes from epidemiologists and statisticians. These commentators argue that, although adoption of the greater-than-two rule by courts is theoretically sufficient to meet the requirements of the civil preponderance-of-evidence standard, it completely ignores the weight that scientists give to relative risk values.[127] Epidemiologists have long considered relative risk values of less than three as indicative of only a weak association between exposure and disease occurrence since it could be due entirely to flaws in either study design or data analysis.[128] These experts argue that epidemiologists do not consider a strong association between exposure and onset of disease to exist until studies demonstrate relative risk values exceeding four,[129] with some experts arguing that epidemiologists only consider relative risk values exceeding eight as indicating a strong correlation.[130] Owing to what they perceive as the disconnect between legally sufficient statistical correlations and scientifically sufficient statistical correlations, these commentators argue that if reliance upon weak associative correlations such as relative risk values below four is to continue, such evidence should not be applied with "certainty" to plaintiffs in the absence of other particularized proof of causation.[131]

In addition to science-based critiques of how courts use epidemiological data and relative risk calculations, a number of commentators have put forward more policy-based objections. Preeminent among such commentators is Michael Dore of

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[127] Thompson, *supra* note 60, at 259.

[128] Bert Black et al., *Expert Evidence: A Practitioner's Guide to Law, Science, and the FJC Manual*, 7 ALI-ABA 115, 130 (2002) (citing David E. Lilienfeld, *Overview of Epidemiology*, 3 SHEPARD'S EXPERT & SCIENTIFIC EVID. Q. 25, 26 (1995)).

[129] *Id.*

[130] Thompson, *supra* note 60, at 289.

[131] *Id.* at 290. Specifically, Thompson argues that moderate associations, meaning those based on relative risk values of between three and eight, are sufficiently probative of causation if such results are "coupled with strength in other epidemiological causation guidelines, such as a 'strong temporal relationship.'" *Id.* at 289. She then argues that with weak associations, "the plaintiff should probably show strength in other epidemiological causation guidelines and, in addition, similarity between the plaintiff and those in the study population." *Id.* Thompson's arguments come close to advocating a return to the "strong" preponderance of the evidence standard discussed *supra* in note 112.

Harvard Law School.[132] Professor Dore's critique is grounded in the observation that epidemiological evidence is proof not of actual, individual *causation*, but is only demonstrative of the relative level of risk to which a defendant's activities exposed members of plaintiff's group and this risk does not specifically relate to any individual plaintiff's illness.[133] Professor Dore argues: "[C]ourts that fail to distinguish the issue of risk from that of actual causation may accordingly, but erroneously, permit the evidence of risk to establish causation."[134] He notes that the inherent limits of epidemiology as a tool for use in the courtroom are a result of its general and statistical nature. Epidemiological studies are general, he argues, because they deal with sources of disease in groups of people rather than in individuals. Moreover, because such studies are statistical, they quantify probabilities and can only show whether a defendant's conduct increased plaintiff's risk to some statistically measurable extent; thus, it is utterly impossible for epidemiology to answer the critical question of whether the defendant's conduct actually injured a specific plaintiff.[135]

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[132] *See* Michael Dore, *A Commentary on the Use of Epidemiological Evidence in Demonstrating Cause-in-Fact*, 7 HARV. ENVTL. L. REV. 429 (1983). Professor Dore's 1983 commentary is cited in virtually every law review article and court opinion critiquing overreliance on relative risk calculations.

[133] *Id. See* Richard W. Wright, *Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof: Pruning the Bramble Bush by Clarifying the Concepts*, 73 IOWA L. REV. 1001, 1011-12 (1988) (arguing that particularistic evidence is a necessary prerequisite for proving actual causation).

[134] Dore, *supra* note 132, at 436. For an earlier iteration of Professor Dore's argument see Louis L. Jaffe, *Res Ipsa Loquitur Vindicated*, 1 BUFF. L. REV. 1, 4 (1951). Professor Jaffe argues:

> Abstract probability may play a role in finding a fact, but what is referred to in the traditional formula is the greater probability in the case at hand. The 'probabilities' in the abstract or statistical sense is only a datum. The jury's quest for the fact can only be undertaken if there is evidence in addition to that upon which the mere abstraction is based which will enable the jury to make a reasoned choice between the competing possibilities. . . . There must be a *rational*, i.e., evidentiary basis on which the jury can choose the competing probabilities. If there is not, the finding will be based . . . on mere speculation and conjecture.

Id. See also, Laurence H. Tribe, *Trial by Mathematics: Precision and Ritual in the Legal Process*, 84 HARV. L. REV. 1329, 1341 (1971) (concluding it is not enough that "the mathematical chances somewhat favor the proposition" to be proved).

[135] Dore, *supra* note 132, at 435-36. Commentators have rebutted Professor Dore's critique by arguing that he incorrectly framed the principles of toxic tort causation. *See* Khristine L. Hall & Ellen K. Silbergeld, *Reappraising Epidemiology: A Response to Mr. Dore*, 7 HARV. ENVTL. L. REV. 441, 445-46 (1983). These authors argue that the real issue in toxic tort litigation is whether sufficient evidence

The strength of Professor Dore's argument—that the statistical nature of epidemiological evidence renders it an improper basis for proving individual causation—was illustrated by Professor Charles Nesson in his famous "blue bus" hypothetical:

> While driving late at night on a dark, two-laned road, a person confronts an oncoming bus speeding down the center line of the road in the opposite direction. In the glare of the headlights, the person sees that the vehicle is a bus, but cannot otherwise identify it. He swerves to avoid a collision, and his car hits a tree. The bus speeds past without stopping. The injured person later sues the Blue Bus Company. He proves, in addition to the facts stated above, that the Blue Bus company owns and operates 80% of the buses that run on the road where the accident occurred. Can he win?[136]

The hypothetical demonstrates the shortcomings of judicial reliance on pure statistical evidence to establish proof of causation in a manner similar to Dore's critique. Both authors are wary of the unfairness that would result if liability were imposed on a non-negligent defendant due solely to reliance on statistical, rather than particularized, causal evidence.[137]

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exists to infer that it is more probable than not that a given exposure caused the plaintiff's disease. *Id.* The statistical evidence generated by epidemiology is highly probative of just that issue. *Id.* at 456. In fact, these authors point out that signature diseases, which are usually not perceived as presenting difficult individual causation issues, are simply cases in which the statistical evidence is highly persuasive because the background incidence of disease is low compared to the incidence in the exposed population. *Id.* at 446-47. *See also* Thomas M. Reavley & Daniel A. Petalas, *A Plea for Return to Evidence Rule 702*, 77 TEX. L. REV. 493, 504 n.72 (1998) (criticizing Professor Dore's position by noting, "Epidemiological evidence can be probative and can yield a basis for assigning explanatory force to an expert's conclusion, despite the individual attribution uncertainty.").

136 Charles Nesson, *The Evidence or the Event? On Judicial Proof and the Acceptability of Verdicts*, 98 HARV. L. REV. 1357, 1378-79 (1985).

137 *See*, *e.g.*, Senn v. Merrell Dow Pharm., Inc., 850 F.2d 611 (9th Cir. 1988) (per curium). In *Senn*, plaintiff sought to recover damages due to the debilitating injuries she suffered from a DPT vaccination. *Id.* at 612-13. Plaintiff joined the only two drug manufacturers that had produced the vaccine. Sean v. Merrell Dow Pharm., Inc., 751 P.2d 215, 259 (Or. 1988). She could not establish which of the two had actually manufactured the particular dose she was given, but the evidence did establish that one defendant had approximately 70% of the market share for the vaccine, while the other defendant had approximately 30% market share. *Id.* at 259 n.1. Plaintiffs urged the Oregon Supreme Court to hold the companies liable under an alternative liability scheme, but the court refused, citing the "violence" such schemes do to the causation element of tort law. *Id.* at 269. Despite the fact that the company with the majority market share had more probably than not caused plaintiff's injury, the court cited the fundamental unfairness that would result from imposing liability against a non-negligent defendant based purely on statistical evidence that it controlled a majority share of the market at the time plaintiff was injured. *See id*.

Other commentators have rebutted the implied conclusion of the blue bus hypothetical by pointing out that the situation presented by Professor Nesson differs significantly from the typical toxic tort case.[138] In the blue bus hypothetical, judicial reliance on statistical proof would be particularly questionable because the data would be used to prove both the identity of the defendant and, by extension, negligence and causation.[139] This contrasts with the typical toxic tort case, such as *In re Hanford*, where statistics are employed only after a defendant has been identified and only after plaintiff's exposure to a particular toxic substance due to the defendant's negligence has been established; thus, in the typical toxic tort case, statistical evidence is used to establish only causation, and, accordingly, is less suspect.[140]

An argument developed more fully in subsequent articles by Professor Nesson concerned the existence of "legal probability" as a distinct concept from pure scientific probability.[141] In this formulation, legal probability is not a strict mathematical concept but one that incorporates within it the idea of justice.[142] Under this line of reasoning, an outcome is legally probable if it "best accomplishes a just and acceptable resolution of the dispute."[143] Professor Nesson thus wrote, "This suggests that the acceptability of a conclusion is not a simple function of mathematical probability, but rather is a complex matter of communication that depends on the nature of the issue, the process of decision,

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138 *See*, *e.g.*, Thompson, *supra* note 60, at 265.

139 *Id.* at 266.

140 Thompson put forth her own hypothetical, asking the reader to ponder the role of statistics in establishing only the issue of causation. *Id.* That scenario is as follows:

> While standing in a large crowd at the bus station, a man becomes aware that a bus from the Blue Bus Company is nearing the crowd. The bus pulls into the station, and the crowd moves to stay clear of the bus. The man, although jostled somewhat by the crowd, does not appear to have been injured. Two years later, he develops pain in his lower back, and x-rays reveal a slipped disc. He sues the Blue Bus Company. He proves, in addition to the facts states above, that statistics indicate it is dangerous for busses to drive near crowds because this can cause injuries to those in the crowd. In addition, he proves that there are more back injures in crowd-related accidents than would be normally expected. Can he win?

Id.

141 Charles Nesson, *Agent Orange Meets the Blue Bus: Factfinding at the Frontier of Knowledge*, 66 B.U. L. REV. 521, 521 (1986).

142 *Id.*

143 *Id.*

and the purposes and audiences the conclusion serves."[144] The detachment of legal probability from pure scientific probability recognizes that courts should still be free to use epidemiological data, even in the absence of particularized proof of causation, in situations where uncertainty concerning proof of causation intersects with underlying principles of justice.

B. Arguments That the Relative Risk of Greater Than Two Is Underinclusive of Compensible Injuries

While a number of commentators argue that the uncertainties upon which epidemiology and risk ratio calculations are based should preclude overreliance on such data, numerous others have argued that requiring relative risk values of more than two prohibits imposition of liability for many injuries that should be compensable under tort law. One critique, published by epidemiologists Sander Greenland and James M. Robins has gained wide recognition among commentators who argue that requiring a relative risk of more than two results in underinclusion of illnesses.[145] Greenland and Robins claim that the fundamental deficiency of relative risk is its failure to distinguish between "etiologic" and "excess" cases of cancer.[146] An etiologic case is one where radiation exposure caused a cancer but the cancer would have occurred without the exposure,[147] while an excess case of cancer is one that would not have occurred without the exposure.[148] For the excess cases, it is clear that the exposure in question caused the cancers whereas the etiologic cases of cancer would have occurred anyway. The relative risk calculation is conceptually limited, Greenland and Robins argue, because it can only account for the excess cases of cancer, while principles of tort recovery support imposition of liability for both the excess cases *and* for those etiologic cases where a defendant's toxic exposure has hastened the onset of a disease.[149] The authors note that, in a non-toxic tort setting when a physician fails to use an obvious treatment for a patient's cancer and this negligence hastens the patient's death, the fact that the patient would have died

144 *Id.* at 522.

145 Sander Greenland & James M. Robins, *Conceptual Problems in the Definitions and Interpretation of Attributable Fractions*, 128 AM. J. EPIDEMIOLOGY 1185 (1988).

146 *Id.* at 1185-86.

147 *Id.*

148 *Id.* at 1190.

149 *See id.* at 1191-93.

anyway does not excuse the physician's negligence.[150]

In addition to the inability of relative risk calculations to account for etiologic cases of disease, commentators point to two additional inherent flaws in epidemiological studies that can also result in an underinclusion of illnesses and a relative risk calculation that is artificially low. The first of these is the "incomplete accrual problem," which is based on the observation that epidemiological studies are typically undertaken for public health purposes and not for the purpose of supporting litigation.[151] Because such studies are done for public health purposes, they aim to identify risks against which protective measures might be taken to promote public health—not to discover whether a relative risk of more than two exists.[152] In pursuing this preventative goal, the positive results of an epidemiological study are usually published as soon as a statistically significant elevated risk is revealed—without waiting for all possible adverse effects to fully develop.[153] For cancers and other illnesses with long latency periods, additional cases routinely develop following the original publication of findings. These post-publication illnesses are generally not counted because there is no scientific reason to do so. As a result, for cancer and other diseases with long latency periods, the published relative risk is often lower than the actual relative risk that exists.[154]

The "healthy worker effect" is another factor resulting in epidemiological underinclusivness, and it is based on the fact that

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150 The doctrine of "loss of a chance" allows for recovery in similar situations. *See* Herskovits v. Group Health Coop., 664 P.2d 474 (Wash. 1983). In *Herskovits*, the Washington Supreme Court considered whether proof of a plaintiff's increased risk of death, due to his decreased chance of survival resulting from a physician's error, was sufficient to present the issue of proximate cause to a jury. *Id.* at 476. The court held: "It is not necessary for a plaintiff to introduce evidence to establish that the negligence resulted in the injury or death, but simply that the negligence increased the *risk* of injury or death. The step from the increased risk to causation is one for the jury to make." *Id.* at 478. The *Herskovitz* court thus concluded that reduction of a chance of survival from 39% to 25% would be sufficient evidence to allow the jury to consider the issue of proximate cause. *Id.* at 479. *See also* Robert S. Bruer, *Loss of a Chance as a Cause of Action in Medical Malpractice Cases*, 59 MO. L. REV. 969 (1994) (analyzing the two different approaches to the loss-of-a-chance doctrine: In one approach courts adopt a relaxed causation standard which treats the underlying injury as the ultimate injury sustained by the plaintiff, usually death; in the other approach, courts treat the actual loss of a chance as a distinct compensable injury).

151 Carruth & Goldstein, *supra* note 121, at 207.

152 *Id.*

153 *Id.*

154 *Id.*

epidemiological studies are often conducted in the workplace.[155] Because workers on the whole are healthier than the general population, their risk of contracting almost any disease is lower.[156] In fact, it is not uncommon for workplace studies to find a relative risk for mortality from all causes to be in the range of 0.7 to 0.8.[157] As a result, comparing studies conducted on exposed workers with individuals in the general population will necessarily result in understating the risk of disease that actually exists.

V

ALTERNATIVES TO THE PREPONDERANCE RULE

A. The Proportionality Rule

Given the divisive scholarly debate concerning the utility and appropriateness of relying on epidemiological data and relative risk calculations as evidence of causation in toxic tort litigation, it is not surprising that commentators have advanced numerous alternatives to the current preponderance-of-evidence rule.[158]

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[155] Kenneth L. Mossman & Gary E. Marchant, *The Precautionary Principle and Radiation Protection*, 13 RISK 137, 145 (2002).

[156] *Id.* at 145 n.15. The principal reasons given by the authors for why workers are more healthy than non-workers are that chronically unhealthy individuals do not enter the work force in large numbers and individuals in the workforce typically have better access to health care than the general population. *See id.*

[157] *Id. See also* Carruth & Goldstein, *supra* note 121, at 207. In fact, the healthy worker effect has been observed in workplace studies conducted at the Hanford Nuclear Reservation. Gerald Woodcock & Michelle R. Fox, *Hanford and Public Health: No Cause for Alarm*, 31 GONZ. L. REV. 69, 73 (1996). In arguing that the "wealth of health and safety data from a variety of agencies provides strong evidence that there has been no increase in radiologically induced health problems in the areas surrounding Hanford," *id.* at 69, Woodcock & Fox note that "no group of people has been closer to the sources of radiation at Hanford than those who have worked there. . . . [yet the] overall health of Hanford workers is significantly better than the general population." *Id.* at 73.

[158] In addition to the alternatives discussed herein, see Troyen A. Brennan & Robert F. Carter, *Legal and Scientific Probability of Causation of Cancer and Other Environmental Diseases in Individuals*, 10 HEALTH POL. POL'Y L. 33 (1985) (recommending that legislatures adopt a policy of compensation proportional to risk); Colin Hugh Buckley, *A Suggested Remedy for Toxic Injury: Class Actions, Epidemiology, and Economic Efficiency*, 26 WM. & MARY L. REV. 497 (1985) (suggesting proportional recovery after cost/benefit analysis); Kenneth A. Cohen, *Class Actions, Toxic Torts, and Legal Rules*, 67 B.U. L. REV. 581 (1987) (book review) (discussing premise that instead of being required to prove individual-level causation, plaintiffs should only be required to prove they are members of a group for which causation has been established for a particular substance); William R. Ginsberg & Lois Weiss, *Common Law Liability for Toxic Torts: A Phantom Remedy*, 9 HOFSTRA L. REV.

Chief among the suggested alternatives is the "proportionality rule," which originally gained prominence through the scholarship of Professor David Rosenberg.[159] The proportionality rule is a burden-shifting scheme that does away with a plaintiff's traditional burden of establishing that it is more likely than not that the defendant's toxic/radioactive substance, and not some independent factor, caused plaintiff's injury.[160] Instead, under the proportionality rule, causation is presumed as soon as a plaintiff brings forward statistical evidence indicating that the defendant caused injury to a known proportion of the individuals within an exposed population.[161] No individualized proof is required; instead, courts are free to impose liability and award compensation to every plaintiff in the exposed population in proportion to the risk that a given defendant created through its negligent actions or omission.[162] If, as would typically be the case, multiple defendants were involved in the litigation, they would each be held liable to the plaintiffs for a pro rata share of the damages.[163]

The following example illustrates the proportionality rule:[164]

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859 (1981) (arguing for an administrative compensation scheme); Christine M. Grant, *Establishing Causation in Chemical Exposure Cases: The Precursor Symptoms Theory*, 35 RUTGERS L. REV. 163 (1982) (advocating for relaxed causation standards where certain medically consistent and individualized precursors are evident); Talbot Page, *On the Meaning of the Preponderance Test in Judicial Regulation of Chemical Hazards*, 46 LAW & CONTEMP. PROBS., Summer 1983, 267 (allowing recovery if probability of causation exceeds a critical value determined by cost/benefit analysis); Glen O. Robinson, *Multiple Causation in Tort Law: Reflections on the DES Cases*, 68 VA. L. REV. 713 (1982) (suggesting a probabilistic approach based on statistical data); Wendy E. Wagner, *Choosing Ignorance in the Manufacture of Toxic Products*, 82 CORNELL L. REV. 773 (1997) (advocating for burden shifting after plaintiffs make a prima facie showing of probability); Wendy E. Wagner, *Trans-Science in Torts*, 96 YALE L.J. 428 (1986) (arguing for adoption of a "qualitative causation standard," with burden shifting and negligence requirements).

159 *See* Rosenberg, *supra* note 60, at 866 ("[T]he proportionality rule is ideally suited to the task of resolving the problem of causal indeterminacy in mass exposure cases."); *see also* David Kaye, *The Limits of the Preponderance of the Evidence Standard: Justifiably Naked Statistical Evidence and Multiple Causation*, 1982 AM. B. FOUND. RES. J. 487, 495-502 (1982) (proposing a shift to the proportionality rule of causation).

160 Shelly Brinker, Comment, *Opening the Door to the Indeterminate Plaintiff: An Analysis of the Causation Barriers Facing Environmental Toxic Tort Plaintiffs*, 46 UCLA L. REV. 1289, 1313 (1999).

161 *Id.* at 1313; Callahan, *supra* note 58, at 669.

162 Brinker, *supra* note 160, at 1313; Callahan, *supra* note 58, at 669.

163 Brinker, *supra* note 160, at 1313; Callahan, *supra* note 58, at 669.

164 Adopted from *In re* "Agent Orange" Prod. Liab. Litig., 597 F. Supp. 740, 838 (E.D.N.Y. 1984). In *In re "Agent Orange"*, Judge Weinstein exhaustively considered

Suppose that in a given population known to have been exposed to radioactive emissions, there is a normal background level of 1,000 cases of thyroid cancer. To investigate the effects of radioactive emissions, an epidemiological study is conducted[165] with the results indicating 1,100 cases of thyroid cancer in the studied population. These 100 "extra" cases of cancer would result in a relative risk calculation well below two, and a corresponding probability of causation of just nine percent.[166] It would be extremely unlikely for a court employing the preponderance rule to admit such evidence (or any expert opinion based upon it) under the relevance prong of *Daubert*;[167] thus, no plaintiff would be able to recover damages.

Suppose instead that all 1,100 of the individuals suffering from thyroid cancer joined in a lawsuit against the five companies responsible for allowing the radioactive emissions—but this time the plaintiffs sued in a court that had adopted the proportionality rule. Further, assume that expert medical opinion could set the level of damages at an average of $1,000,000 per cancer.[168] Under the proportionality rule, the 100 "extra" cases of thyroid

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the pros and cons of several different liability schemes while considering the appropriateness of a proposed settlement agreement between American chemical companies and thousands of Vietnam veterans who claimed injuries resulting from exposure to dioxins in the defoliant Agent Orange. *Id.* at 746-48. Although Judge Weinstein did not adopt any particular liability scheme, he wrote favorably concerning the proportionality rule, dismissing criticism that it would, in all likelihood, result in compensation being paid to individuals whose injuries had not been caused by exposure to toxic substances. *Id.* at 833-43. Concerning this criticism, Judge Weinstein wrote "[n]o matter what system is used, the purpose is to hold a defendant liable for no more than the aggregate loss fairly attributable to its tortious conduct. As long as that goal is met a defendant can have no valid objection that its rights have been violated." *Id.* at 839. *See also* Brinker, *supra* note 160, at 1313-14 (explaining the proportionality rule through hypotheticals); Callahan, *supra* note 58, at 668-70 (using a hypothetical to explain the scheme).

165 Of course, any such studies would have to be done years or decades after the exposure had occurred because of the inevitability of long latency periods.

166 *See supra* text accompanying notes 103-08.

167 *See supra* text accompanying notes 86-91.

168 As Judge Weinstein acknowledged in his *In re "Agent Orange"* opinion, "Putting a dollar amount on the damages suffered by individual plaintiffs is, from a real-world standpoint, a critical part of the solution." *In re* "Agent Orange" Prod. Liab. Litig., 597 F. Supp. 740, 838 (E.D.N.Y. 1984). Judge Weinstein was concerned that the benefits gained from adjudicating toxic tort claims in the form of class action lawsuits under the preponderance rule would be lost in the event that individualized mini-trials were needed to set a specific dollar figure for each plaintiff's injury. *Id.* He noted that the fewer plaintiffs involved in a suit, and the less variation between those plaintiffs' injuries, the more generalized and efficient the process would be. *Id.* at 839. Judge Weinstein concluded that "[a] preferred solution is to pay claims

cancer would be multiplied by the average $1,000,000 in damages and a recovery of $100,000,000 in favor of all the plaintiffs would be awarded. Since no plaintiff would be able to conclusively establish that his or her cancer had actually been caused by the radiation, the total award of $100,000,000 would then be evenly divided between the 1,100 plaintiffs. Thus, each plaintiff would recover $90,000. The percentage of the award paid by each defendant would then depend on factors such as the total amount of radiation emitted while that particular defendant was operating the facility.

B. Variations of the Proportionality Rule

A variation on the compensation structure of the proportionality alternative, known as the "most-likely-victim" approach, is predicated on the fact that not all individuals within an exposed population are equally at risk for developing a given disease.[169] Instead, risk is determined in part by the extent and duration of an individual's exposure and the timing of the exposure during an individual's lifetime, as well as multifarious lifestyle factors, including socioeconomic conditions and independent health variables such as whether an individual smokes and whether he or she lives in a heavily polluted city.[170] Using individualized factors such as these, the most-likely-victim scheme takes a more complex approach to compensation. It alters the compensation structure of the proportionality alternative by dividing members of the exposed population into subgroups composed of individuals with similar characteristics and similar risks of developing cancer from the same general toxic or radiological exposure.[171] This occurs after liability has been established. Starting at the subgroup with the highest risk level and proceeding downward, plaintiffs are compensated fully until a defendant has paid dam-

on a fixed and somewhat arbitrary schedule using a ministerial agency" similar to the process used by Medicaid and Medicare. *Id.*

[169] Daniel A. Farber, *Toxic Causation*, 71 MINN. L. REV. 1219, 1220 (1987). For criticism of the "most likely victim" approach see John G. Culhane, *The Emperor Has No Causation: Exposing a Judicial Misconstruction of Science*, 2 WIDNER L. SYMP. J. 185 (1996) (arguing that the "most likely victim" approach and similar alternatives are the "misguided children of frustration" that promote overreliance on epidemiological data instead of acknowledging the inherent limitations of using category-wide surveys to establish particularized injuries).

[170] Farber, *supra* note 173, at 1243-44.

[171] *Id.* at 1243-44.

ages equal to the attributable risk that it negligently created.[172] Under this scheme, those individuals in the highest risk levels would be fully compensated for their injuries, since it would be more likely that they had in fact contracted their illnesses as a result of the defendant's actions, while those classified into the lower-risk subgroups, whose illnesses are more likely to have alternative causes, would receive no compensation.[173]

Another variation on the proportionality alternative would allow for both apportionment of damages and burden shifting.[174] Under this approach, conceived by Professor Richard Delgado, when a defendant creates an attributable risk that a certain percentage of an exposed population will become ill due to the defendant's activities, the burden would shift to the defendant company to prove that it did not in fact cause each injury.[175] The defendant could rebut the presumption either by proving through scientific studies that the probability of it having caused the injuries to the exposed population was lower than plaintiffs alleged or by demonstrating that plaintiffs injuries had an alternate cause.[176] If the defendant company failed to rebut its presumption, damages would be apportioned among all plaintiffs according to the risk of harm that the defendant created in the exposed population.[177]

The most recognized benefit of the proportionality rule and its variants is the high level of deterrence such schemes would occasion on the manufacturers of toxic substances.[178] Critics of these

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172 *Id.* at 1247-54.

173 *Id.* at 1254. The obvious criticism of the "most-likely-victim" approach is that science lacks the ability to accurately assign a risk-level to any subgroup. *See* Callahan, *supra* note 58, at 669. However, recent articles have also suggested that within the near future, progress in mapping of the human genome will allow scientists to better evaluate an individual's propensity for developing certain diseases resulting from exposure to particular toxic substances. *See* Gary E. Marchant, *Genetics and Toxic Torts*, 31 SETON HALL L. REV. 949 (2001).

174 Richard Delgado, *Beyond* Sindell*: Relaxation of Cause-in-Fact Rules for Indeterminate Plaintiffs*, 70 CAL. L. REV. 881, 886-87 (1982).

175 *Id.* at 900.

176 *Id.*

177 *Id.* at 901.

178 *See*, *e.g.*, *In re* "Agent Orange" Prod. Liab. Litig., 597 F. Supp. 740, 838 (E.D.N.Y. 1984) ("[T]he deterrent effect . . . on producers would be significant."); Brinker, *supra* note 160, at 1315 (noting that the proportionality rule "achieves optimal deterrence and encourages a defendant to invest most efficiently in care and safety"); Delgado, *supra* note 174, at 893 ("[T]he increased costs [under the proportionality rule] deter the defendant from engaging in the liability generating the practice.").

alternatives, however, note that they have the potential to open the door to endless litigation. These critics argue that significantly more lawsuits would be filed under the proportionality rule since recovery would be possible based solely on an increase in the risk of harm, and that settlements would be more unlikely as each side emphatically relied on its competing interpretation of the epidemiological data.[179] The primary criticism of proportionality-type schemes, however, is the fact that in every instance a plaintiff would either be overcompensated or undercompensated for his or her injuries.[180] This criticism is particularly cogent because under- and overcompensation would be even more pronounced in cases where relative risk calculations were less than two—i.e., the exact situation for which the liability scheme would be adopted.

Overcompensation under the proportionality rule would occur when a plaintiff received a windfall by recovering a percentage of damages from a defendant who did not actually cause his or her injuries.[181] When the relative risk of the supporting epidemiological studies was less than two this would statistically be the rule, rather than the exception. Undercompensation in situations where the relative risk was less than two would perhaps be even be more troubling because a plaintiff who had actually developed illnesses due to a defendant's wrongful actions would recover only a fraction of the actual damages he or she suffered—an amount that would doubtlessly fail to make them whole again.[182]

Both Delgado and Rosenberg recognized that proportionality-type schemes were open to criticism due to the inevitability of over- and undercompensation, but both authors argued that fairness to a class of individuals, considered as a whole, was a legitimate alternative to individualized justice.[183] Thus, both authors conceptualized their schemes as necessarily shifting away from individual justice and towards group responsibility and group justice in the unique situation of mass toxic exposure. According to Professor Rosenberg: "The proportionality rule simply holds the

[179] Brinker, *supra* note 160, at 1320.

[180] Delgado, *supra* note 174, at 893-94; Brinker, *supra* note 160, at 1320.

[181] Delgado, *supra* note 174, at 893; Brinker, *supra* note 160, at 1319.

[182] Brinker, *supra* note 160, at 1320.

[183] Delgado, *supra* note 174, at 888; Rosenberg, *supra* note 160, at 884. For an analysis of the group responsibility concept of alternative liability schemes, see Robert A. Baruch Bush, *Between Two Worlds: The Shift From Individual to Group Responsibility in the Law of Causation of Injury*, 33 UCLA L. REV. 1473 (1992).

defendant liable for . . . the losses it has caused in the 'body' of the exposed population. . . . [T]he population as a whole gains no windfall. Essentially, the aggregative conception envisions that courts will assess liability on behalf of the entire exposed population. . . ."[184] When viewed as a system of group responsibility, wherein the defendant's duty is owed to a population as a whole and under which a plaintiff's claim is based on injuries suffered by a group of which he or she was a member, the criticism of proportionality-type schemes for under- and overcompensation is dampened—perhaps only because one realizes the necessity, rather than any innate preference of the group justice concept in the event of mass toxic exposure.

C. Novel Alternatives to the Preponderance Rule

In addition to the proportionality rule and similar alternatives, a number of commentators have proposed more radical solutions for reformulating liability schemes in toxic tort litigation. The most widely discussed of these proposals would entirely do away with the traditional basis of requiring demonstrable evidence that a defendant's toxic or radioactive substance caused a particular plaintiff's illness.[185] Instead, liability in toxic tort litigation would be based on a showing that a defendant failed to develop and disclose information necessary to assess the latent risks of a particular chemical.[186] Such a radical solution may be warranted because conditioning liability on a plaintiff's ability to prove that the defendant's product caused his illness is counterproductive in that it only "creates incentives on the part of corporations not to know and not to disclose" information regarding the health effects and emissions of their chemicals.[187] This proposed alternative would induce corporations to engage in far more scientific research on the health effects of their chemicals and to make this data widely available. This knowledge would in turn provide greater protection against adverse exposure to hazardous substances *before* such exposures happened.[188]

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[184] Rosenberg, *supra* note 60, at 884-85.

[185] Margaret A. Berger, *Eliminating General Causation: Notes Towards A New Theory of Justice and Toxic Torts*, 97 COLUM. L. REV. 2117 (1997).

[186] *Id.* at 2117-18.

[187] *Id.* at 2119.

[188] *Id.* at 2152.

D. Allen v. United States

Although numerous alternatives to the preponderance rule have been proposed, and some such as the proportionality alternative have found their way into judicial opinions,[189] *none* of the schemes discussed above has ever been judicially adopted. However, in the notable case of *Allen v. United States*,[190] Judge Jenkins, writing for the District Court of Utah, adopted a remarkably liberal and pro-plaintiff test to hold the federal government liable for illnesses and deaths caused as a result of radioactive fallout from open-air nuclear bomb tests held in Nevada during the late 1950s and early 1960s.[191]

In his 230-page opinion, Judge Jenkins devoted considerable time to discussing the problems inherent in establishing causation through reliance on scientific data such as epidemiological studies.[192] He indicated, however, that he had no ideological problem concerning the utility of such evidence, and stated that, in answering the question of whether radiation causes cancer, "[w]e simply mean that a population exposed to a certain dose of radiation will show a greater incidence of cancer than that same population would have shown in the absence of the added radiation."[193] To overcome the significant burdens the plaintiffs faced before they could establish causation, the court adopted an analytical framework through which it sought to establish "exclusive factual connections" between the government's actions and plaintiff's injuries.[194] For example, the court sought to determine whether any given plaintiff's injury was consistent with the kind of harm that would result from the defendant's particular risk creating conduct.[195] After noting the burden-shifting scheme as

189 *See*, *e.g.*, *In re "Agent Orange" Prod. Liab. Litig.,* 597 F. Supp. 740, 838-40 (E.D.N.Y. 1984).

190 Allen v. United States, 588 F. Supp. 247 (D. Utah 1984), *rev'd on other grounds*, 816 F.2d 1417 (10th Cir. 1987).

191 *Id.* at 257. The action was brought under the Federal Tort Claims Act by approximately 1200 named plaintiffs who alleged more than 500 deaths and injuries. *Id.* at 257-58. From among the numerous plaintiffs, the district court selected twenty-four representative claims and held a full trial on those claims to develop a framework for managing the remainder. *Id.* at 258-60. In the end, the court entered final judgment in favor of plaintiffs on nine of the representative claims, judgment against the plaintiffs on fourteen of the representative claims, and left one claim unresolved. *Id.* at 420-48.

192 *See id.* at 259-63, 404-19.

193 *Id.* at 405.

194 *Id.* at 406.

195 The court described its rationale by noting that*:* "The more exclusive the fac-

adopted by the California Supreme Court in *Sindell v. Abbot Laboratories*,[196] the *Allen* court strongly criticized the government for its negligent failure to monitor and record the radiation exposures of off-site residents, as well as for its failure to issue warnings about the known risks of radiation or to provide information about appropriate precautions—actions which, in the court's view, were sufficient to justify shifting the burden of proof to the government on the issue of causation.[197]

Based on this rationale, the court adopted a test providing that once a plaintiff established generic causation through "substantial, appropriate, persuasive and connecting" factors, the burden of proof would shift to the government to disprove individual causation.[198] The court thus adopted a burden-shifting, substantial-factor test for establishing causation. The factual connections the plaintiff needed to satisfy the test included: 1) plaintiff's proximity to the test facility; 2) whether plaintiff's injury was consistent with those known to be caused by radiation; and 3) the probability that the plaintiff was exposed to dangerous levels of radiation.[199]

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tual connections that may be established by evidence, the stronger the rational basis for focusing the tools of legal analysis upon a specific defendant's conduct." *Id.* at 406. The specific analysis put forward by the court was:

> That the defendant was engaged in a risk-creating conduct of a particular type, and plaintiff's injuries are consistent with the kind of harm that is predicted and observed when such risks are created, makes the factual connection seem even more exclusive—exclusive of other defendants, other connections, other "causes."

196 See *supra* text accompanying note 58.

197 *Allen*, 588 F. Supp. at 408-15.

198 *Id.* at 415. The precise test adopted by the court was:

> Where a defendant who negligently creates a radiological hazard which puts an identifiable population group at increased risk, and a member of that group at risk develops a biological condition which is consistent with having been caused by the hazard to which he has been negligently subjected, such consistency having been demonstrated by substantial, appropriate, persuasive, and connecting factors, a fact finder may reasonably conclude that the hazard caused the condition absent persuasive proof to the contrary offered by the defendant.

Id. The court restated its substantial factor test later in its opinion, stating:

> Where it appears from a preponderance of the evidence that the conduct of the defendant significantly increased or augmented the risk of somatic injury to a plaintiff and that the risk has taken effect in the form of a biologically and statistically consistent somatic injury, i.e., cancer or leukemia, the inference may rationally be drawn that defendant's conduct was a substantial factor contributing to plaintiff's injury.

Id. at 428.

199 *Id.*

After explicitly rejecting a "but-for" preponderance-of-evidence test, Judge Jenkins held that when a plaintiff relied on statistical evidence in determining the probability that a defendant's act rather than some independent factor caused his injury, "The mechanical application of a 'greater-than-100%-increase' test . . . represents merely the refabrication of the 'but-for' test of causation in mathematical form."[200] The court rejected such an approach and held instead that statistics derived from epidemiological data should be "relied upon as a guide rather than as an answer. . . ."[201] The court thus adopted a malleable definition of statistical relevance that allowed it to assess liability based on what it concluded was "inherently a question of policy," rather than an all-or-nothing determination based on a threshold statistical correlation.[202] Despite the *Allen* court's application of a substantial factor test in the place of a "but-for" preponderance rule, and despite its refusal to require any threshold of statistical proof such as a doubling of the risk, the court denied recovery to the majority of the twenty-four representative plaintiffs.[203]

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[200] *Id.* at 418. The expression "greater-than-100%-risk" has the same meaning as "relative risk greater than two." The court explained the inequities that would result if a relative risk of more than two was strictly required. The court noted that under a strict preponderance of the evidence analysis, a plaintiff who was one of 190 experiencing a disease after exposure, where the background rate of disease showed only one hundred cases, could not recover. *Id.*

[201] *Id.*

[202] *See id.*

[203] *See id*. at 429-43. The court gave varying reasons for rejecting plaintiffs' claims. Some plaintiffs did not suffer the type of cancer that was shown to be caused by radiation exposure. The majority of rejected claims, ten in all, were dismissed because those plaintiffs could not show any increased incidence of their specific type of cancers in the exposed population. Nonetheless, the government did not successfully rebut its presumption regarding the claims of nine separate plaintiffs, including one plaintiff who had contracted leukemia as a child and who introduced studies demonstrating a relative risk of between 2.5 and 3.5 that were consistent with her proximity to the test sites and her age group. *Id.* at 437-39. The district court entered judgment in favor of these nine plaintiffs, with damages set in excess of $2.6 million. *Id.* at 447.

Three years later, the Tenth Circuit Court of Appeals reversed the *Allen* decision, holding that the plaintiffs' claims were non-justiciable under the "discretionary function doctrine." Allen v. United States, 816 F.2d 1417, 1424 (10th Cir. 1987). The discretionary function doctrine is an exception contained within the Federal Tort Claims Act that protects the federal government from liability arising out of the performance of, or failure to perform, a discretionary function or duty. *See* Giselse C. Dufort, *All the Kings' Forces or the Discretionary Function Doctrine in the Nuclear Age:* Allen v. United States, 15 ECOLOGY L. Q. 477, 478 (1988). The Tenth Circuit stressed that every test the government conducted in Nevada had been approved by the Atomic Energy Commission, the National Security Council, and the

CONCLUSION

Much of this Note focuses on what level of epidemiologically-based probability plaintiffs must bring forward on the issue of causation before a court will hold the defendant liable for creating an identifiable level of risk in the exposed population. The opinions of the United States Supreme Court and the Ninth Circuit Court of Appeals in *Daubert v. Merrell Dow Pharmaceuticals*, respectively, established a rigorous standard of relevance for epidemiological evidence; thus the idea of probability is merged with the civil preponderance-of-evidence rule and statistically derived, relative-risk calculations must exceed two before they are admissible. In turn, the district court in *In re Hanford* interpreted the *Daubert* relevance standard as a threshold evidentiary requirement, which meant that, regardless of accepted scientific opinion or plaintiffs' particularized evidence, epidemiological studies detailing illnesses consistent with radioactive exposure were nonetheless ruled inadmissible. The district court's holding was then reversed by the Ninth Circuit, which clarified the *Daubert* standard in its own *In re Hanford* opinion by holding that probabilistic, relative-risk calculations should not be viewed as a threshold requirement in cases where the capability of a toxic/radioactive substance to cause the type of illnesses complained of by a plaintiff has already been established.

In so holding, the Ninth Circuit's *In re Hanford* opinion added to the voluminous scholarship in favor of increased judicial reliance on probability calculations that do not meet the strict requirements of the preponderance of evidence standard. These arguments resonate with particular sensibility in cases of mass toxic exposure where large subpopulations of individuals are exposed to increased levels of risk as a result of government-initiated or government-funded activities undertaken with the purported goal of benefiting the nation as a whole. This argument is typified by Judge Jenkins's conclusion in *Allen* that statistical evidence should act as a guide, not an answer, in reaching conclusions concerning what is essentially a policy determination,

President of the United States. *Allen*, 816 F.2d at 1419. The Tenth Circuit denied each and every claim filed by plaintiffs holding that "[the] acts of subordinates in carrying out the operations of government in accordance with official directions cannot be actionable." *Id.* at 1420 (citing United States v. Varig Airlines, 467 U.S. 797, 811 (1984)); *see also* Barry Kellman, *Judicial Abdication of Military Tort Accountability: But Who Is to Guard the Guards Themselves?*, 1989 DUKE L.J. 1597, 1628-34 (1989).

as well as in Professor Nesson's observation that probability is conceptually different in legal versus mathematical contexts and that, in a legal sense, any "standard" of probability necessarily changes depending on the nature and purposes of the litigation. In fact, in two mass toxic tort cases where plaintiffs achieved some level of success, policy determinations concerning the nature of the duty owed and the purposes underlying the litigation, rather than the sheer weight of statistical probability regarding causation, strongly favored the plaintiffs.[204] In *Allen*, this resulted in citizens exposed to nuclear fallout recovering against the federal government; in *In re Agent Orange*, Judge Weinstein approved a settlement between American chemical companies and Vietnam veterans who had been exposed to the defoliant Agent Orange.

The ongoing *In re Hanford* litigation presents an equally compelling situation for interjecting policy considerations and the "nature and purpose" of plaintiff's claims in making a determination of whether proffered probability of causation evidence is sufficient to hold defendants liable for the known radiological risk they created. This is because the *In re Hanford* plaintiffs were exposed to radiation as a result of government contractors' refinement of weapons-grade plutonium that was used both during World War II and to fuel the Cold War arms race—a situation seemingly as compelling, from a policy point of view, as both *In re Agent Orange* and *Allen*.

Yet, as this Note illustrates, the barriers to recovery traditionally faced by toxic tort plaintiffs were initially applied with particularly unforgiving zeal to the claims of the *In re Hanford* plaintiffs. Although the Ninth Circuit's reversal will narrow application of the preponderance rule on remand, the mere fact that some of plaintiffs' evidence is now likely to survive further pre-trial evidentiary challenges cannot provide much relief, justice, or hope to the thousands of long-suffering Hanford

[204] Judge Weinstein described the plaintiffs' purposes behind the Agent Orange litigation as follows:

> Vietnam veterans and their families desperately want this suit to demonstrate how they have been mistreated by the country they love. They want it to give them the respect they have earned. They want it to protect the public against future harm by the government and chemical companies. They want a jury "once-and-for-all" to demonstrate the connection between Agent Orange and the physical, mental and emotional problems from which many of them clearly do suffer.

In re "Agent Orange" Prod. Liab. Litig., 597 F. Supp. 740, 747 (E.D.N.Y. 1984).

downwinders. The Hanford litigation has crawled along for more than twelve years, and, as the history of the case demonstrates, both sides are likely to contest and appeal each and every incremental ruling—virtually guaranteeing prolongment of the litigation into the next decade. These delays are particularly troubling because most of the injuries *In re Hanford* plaintiffs sustained occurred more than fifty-five years ago. Further negating any hope of recovery that the Ninth Circuit's ruling might have generated for the Hanford downwinders is that *In re Hanford* defendant companies have no incentive to settle the litigation due to the 1957 Price-Anderson Act, which provides each of them with full indemnity from any damages that might be assessed.[205] Another factor that makes settlement unlikely is another provision of the Price-Anderson Act that provides for full reimbursement of all the defendant companies' legal fees that to date exceed forty million dollars.[206]

In light of these realities and the need for an expeditious and just remedy for the *In re Hanford* plaintiffs, federal legislation should be enacted. This legislation would provide damage awards for those plaintiffs who can establish, with a reasonable degree of probability, that their injuries were caused by exposure to radiation emitted from the Hanford facility. To assure adequate compensation, the preferred liability scheme for such legislation would be based on the burden-shifting, substantial-factor

[205] For the indemnity provisions of the Price-Anderson Act, see 42 U.S.C. §§ 2011-2296 (2001). Congress passed the Price-Anderson Act in 1957 after a series of congressional hearings before its Joint Committee on Atomic Energy demonstrated that "the open-ended liability associated with operating nuclear facilities was deterring the participation of private parties in the industry." Chris Addicott, *Double Indemnity for Operators of Nuclear Facilities?* In re Hanford Nuclear Reservation Litigation, *the Price-Anderson Act, and the Government Contractor Defense*, 72 WASH. L. REV. 505, 509 (1997). The Act draws a distinction between contractors who operate commercial power plants for the Nuclear Regulatory Commission and those involved with weapons production for the Department of Energy. Commercial power plant contractors are *not* similarly indemnified by the government under the Price-Anderson Act. *Id.* at 510. The indemnity covers all weapons contractors and subcontractors for all liability arising out of a contract activity, without any deductible, shared liability, or other condition on recovery. 42 U.S.C. § 2210(d)(2). The statute also provides that the government shall indemnify the contractors for any legal expenses they incur in defending suits arising from nuclear incidents at their facilities. *Id.* All of the *In re Hanford* defendants have indemnity agreements with the government that fall under the Price-Anderson Act. Addicott, *supra*, at 511-12.

[206] *See Radiation Exposure Claims Can Proceed Against Contractors*, 16 No. 4 ANDREWS GOV'T CONT. REP. 11, 18 (July 3, 2002).

model adopted by the *Allen* court and not on the preponderance standard adopted by the Ninth Circuit in *In re Hanford*. Such a scheme would provide greater flexibility concerning the use of probabilistic evidence and greater recognition of the underlying policy considerations that necessitate the legislation. In recent years there have been several bills introduced that would have provided relief to individuals exposed to nuclear waste through utilization of schemes similar to the one used in the *Allen* court.[207] While none of these bills were enacted, that fact likely has less to do with any perceived lack of utility for the scheme and more to do with a lack of immediacy felt by legislators in the absence of a discernable group of injured individuals, such as the group that now exists in the case of the *In re Hanford* plaintiffs.

Although a legislative remedy would likely result in the undercompensation of plaintiffs in comparison to the potential damage awards they might receive from successful resolution of the current litigation,[208] the rapidity with which the legislation could be passed makes this solution reasonably equitable. This is especially true because the epidemiological testing and probability calculations that would be utilized to assess liability under the proposed legislation have already been conducted and judicially scrutinized.

Justification for requiring the federal government to enact legislation providing the Hanford downwinders with compensation for their injuries, rather than allowing the current legal battle to meander through the court system for the next decade, comes from the government's own data demonstrating the massive amount of airborne radiation that was released from the Hanford nuclear facility during its four decades of operation. The initial legal success gained by defendant companies should not deter

[207] *See* Toxic Tort Act, H.R. 1049, 96th Cong. §§ 101-101 (1979). Under H.R. 1049, persons claiming injuries from toxic waste exposure needed only to establish a "sufficient" relationship between the toxic exposure, the injury, the geographical proximity, and the temporal extent of their exposure. Similarly, a bill introduced in 1980 provided that once a claimant made a prima facie showing of causal connection, the burden of producing evidence shifted to the defendant to demonstrate that exposure to its toxic chemicals was an insignificant contribution to claimant's injuries. *See* Palma J. Strand, Note, *The Inapplicability of Traditional Tort Analysis to Environmental Risks: The Example of Toxic Waste Pollution Victim Compensation*, 35 STAN. L. REV. 575, 582-84 (1983).

[208] Although it is worth noting that given the huge amount of attorney fees and costs associated with the Hanford plaintiffs' suit, the potential "enormity" of recovery cited by the district court in its original *In re Hanford* opinion is certainly open to speculation.

passage of such legislation since, as this Note demonstrates, their early court victory stemmed largely from rigid imposition of traditional tort rules of causation rather than legitimate questions of fact regarding the high levels of toxic exposure to which individuals living near the Hanford facility were exposed. The suffering of the Hanford downwinders has been prolonged for over half a century, and in the end may only be exacerbated by the legal system. While passage of federal legislation will surely require painful compromises on the part of those who have been injured by radioactive emissions, it is clearly the best hope that exists, given the nearly insurmountable barriers that exist for toxic tort plaintiffs attempting to impose liability by establishing probability of causation through epidemiological data.