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A Relative Risk 2.0: The Ninth Circuit Revisits Daubert's Epidemiological Standard in In re Hanford Nuclear Reservation Litigation

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A RELATIVE RISK 2.0: THE NINTH CIRCUIT REVISITS *DAUBERT'S* EPIDEMIOLOGICAL STANDARD IN *IN RE HANFORD NUCLEAR RESERVATION LITIGATION*

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Between 1944 and 1987, the Hanford Engineering Works (HEW),[1] site of the world's first large-scale plutonium production facility, contaminated the Pacific Northwest with dangerous levels of radioactivity.[2] The litigation that ensued, *In Re Hanford Nuclear Reservation Litigation*[3] and *In re Berg Litigation*,[4] should spark interest in the legal community because of its sheer size. The number of potential plaintiffs could conceivably be in the hundreds of thousands, while the potential costs could measure billions of dollars.[5] More importantly, this litigation should draw attention because of the Ninth Circuit's remarkable treatment of the plaintiffs' scientific evidence under the *Daubert*[6] standard.[7]

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[1] HEW was established in 1943 by the Manhattan Engineer District of the Army Corps of Engineers who were searching for a place to produce plutonium for nuclear weapons. The Hanford site, along the Columbia River in Richland, Washington provided a perfect setting. Plutonium production began in 1944 and continued after World War II into the 1980s. At peak production in the 1960s, the reactors produced one-fourth of the world's plutonium. Hanford Reach Protec. and Mgt. Program, *Interim Action Plan* ch. 2, http://www.co.benton.wa.us/pl/iap/html/ (last updated Apr. 10, 1998). See also the text accompanying *infra* notes 22-35.

[2] Technical Steering Panel of the Hanford Envtl. Dose Reconstruction Project, *Summary: Radiation Dose Estimates from Hanford Radioactive Material Releases to the Air and the Columbia River* 3 (CDC 1994) [hereinafter *Radiation Dose Estimates*].

[3] 292 F.3d 1124 (9th Cir. 2002).

[4] 293 F.3d 1127 (9th Cir. 2002). *Berg*, the companion case to *Hanford*, 292 F.3d 1124, also involved the claims of multiple plaintiffs exposed to radiation from HEW. The plaintiffs in *Berg*, originally part of the group of plaintiffs in *Hanford*, were severed from *Hanford* during the second phase of discovery. The *Berg* court also reversed the lower court's grant of summary judgment on the same day that *Hanford* was decided. *Berg*, 293 F.3d at 1129.

[5] *Hanford*, 292 F.3d at 1128; *infra* n. 65.

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

[7] For a discussion of the *Daubert* standard, review the text accompanying *infra* notes 12-19.

When the District Court for the Eastern District of Washington dismissed the bulk of the plaintiffs' claims,[8] the *Hanford* plaintiffs appeared likely to meet the same fate as many other toxic exposure victims.[9] Despite well-documented evidence of severe and lengthy radiation exposure,[10] the lower court granted the defendant's motion for summary judgment[11] because the plaintiffs' scientific evidence did not meet *Daubert's* rigorous standard for admissibility.[12] However, this standard has been criticized.[13] Some contend that this criterion is an unrealistically high standard to prove generic[14] causation and is not a good

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[8] *In re Hanford Nuclear Reservation Litig.*, 1998 WL 775340 (E.D. Wash. Aug. 21, 1998), *rev'd*, 292 F.3d 1124 (9th Cir. 2002).

[9] Other victims of radiation exposure have fared no better. *See In re TMI Litig.*, 193 F.3d 613, 622-23 (3d Cir. 1999) (dismissed under *Daubert*, 509 U.S. 579); *Allen v. U.S.*, 588 F. Supp. 247 (D. Utah 1984), *rev'd*, 816 F.2d 1417 (10th Cir. 1987) (holding that atomic weapons testing in the 1950s-60s fell under "discretionary function" and was not subject to tort claims).

[10] *See Radiation Dose Estimates*, *supra* n. 2, at 3 (discussing the dangerous levels of contaminants at HEW between 1944 and 1987). *See also* E.J. Antonio et al., *6.0 Potential Radiological Doses from 2000 Hanford Operations* (DOE 2001) (available at http://www.hanford.gov/docs/annualrp00/section6_0.pdf).

[11] *Hanford*, 1998 WL 775340.

[12] The district court in *Hanford* used a standard requiring the plaintiffs to demonstrate that exposure to radioactive toxic agents statistically doubled their risk of contracting a disease. *Id.* at *11. This standard, a relative risk factor of 2.0, also known as a "doubling of the risk" or "doubling dose," was first articulated in *Daubert v. Merrell Dow Pharmaceuticals Inc.*, 509 U.S. 579, and has been adopted in federal courts. *See e.g. TMI*, 193 F.3d 613; *Schudel v. Gen. Elec. Co.*, 120 F.3d 991 (9th Cir. 1997). *See also infra* nn. 135-141.

[13] *See e.g.* Edward K. Cheng & Albert H. Yoon, *Does* Frye *or* Daubert *Matter? A Study of Scientific Admissibility Standards*, 91 Va. L. Rev. 471 (2005) (*Daubert* "become the foundational opinion in the modern law of scientific evidence and arguably one of the most important decisions in the area of tort reform. Over the years, the *Daubert* test for scientific admissibility has spawned countless articles, symposia, and informal discussions about its merits and drawbacks, particularly in contrast to its principal rival, the *Frye* "general acceptance" test. Commentators have extensively debated which test is the stricter standard. . . . In addition, state supreme courts have repeatedly grappled with whether to adopt *Daubert* or maintain *Frye*." (footnotes omitted)); Richard W. Clapp & David Ozonoff, *Environment and Health: Vital Intersection or Contested Territory?*, 30 Am. J.L. & Med. 189 (2004); Daniel E. Fisher, Daubert v. Merrell Dow Pharmaceuticals: *The Supreme Court Gives Federal Judges the Keys to the Gate of Admissibility of Expert Scientific Testimony*, 39 S.D. L. Rev. 141 (1994). For discussion of *Frye v. United States*, 293 F. 1013 (D.C. 1923), review the text accompanying *infra* notes 106-115.

[14] Causation in toxic tort cases is typically discussed in terms of generic and specific causation. "General or 'generic' causation has been defined by courts to mean whether the substance at issue had the capacity to cause the harm alleged." *Hanford*, 292 F.3d at

indicator of individual[15] causation.[16] In addition, the application of the tort "preponderance of the evidence" causation standard[17] to this epidemiological standard[18] is flawed, both as a scientific premise and as a judicial policy decision.[19]

Remarkably, in June 2002 the Ninth Circuit, home of *Daubert II*[20] and originator of the "doubling risk" standard, reversed the district court's summary judgment order.[21] This article seeks to address some of the ramifications of the Ninth Circuit's ruling in *Hanford*. The article will critically study the courts' use of epidemiological evidence in proving causation in toxic tort litigation and examine how the Ninth Circuit applied the *Daubert* standard in *Hanford*. Finally, it will argue that *Hanford* can and should be a trend in the courts to accept scientifically valid evidence.

II. HISTORY OF THE HANFORD NUCLEAR RESERVATION[22]

Despite warnings from leading scientists and rumors of a nuclear weapons program underway in Nazi Germany, in 1939 the United States lacked a coherent nuclear agenda, as the not-yet-named Manhattan Project

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1133. *See also Sterling v. Velsicol Chem. Corp.*, 855 F.2d 1188, 1200 (6th Cir. 1988) (explaining the difference between individual and generic causation).

[15] "[I]ndividual causation refers to whether a particular individual suffers from a particular ailment as a result of exposure to a substance." *Sterling*, 855 F.2d at 1200.

[16] *See* Jean Macchiaroli Eggen, *Toxic Torts, Causation, and Scientific Evidence After* Daubert, 55 U. Pitt. L. Rev. 889 (1994).

[17] According to *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 43 F.3d 1311, 1322 (9th Cir. 1995), proof of causation by a preponderance of the evidence is always required by legal substantive standards, i.e. that a claimed illness which also appears in the general population was more likely than not caused by a certain substance.

[18] Epidemiology is the study of the incidence of disease in large populations, not the specific causation of any particular individual's disease. *See* Fed. Jud. Ctr., *Reference Manual on Scientific Evidence* 125 (1st ed., West 1994) [hereinafter *Reference Manual I*].

[19] Eggen, *supra* n. 16.

[20] *Daubert*, 43 F.3d 1311 (9th Cir. 1995) [hereinafter *Daubert II*].

[21] *Hanford*, 292 F.3d 1124.

[22] *See generally* T.E. Marceau et al., *The Hanford Site Historic District* (Rosalind E. Schrempf & Janet K. Tarantino eds., Battelle Press 2002) (available at http://www.hanford.gov/doe/history/docs/rl-97-1047/index.pdf); Robert Alvarez, *The Legacy of Hanford*, 227 The Nation 31 (Aug. 19, 2003); Michele Stenehjem Gerber, *Legend and Legacy: Fifty Years of Defense Production at the Hanford Site* (Westinghouse 1992); Kit Oldham, *Construction of Massive Plutonium Production Complex at Hanford Begins in March 1943* (HistoryLink 2003) (available at http://historylink.org/essay/output.cfm?file_id=5363).

consisted only of research with limited funding.[23] This situation changed in 1942 when Enrico Fermi[24] conducted the first sustained nuclear reaction capable of producing plutonium.[25] Later that year, Glenn Seaborg[26] discovered a chemical process that would extract plutonium from irradiated uranium.[27] Equipped with the ability to manufacture plutonium, and prompted by the world war, research turned into full-scale industrial production of the first atomic weapon.[28] This became known as the Manhattan Project.[29]

Central to the Manhattan Project was the production of large quantities of plutonium.[30] To that end, in 1943, Lieutenant General Leslie Groves, who was responsible for the rapid development of the Manhattan Project, selected a 670 square-mile area of southeastern Washington for a plutonium production facility that would later become known as the Hanford Engineering Works.[31] The site was ideal from a military standpoint because its remoteness from civilized areas preserved both security and safety.[32] In addition, the nearby Columbia River provided cold, clear water to cool the reactors, and the Bonneville and Grand Coulee Dam electric grids provided enough electricity for what was to become a vast engineering project.[33] During peak construction in 1944, HEW housed over 45,000 workers[34] and was connected by 158 miles of new railway and 386 miles of new roads.[35]

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[23] Marceau et al., *supra* n. 22, at 1.7-1.8.

[24] Fermi, an Italian physicist and Columbia University physics professor, was one of the leaders of the team of physicists on the Manhattan Project for the development of nuclear energy and the atomic bomb. Fermi received the Nobel Prize in Physics in 1938. *See Nobel Lectures, Physics 1922-1941* (Elsevier 1965).

[25] Marceau et al., *supra* n. 22, at 1.9.

[26] Dr. Seaborg, an American chemist, was given a leave of absence from the University of California from 1942 to 1946 in order to head the plutonium work of the Manhattan Project. He received the Nobel Prize in Chemistry in 1951 for his work on transuranium elements. *See Nobel Lectures, Chemistry 1942-1962* (Elsevier 1964).

[27] Marceau et al., *supra* n. 22, at 1.9.

[28] *Id.*

[29] *Id.*

[30] *Id.*

[31] *Id.* at 1.12.

[32] *Id.* at 1.11-1.12.

[33] *Id.*

[34] *Id.* at 1.18.

[35] *Id.* at 1.17.

A. Industrial Nuclear Reactors

Technology proceeded rapidly. HEW's first three nuclear reactors and chemical separation plants began operation in 1944 and early 1945, just two years after Fermi and Seaborg's laboratory work proved production of fissionable material was possible.[36] These reactor models and plants took shape just a few months after the theoretical data became available.[37] The sustained nuclear reactions and the chemical separation processes were based on laboratory models not yet supported by a solid engineering design.[38]

In the rush to create an atomic weapon, engineering technology that prevented toxic chemical releases was not considered or even in existence, and the facility failed to provide adequate safety measures to protect workers from radiation emissions.[39] It was during these early years of plutonium production that HEW's reactors released the greatest amounts of radiation into the air and water.[40] Most of the radiation releases occurred during the ventilation of the chemical separation plants, though other forms of radiation were released via wastewater and through by-products discharged into the Columbia River.[41] Emission levels decreased after 1951 when filtration systems and reactor design improved.[42] However, HEW still continued to play a part in the development of nuclear power and weapons, and the site continued to emit radiation until the reactors were shut down, about one per year, throughout the 1970s and 1980s.[43]

B. Public Concern: Hanford Environmental Dose Reconstruction Project

In 1987, due to public concern, the Department of Energy in conjunction with the Centers for Disease Control and Prevention (CDC), commissioned a study, the Hanford Environmental Dose Reconstruction Project (HEDR), to estimate the levels of human exposure to radionuclide[44] emissions from HEW between 1944 and 1972.[45] HEDR's

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[36] *Radiation Dose Estimates*, *supra* n. 2, at 3.
[37] Marceau et al., *supra* n. 22, at 1.16
[38] *Id.*
[39] *Id.* at 1.5. The majority of workers did not even realize plutonium was being produced at the site. *Id.*
[40] *Radiation Dose Estimates*, *supra* n. 2, at 3.
[41] *Id.*
[42] *Id.*
[43] Marceau et al., *supra* n. 22, at 1.72-1.77.
[44] A radionuclide is a radioactive nuclide, the nucleus of a particular isotope (atoms with the same atomic number and different numbers of neutrons). Almost all elements that are

reconstruction models estimated that peak emissions occurred between 1944 and 1946, when the Hanford facility released an estimated 700,000 curies[46] into the environment.[47] In all, Hanford released over 200 types of radionuclides.[48] The bulk of the emissions, an estimated 88%, was released in the form of radioiodine (Iodine-131),[49] which fortunately degrades harmlessly after just a few days.[50] However, other forms of radionuclides, which were released in smaller amounts, have much longer half-lives, including cerium-144[51] (half-life of 244 days), ruthenium-106[52] (half-life of 370 days), strontium-90[53] (half-life of 29 years), and plutonium 239[54] (half-life of 2,400 years). These emissions spread across a vast area of the Pacific Northwest that encompasses over 75,000 square miles, including most of Washington State, a narrow band of western Idaho, and northern Oregon.[55]

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heavier than bismuth, which has 83 protons, are unstable or radioactive. *See* EPA, *Radiation Information: Radionuclides*, http://www.epa.gov/radiation/radionuclides/ (last modified Nov. 30, 2004).

[45] *Hanford*, 292 F.3d at 1128.

[46] A curie is the unit of measurement of radioactivity. In modern nuclear physics, it is precisely defined as the amount of a substance in which 37 billion atoms per second undergo radioactive disintegration. In the International System of Units, the becquerel is the preferred unit of measure for radioactivity. One curie equals 3.7 x 10^{10} becquerels. *See Encarta Online Encyclopedia* (Microsoft Corp. 2005) (available at http://encarta.msn.com/encyclopedia_761563528/Curie_(measurement).html).

[47] *Radiation Dose Estimates*, *supra* n. 2, at 9.

[48] Wa. St. Dept. of Health, *The Release of Radioactive Materials from Hanford: 1944-1972*, http://www.doh.wa.gov/hanford/publications/history/release.html (last updated July 16, 2004) [hereinafter *Radioactive Materials*].

[49] *Id. See also* EPA, *Radiation Information: Iodine*, http://www.epa.gov/radiation/radionuclides/iodine.htm (last updated Nov. 30, 2004).

[50] *Id.*

[51] *Radioactive Materials*, *supra* n. 48, at http://www.doh.wa.gov/hanford/publications/history/release.html.

[52] *Id.*

[53] *Id. See also* EPA, *Radiation Information: Strontium*, http://www.epa.gov/radiation/radionuclides/strontium.htm (last modified Nov. 30, 2004).

[54] *Radioactive Materials*, *supra* n. 48, at http://www.doh.wa.gov/hanford/publications/history/release.html. *See also* EPA, *Radiation Information: Plutonium*, http://www.epa.gov/radiation/radionuclides/plutonium.htm (last modified Nov. 30, 2004).

[55] *Hanford*, 292 F.3d at 1128.

III. PLAINTIFFS FILE UNDER THE PRICE-ANDERSON ACT[56]

In 1990, HEDR's governing body, the Technical Steering Panel, released a report, *Initial Hanford Radiation Dose Estimates*.[57] This report identified the levels of emissions, doses, and length of exposure to radiation that affected the human population from 1943 to 1971.[58] Humans were exposed through air inhalation, water consumption, swimming in the Columbia River, and through eating contaminated fish, meat, and vegetables.[59] Most of the measurable exposure—of chief concern to researchers—resulted from drinking milk contaminated with Iodine-131.[60]

After the report was released, thousands of plaintiffs filed claims.[61] Plaintiffs alleged a number of theories of liability both in property damage and personal injury including negligence, strict liability, nuisance, trespass, misrepresentation, property devaluation, intentional infliction of emotional distress, negligent infliction of emotional distress, wrongful death, and conspiracy.[62] In 1991, the District Court for the Eastern District of Washington consolidated the claims into one class action.[63] The named defendants were E. I. DuPont de Nemours and Company, General Electric Company, and other entities (hereinafter defendants) that operated the facility between 1943 and 1987 under license agreements with the federal government.[64]

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[56] 42 U.S.C. § 2210 (2000); *The Price-Anderson Amendments Act of 1988*, Pub. L. No. 100-408, 102 Stat. 1066 (1988) (amending 42 U.S.C. § 2210). Congress enacted the Price-Anderson Act to encourage the private sector to invest in nuclear energy generation by providing federal funds to pay for liability above a certain amount, and by establishing a statutory cap on damages. The 1988 Amendments created a federal cause of action in the event of a "public liability action," which is defined as "any liability arising out of, or resulting from, a nuclear incident or precautionary evacuation." 42 U.S.C. § 2014(w) (2000). The Supreme Court upheld the constitutionality of the Price-Anderson Act against Due Process and Equal Protection Clause challenges in *Duke Power Co. v. Carolina Envtl. Study Group, Inc.*, 438 U.S. 59 (1978).

[57] Technical Steering Panel of the Hanford Envtl. Dose Reconstruction Project, *Initial Hanford Radiation Dose Estimates* (Wash. St. Dept. of Ecology 1990). *See also* Genevieve Roessler, *Radiation Dose Estimates from Hanford Radioactive Releases* (Wash. St. Dept. of Health 2004) (available at http://www.doh.wa.gov/hanford/publications/health/mon6.htm).

[58] *Id.*

[59] *Id. See also Hanford*, 292 F.3d at 1128.

[60] *Id.*

[61] *Hanford*, 292 F.3d at 1128.

[62] *Id.* at 1128-29.

[63] *Id.*

[64] *Id.* at 1127.

The district court commented on the magnitude of the litigation:

> 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 plaintiffs' 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.[65]

IV. TOXIC EXPOSURE: THE CAUSATION PROBLEM

Despite the fact that radioactive emissions are recognized as toxic and carcinogenic to humans[66] and that, as a matter of public record, the Hanford site released massive amounts of radiation into the Pacific Northwest,[67] the litigation may leave many, if not all, of the *Hanford* plaintiffs uncompensated.[68] Proving causation is the uphill battle facing *Hanford* plaintiffs.[69] Scientists do not fully understand the molecular and physiological mechanisms of how or why disease results from exposure to toxic substances.[70] Because direct evidence of causation is not yet available in most toxic tort cases, plaintiffs must use circumstantial evidence to infer causation.[71]

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[65] *Id.* at 1128.

[66] *TMI*, 193 F.3d at 643.

[67] *See generally Radiation Dose Estimates*, *supra* n. 2, at 3.

[68] Because the *Hanford* court remanded to the district court for resolution of generic causation issues before individual causation issues could be determined, the *Hanford* plaintiffs have still seen no compensation for their claimed injuries. *Hanford*, 292 F.3d at 1139. "Because discovery in this case had not yet commenced on issues of individual causation, the district court should not have ventured into individual determinations at this stage of discovery when there had not yet been full disclosure of individual plaintiff's circumstances." *Id.* at 1135.

[69] *See Sterling*, 855 F.2d at 1200 ("Although many common issues of fact and law will be capable of resolution on a group basis, individual particularized damages still must be proved on an individual basis.").

[70] Carl F. Cranor & David A. Eastmond, *Scientific Ignorance and Reliable Patterns of Evidence in Toxic Tort Causation: Is There a Need for Liability Reform?*, 64 L. & Contemp. Probs. 5, 32 (Autumn 2001). Cranor and Eastmond comment that science does not yet fully understand the toxicology of aspirin, much less other forms of disease causing substances, the way the courts require. *Id.*

[71] *Id.*

A. Signature Diseases and Temporal Association

Courts have looked favorably on two types of evidence to prove causation.[72] The first of these is a close association between the symptoms and the disease.[73] Certain toxic agents produce a "signature disease," such as mesothelioma, with symptoms clearly pointing to one agent.[74] The second indicator of causation is a close association between exposure and the onset of the disease, a close temporal association.[75]

B. Latency Periods

Neither a close temporal association with a disease nor an easily identifiable symptomology is common in toxic agent exposure, except in cases of extremely high doses.[76] The potential long latency periods between exposure to a toxic substance and the onset of symptoms (often measured in years and decades) makes proving causation difficult.[77] This was the situation with the *Hanford* plaintiffs: their alleged diseases, such as thyroid cancer, non-neoplastic thyroid diseases, and various non-thyroid cancers, may occur years or even decades after exposure and are common in unexposed populations.[78] Further, these diseases are not associated with a single substance such as asbestos.[79]

Long latency periods complicate proving causation in two ways. First, during the latency period a plaintiff can be exposed to the same toxic substance from other sources.[80] For example, the *Hanford* plaintiffs were exposed to naturally occurring radiation from outer space (cosmic radiation) and from radiation occurring naturally in the earth (terrestrial

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[72] *See* Betsy Grey, *Bendectin on Trial: A Study of Mass Tort Litigation*, 40 Jurimetrics 257 (2000).
[73] *Id.* at 260.
[74] *Id.* Mesothelioma, a malignant form of lung cancer, is an often-cited example because it is associated almost exclusively with asbestos exposure. *Id.* at 260 n. 8.
[75] *See e.g. Bonner v. ISP Technologies, Inc.*, 259 F.3d 924, 930 (8th Cir. 2001). The time between exposure and the onset of a disease can establish causation, especially if related to symptomology. *Id. See also Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 154 (3d Cir. 1999) ("A number of courts, including our own, have looked favorably on medical testimony that relies heavily on a temporal relationship between an illness and a causal event."); Jean Macchiaroli Eggen, *Clinical Medical Evidence of Causation in Toxic Tort Cases: Into the Crucible of* Daubert, 38 Hous. L. Rev. 369, 422 (2001).
[76] *See generally* Cranor & Eastmond, *supra* n. 70, at 31 (explaining the difficulty of linking toxic agents with their effects).
[77] *Id.* at 12.
[78] *Id. See also Hanford*, 292 F.3d at 1137-38.
[79] *See* Cranor & Eastmond, *supra* n. 70, at 12. *See also Hanford*, 292 F.3d at 1132.
[80] *See TMI*, 193 F.3d at 643.

radiation).[81] Some persons will suffer disease as the result of this natural exposure.[82] Additionally, there are also natural sources of man-made radiation that do not come from nuclear power plants and weapons manufacturing facilities.[83] Industrial (non-nuclear) power production, medical treatments involving irradiation, and nuclear weapons testing all produce radionuclides similar to the *Hanford* emissions.[84] Once exposure to multiple sources is involved, the plaintiffs' recovery may be barred because of the difficulty in pinpointing one source as the cause of the harm.[85] This is known as the "indeterminate defendant problem."[86] Therefore, it becomes nearly impossible to determine whether a specific toxic agent or another environmental contaminant was the cause of the disease, whether there was an intervening cause, such as exposure to a different toxic substance, or whether the disease occurred naturally.[87]

The second related difficulty is known as the "indeterminate plaintiff problem."[88] The cancers and other diseases *Hanford* plaintiffs contracted occur frequently in unexposed populations and are indistinguishable from those cancers induced by background radiation.[89] An additional complication the *Hanford* plaintiffs face is the mixed reactions humans have to radiation and chemical exposure.[90] Some persons who suffer radiation exposure in low doses will contract cancer or other radiation-

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[81] *Id.* at 644. The average annual dose of radiation from these natural sources is 2.4 millisieverts (a measure of the amount of radiation absorbed by the body). *Id.* at 644 n. 50.

[82] *Id.* at 643.

[83] *Id.* at 647.

[84] *Id.*

[85] *See generally TMI*, 193 F.3d 613.

[86] *See In re Agent Orange Prod. Liab. Litig.*, 597 F. Supp. 740, 819-820 (E.D.N.Y. 1984) ("This case illustrates the inapplicability of burden of proof rules designed for simple two-party cases to mass toxic torts where injury was allegedly caused, but the question of which manufacturer created the harm cannot be answered with precision. . . . Plaintiffs concede that because of the way the defendants' herbicides were mixed by the government in Vietnam before spraying no plaintiff would be able to establish the [cause in fact].").

[87] Eggen, *supra* n. 75, at 434.

[88] *See In re Agent Orange Prod. Liab. Litig.*, 611 F. Supp. 1396, 1408 (E.D.N.Y. 1985) ("Given the lack of scientific basis for general causation and the significant uncertainties involved in proof of individual causation—that is, the indeterminate plaintiff problem—it cannot now be established with any appropriate degree of probability that any individuals who suffer from the diseases listed . . . incurred them as a result of Agent Orange exposure, or that these diseases are more likely than others to be causally related.").

[89] *Hanford*, 292 F.3d at 1130.

[90] *Id.* at 1137.

induced diseases; however, others can be exposed to high levels of radiation with no effects whatsoever.[91] *Hanford's* plaintiffs would need to prove that the Hanford site emissions caused their cancer and disease during a latency period lasting for nearly 50 years, during which they were exposed to various types of natural and man-made radiation from a variety of sources.[92]

C. Advances: Molecular Epidemiology and Genetics

Scientists predict that molecular epidemiology and genetic studies someday will be able to accurately and directly determine causation in toxic exposure cases.[93] Today, however, plaintiffs must rely on expert testimony in the form of epidemiological studies, animal studies, statistical analysis, and chemical analyst comparisons to prove causation in toxic tort claims.[94] A difficulty is that courts often are reluctant to admit circumstantial evidence of causation. This problem was exacerbated in the landmark Supreme Court decisions of *Daubert,*[95] *General Electric Co. v. Joiner,*[96] and *Kumho Tire Co. v. Carmichael,*[97] which define the standards of admissible expert testimony in the federal courts.[98]

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[91] *Id.*

[92] *See TMI*, 193 F.3d at 643 (describing the development of cancer during the latency period); *Radiation Dose Estimates, supra* n. 2, at 3 (establishing the beginning point in time for exposure).

[93] *See generally* Susan R. Poulter, *Genetic Testing in Toxic Injury Litigation: The Path to Scientific Certainty or Blind Alley?*, 41 Jurimetrics 211 (2001) (suggesting that genetic testing could become an important part of causal proof in toxic injury litigation); Christiana P. Callahan, *Molecular Epidemiology: Future Proof of Toxic Tort Causation*, 8 Envtl. Law. 147, 148 (2001) (documenting that molecular epidemiology will be able to pinpoint genetic susceptibility to certain forms of cancer, as well as other diseases). "Molecular epidemiological studies establish that certain toxins cause specific molecular and genetic changes that eventually lead to cancer. These changes can be used as markers for exposure to the toxin. A finding of a marker in an exposed person will allow lawyers to demonstrate causation." *Id.*

[94] *See* Cranor & Eastmond, *supra* n. 70, at 39-40 (1993) (noting sources of circumstantial evidence on which plaintiffs can prove causation).

[95] 509 U.S. 579.

[96] 522 U.S. 136 (1997).

[97] 526 U.S. 137 (1999).

[98] Collectively, these three cases are commonly referred to as the "*Daubert* Trilogy." Some commentators have argued that *Daubert* is simply the vehicle for challenging experts, and that regardless of *Daubert*, the explosion of expert scientific and technical testimony has led litigants to become more adept at challenging expert testimony. Paul R. Rice, *Evidence: Common Law and Federal Rules of Evidence § 8.03[B][1]* 1158, 1178-79 (4th ed., Lexis 2000). One leading evidence commentator has called *Daubert* simply

V. ADMISSION OF EXPERT TESTIMONY IN FEDERAL COURTS: THE *DAUBERT* STANDARD AND RULE 702 OF THE FEDERAL RULES OF EVIDENCE

A. Daubert v. Merrell Dow Pharmaceuticals, Inc.[99]

Daubert is worth examining closely because it was the Supreme Court's first interpretation of Federal Rule of Evidence 702.[100] Also, the Ninth Circuit's treatment of *Daubert* on remand (*Daubert II*) has had a particular impact on toxic tort litigation because it was the first case to apply Rule 702 to epidemiological evidence and the relative risk factor of 2.0.[101]

The plaintiffs in *Daubert* alleged they had suffered prenatal limbic deformation due to their mother's ingestion of Bendectin, a prenatal anti-nausea drug marketed by the defendant, Merrell Dow Pharmaceuticals, Inc.[102] The plaintiffs had no direct evidence Bendectin caused their injuries.[103] To prove Bendectin was in fact a teratogen, a toxic agent that induces birth defects, the plaintiffs attempted to admit various studies, the cumulative effect of which would link Bendectin to birth defects.[104] These studies included *in vivo* tests (tests on living laboratory animals), *in vitro* tests (tests on animal tissues in test tubes), a meta-analysis (a re-analysis and synthesis of data from studies that had previously found Bendectin to be safe), and a chemical analysis comparing the similarities of Bendectin's chemical composition with other known teratogens.[105]

"*Frye* in drag"—look under the skirt, and it is still the old *Frye* standard. *Id. See also infra* nn. 106-115 (discussion of *Frye*).

[99] 509 U.S. 579.

[100] Fed. R. Evid. 702. "Rule 702 has been amended in response to *Daubert*." Fed. R. Evid. 702 advisory committee's notes.

[101] *Id.* at 1320-21.

[102] *Daubert*, 509 U.S. at 582.

[103] *Id.*

[104] *Id.* at 583.

[105] *Id.*

B. Frye *and the General Acceptance Test*

Prior to *Daubert*, federal and many state courts used the *Frye* test[106] to determine the admissibility of scientific testimony, by asking whether the evidence would meet with "general acceptance" in the scientific community.[107] Applying *Frye*, the district court ruled the plaintiffs' experts' testimony inadmissible.[108] The court based its ruling on the fact that there was a consensus among the established scientific community that Bendectin was not a teratogen.[109] The lower court commented that the Food and Drug Administration, which was notoriously cautious in approving new drugs, had approved Bendectin.[110] Between 1957 and 1982, 17.5 million women in the U.S. had used Bendectin.[111] Thirty studies involving over 130,000 patients had concluded Bendectin was safe for women during the first trimester of pregnancy.[112] Because it was not "generally accepted" in the medical community that Bendectin was dangerous, the court excluded the experts.[113] With no evidence of teratogenicity, plaintiffs could not establish that Bendectin had caused their birth defects, so the district court granted Merrell Dow's motion for summary judgment,[114] and the Ninth Circuit affirmed.[115]

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[106] The *Frye* test is named for the test established by the court in *Frye v. United States*, 293 F. 1013 (D.C. Mun. App. 1923), to determine the admissibility of scientific testimony. "While the courts will go a long way in admitting expert testimony, deduced from a well-recognized scientific principle or discovery, the thing from which the deduction is made must be sufficiently established to have gained general acceptance in the particular field in which it belongs." *Id. Frye* was relatively simple to apply, though critics pointed out that it tended to deny legitimate views held by a minority of scientists, and it was not clear exactly who constituted members of the scientific community who would "generally accept" the scientific basis for the expert's conclusions. *See* Holly Davis Thames, Frye *Gone, But Not Forgotten in the Wake of* Daubert*: New Standards And Procedures for the Admissibility of Scientific Expert Opinion*, 63 Miss. L.J. 473, 475 n. 9 (1994); Jeffry D. Cutler, *Implications of Strict Scrutiny of Scientific Evidence: Does* Daubert *Deal A Death Blow to Toxic Tort Plaintiffs?*, 10 J. Envtl. L. & Litig. 189, 191-94 (1995).

[107] *Daubert*, 509 U.S. at 585.

[108] *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 727 F. Supp. 570 (S.D. Cal. 1989).

[109] *Id.* at 574.

[110] *Daubert II*, 43 F.3d at 1314.

[111] *Id.* at 1311.

[112] *Daubert*, 509 U.S. at 582.

[113] *Id.*

[114] *Daubert*, 727 F. Supp. at 580.

[115] *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 951 F.2d 1128, 1129 (11th Cir. 1991).

C. The Court's Interpretation of Rule 702

On appeal, the Supreme Court held that Federal Rule of Evidence 702, and not *Frye*, governed the standards of admissibility of scientific evidence in federal courts.[116] Rule 702 states that, "[i]f 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 . . . ."[117]

In an elaborate reading of the language, the Court found that Rule 702 mandates a two-prong test for evaluating both the reliability and relevance of the expert's testimony.[118] The reliability prong requires the evidence be based on "scientific knowledge" acquired through the "scientific method."[119] Under the "fit," or relevance prong, the evidence must "assist the trier of fact to understand the evidence or determine a fact in issue."[120]

The Court commented that the inquiry into the relevance and reliability of the testimony should be flexible, and listed four non-inclusive "general observations" to assist federal judges in making the determination to admit expert testimony.[121] These observations include: (1) whether the theory or opinion can be tested using reliable scientific methods; (2) whether the theory or opinion has been subject to peer review and publication; (3) whether there is an acceptable rate of error in the study or opinion; and (4) whether the evidence has met with general acceptance in the scientific community.[122] With this last inquiry, the *Daubert* court seemingly includes the old *Frye* standard, but also comments that "general acceptance" is not a prerequisite to admissibility, allowing views held by a minority within the scientific community.[123]

D. What Does Daubert *Really Mean?*

The literal language of *Daubert* supports two conflicting positions. On one hand, the Court stressed that the Federal Rules of Evidence support a policy allowing "liberal admissibility" of all relevant evidence, calling

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[116] *Daubert*, 509 U.S. at 586-87.
[117] Fed. R. Evid. 702.
[118] *Daubert*, 509 U.S. at 589.
[119] *Id.* at 589-90.
[120] *Id.* at 591.
[121] *Id.* at 592.
[122] *Id.* at 592-94.
[123] *Id.* at 597.

Frye too austere for the liberal thrust of the rules.[124] Supporting this view, the Court stated that the procedural devices of directed verdicts[125] and summary judgment,[126] along with the adversarial system would weed out weak and insufficient scientific testimony.[127] "Shaky" but otherwise admissible evidence could be attacked using "[v]igorous cross examination, presentation of contrary evidence, and careful instruction on the burden of proof."[128] On the other hand, the Court stressed that federal judges must act as gatekeepers who are required to exclude shaky evidence from the courtroom.[129]

E. The Daubert *Remand*

The members of the Ninth Circuit expressed a profound trepidation regarding their new role as gatekeepers, anticipating the difficulties judges would have as laypersons in evaluating what constitutes "valid" science.[130] Applying the new test and the new factors, the *Daubert II* court again found, as it had under *Frye*, the testimony to be inadmissible because (1) the plaintiffs' experts had developed their research solely for litigation, (2) they had only the plaintiffs' word that the experts methodology was sound, and (3) despite an enormous interest in Bendectin research and litigation, and the prestige that a published study would bring, none of plaintiffs' experts had submitted their findings for peer review or publication.[131] Taken together, consideration of these factors suggested that the testimony

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[124] *Id.* at 588-89.
[125] Fed. R. Civ. P. 50(a).
[126] Fed. R. Civ. P. 56.
[127] *Daubert*, 509 U.S. at 595-96.
[128] *Id.* at 596.
[129] *Id.* at 596-97. *See also generally* Fisher, *supra* n. 13.
[130] *Daubert II*, 43 F.3d at 1316. Commenting that "scientific validity" was a subjective concept that they were ill equipped to determine, and that scientists often come to conclusions by using methods criticized by other scientists, the court went on to say:

> Our responsibility, then . . . is to resolve disputes among respected, well credentialed scientists about matters squarely within their expertise, in areas where there is no scientific consensus as to what is and what is not 'good science,' and occasionally to reject such expert testimony because it was not derived by the scientific method. Mindful of our position in the hierarchy of the federal judiciary, we take a deep breath and proceed with this heady task. . . . The task before us is more daunting still . . . scientists often have vigorous and sincere disagreements as to what research methodology is proper, what should be accepted as sufficient proof for the existence of a fact . . . .

Id.
[131] *Id.* at 1317-22.

lacked scientific reliability, because the Bendectin litigation had gone on for nearly a decade.[132]

The Ninth Circuit explained that they would have allowed the plaintiffs' experts the opportunity to verify their methodology, but the plaintiffs' epidemiological studies failed to meet the relevance prong of Rule 702.[133] The court reasoned that to meet the "more likely than not" standard that Bendectin had caused the plaintiffs' injuries, the plaintiffs had to demonstrate, using epidemiology's "relative risk" factor of 2.0, that using Bendectin statistically doubled their risk of birth defects.[134]

F. Epidemiology and a Relative Risk of Greater Than 2.0

Epidemiology studies the incidence of disease in populations through statistical analysis.[135] One of the statistical comparisons epidemiologists use is "relative risk," which measures the strength of the association between exposure to a toxic substance and disease.[136] A relative risk of 1.0 indicates no association between a substance and a disease.[137] If one segment of a population is exposed to a substance and another group is not, and both demonstrate the same statistical incidence of a disease, there is no cause and effect shown between the exposure and the disease.[138] In this case, the substance is deemed not harmful, and the relative risk would be a factor of 1.0.[139] On the other hand, if a particular population shows that a toxic agent statistically doubled the group's risk of contracting a disease, the relative risk would be a factor of 2.0.[140] This standard, first articulated in *Daubert*, is also known as a "doubling of the risk" or "doubling dose" standard.[141]

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[132] *Id.* at 1318.
[133] *Id.* at 1320-22.
[134] *Id.* at 1320-21.
[135] *Reference Manual I*, *supra* n. 18, at 125.
[136] *Id.* at 126.
[137] *Id.* at 148.
[138] *Id.*
[139] *Id.*
[140] *Id.*
[141] *Id.*; *see also generally* 509 U.S. 579.

G. Bad Math: Is a Relative Risk of 2.0 Equal to a Preponderance of Evidence Standard?

Notably, *Daubert II* was not the first use of a doubling-of-the-risk standard in toxic tort claims.[142] Federal and state courts previously had used and misused it, but it was stubbornly etched into the Supreme Court's jurisprudence and widely adopted in federal and state courts because of the *Daubert* decision.[143] Although the results in *Daubert II* are frequently criticized, to be fair, the Ninth Circuit's use of the *Daubert* factors in the balancing test was not unreasonable. The court considered the cumulative weight of the *Daubert* factors establishing the reliability of the plaintiffs' experts, including peer review, testability, general acceptance, and publication.[144] The court reasoned that stronger statistical evidence was necessary to overcome major weaknesses in the plaintiffs' case, such as the consensus in the scientific community that Bendectin was safe.[145]

Rather, at issue with the *Daubert* standard is the court's use of the relative risk factor of 2.0 and whether, as the court has claimed, it is equivalent to a preponderance of evidence standard.[146] Judicial logic in using the risk factor of 2.0 as a preponderance of the evidence standard can be explained using *Hanford* as an example: a risk factor of 2.0 would mean that there is a 50% chance that radiation emissions caused the plaintiffs' injuries and a 50% chance that their diseases could have been caused by a background source.[147] According to the courts, a relative risk of 2.0 would equal tort law's preponderance of the evidence standard.[148]

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[142] *See e.g. Maiorana v. Owens-Corning*, 964 F.2d 92 (2d Cir. 1992); *DeLuca v. Merrell Dow Pharmaceuticals, Inc.*, 911 F.2d 941 (3d Cir. 1990); *Landrigan v. Celotex Corp.*, 605 A.2d 1079 (N.J. 1992). *See also* Timothy W. Bouch & G. Hamlin O'Kelly, *Recent Developments in Toxic Torts and Environmental Law*, 36 Tort and Ins. L.J. 629 (2001) (discussing 1999-2000 cases where courts excluded expert testimony under *Daubert*); Russellyn S. Carruth & Bernard D. Goldstein, *Relative Risk Greater Than Two in Proof of Causation in Toxic Tort Litigation*, 41 Jurimetrics 195, 197 (2001) (listing federal courts that applied relative risk factor of 2.0 pre-and post-*Daubert*).

[143] *Id.*

[144] *Daubert II*, 43 F.3d at 1317-22.

[145] *Id.*

[146] *Id.* at 1322.

[147] *See Hanford*, 292 F.3d at 1127 (commenting on the district court's use of the standard). According to the *Reference Manual on Scientific Evidence*, courts still use this logic. Fed. Jud. Ctr., *Reference Manual on Scientific Evidence* (2d ed., Lexis 2000) [hereinafter *Reference Manual II*]. *See e.g. Pozefsky v. Baxter Healthcare Corp.*, 2001 WL 967608, *2-3 (N.D.N.Y. 2001) ("The threshold for concluding that an agent was more likely the cause of a disease than not is a relative risk greater than 2.0. Recall that a relative risk of 1.0 means that the agent has no effect on the incidence of disease. When

The central criticism of *Daubert* is that the Court's equating a relative risk factor of 2.0 to the tort preponderance of the evidence burden of persuasion is simply very bad math.[149] Epidemiologists do not agree that proving toxicity requires such an extraordinarily high statistical showing.[150] Rarely will any disease—even one induced by radiation—show a risk factor greater than 2.0.[151] For example, atomic bomb survivors at Hiroshima and Nagasaki show a relative risk of less than 2.0 for nearly all forms of cancer combined.[152] Also, as will be shown in the *Hanford* example, this risk factor does not take into account a particular plaintiff's susceptibility to contracting a disease; it is therefore a poor indicator of individual and specific causation.[153] One commentator jokes that the Court's math would be inadmissible as "junk science" under *Daubert's* own standard:

> Had an expert filed a report that included the court's math, it could never have survived a serious *Daubert* challenge. Although true for identical red and white balls that are used as specialized examples in basis statistics courses, the theorem is false when applied to toxic substances that may interact in a complex fashion in the body and may cause different impact on individuals depending on their susceptibilities.[154]

H. Further Limitations: Daubert's *Progeny:* Joiner *and* Kumho Tire

The doubling of the risk standard acts as a nearly insurmountable hurdle for toxic tort plaintiffs. In *General Electric Co. v. Joiner*,[155] the Court further restricted how plaintiffs could prove causation by limiting the kinds of evidence courts should accept.[156] This was done by discouraging the use of multiple studies, and, in a marked departure from

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the relative risk reaches 2.0, the agent is responsible for an equal number of cases of disease as all other background cases. Thus, a relative risk of 2.0 implies a 50% likelihood that an exposed individual's disease was caused by the agent.").

[148] *Hanford*, 292 F.3d at 1127.

[149] Jan Beyea & Daniel Berger, *Scientific Misconceptions Among* Daubert *Gatekeepers: The Need for Reform of Expert Review Procedures*, 64 L. & Contemp. Probs. 327, 352 (2001).

[150] *See Reference Manual I, supra* n. 18, at 125.

[151] *Id.*

[152] *Id.*

[153] *Id.*

[154] Beyea & Berger, *supra* n. 149, at 352. For further discussion on how misleading the doubling risk math was when the Ninth Circuit applied it in *Daubert*, see *id.* at 353-55.

[155] 522 U.S. 136.

[156] *Id.* at 139.

Daubert, by allowing judges to question not only the experts' methodology, but their conclusions as well.[157] However, *Kumho Tire*[158] later made explicit what the Court acknowledged in *Daubert*: *Daubert's* standard would encompass all technical and scientific expert testimony, as well as expert opinions based on experience.[159]

I. Joiner *and the Methodology/Conclusion Distinction*

Joiner was the first post-*Daubert* decision dealing with the admissibility of scientific evidence.[160] In *Joiner*, the plaintiff claimed that during his employment with General Electric Company, he had been exposed to polychlorinated biphenyls (PCBs) while repairing electrical transformers and, as a result, contracted small-cell lung cancer.[161] Like the *Daubert* plaintiffs, the *Joiner* plaintiff submitted a number of studies, hoping that the cumulative effect of the studies would prove a causal link between his cancer and his exposure to PCBs.[162] One study was conducted by injecting mice with high concentrations of PCBs.[163] A second statistical study demonstrated a higher rate of lung cancer at a PCB production plant.[164] The third and fourth studies attempted to extrapolate data from two existing studies of workers who had been exposed to potential carcinogens.[165]

The district court disagreed with the plaintiff's conclusion that, collectively, these studies proved his exposure to PCBs could have caused cancer.[166] According to the court, the studies did not rise above "subjective belief" or "unsupported speculation," so it granted the defendant's motion for summary judgment.[167] The Eleventh Circuit reversed, finding that because the Federal Rules of Evidence "display a preference for admissibility," the court should apply a more stringent

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[157] *Id.*
[158] 526 U.S. 137.
[159] *Id.* at 137 (holding that Fed. R. Evid. 702 applies to all expert testimony, technical or otherwise. The 2000 amendments to Rule 702 included *Joiner* and *Kumho*. Therefore, Rule 702 allows testimony to be admitted "if (1) the testimony is based on sufficient facts or data, (2) the testimony is the product of reliable principles and methods, (3) the witness has applied the principles and methods reliably to the case.").
[160] *Joiner*, 522 U.S. at 139.
[161] *Id.*
[162] *Id.*
[163] *Id.* at 142-46.
[164] *Id.*
[165] *Id.*
[166] *Joiner v. Gen. Elec. Co.*, 864 F. Supp. 1310, 1327 (N.D. Ga. 1994).
[167] *Id.* at 1326.

standard of review to the exclusion of the testimony because the exclusion was outcome determinative.[168] The Supreme Court reversed the Eleventh Circuit, holding that "abuse of discretion" is the proper standard of review for an evidentiary ruling, and that the district court had not abused this discretion in dismissing the plaintiff's claims.[169]

J. Effect of Joiner

Joiner put the Supreme Court's jurisprudential stamp of approval on what had already been an existing trend in the federal courts: reluctance in allowing experts to extrapolate from animal studies to humans[170] and discouraging the use of multiple studies to support an overall conclusion of causation.[171] Epidemiological studies were to be used as the primary basis of proof in toxic exposure cases.[172]

In another limitation, which represented a significant departure from *Daubert*, the Supreme Court in *Joiner* abandoned the methodology/conclusion distinction.[173] In *Daubert*, the Court gave deference to an expert's conclusions as long as the methodology was sound.[174] *Joiner*, however, made clear that this distinction was no longer in effect.[175] Courts now could challenge not only the validity of the expert's methodology, but the expert's conclusions as well.[176] As Chief Justice Rehnquist stated flatly:

> Conclusions and methodology are not entirely distinct from one another. Trained experts commonly extrapolate from existing data. But nothing in *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence which is connected to existing data only by the *ipse dixit* of the expert."[177]

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[168] *Joiner v. Gen. Elec. Co.*, 78 F.3d 524, 529 (11th Cir. 1996).
[169] *Joiner*, 522 U.S. at 141.
[170] *See* Erica Beecher-Monas, *A Ray of Light for Judges Blinded By Science: Triers of Science and Intellectual Due Process*, 33 Ga. L. Rev. 1047, 1063-64 (1999) (finding that courts have been reluctant to extrapolate data from animals to humans, despite general scientific acceptance).
[171] *Id.* at 1068-69.
[172] *See e.g. In re Breast Implant Litig.*, 11 F. Supp. 2d 1217, 1224 (D. Colo. 1998) ("epidemiological studies are necessary to determine the cause and effect between breast implants and allegedly associated diseases").
[173] *Joiner*, 522 U.S. at 146.
[174] *Daubert*, 509 U.S. at 595.
[175] *Joiner*, 522 U.S. at 146.
[176] *Id.* at 147.
[177] *Id.* at 146.

The Court had backtracked from *Daubert*, which clearly distinguished between an expert's methodology and his conclusions.[178] A court now could substitute its own judgment for an expert's.[179]

V. THE DISTRICT COURT DISMISSES *HANFORD* CLAIMS USING THE *DAUBERT* STANDARD

A. Bifurcated Discovery: Generic and Specific Causation

In 1991, several thousand *Hanford* plaintiffs were consolidated into a single action.[180] The district court divided the pretrial into three discovery phases.[181] During Phase I, the parties exchanged documents and interrogatories.[182] The second phase dealt with causation issues, and the third phase was used to answer questions of liability.[183] In 1995, the district court bifurcated Phase II into "generic" and "individual" causation inquiries.[184]

The generic causation inquiry determines whether a toxic substance has the capacity to cause harm.[185] Individual, or specific, causation answers the question of whether the toxic substance was the actual or proximate cause of each individual plaintiff's harm.[186] Prior to *Hanford*, courts used the device of dividing causation into generic and specific in an effort to streamline complex mass tort claims.[187] If a plaintiff fails to demonstrate the capacity of a substance to cause harm, then the action is dismissed without needlessly engaging in fact-intensive specific causation questions involving hundreds or thousands of individual claims.[188]

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[178] *Daubert*, 509 U.S. at 595.
[179] *Id.*
[180] *Hanford*, 1998 WL 775340, at *1.
[181] *Id.* at *2.
[182] *Id.*
[183] *Id.*
[184] *Id.*
[185] *Hanford*, 292 F.3d at 1129.
[186] *Id.*
[187] *See e.g. Sterling*, 855 F.2d at 1200; *In re Agent Orange Prod. Liab. Litig.*, 818 F.2d 145, 164-65 (2d Cir. 1987).
[188] *See Sterling*, 855 F.2d at 1200 (stating "However, from this point forward, it became the responsibility of each individual plaintiff to show that his or her specific injuries or damages were proximately caused by ingestion or otherwise using the contaminated water. We cannot emphasize this point strongly enough because generalized proofs will not suffice to prove individual damages. The main problem on review stems from a failure to differentiate between the general and the particular. This is an understandably

Generic causation measures a defendant's potential liability; later, specific causation measures the defendant's liability to each plaintiff.[189] That is, each individual plaintiff must prove the exposure was the cause in fact of his or her injuries.[190] In *Sterling v. Velsicol Chemical Corp.*,[191] for example, the district court required plaintiffs to show that the numerous ultrahazardous chemicals buried under the defendant's waste site were even capable of causing their injuries, rather than extending the litigation of thousands of plaintiffs, each with individual claims.[192] Only after plaintiffs had made a showing of generic causation could they attempt to prove specific causation, i.e., that the defendant had actually contaminated the drinking water, and the chemical contaminants were the proximate cause of each plaintiff's harm.[193]

B. Plaintiffs Appeal to the Ninth Circuit

Once the generic causation phase was completed, the *Hanford* defendants moved for summary judgment.[194] Defendants averred that plaintiffs, in order to survive summary judgment, would have to produce epidemiological evidence showing a relative risk factor of 2.0, as the Ninth Circuit had required in *Daubert II*.[195] The plaintiffs relied on their own understanding of generic causation, arguing they were required to prove only that the radioactive emissions had the capacity to cause harm, not that exposure to radiation had statistically doubled the risk of disease in each individual plaintiff.[196] They believed this determination could be made only during the specific causation phase.[197]

Applying *Daubert*, the lower court established a dose level for each disease and age group that would statistically double his or her risk of disease.[198] The court dismissed any plaintiff who could not link their

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easy trap to fall into in mass tort litigation. Although many common issues of fact and law will be capable of resolution on a group basis, individual particularized damages still must be proved on an individual basis").

[189] *Id.*

[190] *Id.*

[191] 855 F.2d 1188.

[192] *Id.*

[193] *Id.*

[194] *Hanford*, 1998 WL 775340 at *2.

[195] *Id.* at *5.

[196] *Id.* at *9.

[197] *Id.*

[198] *Id.* at *11.

disease to that statistical threshold.[199] Seventeen of the plaintiffs' experts, along with a majority of the plaintiffs, were dismissed in a summary judgment order delivered on August 21, 1998.[200]

C. Critical Distinction

The Ninth Circuit could have easily affirmed the lower court's ruling. In doing so, it would have followed a majority trend using *Daubert's* doubling of the risk standard to both admit causation evidence and to survive summary judgment.[201] Most federal and state courts have used a relative risk of 2.0 as a near absolute requirement to survive summary judgment.[202] However, perhaps because of the magnitude of the litigation, its historical importance, or because the court was troubled by the massive volume of literature critical of *Daubert*, the Ninth Circuit reversed the district court's decision.[203] In doing so, it took a longer look at an important question: What is a proper use of the doubling risk standard?

At first glance, it seemed as though the Ninth Circuit simply found it necessary to correct the district court's procedural misstep (and by so doing, correct the overt hostility the lower court displayed toward the plaintiffs during the proceedings).[204] The district court had not given the plaintiffs notice that they were required to show a relative risk factor of 2.0 during the generic causation phase.[205] Without notice of this requirement, it would have been unfair to grant the defendant's summary judgment motion, and moreover, not to allow the plaintiffs to supplement their discovery with additional data.[206]

The Ninth Circuit made the critical distinction between cases in which a relative risk factor of 2.0 had been used to determine whether a substance was harmful, as in *Daubert II*, and cases in which the substance

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[199] *Id.* at *10.
[200] *Id.* at *1.
[201] *See* Carruth & Goldstein, *supra* n. 142, at 197-200.
[202] *Id.*; *see also Pozefsky*, 2001 WL 967608.
[203] *See Hanford*, 292 F.3d at 1124.
[204] *Id.* at 1131 (commenting on this hostility a number of times: the district court "strictly enforced" deadlines, "intensely scrutinized requirements for extensions of time and leaves to amend, refused to allow updated scientific evidence" [including the latest research from Chernobyl victims]; when the plaintiffs attempted to file additional expert affidavits, Judge McDonald found these requests "intolerable" and threatened to impose sanctions) (citing the district court's order in *Hanford*, 1998 WL 775340).
[205] *Id.* at 1133.
[206] *Id.* at 1134.

was already recognized as a toxic agent and a carcinogen.[207] The defendants cited both *Daubert II* and *Schudel v. General Electric Co.*,[208] in which plaintiffs had not established that the substances to which they were exposed were even capable of causing harm.[209] *Daubert II* required more compelling epidemiological evidence to overcome the scientific consensus that Bendectin was safe.[210] Similarly, the *Schudel* court found there was not enough evidence to determine whether the organic solvents trichloroethylene and perchloroethylene had the capacity to cause plaintiff's neurological and respiratory problems.[211]

The Ninth Circuit found that radiation, by comparison, has long been recognized by the scientific and legal communities as both a toxic agent and a carcinogen, capable of causing disease even in the smallest doses.[212] This has also been codified by the state of Washington: "Radioactive wastes are highly dangerous, in that releases of radioactive materials and emissions to the environment are inimical to the health and welfare of the people of the state of Washington, and contribute to the occurrences of harmful diseases, including excessive cancer, and leukemia."[213]

Implicit in the Ninth Circuit's finding is that once a substance is shown to be harmful, (arguably including any relative risk of over 1.0) the generic causation inquiry is satisfied.[214] The court expressed the idea succinctly: "[C]ommon sense alone mitigates against establishing a bright line threshold for safe irradiation. We do not believe, for example, that a person who is exposed to 10 rem[215] of radiation is at risk for developing a neoplasm, but someone exposed to 9.99 rem is not."[216]

Courts must assess scientific evidence to determine whether a party has met their burden of proof, but the use of the doubling of the risk

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[207] *Id.* at 1136-37.
[208] 120 F.3d 991.
[209] *Hanford*, 292 F.3d at 1136.
[210] *Daubert II*, 43 F.3d at 1311-14.
[211] *Schudel*, 120 F.3d at 997.
[212] *Hanford*, 292 F.3d at 1137.
[213] Wash. Rev. Code § 70.99.010 (2002).
[214] *Hanford*, 292 F.3d at 1137.
[215] A "rem" (radiation equivalent man) is a unit for measuring amounts of radiation that is used in radiation protection and monitoring. It is the equivalent absorbed dose of radiation, which takes into account the varying ways in which ionizing radiations transfer their energy to human tissue. Rems and sieverts measure biological damage. *See Encarta Online Encyclopedia* (Microsoft Corp. 2005) (available at http://encarta.msn.com/dictionary_1861700333/rem.html).
[216] *Hanford*, 292 F.3d at 1137 (citing *TMI*, 193 F.3d at 727 n. 179).

standard is a policy decision that should not be mistaken for judicial objectivity. During a deposition, one of the *Hanford* plaintiffs' experts explained forcefully how the doubling the risk requirement was a judicial, not scientific, construct:

> Plaintiffs have requested that I comment on the defendant's suggestion that the doubling of risk is a *necessary piece of evidence to establish causation.* I am not an attorney and do not know what legal construct may exist regarding this assertion. I do believe it is important for this court to understand that this claim is *not grounded with regard to the basic principles of epidemiology.* As I have indicated . . . excess risk is but one of the guidelines epidemiologists use in assessing a causal relationship. *A doubling of the risk is merely one arbitrary point on the continuum of risk and as such is not required for a finding of generic causation.*[217]

D. Importance of Specific, Individual Factors—The Specific Causation Inquiry

In *Hanford*, the Ninth Circuit drew attention to an important flaw in the doubling risk requirement:[218] Epidemiology is simply a poor predictor of specific causation.[219] The limit of epidemiological evidence in individual causation determinations is readily apparent in studies of radiation exposure. While ionizing radiation is recognized as extremely toxic and a powerful carcinogen, the survivors of the atomic bomb raid on Hiroshima and Nagasaki, for example, show a relative risk of less than 2.0 for nearly all forms of cancer combined.[220] In fact, it would take a very powerful toxic agent or a rare signature disease to show a relative risk factor of 2.0 or more, and thus allow an inference of causation under *Daubert*.[221]

Individualized factors, such as health, lifestyle, and age, uncovered during specific causation inquiries, can play a vital role in increasing the

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[217] *Hanford,* 1998 WL 775340 at *19 (emphasis added) (citing deposition of Lawrence Mayer (1995)).

[218] Hanford, 292 F.3d at 1136.

[219] Beyea & Berger, *supra* n. 149, at 352.

[220] Carl F. Cranor, John G. Fischer & David A. Eastmond, *Judicial Boundary Drawing And The Need For Context-Sensitive Science In Toxic Torts After* Daubert v. Merrell Dow Pharmaceuticals, Inc., 16 Va. Envtl. L.J. 1, 39 (1996).

[221] *Id. See also Reference Manual I, supra* n. 18, at 126 ("[I]n the absence of an understanding of the biological and pathological mechanisms by which disease develops, epidemiological evidence is the most valid type of scientific evidence of toxic causation.").

risk of disease from toxic exposure.[222] Despite its adoption of the 2.0 standard, the Judicial Center's *Reference Manual on Scientific Evidence*[223] supports the view that any relative risk factor over 1.0 will support an inference of causation if the background factors indicate a greater susceptibility to contracting a disease.[224] The *Reference Manual* also comments, in their second edition, that a relative risk greater than 2.0 would permit an inference that an individual's disease more likely than not was caused by the implicated agent, but notes that many statisticians, epidemiologists, and toxicologists "resist" this adaptation.[225]

Because epidemiology cannot predict how toxic agents interact with background factors to cause disease, this question must be answered during a specific causation inquiry. When epidemiological evidence demonstrates a relative risk greater than 1.0 but less than 2.0, a relationship, which may prove to be causal, exists between exposure and disease.[226] In such cases, a plaintiff should have an opportunity to show their particular risk is higher than the relative risk.[227] The limits of epidemiological evidence, and the fact that few diseases will show a risk factor clearly greater than 2.0, should mandate that persons making legitimate claims (like those of the *Hanford* plaintiffs) be allowed to engage in a meaningful inquiry to determine specific causation. By recognizing that a lowered threshold, supported by science and logic, can be used to prove causation, the Ninth Circuit appears willing to allow this to happen.[228]

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[222] *See Agent Orange*, 818 F.2d at 165. "The relevant question, therefore, 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 (e.g. state of health, lifestyle) and the nature of their exposure to Agent Orange." *Id.*

[223] *Supra* n. 18.

[224] *See id.* at 169-70 ("The dose to which the plaintiff was exposed may be greater or lesser than those in the epidemiological study, thereby requiring some extrapolation. In addition, there may be factors peculiar to the plaintiff—excess exposure to another known cause, pathological mechanism, family history of a disease, or conflicting diagnosis, that modify any probability based solely on epidemiological evidence. This additional evidence bearing on causation has led a few courts to conclude that a plaintiff may satisfy his or her burden of production even if a risk of less than 2.0 emerges from the epidemiological evidence.").

[225] *See Reference Manual II*, *supra* n. 147, at 383.

[226] *Id.* at 349.

[227] *Id.*

[228] *See generally Hanford*, 292 F.3d 1124.

VII. EXPERTS AND RELIABILITY UNDER *DAUBERT*

A. Impact of Daubert *on Experts:* Hanford *Defendants Challenge Expert Testimony*

Although lowering the relative risk factor to less than 2.0 will help toxic tort plaintiffs with legitimate claims, these plaintiffs still face another significant hurdle in proving causation: *Daubert*'s reliability prong, which limits admissibility of expert testimony.[229] Federal judges have shown an increased reluctance to allow expert testimony.[230] Additionally, judges have placed further limits on the types of evidence experts are allowed to introduce.[231] An informal study conducted by searching under "*Daubert*" and "expert" on Westlaw found that in a 50 case sample, approximately 90% of the experts were excluded by the district court.[232] Evidence suggests motions in limine challenging an expert's reliability have become so commonplace as to be referred to simply as "*Daubert* motions."[233]

B. Nature of the Daubert *Test*

Daubert effectively limits expert testimony, at least in part because of the nature of the *Daubert* test itself. The Supreme Court gave little guidance on the weight to be applied to each of the four factors: testability, peer review, acceptable rate of error, and general acceptance.[234] Because of this lack of clarity, defendants can argue each factor is equally important. Some have argued the four "general observations" have

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[229] Molly Treadway Johnson, *Expert Testimony in Federal Civil Trials* 1, 4 (Fed. Jud. Ctr. 2000); *see also* Margaret A. Berger, *Setting the Balance Between Adversarial Interests: The Impact of the Supreme Court's Trilogy on Expert Testimony in Toxic Tort Litigation*, 64 L. & Contemp. Probs. 289, 290 (2001) ("The Federal Judicial Center conducted surveys in 1991 and 1998 asking federal judges and attorneys about expert testimony. In the 1991 survey, seventy-five percent of the judges reported admitting all proffered expert testimony. By 1998, only fifty-nine percent indicated that they admitted all proffered expert testimony without limitation. Furthermore, sixty-five percent of plaintiff and defendant counsel stated that judges are less likely to admit some types of expert testimony since *Daubert*. Without the means to prove causation, which is always a crucial element of the plaintiff's case, the plaintiff must lose, and the litigation ends with summary judgment for the defendant. The consequence, according to some observers, is that toxic tort law is being reformulated in the federal courts to the advantage of defendants.").

[230] *See Reference Manual I, supra* n. 18, at 169.

[231] *Id.*

[232] Beyea & Berger, *supra* n. 149, at 358-59.

[233] *Id.*

[234] *Daubert*, 509 U.S. at 593-94.

become a kind of "super *Frye*" test, in which each factor becomes an element that must be satisfied before an expert's opinion can be admitted.[235] As one *Daubert* critic commented, it is simply common sense that a four-part "test" including *Frye* is a more difficult standard to meet than the *Frye* standard alone.[236]

C. Judicial Misconception: Science as a Subjective Process

Another reason *Daubert* challenges have so effectively excluded experts is that judges have failed to appreciate science as a subjective process.[237] A common criticism of both federal and state judges is that judges fail to understand that scientific opinions will have elements of subjectivity, especially when scientists attempt to explain an event as large as the *Hanford* contamination.[238] Subjectivity in a complex scientific theory involves assumptions, judgments, inferences, and creative hypotheses; judges have tagged this process as "unreliable" under *Daubert*.[239] While an expert opinion can and almost certainly will contain these subjective elements,[240] the opinion can still be perfectly valid and subject to legitimate critiques of its methodology.[241]

Judges are often victims of the cultural myth that science is based on flawless logic; this leads to the requirement that expert testimony be free from any subjective element.[242] Scientific "truths" are not based on absolute proofs; rather they frequently involve reaching a consensus through testing, criticizing competing theories, and applying different methodologies.[243]

Ironically, the Supreme Court addressed this very concern in *Daubert*. "Indeed, scientists do not assert that they know what is immutably 'true'—they are committed to searching for new, temporary theories to explain, as

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[235] Beyea & Berger, *supra* n. 149, at 328.

[236] *See id.* ("It also can, in effect, raise the burden of proof in science-dominated cases from the acceptable 'more likely than not' standard to the nearly impossible burden of 'beyond a reasonable doubt.'").

[237] *See generally* Beecher-Monas, *supra* n. 170, at 1063; Cranor & Eastmond, *supra* n. 70, at 10-11; Beyea & Berger, *supra* n. 149, at 372; Judge Harvey Brown, *Eight Gates for Expert Witnesses*, 36 Hous. L. Rev. 743 (1999).

[238] Beyea & Berger, *supra* n. 149, at 371-72.

[239] *Id.*

[240] *Id.*

[241] *Id.*

[242] *Id.*

[243] *Id.*

best they can, phenomena."[244] An appropriate method for evaluating expert testimony would be a judicial understanding of the subjective element in the scientific process and an evaluation of testimony based on how scientists reach their conclusions.[245]

D. "Corpuscular Attacks" on Methodology

This pervasive judicial view of science has allowed defendants to become adept at challenging expert testimony by using what has been termed the "corpuscular approach."[246] Rather than attacking an expert's overall conclusions, defendants will focus on each "corpuscle" of an expert's methodology.[247] This attack focuses upon an expert's inferences each time he supplements data with an estimation, best guess, or simple intuition. Once the attack has taken root, it throws into question any conclusion flowing from the data, making a plaintiff's argument susceptible to a *Daubert* challenge.[248]

E. Dr. Mayer, the Hanford *Example*

The bulk of the district court opinion granting the defendant's summary judgment in *Hanford* consists of corpuscular attacks on virtually every aspect of each expert's methodology.[249] The defendants also attack expert reliability when the expert ventures even slightly away from an area of expertise, when an expert extrapolates from one study to another, or when an expert makes an inference based on simple intuition.[250] Ultimately, the district court determined the majority of the plaintiffs' experts did not meet Rule 702 reliability requirements.[251]

Defendants' treatment of Dr. Lawrence Mayer, an expert in medical statistics, illustrates how the nature of the *Daubert* test, judicial misconceptions about science, and the susceptibility of an expert's

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[244] *Daubert*, 509 U.S. at 590 (quoting an amicus brief).

[245] *See* Beyea & Berger, *supra* n. 149, at 328 ("A synthesis of the two views of science can be achieved by recognizing that subjective assumptions and inferences can never be completely eliminated from expert testimony.").

[246] Thomas O. McGarity, *Proposal for Linking Culpability and Causation to Ensure Corporate Accountability for Toxic Risks*, 26 Wm. & Mary Envtl. L. & Policy Rev. 1, 18-19 (2001).

[247] *Id.*

[248] *Id.*

[249] *See generally Hanford*, 1998 WL 775340.

[250] *Id.*

[251] *Id.* at *18.

conclusions can lead to exclusion of reliable evidence.[252] Mayer was asked to provide statistical evidence of the dose/response relationship for radiation on hypothyroidism.[253] He ultimately concluded the dose level of Iodine-131 that would double the risk of hypothyroidism in the general population was approximately 50 rads.[254] Due to the size of the sample, Mayer used a "bounding" of the data, a common bioengineering practice used to ascertain an error rate at which the general population could be subjected to hypothyroidism.[255] This was the kind of estimation, or guess, that courts have found impermissibly unreliable under *Daubert*: "Mayer's dose-response analysis does not distinguish between biochemical and clinical cases of hypothyroidism, nor between antibody positive and antibody negative cases of hypothyroidism. He asserts the distinction can be left for 'a later stage of the proceedings,' based upon specific information for individual plaintiffs."[256]

Mayer based his "subjective" opinion on hundreds of sources, including studies of the natives of the Marshall Islands,[257] the survivors of Nagasaki, and the effects of therapeutic doses of radiation on the thyroid.[258] Although his conclusions were based on these and many other sources, as well as extensive clinical experience, Mayer was ultimately dismissed from the litigation because he made the kinds of inferences found impermissible under *Daubert*.[259]

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[252] *Id.* at **18-22.

[253] *Id.*

[254] *Id.* at *20. A rad (radiation absorbed dose) is the unit of radiation absorption used to measure the level of ionizing radiation absorbed by something. *See Encarta Online Encyclopedia* (Microsoft Corp. 2005) (available at http://encarta.msn.com/dictionary_/rad.html).

[255] *Hanford,* 1998 WL 775340, at *20.

[256] *Id.*

[257] The Marshall Islands were exposed to radiation during a thermonuclear bomb test in 1954. In his studies, Mayer cites a report on the thyroid conditions among exposed Marshall Islanders. *Hanford,* 1998 WL 775340 at *27 (citing R. Larsen et al., *Thyroid Hypofunction Appearing as a Delayed Manifestation of Accidental Exposure to Radioactive Fallout in a Marshallese Population* vol. 1 (Intl. Atomic Energy Agency 1978) (proceedings of the Symposium of the Late Biological Effects of Ionizing Radiation)).

[258] *Hanford,* 1998 WL 775340 at *19.

[259] *Id.* at *37. Mayer made judgments regarding what kinds of hypothyroidism (clinical and autoimmune) to use to determine the dose level. The court found his testimony unreliable in part because the doses and types of radiation in the study of the Marshall Islanders were not similar to the *Hanford* plaintiffs. *Id.*

It is paradoxical that courts require epidemiological studies "as the best evidence" to prove causation, yet as a practical matter, these studies rarely meet *Daubert* standards.[260] Epidemiological studies on radiation are particularly vulnerable to *Daubert* attacks.[261] In practical and ethical terms, no study could cover dose/response relationships of every kind of radiation, for every age group and environmental pathway, and take into account long latency periods of many years.[262] Even though radiation exposure has been studied extensively, no single body of evidence exists regarding every kind of radiation at every dose.[263] Thorough epidemiological studies take years, ideally consider large populations, and are extremely expensive.[264] Controlled studies involving radiation exposure to humans are simply not feasible for obvious ethical reasons, and studies of a variety of radiation emissions, with various doses, on large population samples are not available.[265] When a court unreasonably insists that studies contain no flaws, legitimate expert testimony is left out of the federal courts.

One toxicologist has commented that to label a substance a carcinogen under *Daubert* would take "multiple epidemiological studies, multiple animal studies subject to strict experimental conditions, and multiple short term studies."[266] In the absence of this information, a toxicologist might be reluctant to call something carcinogenic to humans by *Daubert* standards.[267] Science, though, recognizes harmful substances before their toxicity is fully understood. For example, scientists warned of the dangers of benzene as early as the 1890s.[268] Early reports suggested exposure could cause leukemia in the 1920s; yet a scientific consensus about benzene did not develop until the 1970s, when the World Health Organization published a study on benzene's harmful properties.[269]

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[260] McGarity, *supra* n. 246, at 36-37.
[261] *Id.*
[262] *Id.*
[263] *Id.*
[264] *Id.* at 11.
[265] *Id.*
[266] Cranor & Eastmond, *supra* n. 70, at 20-21.
[267] *Id.*
[268] *Id.* at 16.
[269] *Id.*

F. Flexible Standards for Admissibility: Bonner v. ISP Technology[270]

The Ninth Circuit's reversal of the *Hanford* district court, and its reliance on the Eighth Circuit in *Bonner*, suggests that the Ninth Circuit will not be as restrictive in admitting expert evidence. The standard for admission of experts is quite generous, as articulated in *Bonner*: "Only if the expert's opinion is *so fundamentally unsupported* that it can offer no assistance to the jury must such testimony be excluded."[271] The *Bonner* court went on to comment:

> [E]ven if the judge believes there are better grounds for some alternative conclusion, and that there are some flaws in the scientist's methods, if there are good grounds for the expert's conclusion, it should be admitted. . . . [T]he district court could not exclude [scientific] testimony simply because the conclusion was "novel" if the methodology and the application of the methodology

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[270] 259 F.3d 924.

[271] *Id.* at 929-30 (emphasis added). *Bonner* is one of many Eighth Circuit decisions that has shown greater flexibility in admitting scientific evidence and has given toxic tort plaintiffs the chance to present their claims to a jury. *See e.g. Mattis v. Carlon Elec. Prods.*, 295 F.3d 856, 860-61 (8th Cir. 2002) ("To meet his burden of proving causation, Mattis presented the testimony of two expert witnesses, Dr. Hansen, his treating pulmonologist, and Roger Wabeke, an industrial hygienist. The district court found that this testimony, in addition to the testimony by appellants' expert, Dr. Kapp, was sufficient for a reasonable jury to find that Mattis's exposure to the organic solvents in Carlon cement was capable of causing RADS and that exposure to those solvents did, in fact, cause his illness. We agree. Dr. Kapp admitted that the organic solvents in Carlon cement were capable of causing RADS at high exposure levels. Wabeke's testimony established that Mattis was exposed to dangerous levels of those organic solvents, and Dr. Hansen's testimony provided evidence that Mattis's exposure to the organic solvents in the cement caused him to develop RADS.

Appellants argue, however, that Dr. Hansen's and Wabeke's testimony was insufficient as a matter of law to establish causation. They fault Wabeke's testimony because he could not determine Mattis's exact exposure level. To prove exposure levels, plaintiffs need not produce a 'mathematically precise table equating levels of exposure with levels of harm' [citations omitted]. Rather, a plaintiff need only make a threshold showing that he or she was exposed to toxic levels known to cause the type of injuries he or she suffered [citation omitted]. Wabeke testified that experts have known for a long time that the organic solvents in Carlon cement are respiratory irritants capable of injuring respiratory mucous membranes in the nostrils, throat, trachea, and lungs. In addition, Wabeke used a vapor concentration test to determine whether Mattis was exposed to a dangerous level of fumes. . . . This evidence is admissible and created a question of fact for the jury about whether Mattis was exposed to an unsafe level of fumes, capable of causing respiratory problems."). Other courts look favorably on *Bonner*. *See e.g. Martin v. Shell Oil Co.*, 180 F. Supp. 2d 313, 320 (D. Conn. 2002); *Archer Daniels Midland Co. v. Aon Risk Servs., Inc.*, 356 F.3d 850, 858 (8th Cir. 2004).

> were reliable. . . . Both our cases and the decisions of the Supreme Court make clear that it is the expert witnesses' methodology, rather than their conclusions, that is the primary concern of Rule 702."[272]

The *Bonner* court noted important limitations on *Daubert's* "general acceptance" and peer review factors as applied to toxic torts, commenting that the first victims of a toxic tort "should not be barred from having their day in court" for want of medical literature.[273] The court recognized that even the best-studied substances, like aspirin, will have gaps in the analysis of their effects.[274] Frequently, the first time a substance will be subjected to a study will be for purposes of litigation.[275]

G. Constitutional Questions: Usurping the Jury Function

Judges have broad discretion under both the Federal Rules[276] and common law[277] to admit or exclude evidence. However, *Daubert* insists courts use Rule 702 to first disallow plaintiff's experts, and then grant summary judgment to defendants because the plaintiffs failed to provide sufficient evidence to establish causation.[278] This practice is constitutionally questionable because it denies plaintiffs the Seventh Amendment's guarantee of a jury trial.[279] Judges usurp the jury's function

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[272] *Bonner*, 259 F.3d at 929 (quoting *Heller*, 167 F.3d at 152 and citing *Kumho Tire*, 526 U.S. at 152).

[273] *Id.* at 928 (quoting *Turner v. Iowa Fire Equip. Co.*, 229 F.3d 1202, 1208-09 (8th Cir. 2000)).

[274] *See* Cranor & Eastmond, *supra* n. 70, at 12 n. 5 ("Typical text notes that aspirin has numerous therapeutic effects. At high doses it can also cause a number of adverse or toxic effects. At higher doses, there is 'direct stimulation of the respiratory center.'").

[275] *Id.* at 12.

[276] Fed. R. Evid. 104.

[277] *See e.g. Jackson v. Denno*, 378 U.S. 368 (1964) (admissibility of confessions); *Watkins v. Sowders*, 449 U.S. 341 (1981) (allowing judicial determination outside of the jury's presence as to admissibility of evidence); Edmund M. Morgan, *Basic Problems of Evidence* 45-46 (Inst. CLE 1961); John W. Strong et al., *McCormick's Hornbook on Evidence* § 53 (5th ed., West 1999).

[278] This is precisely what happened in *Hanford*, 292 F.3d at 1138 (defendant's motions in limine were linked to motion for summary judgment).

[279] U.S. Const. amend VII, cl. 2 ("[T]he right of a trial by jury shall be preserved, and no fact tried by a jury shall otherwise be reexamined in any court of the United States, than according to the rules of common law").

when they effectively dismiss claims based on what typically are considered factual issues.[280]

Daubert originally focused on this point by stressing Rule 702's requirement that the evidence, to be sufficiently reliable and relevant, must assist the jury's determination of a disputed issue.[281] *Daubert* thus has become a substantive standard to survive summary judgment and not a standard of admissibility for evidence.[282]

VIII. RELIABLE EVIDENCE: THE COURT'S FAILURE TO CONSIDER ALL EVIDENCE

Tort law acts as a deterrent. Judicial failure to properly apply the doubling of the risk standard in toxic tort cases allows egregious violators to escape liability, and provides little incentive to correct risky behavior.[283] This problem is exacerbated when judges disallow relevant evidence. Had the Ninth Circuit affirmed the district court's ruling in *Hanford*, the message would have been clear to nuclear material processing facilities. Little danger of liability would exist, and no incentive to conduct studies showing the effects of radiation emissions would arise.

> A legal system that demands a well-conducted epidemiological study demonstrating a relative risk greater than two as a necessary condition to a plaintiff's toxic tort recovery is a legal system that is willing to tolerate or even encourage a high degree of uncertainty

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[280] *Cf. Wright v. Willamette*, 91 F.3d 1105 (8th Cir. 1996) (The plaintiffs alleged injury from the exposure to formaldehyde from defendant's wood processing plant. The plaintiffs proved they had been exposed to formaldehyde—there was formaldehyde in the wood fibers from defendant's processing plant in their house, their sputum, and their urine—and won at trial. Despite the evidence, on appeal the Eighth Circuit disallowed the testimony from one of plaintiff's key expert witnesses who had extrapolated the levels of formaldehyde the plaintiffs had been exposed to from studies of gaseous formaldehyde. The court found it "simply speculation" that the experts had extrapolated being injured from gaseous formaldehyde to the type of exposure the Wrights had suffered, which was from wood chips impregnated with formaldehyde, despite a vociferous dissent from Judge Heaney. The trial record showed that one of the defendant's experts had testified that the plaintiff's complaints were more probably than not related to exposure to formaldehyde. The court characterized the testimony as "simply speculation." The *Bonner* line of cases may have been a reaction to the results reached in *Wright*.).

[281] *Daubert*, 509 U.S. at 589-90.

[282] *See* Carruth & Goldstein, *supra* n. 142, at 197 (finding majority of federal cases between 1991 and 1998 required relative risk greater than 2.0 to establish causation or to admit evidence).

[283] *See generally* McGarity, *supra* 246.

about the dangers of some products, and to reward manufacturers for ignoring the risk.[284]

A. "Weight of the Evidence" Approach: Is it Valid?

Given the difficulty in establishing causation, some commentators have urged that tort laws should be altered in the area of toxic tort causation.[285] However, plaintiffs can prove causation without the need for alternative theories, under existing tort law, if judges learn to recognize and admit reliable, scientifically valid patterns of evidence.[286] This evidence would include the cumulative effect of multiple studies, extrapolation from animal studies, and chemical analysis—all of which can be reliable and valid ways to prove causation.[287]

Reliance on evidence other than epidemiology to show risk and causation is not new or radical. Federal and international agencies such as the Environmental Protection Agency (EPA), the Occupational Health and Safety Administration (OSHA), and the International Agency for Research on Cancer (IARC) assess the hazard of toxic substances by using a method similar to the "weight of the evidence" approach.[288] The EPA considers "all relevant, available evidence" in making a hazard assessment.[289] This assessment includes the kinds of evidence the federal courts have implicitly discouraged or rejected: animal studies (*Joiner*), meta-analysis (*Daubert II*), chemical structure comparisons (*Daubert II*), in vitro testing, and extrapolation of data from similar studies (*Hanford*).[290]

B. Animals Studies Applied to Humans

Animal studies are performed by exposing animals to toxic chemicals at various doses, times, and pathways, and then extrapolating the results to humans.[291] The EPA relies on animal studies to conduct risk assessments for hazardous substances.[292] Animal studies, by definition, take into

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[284] *Id.* at 38.

[285] Michael D. Green, *Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of Agent Orange and Bendectin Litigation*, 86 Nw. U. L. Rev. 643, 644 (1992).

[286] Cranor & Eastmond, *supra* n. 70, at 30.

[287] *Id.*

[288] *See* Beecher-Monas, *supra* n. 170, at 1049 (citing EPA's *Proposed Guidelines for Carcinogen Risk Assessment*, 61 Fed. Reg. 17960, 17960 (Apr. 23, 1996)).

[289] *Id.*

[290] *Id.*

[291] *Id.* at 1063.

[292] McGarity, *supra* n. 246, at 26-27.

account the different physiologies of humans and animals.[293] Proponents argue differences between human physiology and animal physiologies are not as important as the similarities.[294] Of approximately 40 known carcinogens, all cause cancer in animals.[295] Animal studies can be conducted in controlled environments, repeated and verified, and ultimately critiqued and refuted if invalid under *Daubert* more objectively than can other kinds of studies.[296]

Such studies of radiation exposure on animals would be useful to establish causation, yet unlike the EPA, courts have refused to allow experts to extrapolate these studies to similar studies on humans under *Daubert's* relevance prong.[297] The court in *In re Agent Orange Product Liability Litigation*[298] expressed a position commonly held in federal courts, which is simply that animal studies require too much "surmise" to be applied to humans.[299] As one judge stated, "Humans are not rats."[300]

C. Thresholds and Causation

Another argument against using the weight of the evidence approach in judicial settings is that the EPA will issue a lower threshold to label a substance hazardous, and that it therefore would be an unsuitable tort standard.[301] This position confuses the validity of the inquiry with the threshold required to establish risk or capacity to harm.[302] Regulatory agencies, of course, consider the long term, broad consequences of toxic exposure, as well as long-term litigation costs, so they tend to label substances hazardous at lower thresholds and exposures.[303] Yet, one could conclude that the approach that the EPA uses is more scientifically valid than is the rigid approach taken by the federal courts, as exemplified in the

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[293] Cranor & Eastmond, *supra* n. 70, at 30.
[294] *Id.* at 44.
[295] Beecher-Monas, *supra* n. 170, at 1065.
[296] *Id.* at 1064-65.
[297] McGarity, *supra* n. 246, at 26-27.
[298] *In re Agent Orange Prod. Liab. Litig.*, 611 F. Supp. 1223 (E.D.N.Y. 1985).
[299] *Id.* at 1231.
[300] *Intl. Union v. Pendergrass*, 878 F.2d 389, 394 (D.C. Cir. 1989).
[301] *See e.g. Allen v. Pa. Engr. Corp.*, 102 F.3d 194, 198 (5th Cir. 1996) (The court declined to use the weight of the evidence approach, finding that cellular studies and animal studies were suited to regulatory agencies, but not to tort norms.).
[302] *Id.*
[303] *See generally* Beecher-Monas, *supra* n. 170, at 1079 (discussing the legitimacy of setting a low exposure standard and the political and market consequences of unwarranted regulation).

district court's *Hanford* ruling.[304] The cumulative impact of a variety of relevant studies is critical to making realistic causation determination in toxic torts, which by definition must rely on a number of studies to portray an accurate picture of a toxic agent.[305] This fact was not lost on the Eleventh Circuit, before the Supreme Court in *Joiner* reversed the lower court's decision.[306] "Opinions of any kind are derived from individual pieces of evidence, each of which by itself might not be conclusive, but when viewed in their entirety are the building blocks of a perfectly reasonable conclusion."[307]

IX. CONCLUSION

The Ninth Circuit seems to intend the litigation to continue, at least through the individual causation phase.[308] The Court of Appeals urged the district court to rule on class certification and suggested the court reconsider the in limine motions in light of the "doubling of the risk" ruling.[309] Defendants likely will increase the intensity of *Daubert* challenges under Rule 702.

The Ninth Circuit's treatment of this major litigation should help guide other courts in applying *Daubert* reasonably.[310] Also, good reasons exist to allow the litigation to continue even if the plaintiffs fail to prove a present injury. Long-term exposure to radiation is a particularly nightmarish idea for everyone and it deserves special attention by the courts, both in terms of emotional distress claims[311] and possible awards of medical monitoring costs.[312] If the Supreme Court eventually grants certiorari in *Hanford*, it should take a more critical look at how it has addressed medical monitoring claims, which help defray expenses incurred from ongoing

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[304] *Hanford*, 292 F.3d at 1139.

[305] Beecher-Monas, *supra* n. 170, at 1068.

[306] *Joiner*, 78 F.3d at 540.

[307] *Id.* at 532.

[308] *Hanford*, 292 F.3d at 1139 (remanding to district court for resolution of generic causation issues before determining individual causation issues).

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

[310] *Id.* (The Ninth Circuit seemed to imply that though the district court ruled on the defendant's in limine motions without consulting the special master, the special master might assist in making a more objective scientific determination.).

[311] *See e.g.* Conrad G. Tuohey & Ferdinand V. Gonzalez, *Emotional Distress Issues Raised By the Release of Toxic and Other Hazardous Materials*, 41 Santa Clara L. Rev. 661 (2001).

[312] *See generally* Arvin Maskin, Konrad L. Cailteux & Joanne M. McLaren, *Medical Monitoring: A Viable Remedy For Deserving Plaintiffs Or Tort Law's Most Expensive Consolation Prize?*, 27 Wm. Mitchell L. Rev. 521 (2000).

medical examinations required after toxic exposure.[313] Many states require that the plaintiff either prevail to award such costs, or to show symptoms of a disease.[314] This approach does not make much sense in radiation exposure cases, because the diseases that radiation cause can have latency periods of years or even decades.[315] Radiation releases exceeding regulatory limits should be compensated for medical monitoring costs. At the very least, the Price-Anderson Act[316] should be amended to allow plaintiffs to recover such costs.

[313] The Court's treatment of medical monitoring is at odds in an increasing number of state courts. In *Metro-North Commuter RR Co. v. Buckley*, 521 U.S. 424 (1997), the Court denied an award of medical monitoring costs, holding that to grant such a claim required both physical impact, and an identifiable symptomology. *See also generally* Maskin, Caliteux & McLaren, *supra* n. 312.

[314] *Id.* at 535.

[315] *Id.* at 526.

[316] 42 U.S.C. § 2210.