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Who's to Blame: How Genetic Information Will Lead to More Accurate Decisions in Toxic Tort Litigation

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**WHO'S TO BLAME?:
HOW GENETIC INFORMATION WILL LEAD TO MORE ACCURATE DECISIONS
IN TOXIC TORT LITIGATION**

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I. INTRODUCTION

What happens when an individual is diagnosed with a disease for which there is no specific discernable cause? While scientists and physicians are busy conducting research and searching for diagnoses to answer this question, American courtrooms are struggling to determine the answer so that they can properly assign liability in toxic tort litigation.¹ In the absence of the exacting science that courts crave,² decisions in toxic tort cases are often based on mere inferences.³ Professor Gary Marchant describes toxic tort litigation as a “black hole of ignorance and uncertainty that judges and juries must venture to resolve whether a particular exposure caused an individual plaintiff’s illness.”⁴ Genetic research has the ability to supply courts with conclusive evidence of plaintiffs’

1. See Albert C. Lin, *Deciphering the Chemical Soup: Using Public Nuisance to Compel Chemical Testing*, 85 NOTRE DAME L. REV. 955, 961 (2010) (“The application of *Daubert* in toxic tort cases has led many courts to reject proposed expert testimony, particularly that proffered by plaintiffs, as too unreliable in light of scientific uncertainty and incomplete scientific knowledge.”).

2. See Christiana P. Callahan, Note, *Molecular Epidemiology: Future Proof of Toxic Tort Causation*, 8 ENVTL. LAW. 147, 161 (2001) (citing *Chambers v. Exxon Corp.*, 81 F. Supp. 2d 661, 664 (M.D. La. 2000)) (stating that epidemiological evidence does “not directly demonstrate [the] causal link,” but instead “show[s] that occurrence of the disease is more common among those exposed to the toxin than those who are not”).

3. See Gary E. Marchant, *Genetic Data in Toxic Tort Litigation*, 14 J.L. & POL’Y 7, 7–8 (2006) (stating that “[m]ost disease conditions have multiple potential etiologies, and there is usually no direct evidence of which possible cause produced the disease in a specific individual,” and that “the outcome in such toxic tort cases is often uncertain, contentious, and unjust”); see also *Smith v. Wyeth–Ayerst Labs. Co.*, 278 F. Supp. 2d 684, 696 (W.D.N.C. 2003) (finding that general causation could reasonably be inferred from expert testimony offered by plaintiff (citing Michael D. Green et al., *Reference Guide on Epidemiology*, in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 333, 375 (Fed. Judicial Ctr. 2d ed. 2000))).

4. Marchant, *supra* note 3, at 7.

injuries, thereby improving courts' ability to accurately measure and assign liability in toxic tort litigation.⁵

Parties involved in toxic tort litigation face unique challenges in establishing, and defending against, the claims involved.⁶ Unlike traditional products liability cases where a plaintiff alleges that a defect in a durable good, such as an automobile, caused her injury,⁷ toxic tort cases deal with alleged defects in products like prescription drugs⁸ and injuries from diseases like cancer.⁹ Thus, even in the most complex products liability case, a court is much more likely to have access to tangible evidence to establish causation than it is when dealing with a toxic tort case in which it must determine the cause of a complex medical disease for which there may be numerous potential causes.¹⁰ For these reasons, toxic tort litigation is uniquely dependent on scientific advances that can provide courts with an understanding of the relationship between the human body and exposure to outside substances, like prescription drugs.¹¹ Given the fact that people are increasingly exposed to thousands of chemicals in the environment,¹² toxic tort litigation is naturally growing¹³ as

5. See *id.* at 8.

6. See Lin, *supra* note 1, at 965 ("Plaintiffs in toxic tort cases, however, face numerous obstacles to bringing successful claims, particularly as to issues of causation.").

7. See, e.g., *Collazo-Santiago v. Toyota Motor Corp.*, 149 F.3d 23, 24 (1st Cir. 1998) (plaintiff brought product liability action against automobile manufacturer alleging that the air bag was defectively designed).

8. See, e.g., *Smith*, 278 F. Supp. 2d at 688, 691 (plaintiff brought claim against prescription drug manufacturer alleging that drug caused her to develop primary pulmonary hypertension).

9. See, e.g., *In re Hanford Nuclear Reservation Litig.*, No. CY-91-3015-AAM, 1998 WL 775340, at *2 (E.D. Wash. Aug. 21, 1998) (plaintiffs brought claim seeking damages for injuries including cancer), *rev'd*, 292 F.3d 1124 (9th Cir. 2002).

10. See Marchant, *supra* note 3, at 7; see also John C. Childs, *Toxicogenomics: New Chapter in Causation and Exposure in Toxic Tort Litigation*, 69 DEF. COUNS. J. 441, 441 (2002) ("Most toxic tort litigation involves diseases, such as cancer, for which there are numerous possible causes and where there is little objective, scientific proof that the claimant's disease was caused by the alleged exposure.").

11. See Steve C. Gold, *The More We Know, the Less Intelligent We Are?—How Genomic Information Should, and Should Not, Change Toxic Tort Causation Doctrine*, 34 HARV. ENVTL. L. REV. 369, 397 (2010) ("[T]he legal significance of advancing scientific knowledge will continue to be tested in personal injury actions brought by people who allege that their exposure to drugs, workplace chemicals, pollutants, or other agents caused their disease.").

12. See Elizabeth B. Forsyth, Note, *Solving Widespread Toxic Chemical Exposure: A Taxing Job*, 29 VA. ENVTL. L.J. 115, 118 (2011) (citing Gertrud S. Berkowitz et al., *The Rationale for a National Prospective Cohort Study of Environmental Exposure and Childhood Development*, 85 ENVTL. RES. 59, 60 (2001)) (stating that "[t]here are more than 80,000 synthetic chemicals on the market in the United States, the majority of which have been developed since World War II"); see also Alan Heaton, *Introduction*, in *THE CHEMICAL INDUSTRY* 1, 2 (Alan Heaton ed., 2d ed. 1994) (noting that the chemical industry "makes thousands of different chemicals which the general public only usually encounter as end or consumer products").

13. Martha A. Churchill, *Toxic Torts: Proof of Medical Monitoring Damages for Exposure to Toxic Substances*, in 25 AM. JUR. 3D *Proof of Facts* 313, 322–24 (1994) ("In less than a decade, environmental toxic tort law has developed from an unknown and obscure branch of litigation to a full blown legal specialty. Toxic tort litigation is increasing rapidly . . .").

individuals seek to hold potential wrongdoers responsible for causing their injuries.

This Note will show how genetic evidence can improve the accuracy of courts' decisions in toxic tort cases by providing specific information about plaintiffs' exposures to certain substances and any effects of such exposures. Part II of this Note provides background information on the study of human genetics. Part III then analyzes the challenges that exist in toxic tort litigation today and discusses the ways it will be impacted by the introduction of genetic evidence. Finally, Part IV briefly discusses the ongoing debate about the use of genetic evidence in toxic tort litigation and concludes with recommendations for its use. Ultimately, as genetic research provides new information on the causes of human diseases,¹⁴ toxic tort law stands to benefit from courts' improved ability to use this information to more accurately assign responsibility for injuries.

II. BACKGROUND ON HUMAN GENETICS

In 2003, scientists announced the completion of the Human Genome Project (HGP)¹⁵ and presented the world with the first complete map of the human genome.¹⁶ Designed "to improve our understanding of health and genetic disease"¹⁷ by determining the causes of widespread diseases like cancer,¹⁸ the HGP is considered to be one of the greatest scientific achievements in history.¹⁹ So far, the HGP has led to the discovery of over 1,800 disease genes²⁰ and has revealed connections between certain genetic mutations and over 6,000

14. See Callahan, *supra* note 2, at 148 ("[E]pidemiological studies could help establish the causal link between exposure and injury . . .").

15. See Jessica L. Roberts, *Preempting Discrimination: Lessons from the Genetic Information Nondiscrimination Act*, 63 VAND. L. REV. 439, 442 (2010) (citing *Major Events in the U.S. Human Genome Project and Related Projects*, OAK RIDGE NAT'L LAB., http://www.ornl.gov/sci/techresources/Human_Genome/project/timeline.shtml (last modified Sept. 19, 2011)).

16. See Michael J. Malinowski et al., Acknowledgement, *Symposium: Proceedings of "The Genomics Revolution? Science, Law and Policy"*, 66 LA. L. REV. (SPECIAL ISSUE) 1, 2 (2005).

17. Diane E. Hoffmann & Karen H. Rothenberg, *Judging Genes: Implications of the Second Generation of Genetic Tests in the Courtroom*, 66 MD. L. REV. 858, 860 (2007) (citing *An Overview of the Human Genome Project*, NAT'L HUMAN GENOME RESEARCH INST., <http://www.genome.gov/12011238> (last updated Oct. 13, 2011)).

18. See Nicholas Wade, *Disease Cause is Pinpointed With Genome*, N.Y. TIMES, Mar. 10, 2010, <http://www.nytimes.com/2010/03/11/health/research/11gene.html>.

19. See Lauren Elizabeth Nuffort, *The Genetic Information Nondiscrimination Act of 2008: Raising a Shield to Genetic Discrimination in Employment and Health Insurance*, 21 HEALTH LAW, no. 5, June 2009 at 1, 1 (quoting *International Consortium Completes Human Genome Project*, U.S. DEP'T OF HEALTH & HUMAN SERVS. (Apr. 14, 2003, 1:00 PM), <http://www.nih.gov/news/pr/apr2003/nhgri-14.htm>) (describing the Human Genome Project as a "thirteen-year milestone in genomics").

20. Cynthia Marietta & Amy L. McGuire, *Direct-to-Consumer Genetic Testing: Is It the Practice of Medicine?*, 37 J.L. MED. & ETHICS 369, 369 (2009).

biological disorders, including multiple cancers.²¹ Additionally, the National Institute of Environmental Health Sciences launched a similar project in 1998, the Environmental Genome Project (EGP), “to understand the relationship between environmental exposures and disease.”²² The EGP has successfully “identified over 500 putative environmental susceptibility genes” and continues to study changes in genes that cause individuals to become susceptible or resilient to certain toxic substances.²³

Discoveries in genetic research are advancing our understanding of the interaction between the human body and outside substances, such as chemicals found in the water we drink, the food we eat, and the medicines we take.²⁴ Scientists have discovered certain “[g]ene expression biomarkers” that provide evidence of a person’s exposure to particular substances,²⁵ and a field of study known as “molecular epidemiology”²⁶ has developed to evaluate these biomarkers in order “to evaluate the damage done by toxic substances.”²⁷ Science has further uncovered the existence of certain types of biomarkers that can go beyond supplying proof that an individual has been exposed to a particular substance.²⁸ These biomarkers can measure the level of exposure that occurred,²⁹ determine the effects of the exposure,³⁰ and provide information

21. See Roberts, *supra* note 15, at 443 n.6 (citing Carolyne Park, *Genetics Offers Tool in Combat of Cancer: Field Young, Pays Off in Early Detection*, ARK. DEMOCRAT-GAZETTE, Aug. 24, 2008, at A1); see also Callahan, *supra* note 2, at 154 (citing Harri Vainio, *Promise of Molecular Epidemiology—Epidemiologic Reasoning, Biological Rationale and Risk Assessment*, 25 SCANDINAVIAN J. WORK ENVTL. HEALTH 498, 500 (1999)) (stating that “[t]oxic substances can also cause mutations at the genetic level,” and that “[m]utations in certain genes are specifically associated with cancer causation”).

22. See Callahan, *supra* note 2, at 165 (citing *Environmental Genome Project: Program Description*, NAT’L INST. OF ENVTL. HEALTH SCIENCES, <http://www.niehs.nih.gov/research/supported/programs/egp/> (last viewed Feb. 6, 2009)).

23. Marchant, *supra* note 3, at 9 (citing Jocelyn Kaiser, *Tying Genetics to the Risk of Environmental Diseases*, 300 SCIENCE 563, 563 (2003); Julie Wakefield, *Environmental Genome Project: Focusing on Differences to Understand the Whole*, 110 ENVTL. HEALTH PERSP. A757, A758 (2002)).

24. See Gold, *supra* note 11, at 397 (“[T]he legal significance of advancing scientific knowledge will continue to be tested in personal injury actions brought by people who allege that their exposure to drugs, workplace chemicals, pollutants, or other agents caused their disease.”).

25. See Marchant, *supra* note 3, at 24.

26. Callahan, *supra* note 2, at 147 (citing Frederica P. Perera, *Molecular Epidemiology: On the Path to Prevention?*, 92 J. NAT’L CANCER INST. 602, 602 (2000)) (describing molecular epidemiology as “an area of research founded in the early 1980s for the purpose of investigating cancer risk at the molecular or genetic level”).

27. *Id.* at 151 (citing Vainio, *supra* note 21, at 498).

28. See *id.* at 151–55 (citations omitted) (describing various types of biomarkers).

29. See *id.* at 152 (citing Frederica P. Perera & I. Bernard Weinstein, *Molecular Epidemiology: Recent Advances and Future Directions*, 21 CARCINOGENESIS 517, 518 (2000)) (“The most common biomarkers are those that are used to measure the internal dose of a toxin. . . . These markers have been used to measure exposure to several types of toxins, including cigarette smoke, polycyclic aromatic hydrocarbons (PAHs) and aflatoxin.”).

30. See *id.* at 153–54 (citing Vainio, *supra* note 21, at 499–501) (describing the ability of environmental toxins and toxic substances to cause chromosomal and genetic mutations).

about whether an individual's disease can be attributed to such exposure.³¹ Additionally, aside from uncovering new information linking human disease to toxic substances, genetic research is providing information about human genes themselves and their own propensity to cause diseases.³²

Moreover, genetic research has also revealed information about various types of genetic susceptibilities to disease that may predispose certain individuals to developing particular diseases³³ or increase their chances of experiencing adverse reactions to particular substances.³⁴ While an individual's genetic composition alone may lead him or her to develop a disease, "environmental susceptibility genes" increase an individual's susceptibility to outside environmental toxins.³⁵ Thus, these environmental susceptibility genes can cause genes to act in ways that they would not otherwise act when they are exposed to outside factors.³⁶ Additionally, there are genes that protect the body by "enhancing tumor suppression, DNA repair, or detoxification, or interfering with activation pathways."³⁷ Taken together, scientific information about these genes will increase our understanding of the effects of toxic substances and allow courts to better determine the cause of injuries presented in toxic tort claims.

III. TOXIC TORT LITIGATION AND ITS FUTURE WITH GENETIC INFORMATION

Penny Plaintiff is a fifty-six-year-old resident of South Carolina who was recently diagnosed with a rare form of leukemia. While the cause of her leukemia has so far eluded the medical community, Penny is convinced that her use of the prescription drug Valtor is to blame. Therefore, Penny wishes to bring a toxic tort claim against the manufacturer of Valtor, Columbia Pharmaceuticals, Inc. (Columbia). How can Penny establish this claim? Her success will greatly depend not only on her ability to access scientific information about the general use of Valtor, but more importantly, it will depend on her ability to present conclusive evidence about Valtor's effect on her body.³⁸

31. See *id.* at 153 (citing Perera, *supra* note 26, at 605).

32. See generally Susan R. Poulter, *Genetic Testing in Toxic Injury Litigation: The Path to Scientific Certainty or Blind Alley?*, 41 JURIMETRICS J. 211, 216 (2001) (discussing the development of tests for genetic predispositions to disease).

33. *Id.* at 214–15.

34. See *id.* at 219 n.41 (discussing the role of genetics in increasing one's susceptibility to allergies).

35. See *id.* at 214–15 (citing NAT'L CANCER INST., NAT'L INST. OF HEALTH, UNDERSTANDING GENE TESTING 11–12 (1997)) ("[A] number of specific genetic mutations or variations have been associated with various diseases, from Alzheimer's disease to cancers of various kinds.").

36. See *id.*

37. *Id.*

38. See Callahan, *supra* note 2, at 156–57 (citing GERALD W. BOSTON & M. STUART MADDEN, LAW OF ENVIRONMENTAL AND TOXIC TORTS 342–44 (1994)) (discussing requirement that plaintiffs must prove generic and individual causation).

Toxic Tort Litigation Today

Americans face increasing health risks due to the growing presence of toxic substances in the environment,³⁹ and new evidence continues to reveal associations between individuals' exposures to certain substances and the development of particular diseases.⁴⁰ Not surprisingly, toxic tort litigation has filled court dockets throughout the United States as injured parties, like Penny, seek relief for injuries allegedly caused by their exposure to certain substances.⁴¹ Unfortunately, while there is increasing evidence connecting chemical exposure to certain diseases, toxic tort litigation continues to be plagued by uncertainties⁴² and evidence that fails to establish the products' alleged defects and causation between these defects and the plaintiffs' injuries.⁴³

Penny's claim will be especially difficult to establish for three reasons. First, prescription drug manufacturers are widely protected from defective design claims⁴⁴ because prescription drugs are considered to be "unavoidably unsafe products."⁴⁵ Thus, courts have generally determined that, while prescription drugs carry inherent dangers of harming users, their benefits to human health outweigh the risks of such dangers.⁴⁶ Thus, in the absence of manufacturing and warning defects, prescription drugs are normally considered not defective and manufacturers are protected from related strict liability and negligence claims.⁴⁷

39. See *supra* note 12.

40. See Forsyth, *supra* note 12, at 120 ("There is also increasing evidence of association between chemical exposure and diseases such as cancer and conditions such as autism, infertility, and birth defects. Advances in science have shown both new links between specific diseases and specific chemicals, and a general increase in disease and disorders potentially linked to widespread pervasive exposure to toxic chemicals." (footnotes omitted)).

41. See DANIEL J. SMITH, *Products Liability—Formaldehyde Fumes Emitted by Building Materials*, in 3 AM. JUR. 3D *Proof of Facts* 225, 287 (1989) (describing "[t]he explosion of toxic tort litigation").

42. See Marchant, *supra* note 3, at 7 ("When the question in a toxic tort case is whether a particular toxic substance caused injury in a specific individual, the data gaps and uncertainties are even greater.").

43. See Forsyth, *supra* note 12, at 122 (listing "insurmountable obstacles to proving causation" as one of the "three main obstacles the tort system presents that have made it unable to offer relief from widespread chemical exposure").

44. See RESTATEMENT (THIRD) OF TORTS: PRODS. LIAB. § 6 cmt. f (1998) ("Given this very demanding objective standard, liability is likely to be imposed only under unusual circumstances.").

45. See RESTATEMENT (SECOND) OF TORTS § 402A cmt. k (1965) (describing "unavoidably unsafe products" as "products which, in the present state of human knowledge, are quite incapable of being made safe for their intended and ordinary use" and finding that "[t]hese are especially common in the field of drugs").

46. See RESTATEMENT (THIRD) OF TORTS § 6 cmt. b ("The traditional refusal by courts to impose tort liability for defective designs of prescription drugs and medical devices is based on the fact that a prescription drug or medical device entails a unique set of risks and benefits. What may be harmful to one patient may be beneficial to another.").

47. See RESTATEMENT (SECOND) OF TORTS § 402A cmt. k ("Such a product, properly prepared, and accompanied by proper directions and warning, is not defective, nor is it *unreasonably* dangerous. . . . The seller of such products, again with the qualification that they are

Second, while defective warning claims dominate prescription drug litigation,⁴⁸ toxic tort plaintiffs must combat the fact that a drug manufacturer's duty to warn is generally limited to warning about foreseeable risks of harm posed by its product.⁴⁹ Based on this limited duty to warn, drug manufacturers have traditionally been shielded from any duty to warn of unknowable dangers,⁵⁰ as well as from any duty to warn individuals with unusual susceptibilities to their drugs.⁵¹ Regarding the duty to warn only of foreseeable dangers, a drug manufacturer will only be held liable for design and warning defect claims if a court determines that, prior to selling its product, the manufacturer failed to carry out its responsibility to perform reasonable testing to discover risks that such testing would have revealed.⁵² Thus, liability for any dangers that are discovered after the manufacturer sells the drug will be viewed in this context, and a plaintiff like Penny will be faced with the difficult burden of establishing that the danger could have been discovered prior to such sale.

Additionally, many plaintiff's claims are significantly damaged by evidence that their unusual susceptibilities are partly to blame for causing their injuries.⁵³ Just as a drug manufacturer is not required to discover every possible danger associated with its product before placing it on the market,⁵⁴ it is also not required to discover every potential person that might be harmed by using the product.⁵⁵ The *Restatement (Third) of Torts: Products Liability* provides that "[t]he general rule in cases involving allergic reactions is that a warning is required when the harm-causing ingredient is one to which a substantial number

properly prepared and marketed, and proper warning is given, where the situation calls for it, is not to be held to strict liability for unfortunate consequences attending their use . . .").

48. DAVID G. OWEN, *PRODUCTS LIABILITY LAW* 627 n.3 (2d ed. 2008) ("Failure to warn or instruct is the major basis of liability for manufacturers of prescription drugs and medical devices." (quoting *RESTATEMENT (THIRD) OF TORTS* § 8 cmt. d) (internal quotation marks omitted)).

49. See *RESTATEMENT (THIRD) OF TORTS* § 2(c); see also *Wooderson v. Ortho Pharm. Corp.*, 681 P.2d 1038, 1049 (Kan. 1984) ("The duty of the ethical drug manufacturer to warn is limited to those dangers which the manufacturer knows, or has reason to know, are inherent in the use of its drug." (quoting *McEwen v. Ortho Pharm. Corp.*, 528 P.2d 522, 528 (Or. 1974)) (internal quotation marks omitted)).

50. See DAVID G. OWEN, *PRODUCTS LIABILITY IN A NUTSHELL* 315 (8th ed. 2008) ("[T]he vast majority of courts today refuse to impose a duty on manufacturers to warn of unknowable risks . . .").

51. See OWEN, *supra* note 48, at 738 ("[C]omment k [to the *RESTATEMENT (THIRD)*] provides that there is no duty to warn of risks of unforeseeable allergic reactions.").

52. See *RESTATEMENT (THIRD) OF TORTS* § 2 cmt. a.

53. See Marchant, *supra* note 3, at 14 ("Another set of legal issues will revolve around the duty of a product manufacturer to protect or warn genetically susceptible individuals in the population.").

54. See *RESTATEMENT (THIRD) OF TORTS* § 2 cmt. a.

55. See Marchant, *supra* note 3, at 15 ("The Court of Appeals for the Fourth Circuit held that liability can only be imposed for adverse effects that would be suffered by a 'normal' person, and thus the plaintiff's own allegation that she was unusually susceptible precluded her claim." (citing *Cavallo v. Star Enter.*, 100 F.3d 1150, 1154 (4th Cir. 1996))).

of persons are allergic.”⁵⁶ Therefore, despite a couple of exceptions to this rule, which are discussed below,⁵⁷ if Penny is found to be unusually susceptible to harm caused by Valtor, Columbia will likely prevail against any claim of defective design or warning by asserting the “‘idiosyncratic response’ defense,” i.e., by arguing that manufacturers have no duty to warn of risks that are remotely possible to individuals with rare allergies or susceptibilities.⁵⁸

Finally, toxic tort plaintiffs experience the most difficulty in establishing causation.⁵⁹ Here, the burden of proof is often insurmountable due in large part to the lack of available scientific information to show that a particular drug is capable of causing the injury at issue and that it, in fact, caused the plaintiff’s particular injury.⁶⁰ Toxic tort plaintiffs and defendants have been forced to defend their arguments using scientific methodologies that are incapable of providing concrete evidence of causation.⁶¹ Consider, for instance, epidemiological studies, which are widely considered to be the preferred method of proving causation in toxic tort litigation.⁶² Because these studies present information about “the incidence, distribution, and etiology of disease in human populations,”⁶³ they can be used to show general causation by presenting evidence of a general causal relationship between exposure to a substance and development of a particular disease.⁶⁴ However, because epidemiological studies cannot directly attribute the cause of an individual’s disease to her exposure to a specific substance, they cannot effectively establish specific causation.⁶⁵

56. RESTATEMENT (THIRD) OF TORTS § 2 cmt. k.

57. See *infra* Part III.A.1.

58. See Marchant, *supra* note 3, at 14 (describing the idiosyncratic response defense as traditionally applying “to protect a manufacturer from liability for a product . . . that appears safe to the general population but may cause an unusual response in individuals with a rare allergy or sensitivity to the product”).

59. See, e.g., Craig A. Barr, *A Practical Guide to Proving and Disproving Causation in Radiation Exposure Cases: Hanford Nuclear Site and Radioactive Iodine*, 31 GONZ. L. REV. 1, 2 (1996).

60. See Lin, *supra* note 1, at 965–66 (“Because of the extensive research required, the probabilistic nature of research results, and the uncertainty often associated with those results, plaintiffs are rarely in a position to prove either general or specific causation.”).

61. See *id.*

62. See *Chambers v. Exxon Corp.*, 81 F. Supp. 2d 661, 664 (M.D. La. 2000) (“[T]he most conclusive type of evidence of causation is epidemiological evidence.” (citing *Brock v. Merrell Dow Pharms., Inc.*, 874 F.2d 307, 311 (5th Cir.), *modified*, 884 F.2d 166 (5th Cir. 1989))).

63. Michael D. Green et al., *Reference Guide on Epidemiology*, in ANNOTATED REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 469, 471 (Michael J. Saks et al. eds., 2d ed. 2004).

64. Callahan, *supra* note 2, at 157.

65. See, e.g., Barr, *supra* note 59, at 14 (“Another disadvantage is that epidemiological studies are generalistic in nature.” (citing *DeLuca v. Merrell Dow Pharms., Inc.*, 911 F.2d 941, 945 & n.6 (3rd Cir. 1990))).

Next, consider toxicological studies, which are often used in conjunction with epidemiological and other studies to establish general causation.⁶⁶ Because toxicological research is performed on animals and in test tubes,⁶⁷ and not often on humans,⁶⁸ courts have viewed them with skepticism⁶⁹ and often exclude them from evidence in toxic tort cases.⁷⁰ Parties have additionally relied on clinical, or case, reports offered by their expert medical witnesses to establish causation.⁷¹ Physicians publish these reports and their focus is usually limited to an individual's reaction to a particular substance.⁷² Therefore, as opposed to providing a broad understanding of a substance's effect on a large group of people, clinical reports only provide the court with one example of how a particular substance could affect a person.⁷³ Therefore, just like toxicological studies,⁷⁴ courts often consider these reports to be unreliable as scientific evidence of causation, and they are generally never sufficient to establish causation on their own.⁷⁵

While the above three scientific methodologies, if admitted in toxic tort litigation, are generally limited to serving as evidence of general causation,⁷⁶ differential diagnosis is commonly employed by expert witnesses to establish

66. See *id.* at 19 (“[A]nimal studies, much like epidemiological studies, can only demonstrate general causation.”).

67. See 3 DAVID L. FAIGMAN ET AL., MODERN SCIENTIFIC EVIDENCE: THE LAW AND SCIENCE OF EXPERT TESTIMONY 113 (2011).

68. See *id.*

69. See, e.g., *Conde v. Velsicol Chem. Corp.*, 804 F. Supp. 972, 1026 (S.D. Ohio 1992) (“[A]nimal studies alone are not, under the circumstances of this case, sufficiently reliable medical or scientific evidence to prove that a chemical causes human illness or disease.” (citing *Turpin v. Merrell Dow Pharms., Inc.*, 959 F.2d 1349, 1360 (6th Cir. 1992); *Novak v. United States*, 865 F.2d 718, 721–23 (6th Cir. 1989); *In re “Agent Orange” Prod. Liab. Litig.*, 611 F. Supp. 1223, 1241 (E.D.N.Y. 1985))).

70. See Callahan, *supra* note 2, at 158; see also Barr, *supra* note 59, at 18 (“The general trend is to exclude animal studies from evidence at trial.”).

71. See Callahan, *supra* note 2, at 159 (“Plaintiffs also use clinical or case reports to show that exposure to a particular toxin caused injury.”).

72. See *id.* (citing *Allison v. McGhan Med. Corp.*, 184 F.3d 1300, 1316 (11th Cir. 1999)); see also *Smith v. Wyeth–Ayerst Labs. Co.*, 278 F. Supp. 2d 684, 695 (W.D.N.C. 2003) (“These are reports in medical journals describing clinical events involving one individual or a few individuals. They report unusual or new disease presentations . . . or external causes of diseases.”).

73. See Callahan, *supra* note 2, at 159 (“These reports are problematic as proof of causation because they are only about one patient, who could have had an unusual reaction to the exposure.” (citing *Allison*, 184 F.3d at 1316)).

74. See *id.* at 158.

75. See, e.g., *Siharath v. Sandoz Pharms. Corp.*, 131 F. Supp. 2d 1347, 1361 (N.D. Ga. 2001) (quoting *Casey v. Ohio Med. Prods.*, 877 F. Supp. 1380, 1385 (N.D. Cal. 1995)) (finding case reports insufficient to establish causation); *Hollander v. Sandoz Pharms. Corp.*, 95 F. Supp. 2d 1230, 1237 (W.D. Okla. 2000) (finding the case reports relied upon by plaintiffs’ experts insufficient to establish causation), *aff’d in part*, 289 F.3d 1193 (10th Cir. 2002).

76. See Callahan, *supra* note 2, at 157 (“Although these studies are useful in proving that the toxin is capable of harm, courts have found them inadmissible under *Daubert* as their inherent weaknesses make it difficult to prove the specific toxin exposure caused the plaintiff’s injuries.”).

specific causation.⁷⁷ Physicians use differential diagnosis to determine the cause of a patient's disease by ruling out probable causes until only one cause is left.⁷⁸ While courts prefer—and often demand—evidence derived from differential diagnosis as proof of specific causation,⁷⁹ this methodology, nonetheless, falls short of definitively proving the cause of a plaintiff's injury.⁸⁰ Consider, for example, *Doe v. Ortho-Clinical Diagnostics, Inc.*,⁸¹ a case that illustrates the problems associated with using differential diagnosis to prove specific causation. In *Doe*, the parents of an autistic child alleged that thimerosal contained in the defendant drug manufacturer's vaccine caused their child to develop autism.⁸²

While the plaintiffs were unable to satisfy the burden of proving general causation,⁸³ usually a prerequisite to a court's consideration of specific causation,⁸⁴ the court nonetheless considered whether the plaintiffs could prove specific causation.⁸⁵ As evidence of specific causation, the plaintiffs presented an expert medical witness who testified on his use of differential diagnosis to determine that thimerosal was the cause of the child's autism.⁸⁶ However, based on its finding that the expert "fail[ed] to take into account the existence of such a

77. See Joseph Sanders & Julie Machal-Fulks, *The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law*, 64 LAW & CONTEMP. PROBS., no. 4, Autumn 2001, at 107, 111 ("Because courts have generally refused to relieve the plaintiff from proving specific causation, differential diagnosis evidence is often a crucial component of the plaintiff's case.").

78. See *id.* at 107–08; see also Wendy Michelle Ertmer, Note, *Just What the Doctor Ordered: The Admissibility of Differential Diagnosis in Pharmaceutical Product Litigation*, 56 VAND. L. REV. 1227, 1240 (2003) ("As with differential diagnosis of a disease, clinicians attempting to determine the cause of the disease make a list of potential causal agents and, through a process of elimination, identify the agent that remains on the list as the most likely cause of the disease.").

79. See Sanders & Machal-Fulks, *supra* note 77, at 111.

80. See *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 265 (4th Cir. 1999) ("A differential diagnosis that fails to take serious account of other potential causes may be so lacking that it cannot provide a reliable basis for an opinion on causation."); see also Ertmer, *supra* note 78, at 1252–53 (citing *Glastetter v. Novartis Pharms. Corp.*, 107 F. Supp. 2d 1015, 1019, 1028 (E.D. Mo. 2000), *aff'd*, 252 F.3d 986 (8th Cir. 2001)) (describing case where plaintiff's experts "relied on differential diagnosis methodology to form their opinion that [the prescription drug] caused the plaintiff's stroke" and "[t]he court suggested that a differential diagnosis opinion on causation is unreliable whenever that expert has not also formed an opinion, based on scientific studies, on general causation").

81. 440 F. Supp. 2d 465 (M.D.N.C. 2006).

82. *Id.* at 468.

83. *Id.* at 476.

84. See *id.* (finding that "the Court need not go further"); see also *Mack v. AmerisourceBergen Drug Corp.*, 671 F. Supp. 2d 706, 710 (D. Md. 2009) (describing general causation as "an essential prerequisite to proving specific causation" (quoting *Foster v. Legal Sea Foods, Inc.*, No. CCB-03-2512, 2008 WL 2945561, at *10 (D. Md. July 25, 2008)) (internal quotation marks omitted)).

85. *Doe*, 440 F. Supp. 2d at 476 ("Nevertheless, for sake of completeness, the Court will also examine [the medical expert's] methodology concerning specific causation, that is, whether RhoGAM specifically caused Minor Child Doe's autism.").

86. *Id.*

strong likelihood of a currently unknown genetic cause of autism,”⁸⁷ the court concluded that the expert’s differential diagnosis was not properly performed and could not be admitted as evidence of causation.⁸⁸ Thus, as courts seek more precise evidence of causation in toxic tort litigation, even evidence obtained using the best available scientific methodologies, like differential diagnosis, may be excluded.⁸⁹

An understanding of these deficiencies in available scientific evidence provides the basis for establishing a court’s need for advanced scientific methodologies to determine causation in toxic tort litigation, especially where prescription drugs are alleged to cause plaintiffs’ injuries.⁹⁰ As Professor David Owen explains, “[d]rugs . . . are different,”⁹¹ and they provide unique and difficult challenges to the American tort system.⁹² Fortunately, genetic research is rapidly advancing⁹³ and producing the very information about the causes of human disease that courts desire.

Using Genetic Research to Improve Courts’ Decisions in Toxic Tort Cases

Genetic research towers over its rival scientific methodologies with its ability to provide answers to many of the questions that have, so far, stumped the courts.⁹⁴ As this research continues to develop, and as courts begin to admit it into toxic tort litigation, plaintiffs and defendants alike will benefit,⁹⁵ as

87. *Id.* at 478.

88. *Id.*

89. *See, e.g.,* Westberry v. Gislaved Gummi AB, 178 F.3d 257, 265 (4th Cir. 1999) (making it clear that the methodology used by a plaintiff to prove causation cannot “fail[] to take serious account of other potential causes [such that it would be] so lacking that it cannot provide a reliable basis for an opinion on causation”).

90. *See* Ertmer, *supra* note 78, at 1241 (“The diagnosis of adverse drug reactions is substantially more complex, however, precisely because the association between the causal agent (the drug) and the disease is not well established.”).

91. OWEN, *supra* note 48, at 572 & n.33.

92. Professor Owen continues:

Many have been bewitched, bedazzled, and bewildered in attempting to figure just how principles of design defectiveness should be applied to prescription drugs Whether and how prescription drugs in particular should be treated differently from other types of products has consumed more time and effort, and resulted in the gnashing of more teeth, than about any other particularized issue in all of products liability law.

OWEN, *supra* note 48, at 566–67 (footnote omitted). *See* Ertmer, *supra* note 78, at 1263 (“The admissibility of expert testimony to prove causation is one of the most difficult issues facing trial courts in pharmaceutical product liability litigation.”).

93. *See* Rochelle C. Dreyfuss & James P. Evans, *From Bilski Back to Benson: Preemption, Inventing Around, and the Case of Genetic Diagnostics*, 63 STAN. L. REV. 1349, 1353 (2011).

94. *See, e.g.,* Marchant, *supra* note 3, at 8 (“New genetic methods and data have the potential to fill some of the scientific uncertainties and data gaps in toxic tort litigation, thus making toxic tort litigation more accurate and fair.”); Callahan, *supra* note 2, at 163–64 (“Molecular epidemiological studies are perfectly suited to prove individual causation because . . . the use of biomarkers identifies the *specific* cause of the plaintiff’s disease.”).

95. *See* Callahan, *supra* note 2, at 165.

illustrated by the following discussion of Penny's potential claims and Columbia's potential defenses.

To begin, consider the ability of genetic research to reveal new information about how certain substances affect the human body.⁹⁶ Suppose that genetic research is conducted to expose human genes to the active ingredient found in Valtor, and the research results in new information showing that when healthy genes are exposed to this ingredient, they mutate in a way that leads to the development of leukemia. Of course, assuming that it was not possible for Columbia to discover this information prior to placing Valtor on the market, Columbia will not be liable for defective design or warnings claims.⁹⁷ However, this discovery transforms a once unknown danger into a foreseeable one, and one where federal regulators, as well as Columbia, must balance the risks and benefits of Valtor in determining whether to keep the drug on the market and what warnings to include with the drug.⁹⁸ This new information will undoubtedly tilt the scale in the direction of greater risks. Thus, if Columbia continues to market Valtor after learning of this potential danger and fails to take any available and reasonable precautions to make the drug safer or to warn about the danger,⁹⁹ it risks a court's determination that upon discovery of this danger, Valtor became unreasonably dangerous and Columbia became liable for defective design and warning claims.¹⁰⁰ Where future plaintiffs are able to connect this defect to their development of leukemia, Columbia will face significantly increased liability in toxic tort claims.¹⁰¹

Next, consider a scenario in which genetic research reveals information that the active ingredient in Valtor, while once believed to have the capacity to cause leukemia in only a small number of individuals, is found to have the potential to cause this disease in many more individuals. For example, it is possible that scientists once believed that very few individuals were genetically susceptible to harm caused by Valtor. However, genetic research then reveals information showing that numerous individuals carry a genetic susceptibility to harm caused by Valtor. Now, the general rule that a "product [must] contain[] an ingredient

96. See Callahan, *supra* note 2, at 147 (citing David J. Hunter, *The Future of Molecular Epidemiology*, 28 INT'L J. EPIDEMIOLOGY S1012, S1014 (1999)) ("[M]olecular epidemiology links exposure and the onset of disease.").

97. See RESTATEMENT (THIRD) OF TORTS: PRODUCTS LIABILITY § 2 cmt. a (1998) ("Most courts agree that, for the liability system to be fair and efficient, the balancing of risks and benefits in judging product design and marketing must be done in light of the knowledge of risks and risk-avoidance techniques reasonably attainable at the time of distribution.").

98. See *id.* § 2 cmt. k ("[T]his reflects the same risk-utility balancing undertaken in warnings cases generally.").

99. See *id.* § 2(b)–(c).

100. See *id.* § 2 cmt. k.

101. See *id.* ("Clearly the plaintiff in most cases must show that the allergic predisposition is not unique to the plaintiff. . . . [H]owever, the court may properly consider the severity of the plaintiff's harm. The more severe the harm, the more justified is a conclusion that the number of persons at risk need not be large to be considered 'substantial' so as to require a warning.").

to which a substantial number of the population are allergic”¹⁰² in order for a seller to be required to warn of the danger, may be replaced with a new duty imposed upon Columbia to warn of the danger.¹⁰³ This is especially true given the general rule that the “degree of substantiality is not precisely quantifiable”¹⁰⁴ and courts’ previous decisions holding manufacturers liable for their failure to warn, even where only small percentages of people were known to be susceptible to the product’s risks at issue.¹⁰⁵ Considering that a court held a manufacturer responsible for a danger known to affect less than two percent of the general population,¹⁰⁶ any new information increasing the number of individuals susceptible to developing leukemia by using Valtor will increase the likelihood that Columbia will be held liable for its failure to warn of the danger.¹⁰⁷

It is also important to consider a scenario in which genetic research does not reveal information about new health dangers associated with Valtor or increased numbers of individuals susceptible to harm from use of the drug, but instead provides additional evidence showing that the development of leukemia from the use of Valtor is limited to extremely rare cases in which individuals have unusual genetic susceptibilities. In this scenario, if Columbia is able to obtain genetic testing of Penny, and if such testing reveals that she is one of the rare individuals carrying this unique susceptibility to Valtor, Columbia’s defense—based in the idiosyncratic response doctrine—will be strengthened.¹⁰⁸

Additionally, as mentioned in Part I, genetic testing stands to have the greatest impact on the element of causation.¹⁰⁹ And, as discussed in Part II above, scientists are currently engaged in molecular epidemiological studies that assess the effects of certain substances on the human body.¹¹⁰ These studies continue to reveal new biomarkers that show that genes have been exposed to particular substances,¹¹¹ thus unveiling “‘fingerprints’ of specific carcinogens,

102. RESTATEMENT (SECOND) OF TORTS § 402A cmt. j (1965).

103. See Marchant, *supra* note 3, at 16 (“These cases are the first in what is likely to become an increasingly frequent type of legal claim in which a plaintiff contends that a manufacturer has a duty to identify and warn about possible genetic susceptibilities to its products.”).

104. RESTATEMENT (THIRD) OF TORTS § 2 cmt. k.

105. See, e.g., *Holmes v. Grumman Allied Indus.*, 478 N.Y.S.2d 143, 144–45 (N.Y. App. Div. 1984) (affirming denial of summary judgment motion by a bus manufacturer liable for its failure to warn of a danger caused by a chemical found in the dashboard of the bus which was known to affect less than two percent of the population).

106. See *id.* at 144.

107. See RESTATEMENT (THIRD) OF TORTS § 2 cmt. k.

108. See Marchant, *supra* note 3, at 12 (“Additionally, defendants could also seek to test plaintiffs for the presence of other genetic traits that might predispose the plaintiffs to the illnesses they have developed. Defendants would use such findings to support alternative causation arguments, namely, that the plaintiffs’ own genotypes, rather than exposure to the defendants’ toxic substances, caused or contributed to the plaintiffs’ illnesses.”).

109. See *supra* text accompanying notes 1–11; see also Marchant, *supra* note 3, at 8.

110. See *supra* text accompanying notes 15–23.

111. See Callahan, *supra* note 2, at 151 (citing Perera & Weinstein, *supra* note 29, at 517).

associating a specific carcinogen with a specific mutation.”¹¹² Furthermore, not only can biomarkers present evidence that a person has been exposed to a particular substance, but they can also provide direct proof that such exposure led to development of a disease like cancer.¹¹³ To understand the implications of this research on a toxic tort claim, consider a situation in which genetic research produces information showing that a particular inherited genetic characteristic can lead to the development of leukemia without any interference from outside substances.¹¹⁴ In this hypothetical, Columbia will likely seek genetic testing to determine whether Penny has this genetic characteristic, and where the results of this testing show that Penny carries the genetic trait in question, Columbia will defend itself against Penny’s claim by arguing that Penny’s own genes are to blame for causing her leukemia.¹¹⁵

Moreover, just as proof that Penny carried the genetic characteristics at issue here would increase Columbia’s chances of successfully defending against her claim, proof that Penny does not carry a genetic characteristic that she claims to carry can make or break her claim.¹¹⁶ For example, consider a situation in which Penny claims to carry a genetic mutation known to lead to the development of leukemia and known to be caused by exposure to Valtor’s active ingredient.¹¹⁷ Genetic testing will provide definitive proof favoring Penny’s claim or Columbia’s defense, depending on whether it offers proof that Penny carries the genetic characteristic or provides for the absence of such proof.¹¹⁸ This proposition is illustrated in *Tompkin v. Philip Morris USA, Inc.*,¹¹⁹ where the

112. See *id.* at 154 (citing Perera & Weinstein, *supra* note 29, at 519).

113. See *id.* at 154–55 (citing Perera & Weinstein, *supra* note 29, at 519) (“Recent studies suggest mutations in oncogenes . . . and in tumor suppressor genes . . . may be used as biomarkers to directly prove exposure to a specific carcinogen leading to cancer.”).

114. See Marchant, *supra* note 3, at 13 (citing Bowen v. E.I. Du Pont de Nemours & Co., No. Civ.A. 97C-06-194 CH, 2005 WL 1952859 (Del. Super. Ct. June 23, 2005)) (describing a case in which a “defendant obtained genetic testing of a plaintiff whose birth defect was allegedly caused by prenatal exposure to Benlate and demonstrated, to the satisfaction of both the plaintiff’s lead expert and the court, that the disability was caused by a specific inherited genetic mutation rather than chemical exposure”).

115. See, e.g., Hoffman & Rothenberg, *supra* note 17, at 867 n.20 (listing cases in which defendants argued that plaintiffs’ injuries were caused by their own genetics). But see Marchant, *supra* note 3, at 12 n.17 (listing cases where defendants’ claims that plaintiffs’ genetics caused their injuries failed due to insufficient evidence to substantiate the defenses).

116. See Marchant, *supra* note 3, at 11–12 (citing Easter v. Aventis Pasteur, Inc., 358 F. Supp. 2d 574, 575, 579 (E.D. Tex. 2005)) (discussing *Easter*, in which genetic testing revealed that the plaintiff did not have the pertinent genetic susceptibility and suggesting that “defendants might use the absence of the pertinent susceptibility genes in a plaintiff to buttress their arguments against causation”).

117. See Barry B. Cepelewicz & Eric Watt Wiechmann, *Genetic Injury in Toxic Tort Cases: What Science Can and Cannot Prove*, 62 DEF. COUNS. J. 201, 202 (1995) (“In an increasing number of instances, plaintiffs have argued that exposure to a toxin created a change in their chromosomes, thereby increasing their risk of some disease, usually cancer, in the future.”).

118. See Marchant, *supra* note 3, at 8.

119. 362 F.3d 882 (6th Cir. 2004).

widow of a former smoker brought a products liability claim against several cigarette manufacturers, alleging that her husband died as a result of smoking their cigarettes.¹²⁰ The defendant manufacturers presented expert testimony that tissue samples obtained from the husband's body did not show the genetic changes associated with smoking,¹²¹ but instead provided evidence of damage caused by the husband's exposure to asbestos and mineral fibers.¹²² Thus, the court of appeals refused to overturn the lower court's decision favoring the defendant cigarette manufacturers.¹²³

Alternatively, suppose that in response to Penny's claim that Valtor caused her leukemia, Columbia obtains information about other prescription drugs that Penny has taken and, additionally, finds scientific information showing that the use of another drug, "Drug B," can cause a certain gene mutation that has a high likelihood of leading to the development of leukemia. Suppose also that Penny confirms that she has used Drug B and agrees to undergo genetic testing. Where the testing reveals that she has been exposed to Drug B, Columbia will defend against liability for Penny's injury by arguing that this information points to Drug B as the cause of her leukemia.¹²⁴

In addition to providing direct evidence of causation by using genetic evidence, Penny and Columbia may both benefit by presenting genetic evidence in support of inferences. For example, consider a case in which an individual brought a workers' compensation claim, alleging that his exposure to benzene caused him to develop chronic myelogenous leukemia (CML).¹²⁵ While the plaintiff in this case was not subject to the same standards of proof of causation that are imposed on toxic tort litigants,¹²⁶ the case illustrates an important avenue for plaintiffs and defendants to pursue using genetic evidence in toxic tort litigation. Here, the plaintiff prevailed by presenting expert testimony linking benzene to one form of leukemia and subsequently using this causal link to infer that benzene caused the plaintiff's particular leukemia.¹²⁷ Similarly, should Penny be able to present genetic information showing that Valtor's active ingredient has a significant likelihood of causing another type of leukemia,

120. *Id.* at 885.

121. *Id.* at 890 & n.5.

122. *Id.* at 890.

123. *Id.* at 896.

124. See DAVID G. OWEN ET AL., MADDEN & OWEN ON PRODUCTS LIABILITY 768 & n.67 (3d ed. 2000) (listing cases in which defendant attempted to prove its product was not a substantial factor for causing plaintiff's harm).

125. See *Casdorff v. W. Va. Office Ins. Comm'r*, 690 S.E.2d 102, 105 (W. Va. 2009).

126. See *id.* at 113 (observing that "the Rules of Evidence do not strictly apply to workers' compensation claims").

127. *Id.* at 109 ("[The plaintiff's expert] noted that it is generally accepted by the scientific community that benzene exposure can cause acute myelogenous leukemia . . . and after analyzing several case studies, he believed that they were persuasive enough to allow him to state to a reasonable degree of medical certainty that exposure to benzene and other hydrocarbons were probably causative in the development of the Appellant's CML.").

Penny may be able to provide genetic evidence of her exposure to Valtor in order to present a strong inference that the drug caused her type of leukemia.¹²⁸

Alternatively, Columbia may build its own inferences of alternative causes of Penny's leukemia by presenting genetic evidence that Penny carries a genetic mutation that causes similar forms of leukemia without any interference from outside substances,¹²⁹ or by showing that other factors, for example, chemicals found in the water that Penny drinks, are known to cause similar types of leukemia.¹³⁰ These examples reveal Columbia's ability to use inferences to establish arguments for alternative causes of Penny's leukemia.

While concededly simplistic, the examples above illustrate the capacity of genetic research to provide the conclusive evidence¹³¹ that other scientific methodologies have been unable to produce and that courts faced with toxic tort claims have long desired.¹³² However, despite the incredible potential for genetic research to transform toxic tort litigation, the degree to which courts will be willing to admit genetic evidence in toxic tort litigation is unclear.¹³³ Admittedly, for any of the benefits of genetic research to be realized by courts and toxic tort litigants, trial court judges, serving in their capacity as "gatekeepers,"¹³⁴ must first admit genetic evidence. However, given the fact that courts seem to have set epidemiological studies and differential diagnoses as the baseline for determining whether scientific evidence is reliable,¹³⁵ it seems unlikely that evidence obtained through advances in genetic research will be excluded.¹³⁶ Furthermore, it is important to note that federal and state courts

128. See Callahan, *supra* note 2, at 164–65.

129. See Marchant, *supra* note 3, at 12.

130. See OWEN ET AL., *supra* note 124, at 768.

131. See Callahan, *supra* note 2, at 164 (“[U]nlike differential diagnosis that only rules out other possible causes of disease, the use of biomarkers identifies the *specific* cause of the plaintiff's disease.”).

132. See Marchant, *supra* note 3, at 7–8.

133. See Callahan, *supra* note 2, at 148 (“Although a few authors have suggested the use of this type of genetic evidence in toxic tort litigation, they have not addressed admissibility of the evidence under the *Daubert* standard.”); see also Childs, *supra* note 10, at 443 (“[W]hether courts are willing to recognize and accept toxicogenomics as a new science that can objectively demonstrate how chemicals impact a disease process early in the chain of causation is still an open question.”); Mark S. Ellinger, *DNA Diagnostic Technology: Probing the Problem of Causation in Toxic Torts*, 3 HARV. J.L. & TECH. 31, 50 (1990) (discussing the use of DNA tests in toxic tort litigation and its potential admissibility under the *Frye* standard); Randi B. Weiss et al., *The Use of Genetic Testing in the Courtroom*, 34 WAKE FOREST L. REV. 889, 910–12 (1999) (discussing the use of genetic changes as proof of causation in toxic torts).

134. See *Watson v. Ford Motor Co.*, 389 S.C. 434, 445, 699 S.E.2d 169, 174 (2010).

135. See Callahan, *supra* note 2, at 160.

136. See *id.* at 163 (“Courts will probably find molecular epidemiological studies reliable under *Daubert* as these studies prove individual causation by linking exposure with the plaintiff's disease.”). But see Niccol Kording & Janine P. DuMontelle, *An Overview of Admissibility of Genetic Test Results in Federal Civil Actions: An Uncertain Destiny*, 19 WHITTIER L. REV. 681, 690 (1998) (citing *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579 (1993)) (“[G]enetic testing . . . is speculative and can not pass the relevancy requirement of *Daubert* for scientific evidence.”).

routinely use genetic information,¹³⁷ particularly in criminal and paternity cases,¹³⁸ and courts have already begun to admit genetic information in toxic tort litigation.¹³⁹ Moreover, some courts have specifically pointed to the lack of evidence of genetic information in toxic tort cases, highlighting their interest in considering such information in the future.¹⁴⁰

IV. ADVANCING WITH SCIENCE: WHY COURTS SHOULD INCORPORATE GENETIC EVIDENCE INTO TOXIC TORT LITIGATION

As genetic research has advanced, arguments for and against the use of genetic evidence in toxic tort cases have grown louder.¹⁴¹ Proponents of courts' use of genetic evidence argue that it holds the key to conclusively identifying causation in toxic tort litigation,¹⁴² while critics of such use argue that the touted benefits of genetic evidence are mostly yet to be realized.¹⁴³ Additionally, some critics have raised concerns that the introduction of genetic information could adversely affect plaintiffs' lives outside of the courtroom if personal genetic information becomes public.¹⁴⁴ However, review of federal legislation enacted in recent years¹⁴⁵ suggests that these concerns have been addressed by the

137. See Weiss et al., *supra* note 133, at 906 & n.187, 913.

138. See Jordan K. Garrison, *Courts Face the Exciting and the Inevitable: DNA in Civil Trials*, 23 REV. LITIG. 435, 435–36 (2004); see also *Andrews v. State*, 533 So. 2d 841, 850 (Fla. Dist. Ct. App. 1988) (affirming the trial judge's decision to admit the results of DNA testing in criminal case).

139. See Hoffman & Rothenberg, *supra* note 17, at 867; Anthony S. Niedwiecki, *Science Fact or Science Fiction? The Implications of Court-Ordered Genetic Testing Under Rule 35*, 34 U.S.F. L. REV. 295, 296 (2000) ("The use of genetics has now made its way into the civil courtroom as well.").

140. See *Doe v. Ortho-Clinical Diagnostics, Inc.*, 440 F. Supp. 2d 465, 477 (M.D.N.C. 2006).

141. See, e.g., Gold, *supra* note 11, at 393 ("So far, the scholarship has been split between a prophetic and a skeptical view of the new science's likely impact [on toxic tort litigation].").

142. See, e.g., Garrison, *supra* note 138, at 460 ("Within the legal system, the courts must face genetic evidence as the newest scientific advance and embrace it for the information it can provide."); Gold, *supra* note 11, at 401 ("The touted promise of toxicogenomics for providing particularistic evidence—at long last opening the black box—may lead courts to conclude that the grail is found. In some cases it probably will be." (footnote omitted)).

143. See, e.g., Poulter, *supra* note 32, at 216 ("[U]ncertainties suggest caution in using genetic information in analyzing the role of toxic substances in causing a particular instance of disease.").

144. See, e.g., Weiss et al., *supra* note 133, at 913 ("As genetic testing becomes more sophisticated, it will have many applications in the legal system. Complex legal questions regarding privacy, discrimination, and insurance coverage are likely to arise with wider use of genetic testing."); see also Rohan Hebbur, *The Impact of the Genetic Information Nondiscrimination Act on Sports Employers: A Game of Balancing Money, Morality, and Privacy*, 8 WILLAMETTE SPORTS L.J. 52, 54 (2011) (citing Paul Steven Miller, *Is There a Pink Slip in My Genes? Genetic Discrimination in the Workplace*, 3 J. HEALTH CARE L. & POL'Y 225, 226 (2000)) ("Health insurance companies and employers who have access to the results of genetic tests may use these results to deny coverage or refuse to hire someone based on their genetic defects.").

145. See, e.g., Genetic Information Nondiscrimination Act (GINA) of 2008, Pub. L. No. 110-233, 122 Stat. 881 (codified in scattered sections of 26, 29, and 42 U.S.C.) (prohibiting discrimination in insurance based on genetic information); Health Insurance Portability and

imposition of significant new protections for individuals' personal genetic information which prohibit access to, and discriminatory use of, this information by health insurers,¹⁴⁶ employers,¹⁴⁷ and others.¹⁴⁸ Furthermore, trial court judges share a long history of evaluating scientific evidence for admissibility purposes, and the Federal Rules of Evidence require that they "ensure that any and all scientific testimony or evidence admitted is not only relevant, but reliable."¹⁴⁹ Therefore, where questions regarding the reliability of genetic evidence are at issue, the trial courts are well-positioned to render admissibility determinations.

Overall, the above concerns are far outweighed by the benefits that toxic tort litigation stands to reap as scientists continue to discover genetic evidence linking toxic substances to widespread diseases. Information gleaned from this research will allow plaintiffs and defendants to break through evidentiary barriers that have long frustrated their arguments¹⁵⁰ and will put to rest many of the uncertainties that continue to plague toxic tort cases. Genetic evidence should play a key role in future toxic tort litigation by providing the conclusive evidence courts need to effectively determine fault.

Allison Hite

Accountability Act (HIPPA) of 1996, 29 U.S.C. §§ 1181(b)(1)(B), 1182(a)(1)(F) (2006) (same); Pauline T. Kim, *Regulating the Use of Genetic Information: Perspectives From the U.S. Experience*, 31 COMP. LAB. L. & POL'Y J. 693, 693 (2010) (citing GINA, Pub. L. No. 110-233, 122 Stat. 881) (discussing the purpose and impact of GINA); Roberts, *supra* note 15, at 442 ("Congress passed GINA in response to scientific advancements that advocates feared could result in a new form of discrimination, left uncovered by existing legal protections.").

146. See Roberts, *supra* note 15, at 443, 451 (citing HIPPA §§ 1181-82) (discussing that HIPPA "prohibits group health insurers from using genetic information in determining eligibility or setting premiums and from treating genetic information as a preexisting condition," and that GINA prohibits genetic information discrimination in health insurance).

147. See Hebbard, *supra* note 144, at 60 (citing 42 U.S.C.A. § 2000ff-1(b) (West Supp. 2011)) ("GINA makes it illegal for employers to request, require, or purchase genetic information about an employee or any member of the employee's family.").

148. See Roberts, *supra* note 15, at 451 (describing prohibitions on discrimination based on genetic-information for health insurers, employers, and others).

149. *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 589 (1993).

150. See Childs, *supra* note 10, at 441 ("Toxic tort litigation as most trial attorneys know it today is about to undergo a drastic, irrevocable change. Genetic evidence has the potential to revolutionize in this area.").

FOURTH CIRCUIT SURVEY

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