

PLAINTIFFS WITH HOPELESS CAUSES: IDIOPATHIC CAUSES IN TOXIC TORT LITIGATION – ANALYZING HOW COURTS ADDRESS SCIENTIFIC UNCERTAINTY

Julia Peebles, JD, LLM

INTRODUCTION

After five years of working as a gasoline tanker truck driver, Plaintiff—a male, outdoor enthusiast, in his early-thirties—begins experiencing dramatic weight loss, constant fatigue, and severe chest pain. Concerned at his persistent symptoms, Plaintiff visits his primary care physician. Plaintiff's physician takes extensive medical and family histories, questions him about his smoking history and alcohol habits, and orders extensive lab work. Based on his findings, physician concludes that Plaintiff has Acute Myeloid Leukemia ("AML"), a form of leukemia. Physician indicates AML is linked to the chronic exposure of benzene—a component of gasoline, one of the most widely utilized chemicals in the United States, and a known carcinogen.¹

Based on his diagnosis, Plaintiff contacts you—a plaintiffs' attorney—seeking to sue his employer and the manufacturers' of the gasoline. In theory, the representation seems promising, given Plaintiff's young age, active lifestyle, and employment setting. However, the case presents one, very *specific* challenge—proving specific causation, i.e. that Plaintiff's workplace exposures actually

¹ *Benzene and Cancer Risk*, AMERICAN CANCER SOCIETY, <http://www.cancer.org/cancer/cancercauses/othercarcinogens/intheworkplace/benzene> (last visited Dec. 5 2016).

caused his injury. Namely, Plaintiff's relatively short term of employment as a truck driver poses complications because of the requirement that expert testimony support that Plaintiff's exposure was significant enough to result in the formation of the injury. Because between 70 and 80 percent of cases of AML are idiopathic – having no known cause² – and because Plaintiff did not work with benzene over an extensive duration of time, you decline representation.

In a society where over 85,000 commercial chemicals are utilized in the United States alone, the challenge in determining which chemical or exposure *caused* a plaintiffs' injury becomes akin to a search for "a needle in a haystack."³ Because of various factors, it becomes increasingly difficult to demonstrate that exposure to any *one* toxic chemical caused a specific injury. Notably, even if one has a disease with a high percentage due to idiopathic causes, yet is exposed to a certain substance known to cause an offending effect, it is still not enough in today's toxic tort system because courts uniformly conclude that "the presence of a known risk factor is not a sufficient basis for ruling out idiopathic origin in a particular case[.]"⁴ Thus, idiopathic causes place enormous burdens on plaintiffs and their attorneys, often resulting in the dismissal of a case at the causation stage. Law disfavors uncertainty, and uncertainty is the hallmark of injuries due to idiopathic causes. Court treatment and categorical dismissal for cases involving plaintiffs with injuries due to idiopathic causes, illuminates the heavy burdens placed on plaintiffs to successfully prove causation. This paper investigates how courts address scientific uncertainty and considers such uncertainty by examining court treatment of expert scientific causation testimony for idiopathic causes.

² *Milward v. Acuity Specialty Prod. Grp., Inc.*, 969 F. Supp. 2d 101, 109 (D. Mass. 2013), *aff'd sub nom*, *Milward v. Rust-Oleum Corp.*, 820 F.3d 469 (1st Cir. 2016).

³ Tracy Bach, *Better Living Through Chemicals (Regulation)? The Chemical Safety Improvement Act Of 2013 Through An Environmental Public Health Law Lens*, 15 VT. J. ENVTL. L. 490 (2014) (citing John M. Broder, *New Alliance Emerges to Tighten Chemical Rules*, N.Y. TIMES (May 24, 2013), http://www.nytimes.com/2013/05/25/us/politics/lautenbergs-chemical-safety-bill-gainmomentum.html?smid=pl-share&_r=0).

⁴ *Henricksen v. ConocoPhilips Co.*, 605 F. Supp. 2d 1142, 1162 (E.D. Wash. 2009).

LEGAL AND SCIENTIFIC PRINCIPLES IN TOXIC TORT CASES

Toxic tort cases have distinct characteristics and present unique issues that differentiate them from other forms of tort litigation, namely the challenges of proving causation using expert testimony. A toxic tort is “any injury attributable to exposure to a toxic substance where injury is not immediately manifest.”⁵ Toxic torts arise in a variety of exposure contexts, including occupational, environmental, and consumer products. Once the type of exposure context is determined, plaintiffs proceed to initiating a toxic tort action.

In a toxic tort action, plaintiffs must prove: (1) exposure to a toxic substance is connected to the defendant’s worksite or product, over which the defendant had control; (2) injury or illness occurred to the plaintiff; and (3) a causal connection between the exposure and the injury. Exposure, injury, and causation are often more complex in a toxic tort action compared to the theories asserted in a traditional tort action because providing evidence that the court deems insufficient at each step can derail a case, often at the summary judgment stage.⁶ The latency period, or the time between the exposure to the toxic substance and the manifestation, is the *sine qua non* of toxic torts.⁷ Thus, the central problem underlying most toxic tort cases is overcoming causation.⁸

The proffering party is required to meet the causation standard applicable to the jurisdiction and satisfy a two-part burden of proof on the question of causation. First, the proffering party must demonstrate general causation—that a toxic substance is generally capable of causing the injury in question.⁹ Second, the proffering party must

⁵ Alexander J. Bandza, *Epidemiological-Study Reanalysis and Daubert: A Modest Proposal To Level The Playing Field in Toxic Tort Litigation*, 39 *ECOLOGY L. Q.* 247 (2012) (citing Victor Schwartz & Thomas Means, *The Need For Federal Product Liability and Toxic Tort Legislation: A Current Assessment*, 28 *VILL. L. REV.* 1088, 1094 (1983)).

⁶ See 2 JAMES T. O’REILLY, *ISSUES IN ESTABLISHING PROOF OF EXPOSURE, INJURY, AND CAUSATION*, TOXIC TORTS PRAC. GUIDE § 15:3 (2016); see also Michael B. Kent Jr., *Daubert, Doctors and Differential Diagnosis: Treating Medical Causation Testimony as Evidence*, 66 *DEF. COUNS. J.* 525 (1999) (discussing a classic tort action, where plaintiffs must show duty, negligence, causation, and damage).

⁷ Michael B. Kent Jr., *Daubert, Doctors and Differential Diagnosis: Treating Medical Causation Testimony as Evidence*, 66 *DEF. COUNS. J.* 525 (1999),

⁸ *Id.*

⁹ *Knight v. Kirby Inland Marine Inc.*, 482 F.3d 347, 351 (5th Cir. 2007).

prove specific causation—that the toxic substance caused the specific injury at issue in the litigation.¹⁰ Idiopathic causes are usually scrutinized during the plaintiff’s offering of specific causation and present severe barriers at this stage because courts disfavor uncertainty.

Given the high burden of proving both general and specific causation in toxic tort cases, the use of expert testimony, including toxicologists and physicians with specialized scientific or technical knowledge, is required.¹¹ To demonstrate a causal relationship between a toxic substance and an offending agent, experts rely on different types of scientific evidence to support their conclusions. However, federal district and circuit courts vary on how they weigh or assess specific types of scientific evidence, and as the extent they review the reliability of evidence in determining whether an expert’s methodology is sound. Thus, before evidence is submitted to the factfinder, parties—upon motion¹²—present their evidence to the judge in the form of a *Daubert* hearing. *Daubert* hearings are screening mechanisms that grant judges immense discretion to review expert testimony ensuring that proffered testimony is both relevant and reliable. Once the evidence is permitted, judges determine what expert testimony and evidence, if any, is submitted to the factfinder. Therefore, understanding the *Daubert* process, specifically, how courts weigh expert testimony, in toxic tort actions is vital.

SCIENCE IN THE COURTROOM: EXPERT TESTIMONY AND *DAUBERT* HEARINGS

Daubert gave the U.S. Supreme Court an opportunity to examine the “junk science”¹³ issue in a critical legal context for determining the

¹⁰ *Id.*

¹¹ Kent, *supra* note 6.

¹² See generally 5 MICHAEL H. GRAHAM, RELIABILITY “GATEKEEPING” UNDER DAUBERT/KUHMO/RULE 702: HISTORICAL DEVELOPMENT AND ASSESSMENT, HANDBOOK OF FED. EVID. § 702:5 (7th ed. 2016).

¹³ See Howard B. Rocklman, *The Attorney’s Ethical Responsibility In Offering Expert Technical And Scientific Evidence In Patent Trials*, C876 ALL-ABA 143 , 145-46 (1993) (defining “junk science” as the following: “[b]roadly defined as testimony from an expert witness willing to testify to any theory for a price; and [a] technical witness is biased toward testimony favoring the party who hired him.”).

reliability of expert testimony.¹⁴ Notably, the Supreme Court found that district court judges must assess expert testimony for relevancy and reliability, acting as “gatekeepers.” The Court clarified that scientific evidence is reliable if it is based on an assertion that is established in the scientific method. Thus, *Daubert* defined a standard that courts use to screen expert testimony before it is submitted for review by the factfinder. The standards are in place to ensure that experts are qualified to testify on certain issues, safeguard against unreliable testimony, and prevent giant logical leaps in reasoning that could otherwise sway the jury. An understanding of the standards set forth in *Daubert* becomes particularly important in studying toxic torts because toxic tort claim rely heavily on expert testimony in establishing and refuting causation.¹⁵

THE EFFECT OF *DAUBERT* IN PROVING CAUSATION AND THE PLAINTIFF’S BURDEN OF PROOF IN TOXIC TORT LITIGATION

There is a distinction between the difficulty in demonstrating causation in toxic tort cases and the relative ease associated with establishing causation in most other tort actions.¹⁶ This is due to a number of reasons, the most notable being, the long latency period between the exposure and resulting disease, calculating the amount or dose of the toxic substance the plaintiff was exposed to, and the fact that most injuries have many other known and unknown causes, besides exposure.¹⁷ Thus, proof of causation in toxic tort cases is typically dependent on a hodgepodge of evidence and expert testimony – each aimed at addressing the ambiguity associated with exposure to toxic torts.

To prove factual causation, a toxic tort plaintiff must demonstrate factual causation in two ways. First, a plaintiff must show general

¹⁴ See Thomas O. McGarity, *Proposal for Linking Culpability and Causation to Ensure Corporate Accountability for Toxic Risks*, 26 WM. & MARY ENVTL. L. & POL’Y REV. 1 (2001).

¹⁵ Jean Eggen, *Toxic Torts and Causation: The Challenge of Daubert After the First Decade*, NAT RESOURCES & ENV’T, Spring 2003, at 213.

¹⁶ Jean Eggen, *Toxic Torts, Causation, and Scientific Evidence After Daubert*, 55 U. PITT. L. REV. 889, 895 (1994).

¹⁷ 3 DAVID L. FAIGMAN ET AL., MODERN SCIENTIFIC EVIDENCE: THE LAW AND SCIENCE OF EXPERT TESTIMONY, § 29:3 (2014-2015).

causation, or that the toxic substance is generally capable of causing the injury in question.¹⁸ If the plaintiff is successful in proving general causation, they then must prove specific causation, or that “a particular individual suffers from a particular ailment as a result of exposure to a substance.”¹⁹ Traditional tort theory mandates that in order for a successful plaintiff to prevail, they must prove both issues by a preponderance of the evidence.²⁰ This is a high burden that often disposes of cases with weak causal relationships.

To carry this heavy burden, plaintiffs must rely heavily on expert testimony. A toxic tort plaintiff will often need expert medical and scientific testimony to establish the link between the exposure and the injury. The most common studies relied upon by experts are epidemiological studies of exposed populations²¹ and toxicological studies,²² which include animal studies and other laboratory tests, such as structure activity and in vitro studies.²³ Both epidemiological and toxicological studies are principally utilized in proving or disproving general causation. However, neither epidemiology nor toxicology, alone, is satisfactory to prove specific causation in individual plaintiffs. Therefore, plaintiffs must rely on different types of evidence to satisfy specific causation.

The most crucial type of evidence offered by a plaintiff in support of specific causation is the use of “differential etiology” or “differential diagnosis.”²⁴ Differential diagnosis is a form of clinical methodology

18 David Bernstein, *Getting to Causation in Toxic Tort Cases*, 74 BROOK. L. REV. 51, 52 (2008).

19 *In re Bextra & Celebrex Mktg. Sales Practices & Prod. Liab. Litig.*, 524 F. Supp. 2d 1166, 1172 (N.D. Cal. 2007).

20 See FAIGMAN, *supra* note 17, at § 21:2; see, e.g., *Sterling v. Velsicol Chem. Corp.*, 855 F.2d 1188 (6th Cir. 1988); *Chaney v. Smithkline Beckman Corp.*, 764 F.2d 527 (8th Cir. 1985).

21 See Jeffrey Dintzer & Jonathan Mosher, *Epidemiologic Evidence in Toxic Tort Cases*, NAT. RESOURCES & ENV'T, Spring 2003, 222 (defining epidemiology as “the study of the distribution and determinants of disease in human populations.”).

22 See 3 LAWRENCE G. CETRULO, TOXIC TORTS LITIGATION GUIDE: TOXICOLOGY/ANIMAL TESTS, § 5:38 (2016) (defining toxicology as “the study of the adverse effects of chemical agents on biological systems.”).

23 See Douglas Danner & Larry L. Varn, *Toxic Experts*, 52 AM. JUR. TRIALS 473 (2016) (“The methodology for studies characterized as ‘animal studies’ basically consists of experiments in which doses of the substance being investigated are administered to test animals and the responses of the animals are observed, recorded and quantified at specific time intervals.”).

24 See Joseph Sanders & Julie Machal-Fulks, *The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law*, LAW &

“used in internal medicine whereby a treating physician formulates a hypothesis as to likely causes of a patient’s presented symptoms and eliminates unlikely cases by deductive process of elimination.”²⁵ The first step requires that a physician—either the treating physician or one retained as an expert—“rule in” possible causes by considering all relevant potential causes of a patient’s symptoms.²⁶ The second step requires that the physician “rule out” or eliminate all but one potential cause or set of integrated causes.²⁷ The second stage causes the most trouble in toxic tort cases because while physicians do not need to rule out every conceivable cause in order for a differential diagnosis to be admissible,²⁸ “if other possible causes of an injury cannot be ruled out, or at least the possibility of their contribution to causation minimized, then the ‘more likely than not’ threshold for proving causation may not be met.”²⁹ Thus, the use of a differential diagnosis is most compelling when the causes of a substantial proportion of the injury are known, as the presence or absence of the causes applicable to a specific plaintiff are easier to properly eliminate or address.³⁰ On the flipside, complications arise when the causes of a disease are largely unknown or uncertain, rendering differential diagnosis virtually useless.³¹ Thus, it is extremely important for experts to ensure that they have sufficient reliable and relevant evidence to prove that exposure to the toxic substance, rather than an idiopathic cause, caused the plaintiff’s injury.

CONTEMP. PROBS., Autumn 2001, at 107-08 (stating “[p]hysicians are frequently called upon to offer opinions identifying an injury’s cause based both on a physical examination of a patient and the exclusion of other causes of the patient’s condition. When this type of testimony is presented by physicians, it frequently goes by the name of ‘differential diagnosis,’ although some courts have more appropriately called it ‘differential etiology’”).

²⁵ See Harvey Brown & Melissa Davis, *Eight Gates for Expert Witnesses: Fifteen Years Later*, 52 HOUS. L. REV. 1 (2014) (citing *Transcon. Ins. Co. v. Crump*, 330 S.W.3d 211, 216 (Tex. 2010)).

²⁶ *Id.* at 251.

²⁷ *Id.* at 251-52.

²⁸ *Best v. Lowe’s Home Centers, Inc.*, 563 F.3d 171 (6th Cir. 2009).

²⁹ See Stout et al., *supra* note 44, at 877-78 (quoting *Moore v. Ashland Chem. Inc.*, 151 F.3d 269, 278 (5th Cir. 1998)).

³⁰ See Green, *supra* note 43, at 158.

³¹ *Id.*

TREATMENT OF IDIOPATHIC CAUSES BY COURT: THE CHALLENGES OF SPECIFIC CAUSATION

The triggering cause and origin of many cancers and other illnesses are not completely understood in the medical community.³² Specifically, the causes of diseases such as blood cancers, Parkinson's disease, and autism spectrum disorders, are principally unknown or idiopathic, notwithstanding the extensive amount of causation concentrated scientific studies.³³ For example, lung cancer may appear in the population affecting those without any toxic exposure, such as when nonsmokers develop lung cancer. Thus, even though the chemicals in cigarettes are known to cause lung cancer in smokers, exposure to those chemicals is not the triggering event in *all* cases of lung cancer.³⁴ The problems facing legally redressable injuries with idiopathic causes are illuminated when compared to the exposure of a specific substance with a resultant "signature disease."³⁵ The most well-known signature disease is mesothelioma, also known as asbestosis, where diagnosis in persons not exposed to asbestos is extremely rare.³⁶ Thus, in a case where the plaintiff is plagued with a disease that has a reputation for being idiopathic in origin, but the plaintiff has a history of exposure to a toxic substance, the difficulty

³² See Eggen *supra* note 43, at 214.

³³ Padgett, *supra* note 47, at 56.

³⁴ See Erica Beecher-Monas, *Lost in Translation: Statistical Inference in Court*, 46 ARIZ. ST. L. J. 1057, 1078-9 (2014); see also David L. Faigman, *A Preliminary Exploration of the Problem of Reasoning from General Scientific Data to Individualized Legal Decision-Making*, 75 BROOK. L. REV. 1115 (2010) ("If tobacco smoke causes lung cancer, but many other things, known and unknown, do so as well, we cannot say with certainty that the person's lung cancer was caused by tobacco smoke. The degree of certainty that the science provides, of course, is the operative question. Indeed, sometimes even very good science will not demonstrably improve the accuracy of individual decision-making, though it might nonetheless be relevant and admissible because it provides the triers of fact with contextual information that will help them understand other evidence in the case").

³⁵ See Steve C. Gold, *The "Reshapement" of the False Negative Asymmetry in Toxic Tort Causation*, 37 WM. MITCHELL L. REV. 1507, 1516 (2011).

³⁶ See Anita Bernstein, *Asbestos Achievements*, 37 SW. U. L. REV. 691 (2008) ("Mesothelioma almost never occurs absent asbestos exposure; it is, or is at least very close to, what epidemiologists call a signature disease"); see also Michelle J. White, *Why the Asbestos Genie Won't Stay in the Bankruptcy Bottle*, 70 U. CIN. L. REV. 1319 (2002) ("Mesothelioma is cancer of the pleural membrane around the lungs and organs. Asbestosis is non-cancerous scarring of the lungs, which reduces lung capacity").

arises in determining what caused the plaintiff's disease. The challenges that emerge from idiopathic causes are most prevalent in proving specific causation through differential diagnosis.

When the etiology of illness or disease is not well understood, the amount of weight credited to idiopathic causes increases. This situation places an enormous weight on an expert's differential diagnosis because it may be almost impossible to demonstrate by a preponderance of the evidence that the toxic substance in question caused the plaintiff's injury. Thus, as part of the differential diagnosis, and depending on the disease and the individual's factual circumstances, a trend has arisen among courts to require medical causation experts to consider and rule out the idiopathic cause as part of conducting their differential diagnosis. This begs the question, "how do [experts] rule out the unknown?"³⁷ Below, I provide an examination of three cases that attempted to confront this very question. After analyzing the cases, I briefly summarize the factors that courts use to determine whether an expert has sufficiently addressed idiopathic causes in their specific causation testimony.

SPECIFIC TREATMENT OF IDIOPATHIC CAUSES BY COURTS: AML AND BENZENE EXPOSURE³⁸

A growing number of mass tort lawsuits assert that occupational and environmental exposure to the chemical benzene causes and leads to diseases, ranging from leukemia to aplastic anemia.³⁹ However, the relationship between benzene, and Acute Myeloid Leukemia ("AML") and its subtypes, is perhaps the best example to demonstrate the challenges posed by idiopathic causes in toxic tort litigation. Due to the high percentage of AML incidences that are idiopathic in nature,

³⁷ See *Soldo v. Sandoz Pharm. Corp.*, 244 F. Supp. 2d 434 (W.D. Pa. 2003).

³⁸ In conducting research, I conducted several Westlaw searches to get a general sense on the case law available addressing idiopathic cases in benzene related cases. When using the search terms "benzene" and "idiopathic" 25 cases emerged. Further, when I searched for "AML," "benzene," and "idiopathic" only ten searches emerged. Additionally, when I used the terms "AML," "benzene," and "unknown cause" only eight searches arose. Finally, I searched "benzene," "AML," and "ideopathic," as I noticed some scholars spelled it this way, and one case emerged.

³⁹ See *CETRULO*, *supra* note 22, at 35:1 (citing *Indus. Union Dep't, AFL-CIO v. Am. Petroleum Inst.*, 448 U.S. 607 (1980)).

and the relative strength of association between benzene exposure and AML occurrence, demonstrating causation proves to be difficult, leading to inconsistencies among courts. Below, are examples of court treatment of the association between benzene exposure and AML.

OVERVIEW: THE RELATIONSHIP BETWEEN BENZENE, AML, AND ITS SUBTYPES

Benzene, also referred to as benzol, is a colorless liquid with a sweet odor.⁴⁰ Benzene evaporates into the air quickly, dissolves slightly in water, and is highly flammable.⁴¹ Benzene is found in air, water, and soil, and it produced by both industrial and natural sources.⁴² Benzene was first discovered in the 1800s, when it was isolated from coal tar.⁴³ Today, benzene is widely used and ranks within the top 20 chemicals produced in the United States. Benzene is a component of gasoline, is used to create other chemical compounds, and is used in the manufacturing of certain types of dyes, detergents, and pesticides.⁴⁴ Additionally, benzene is found in natural sources, often from gas emissions from volcanoes and forest fires.⁴⁵

Everyone is exposed to trace amounts of benzene on a daily basis. Common exposure to benzene includes ingestion of food or beverages containing benzene and inhalation of benzene vapor.⁴⁶ However, exposure to hazardous concentrations of benzene most often occur from occupational exposure, usually from workplaces that use or produce benzene-containing materials such as industrial solvents or

⁴⁰ *Toxicology Profile for Benzene*, U.S. DEP'T OF HEALTH & HUMAN SERVS. 1.1 (August 2007) <https://www.atsdr.cdc.gov/toxprofiles/tp3.pdf> (last visited Dec. 9, 2016) (“Most people can begin to smell benzene in air at approximately 60 parts of benzene per million parts of air (ppm) and recognize it as benzene at 100 ppm. Most people can begin to taste benzene in water at 0.5– 4.5 ppm. One part per million is approximately equal to one drop in 40 gallons”).

⁴¹ *Id.*

⁴² *Id.* at 1.2.

⁴³ *Id.*

⁴⁴ *See id.* (stating that benzene is used in the production of chemicals used to create other products such as, “styrene (for Styrofoam® and other plastics), cumene (for various resins), and cyclohexane (for nylon and synthetic fibers)”).

⁴⁵ *Id.*

⁴⁶ *See* CETRULO, *supra* note 22, at 35:3 (discussing the common ways people are exposed to benzene).

gasoline.⁴⁷ Industries commonly using benzene include: benzene production, rubber tire manufacturing, and storage or transport of benzene, and petroleum products containing benzene.⁴⁸ The inhalation of benzene, or benzene containing products, is the most dangerous type of exposure.⁴⁹

The carcinogenicity of benzene is well documented in exposed workers. Chronic or long-term exposure to benzene can cause leukemia, a form of cancer in the blood-forming organs.⁵⁰ There are different types of leukemia that are often classified under two main categories:

The first classification is between leukemia's acute and chronic forms: acute leukemia is characterized by a rapid increase in the number of immature blood cells, while chronic leukemia is characterized by the excessive buildup of relatively mature but abnormal white blood cells. The second classification is between the types of stem cells affected: leukemia can be either 'myeloid' or 'lymphoid'.⁵¹

Epidemiological studies and case reports provide evidence of a causal relationship between occupational exposure to benzene, benzene containing substances, and the occurrence of AML, the acute form of leukemia.⁵² The only form of leukemia linked to benzene exposure is AML.⁵³ AML is also one of the most common types of leukemia, affecting people of all ages, though the risk increases with age.⁵⁴ While benzene is certainly a risk factor for AML, there are various other causes of AML, both known and unknown.⁵⁵ However, the majority of AML cases have no known cause, and are therefore

⁴⁷ *Id.* at 35:9.

⁴⁸ See *Toxicology Profile for Benzene*, *supra* note 40, at 1.3.

⁴⁹ See *id.* at 2.2.

⁵⁰ *Id.*

⁵¹ *Milward v. Acuity Specialty Prod. Grp., Inc.*, 639 F.3d 11, 16 (1st Cir. 2011).

⁵² See *Toxicology Profile for Benzene*, *supra* note 40, at 2.2.

⁵³ See LAWRENCE G. CETRULO, *supra* note 22, at 35:7 (citing H. Kantarjian H & S. O'Brien *The Chronic Leukemias*, in GOLDMAN'S CECIL MEDICINE (L. Goldman & D. Ausiello eds., 24th ed. 2011).

⁵⁴ See *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1149 (E.D. Wash. 2009).

⁵⁵ *Id.*

idiopathic in origin.⁵⁶ It is also important to note that there are different subtypes of AML, including Acute Promyelocytic Leukemia (“APL”).⁵⁷ Additionally, AML is further classified based on the cause of the AML. “Cases can either be termed idiopathic or *de novo* (primary, endogenous), meaning onset without external or environmental stimulus, or *secondary* (event-related, exogenous) events which could be related to exposure to chemotherapy or radiation interventions in addressing preexisting hematologic disorders, or exposure to environmental toxins, including benzene.”⁵⁸ Notably, the different categories of AML cause different biological repercussions, mainly that chromosomal aberrations occur more frequently in secondary AML, while idiopathic AML chromosomal aberrations occur less frequently.

CASE ANALYSIS: COURT TREATMENT OF IDIOPATHIC CAUSES IN BENZENE LITIGATION

Milward v. Rust-Oleum Corporation, decided in 2009, is a more recent case highlighting court treatment of idiopathic causes of AML for benzene exposure. The case’s procedural history is extensive.⁵⁹ For purposes of this analysis, however, the most relevant portion is the First Circuit’s affirmation of the district court’s exclusion of the plaintiff’s specific causation testimony.⁶⁰

In *Milward*, the plaintiff brought a toxic tort action against a paint manufacturer after he developed APL, a rare subtype of AML, asserting that workplace exposure to benzene containing paint

⁵⁶ *Id.*

⁵⁷ See Joseph Sanders, *Milward v. Acuity Specialty Products Group: Constructing and Deconstructing Science and Law in Judicial Opinions*, 3 WAKE FOREST J.L. & POL’Y 141 (2013).

⁵⁸ *Henricksen*, 605 F. Supp. 2d at 1149.

⁵⁹ See *Milward v. Acuity Specialty Prod. Grp., Inc.*, 664 F. Supp. 2d 137 (D. Mass. 2009) (finding that testimony by toxicology expert was inadmissible, and dismissed the action); see also *Milward v. Acuity Specialty Prod. Grp., Inc.*, 639 F.3d 11 (1st Cir. 2011) (holding that the testimony regarding causation between exposure to benzene in the workplace and development of APL was admissible); *U.S. Steel Corp. v. Milward*, 132 S. Ct. 1002 (2012) (denying petitioner-defendant’s writ of certiorari); *Milward v. Acuity Specialty Prod. Grp., Inc.*, 969 F. Supp. 2d 101 (D. Mass. 2013) (expert’s specific causation opinion that worker’s APL was caused by exposure to benzene in defendant’s product was inadmissible).

⁶⁰ *Milward v. Rust-Oleum Corp.*, 820 F.3d 469 (1st Cir. 2016).

products caused his disease.⁶¹ For over 30 years, the plaintiff worked as a pipefitter and refrigerator technician and regularly worked with products with various levels of benzene, including products manufactured by the defendant.⁶² To prove specific causation, the plaintiff retained expert Dr. Shelia Butler, an occupational medicine physician.⁶³ Dr. Butler presented three theories: (1) she advocated for the “not safe threshold” argument, alleging that no level of exposure to benzene is safe,⁶⁴ (2) she found that, beyond the no safe threshold argument, epidemiological studies establish that individual’s risk for developing APL increases when exposed to specified amount of benzene and that the plaintiff was allegedly exposed to higher than the amount to be hazardous,⁶⁵ and (3) she conducted a differential diagnosis that “ruled out” common factors associated with APL, which led her to conclude that because benzene exposure was present, she could “rule out” idiopathic causes.⁶⁶ Specifically, Dr. Butler argued that the presence of the risk factor, i.e. exposure to benzene, negated the possibility that APL was caused by an idiopathic origin.⁶⁷ Because of these theories, Dr. Butler concluded that it was more probable than not that the plaintiff’s APL was caused by his exposure to benzene, as opposed to an idiopathic cause.⁶⁸

The First Circuit disagreed, affirming the district court’s exclusion–discounting both Dr. Butler’s relative risk theory and differential diagnosis. First, the court rejected Dr. Butler’s relative risk theory, criticizing her method of choosing studies to support her theory, because she did not describe to the court why she chose certain

⁶¹ *Id.* at 471.

⁶² *Id.*

⁶³ *Id.*

⁶⁴ Dr. Butler reached this conclusion by examining “the biology, the pathophysiology, what the substance does to the person and the disease process.” *Id.*

⁶⁵ *Id.* at 472 (“Dr. Butler rather cursorily concluded that even beyond the no-safe level hypothesis, certain epidemiological studies have established that an individual’s ‘relative risk’ of developing APL increases when exposed to specified amounts of benzene. She then compared Milward’s exposure levels to those that had been found to be dangerous in that research. Since Milward’s exposure was higher than the amounts found to be hazardous, Dr. Butler reasoned that benzene exposure was likely the cause of his APL.”).

⁶⁶ *Id.*

⁶⁷ *Id.* at 475 (“[Dr. Butler] ‘ruled out’ an idiopathic APL by ‘ruling in’ benzene as a cause).

⁶⁸ *Id.* at 474.

studies over others.⁶⁹ Notably, because there are studies that do and do not support a correlation between APL and benzene exposure, Dr. Butler did not adequately address *how* and *why* contrary studies were irrelevant in comparison to the plaintiff's APL. The court stated that the reliability of a relative risk theory requires that an expert consider the reliability and applicability of contradictory studies.⁷⁰ Next, the court addressed the sufficiency of Dr. Butler's differential diagnosis. Focusing on the high percentage of AML due to idiopathic causes—between 70 to 80 percent of cases—the court disapproved Dr. Butler's "circular" idiopathic argument.⁷¹ The court agreed that Dr. Butler "ruled out" certain risk factors, such as obesity and smoking; however, the court also determined that the mere presence of benzene exposure as a risk factor, does not abolish the possibility that the plaintiff's APL was due to idiopathic causes.⁷² Thus, the court questioned the reliability of Dr. Butler's testimony because she "ruled out" or eliminated a number of potential causes without properly "ruling in" a cause, citing the Restatement (Third) of Torts: Physical and Emotional Harm.⁷³ As Dr. Butler did not provide a reliable method to "rule out" idiopathic causes, her differential diagnosis was excluded.⁷⁴

Milward confirms courts' tendency to exclude expert testimony when there is the existence of an injury—a high percentage of which are due to idiopathic causes—regardless of the presence of an acknowledge risk factor. The take away from both cases is that in order to be successful in demonstrating specific causation, expert testimony must

⁶⁹ *Id.* at 472.

⁷⁰ *Id.* at 475.

⁷¹ *Id.* at 475-76 ("Since Dr. Butler was only able to 'rule out' an idiopathic APL because she had 'ruled in' benzene as a cause, the validity of her differential diagnosis turns on the reliability of that latter conclusion").

⁷² *Id.* at 476.

⁷³ *See id.* (citing a passage from the Restatement (Third) of Torts: Physical and Emotional Harm § 28, comment c(4)(2010), "The underlying premise [of differential diagnosis] is that each of these known causes is independently responsible for some proportion of the disease in a given population. Eliminating one or more of these as a possible cause for a specific plaintiff's disease increases the probability that the agent in question was responsible for that plaintiff's disease.... This technique is ... most useful when the causes of a substantial proportion of the disease are known. Then, the presence (or absence) of these causes for the specific plaintiff affects the probability that the agent in question caused the plaintiff's illness. When the causes of a disease are largely unknown, however, differential etiology is of little assistance.").

⁷⁴ *Milward v. Rust-Oleum Corp.*, 820 F.3d 469, 476 (1st Cir. 2016).

be meticulously completed, requiring strong epidemiological studies and rebuttals to contrary studies. Furthermore, experts must directly address idiopathic causes, and provide compelling evidence discounting that such causes were the cause in the plaintiff's case. In order to do so, experts should look into the patterns of the disease present in the plaintiff and compare them to recognized, non-idiopathic examples of the disease. For example, to establish that the plaintiff's AML was caused by exposure to benzene, experts should be prepared to testify about the level of exposure or dosage, and should present evidence of the plaintiff's chromosomal aberrations, and how those patterns are consistent with AML caused by secondary events. However, without the presence of a known high exposure rate and/or the existence of chromosomal aberrations, most courts will exclude the testimony as unreliable.

POTENTIAL SOLUTIONS TO RECENT TRENDS IN THE TREATMENT OF IDIOPATHIC CAUSES

Courts repeatedly emphasize that—regardless of the presence of a known risk factor—when most cases of a disease are due to idiopathic causes, experts must go beyond the differential diagnosis to prove specific causation.⁷⁵ While courts attempt to suggest means of combating this issue, such as comparing the presentation of a plaintiff's injury, i.e. symptoms and growth patterns, to non-idiopathic examples of the injury seen in others, courts seem to immediately denounce the application of such a method to facts of the specific case.⁷⁶ This is the exact issue at hand: there is no consensus on *what* a

⁷⁵ See *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1162 (E.D. Wash. 2009). (“Standing alone, the presence of a known risk factor is not a sufficient basis for ruling out idiopathic origin in a particular case, particularly where most cases of the disease have no known cause. This is not to say that where most diagnoses of a disease are idiopathic it is impossible to prove specific causation. But in those cases, analysis beyond a differential diagnosis is required”).

⁷⁶ See *Perry v. Novartis Pharm.*, 564 F.Supp. 2d 452, 470 (“This is not to say that where most diagnoses of a disease are idiopathic it is impossible to prove specific causation. But in those cases, analysis beyond a differential diagnosis will likely be required. Here, for example, because lymphoma caused by immunosuppressant drugs is well-understood, Drs. Smith and Kolb could have compared the presentation of Andreas Perry's symptoms with those common in post-transplant lymphoma cases. *Doing so, however, would not have served plaintiffs' purposes.*”) (emphasis added).

specific causation expert needs to present above a differential diagnosis to effectively “rule out” idiopathic causes. Commentators propose numerous options for confronting causation problems in toxic torts, many of which could be applicable to those cases that involved the presence of known risk factors and injuries with a high percent of idiopathic causes. Discussed briefly *infra*, there are three main proposals that commentators suggest may address the causation controversy: (1) allowing recovery based on “loss of chance”; (2) adopting an administrative and legislative “weight of the evidence” approach; and (3) applying a proportional recovery method.

LOSS OF CHANCE DOCTRINE

The loss of chance doctrine allows a finding of liability when causation and actual harm may not be provable, by permitting evidence of statistical probabilities of causation and harm to be used instead.⁷⁷ The loss of chance doctrine is a relatively new theory of recovery, utilized principally in medical malpractice cases, that permits a plaintiff to recover for a physician’s negligence that results in the loss of some probability of recovery or of a better outcome.⁷⁸ A loss of chance claim allows the plaintiff to present evidence that the defendant’s malpractice, to a reasonable degree of medical certainty, proximately caused the increased risk of harm or lost chance of recovery.⁷⁹ Therefore, loss of the opportunity to survive, by itself, is a compensable injury although the opportunity must be “lost,” i.e. the bad result must occur, in order for a claim to accrue.⁸⁰ Proponents of the loss of chance doctrine argue that application of this doctrine to toxic torts would further the goal of deterrence, by requiring that defendants pay for the damage they caused.⁸¹ As plaintiffs face

⁷⁷ Boaz Shnoor, *Loss of Chance: A Behavioral Analysis of the Difference Between Medical Negligence and Toxic Torts*, 33 AM. J. TRIAL ADVOC. 71, 71-72 (2009).

⁷⁸ Brian Casaceli, *Losing a Chance to Survive: An Examination of the Loss of Chance Doctrine Within the Context of a Wrongful Death Action*, 9 J. HEALTH & BIOMEDICAL L. 521 (2014).

⁷⁹ See Richard E. Kaye, *Damages for Loss of Chance Cure*, 154 AM. JUR. PROOF OF FACTS 3d 273 (2016) (discussing *Mays v. U.S.*, 608 F. Supp. 1476, 1483 (D. Colo. 1985)).

⁸⁰ *Id.* (citing *Stone v. Williamson*, 482 Mich. 144, 753 N.W.2d 106 (2008)).

⁸¹ See Shnoor, *supra* note 78, at 79 (discussing Byron G. Stier, *Jackpot Justice: Verdict Variability and the Mass Tort Class Action*, 80 TEMP. L. REV. 1013, 1061 (2007)).

difficulty in proving, that defendants actually caused their physical harm, by a preponderance of the evidence, they would only have to prove that defendants actually caused them to be in a statistically less advantageous situation.⁸² The loss of chance doctrine would serve as a different form of recovery and could address certain situations where risk factors and potential idiopathic causes are both present.

UTILIZING A WEIGHT OF THE EVIDENCE APPROACH

Realizing that scientific uncertainty burdens both courts and regulatory agencies, commentators suggest that, in toxic tort cases, courts should adopt the weight of the evidence approach utilized by regulatory agencies.⁸³ Under the weight of the evidence approach, experts render a cause and effect determination based on the totality of the evidence available within the scientific community.⁸⁴ “The expert considers all available studies and determines the weight to be afforded to each on the basis of the strengths and weaknesses of the individual studies.”⁸⁵ Thus, if a specific study is compelling on one aspect, but is weak or flawed in some other respect, such as its general applicability, then it may be given a lower “weight” than a study that validates a certain position in its entirety. Using this method would allow experts to testify using all available evidence, including epidemiological evidence, to prove specific causation.⁸⁶ Therefore, in addressing idiopathic causes, experts could use all available evidence in order to effectively “rule out” idiopathic causes for the plaintiff’s disease, which would help resolve several of the concerns courts have expressed, namely the use of other types of evidence, besides a differential diagnosis, to prove specific causation testimony.

⁸² See *id.* (discussing Aaron Twersky & Anthony J. Sebok, *Liability Without Cause? Further Ruminations on Cause-in-Fact as Applied to Handgun Liability*, 32 CONN. L. REV. 1379 (2000)).

⁸³ Thomas O. McGarity & Sidney A. Shapiro, *Regulatory Science in Rulemaking and Tort: Unifying The Weight of The Evidence Approach*, 3 WAKE FOREST J. L. & POL’Y 65 (2003).

⁸⁴ *Id.* at 78.

⁸⁵ *Id.*

⁸⁶ See *Estate of George v. Vermont League of Cities & Towns*, 993 A.2d 367 (Vt. 2010).

USING A METHOD OF PROPORTIONAL RECOVERY

Certain scholars advocate for a proportional theory of recovery, or a “probabilistic causal contribution model.”⁸⁷ This approach considers any exposure as a cause of an injury, if it contributed to an injury.⁸⁸ Thus, this determination requires plaintiffs to prove, by a preponderance of the evidence that the exposure added to the incremental risk of the plaintiff’s injury.⁸⁹ Therefore, damages would vary according to the proportion of risk created by the exposure.⁹⁰ Proportional recovery would ease the burden of proof for specific causation in proving injuries stemming from idiopathic causes because it would allow plaintiffs to recover based on the likelihood that exposure caused plaintiff’s injuries.⁹¹ Thus, under this theory, defendants are required to compensate injured parties exposed to demonstrably toxic substances in proportion to the established likelihood that the substance caused the plaintiffs’ injuries.⁹² However, the proportional method of recovery, however, would be accompanied by several repercussions on the toxic tort system. It would permit plaintiffs, who have not been injured, recovery, leaving companies and manufacturers vulnerable to immense liability.⁹³ Further, this theory presents challenges in calculating the proportion of liability.

⁸⁷ Kerriann Laubach, *Epigenetics and Toxic Torts: How Epidemiological Evidence Informs Causation*, 73 WASH. & LEE L. REV. 1019 (2016) (discussing Steve C. Gold, *When Certainty Dissolves into Probability: A Legal Vision of Toxic Causation for the Post-Genomic Era*, 70 WASH. & LEE L. REV. 237, 244 (2013)).

⁸⁸ Steve C. Gold, *When Certainty Dissolves into Probability: A Legal Vision of Toxic Causation for the Post-Genomic Era*, 70 WASH. & LEE L. REV. 237, 281 (2013).

⁸⁹ Laubach, *supra* note 88, at 1049-50.

⁹⁰ *Id.*

⁹¹ See generally David Rosenberg, *The Causal Connection in Mass Exposure Cases: A ‘Public Law’ Vision of the Tort System*, 97 HARV. L. REV. 849 (1984).

⁹² *Id.* at 39-40.

⁹³ *Id.*

CONCLUSION

“Absolute certainty in science is rarely an option; uncertainty is the norm, not the exception.”⁹⁴ Courts disfavor uncertainty. Uncertainty is a hallmark of science. As seen in the case analysis above, accounting for idiopathic causes in differential diagnosis is extremely challenging. In order to decrease the chances of expert exclusion, plaintiffs should ensure that their experts account for idiopathic causes in their differential diagnosis. However, regardless of the thoroughness of a differential diagnosis, courts may still exclude plaintiff’s specific causation testimony if the plaintiff has a disease that has a history of a high percentage due to idiopathic causes. If, however, a relatively low percentage of cases of the disease are idiopathic, and the expert has properly excluded a substantial number of known alternative causes, then a court is unlikely to rule that the expert’s specific causation, and differential diagnosis, are unreliable. It is sufficient to say that in many cases, plaintiffs “with hopeless causes” will have little to no expectation of a legal remedy.

⁹⁴ David Michaels, *DOUBT IS THEIR PRODUCT: HOW INDUSTRY’S ASSAULT ON SCIENCE THREATENS YOUR HEALTH*, 165 (2008).