

**GENETIC TESTING AND TESTIMONY IN TOXIC TORT LITIGATION:
“ADMISSIBILITY AND EVALUATION”**

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Official guidelines must be promulgated in order to assist with case-by-case judicial admissibility determinations of genetic testing evidence in toxic tort litigation. Emerging technology, specifically advancements in genetic testing, could prove highly influential in toxic tort litigation. Genetic testing data can, in many cases, provide evidence of both (i) proof of exposure to a toxic substance and (ii) proof of genetic susceptibility of an individual plaintiff to a specific illness. Thus, genetic testing data may be helpful in proving an individual plaintiff’s exposure to the alleged toxic substance, as well as in establishing or disproving the plaintiff’s genetic susceptibility to illness alleged to have resulted from exposure to said toxic substance. As such, genetic testing data could have a drastic impact on a jury’s causation analysis in toxic tort litigation. It is imperative that such evidence be admitted only where there is clear scientific significance of the evidence in question and the impact of such data can be sufficiently explained to the jury. This article proposes that formal guidelines be promulgated to ensure the probative value of genetic testing evidence and testimony in toxic tort litigation is properly weighed against the potential harm such evidence poses to individual plaintiffs and defendants, as well as the potential of the evidence to confuse the jury’s causation analysis.

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I. INTRODUCTION

Proof of causation in toxic tort litigation remains one of the most challenging hurdles for plaintiffs to overcome, as plaintiffs must prove actual exposure to the alleged toxic substance as well as specific causation of plaintiff's injury, disease, or health ailment.¹ The potential presence of numerous disease-causing toxic substances in our everyday lives leads to uncertainties in determining which, if any, substance the plaintiff was exposed to and if that substance is the cause of the plaintiff's illness.² Given these uncertainties, the field of toxic tort litigation is a prime candidate for the use of emerging genetic testing to facilitate the analysis of causation.³ Genetic data most relevant to toxic tort litigation can be broken down into two main groups. The first group includes "inherited genetic variations that affect an individual's susceptibility to disease as a result of toxic exposures," and the second group includes "genetic changes that occur in individual cells as a result of toxic exposures during the person's lifetime."⁴ The second group of relevant genetic data includes cellular changes that represent genetic mutations not generally inherited by future generations; however, they can act as unique biomarkers of exposure and can indicate exposure to specific toxic substances.⁵ The way each individual responds to toxic substance exposure may vary as a result of a person's unique

¹ See Gary E. Marchant, *Genetic Data in Toxic Tort Litigation*, 14 J.L. & POL'Y 7, 9 (2006).

² See *id.*

³ See *id.*

⁴ Gary E. Marchant, *Genetics in the Courtroom: Genetics and Toxic Torts*, 31 SETON HALL L. REV. 949, 949–50 (2001).

⁵ See *id.*; Marchant, *supra* note 1, at 18–19 (explaining that biomarkers are genetic changes in an individual that can indicate exposure to a toxic substance: "A biomarker is a molecular change in blood or some other tissue of a person exposed to a toxic substance which can be used to qualitatively or quantitatively evaluate the individual's exposure (biomarker of exposure) or the early pre-symptomatic progression of the disease process (biomarker of effect)."). Marchant explains that there are several types of biomarkers and states that "the most promising types of genetic biomarkers for the future, because of both their potential sensitivity and specificity, are toxicogenomic changes consisting of changes in gene expression, protein concentrations, or metabolite profiles." Marchant, *supra* note 1, at 18.

genetic makeup.⁶ For example, about half of the Caucasian population possesses a genetic variation associated with an increased risk of bladder and lung cancer that could cause those individuals to be more susceptible to toxic substances linked to either bladder or lung cancer.⁷

These types of genetic data can be highly informative. However, the interpretation of such genetic data varies based on a combination of non-genetic factors, such as ethnicity and geographic location; thus variations may have different impacts on different populations.⁸ Therefore, interpretation of genetic data is a complex matter, which must take into account a combination of factors and can have questionable predictive value.⁹ Additionally, jurors typically place high value on scientific evidence, especially DNA evidence.¹⁰ Therefore, an unclear representation of genetic data as conclusive could severely impact a jury's legal analysis and decision in toxic tort litigation.¹¹ As a result of the drastic potential for impact on jury's analysis of causation, it is imperative that the utmost precautions be taken when admitting genetic data in toxic tort litigation.¹² Judges should be required to act as strict gatekeepers of admissibility of genetic information and ensure that juries are provided proper guidelines for the interpretation of such data.¹³

The preliminary judicial assessment of genetic evidence in toxic tort litigation should use a case-by-case analysis in which the probative value of genetic information is to be weighed against its

⁶ See Marchant, *supra* note 1, at 7.

⁷ See Marchant, *supra* note 4, at 951 (“In approximately fifty percent of the Caucasian population, a gene (GSTM1) coding for one in another set of metabolic enzymes (the glutathione S-transferases) is completely deleted, which is associated with an increased risk of bladder and lung cancer from exposure to several toxic substances normally detoxified by the GSTM1 enzyme.”).

⁸ See *id.* at 953.

⁹ See *id.*

¹⁰ See Hon. Donald E. Shelton, *Twenty-First Century Forensic Science Challenges For Trial Judges In Criminal Cases: Where The “Polybutadiene” Meets The “Bitumen,”* 18 WIDENER L.J. 309, 377–78 (2009).

¹¹ See *id.*

¹² See *id.* at 378–81.

¹³ See *id.*

potential harm to both the plaintiff and defendant, as well as its potential to confuse the jury.¹⁴ The Supreme Court has established extensive standards for admissibility of scientific evidence, which are binding on federal judges only.¹⁵ Therefore, this article strongly recommends that state courts adopt the standards set forth by the Supreme Court and the applicable Federal Rules of Evidence necessary to apply those standards. Finally, this article proposes that formal guidelines be promulgated to outline in detail the process by which judicial assessments of admissibility of genetic data in toxic tort litigation should be made. The promulgated guidelines should also clearly describe the appropriate standards of admissibility to be applied and factors to be considered. In order to prevent the premature admission of genetic data in toxic tort litigation, when the interpretation of such data may not be easily applied to the causation analysis, this article proposes that judges follow a universal method for analysis of genetic evidence in toxic tort litigation.

This article proceeds as follows. Part II discusses current and past uses of genetic data in the courtroom, and outlines the potential uses and complications of applying genetic data to the causation analysis in toxic tort litigation. Part III examines potential hurdles in toxic tort litigation for the use of genetic data, specifically the analysis of admissibility and potential privacy concerns. Finally, Part IV proposes a universal method of judicial analysis for admissibility determinations of genetic evidence in toxic tort litigation, in addition to the promulgation of formal guidelines for such admissibility determinations.

II. APPLICATION OF GENETIC TESTS IN TOXIC TORT LITIGATION

Genetic testing data has become a common and useful form of evidence admitted in courtrooms today. For example, there is widespread use of DNA evidence and fingerprinting in criminal courts to link a defendant to a crime scene.¹⁶ Genetic tests are also

¹⁴ *See id.* at 313

¹⁵ *See id.*

¹⁶ *See* Shelton, *supra* note 10, at 310.

frequently compelled in paternity litigation; for example, blood tests are routinely required to determine the identity of the child's father.¹⁷ In addition, courts have also relied on genetic evidence in custody battles involving surrogate mothers.¹⁸ In *Johnson v. Calvert*¹⁹ the court relied on genetic evidence in determining whether custody of an infant should be granted to the genetic parents or the surrogate mother.²⁰ In the examples discussed in criminal, paternity, and custody litigation, the genetic data involved has mostly revolved around genetic evidence establishing identity rather than the genetic susceptibility and predisposition data.²¹ Genetic susceptibility data, on the other hand, is much less frequently admitted into evidence, even in the criminal context where genetic evidence is heavily relied upon.²² While there have been attempts to admit evidence of genetic predisposition data in criminal trials, the majority of those attempts have proven unsuccessful due to insufficient correlations between the genetic

¹⁷ See *People ex rel. Coleman v. Ely*, 390 N.E.2d 140, 141 (Ill. App. Ct. 1979) (compelling blood test to determine paternity, and holding that defendant could be held in contempt for failure to comply); *State v. Shadinger*, 702 So.2d 965, 967 (La. App. 5th Cir. 1997) (ordering paternity blood testing in accordance with a state paternity statute); *Lucas v. Becks*, 52 Va. Cir. 338, 339 (2000) (ordering blood test to establish that defendant was not the father of a child, as the mother alleged).

¹⁸ Dorothy Nelkin, *After Daubert: The Relevance and Reliability of Genetic Information*, 15 CARDOZO L. REV. 2119, 2120 (1994).

¹⁹ 851 P.2d 776 (Cal.1993).

²⁰ *Id.* at 779–82 (ruling in favor of the genetic parents over surrogate mother).

²¹ See Nelkin, *supra* note 18.

²² See Diane Hoffmann & Karen Rothenberg, *Judging Genes: Implications of the Second Generation of Genetic Tests in the Courtroom*, 66 MD. L. REV. 858, 870 (2007); Marchant, *supra* note 1, at 8–9 (explaining how genetic susceptibility data can be utilized to demonstrate an individual's increased or decreased susceptibility to a toxic substance based on their genetic profile: “[t]he genes that code for enzymes involved in the metabolism of foreign substances entering the boy, including pollutants and other toxic substances, appear to be highly variable between individuals. Genetic variations (‘polymorphisms’) that affect susceptibility have been identified for most toxic substances that have received significant regulatory scrutiny.”).

trait and the behavior in question.²³ Genetic predisposition data has, however, been successfully admitted and even compelled in several civil cases.²⁴

In medical malpractice cases, for example, genetic testing has proven useful and has been compelled where the results could show that the physician was not responsible for disabilities of a newborn infant.²⁵ In contrast, the admission of genetic susceptibility and predisposition data as evidence has been limited in the context of toxic tort litigation.²⁶ Family history and expert testimony, however, is often used in toxic tort litigation to establish a genetic predisposition/susceptibility argument.²⁷ For example, in *Wintz v. Northrop Corp.*, the court in a toxic tort case permitted the use of expert testimony to establish that a child's injury was the result of a genetic defect rather than the mother's exposure to bromide during pregnancy.²⁸ Generally in tort cases, the defendant takes the plaintiff as he finds him.²⁹ The potential use of genetic susceptibility/predisposition data, however, could allow defendants in toxic tort cases to argue that due to the plaintiff's high susceptibility or predisposition to a disease that there existed an

²³ See Hoffman & Rothenberg, *supra* note 22, at 871–72 (discussing failed attempts by defense attorneys to use an XYY chromosome defense arguments based on a plaintiff's predisposition to violence).

²⁴ See *id.* at 866–68 (stating that unlike genetic susceptibility data indicative of an individual's increased or decreased susceptibility to a toxic substance, genetic predisposition data could be argued by the defendant to be an alternative cause "i.e., the plaintiff had a genetic predisposition to the disease at issue and likely would have developed the disease independently of the exposure to the toxic substance . . .").

²⁵ See *Harris v. Mercy Hosp.*, 596 N.E.2d 160, 163 (Ill. App. Ct. 1992); Hoffman & Rothenberg, *supra* note 22, at 866.

²⁶ Hoffman & Rothenberg, *supra* note 22, at 868.

²⁷ *Wintz v. Northrop Corp.*, 110 F.3d 508, 510 (7th Cir. 1997); *Kaplowitz v. Borden, Inc.*, 594 N.Y.S. 2d 744 (N.Y. App. Div. 1993) (allowing defendants to compel genetic testing of a child to determine whether mother's in utero exposure to spray paint caused child's illness).

²⁸ *Wintz*, 110 F.3d at 510.

²⁹ See *Benn v. Thomas*, 512 N.W. 2d 537, 539 (1994) (describing the "eggshell plaintiff" rule, which enables the tortfeasor defendant to be liable for all damages even if the plaintiff had an existing condition that might have exacerbated the injuries).

alternate means of causation or a reason to limit damages, such as in the *Wintz* case.³⁰ Plaintiffs alternatively could use genetic data to bolster their cases for causation by demonstrating definitive proof of exposure as well as an increased likelihood of being affected by exposure due to increased genetic susceptibility.³¹ Genetic susceptibility evidence can be utilized in a plaintiff's causation argument by arguing that due to their genetic susceptibility to the alleged toxic substance, their exposure to the substance posed a heightened risk and thus was more likely to cause their health affliction.³²

A. *Application of Genetic Testing Data to Causation*

There are two main areas where genetic testing could provide data potentially relevant to toxic tort litigation: (i) proof of exposure to a toxic substance, and (ii) proof of “genetic susceptibility of individual plaintiffs.”³³ In the first area, genetic data can be helpful in proving exposure to a particular toxic substance through testing for genetic biomarkers.³⁴ The second area utilizes genetic susceptibility data to assist in establishing or disproving specific causation.³⁵ Genetic biomarkers represent molecular changes that occur to cells after exposure to a toxic substance, and indicate whether or not exposure to specific substances has occurred.³⁶ Some biomarkers indicate exposure to a toxic substance generally; however, certain biomarkers can also involve specific mutations in an individual's genes that can be used

³⁰ See Marchant, *supra* note 1, at 8.

³¹ See *id.*

³² 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, 407 (stating “[t]he plaintiffs contend that some children are genetically susceptible to mercury poisoning and cannot excrete or otherwise eliminate the mercury in the vaccine preservative” (citing *Easter v. Aventis Pasteur, Inc.*, 358 F. Supp. 2d. 574, 575 (E.D. Tex. 2005))).

³³ Marchant, *supra* note 1, at 8.

³⁴ *Id.* at 8.

³⁵ *Id.*

³⁶ *Id.* at 19.

to determine exactly which toxic substance caused the mutation.³⁷ Thus, this genetic data could be highly useful for both plaintiffs and defendants in either proving or disproving exposure to the toxic substance, thereby facilitating the causation analysis.³⁸ While biomarkers present a promising tool in toxic tort litigation, this data would be used primarily to establish proof of exposure.³⁹ Even with the assistance of biomarker data, questions remain as to the quantity and effect of the exposure, as well as whether the plaintiff's exposure to the toxic substance was the specific cause of the plaintiff's illness.⁴⁰ Similar exposure to a toxic substance can occur in two different individuals and lead to the development of disease in the first individual while having little to no negative health impact on the second individual.⁴¹ The strongest explanation for this difference resides in differing individual susceptibility to disease, either due to increased susceptibility to the particular toxic substance or a genetic predisposition to the related disease.⁴² Throughout the human population, levels of susceptibility vary as a result of individual "genetic variations," that are variations in the nucleotide sequence of genes.⁴³ The

³⁷ See Marchant, *supra* note 4, at 971–72; Ian C. Semeza & Lisa H. Weasel, *Molecular Epidemiology in Environmental Health: The Potential of Tumor Suppressor Gene p53 as a Biomarker*, 105 ENVTL. HEALTH PERSP. 155, 155–56 (1997); Steven J. Smith et al., *Molecular Epidemiology of p53 Protein Mutations in Workers Exposed to Vinyl Chloride*, 147 AM. J. EPIDEMIOLOGY 302, 302–03 (1998).

³⁸ See Marchant, *supra* note 1, at 20.

³⁹ See *id.* at 18–20.

⁴⁰ See *id.*

⁴¹ See Susan R. Poulter, *Genetic Testing in Toxic Injury Litigation: The Path to Scientific Certainty or Blind Alley?*, 41 JURIMETRICS J. 211, 214 (2001).

⁴² See *id.* at 214–15.

⁴³ See *id.* at 214 ("Genetic variation among individuals is characterized as either a mutation, when less than 1% of the population carries a particular form of the gene, or as a genetic polymorphism, when the less common form occurs at greater than 1%."); Gold, *supra* note 32, at 384 (noting that a variation in a single nucleotide base can cause disease or affect likelihood of disease development: "A gene is a segment of DNA, found at a particular location ('locus') on a chromosome, which codes for a particular sequence of amino acids as determined by the arrangement of the four nucleotide bases of which DNA is made.").

likelihood that a genetic variation will actually result in a disease is referred to as penetrance.⁴⁴

1. *Genetic Data and the Plaintiff's Burden of Causation*

Genetic data can be best used to bolster the plaintiff's case in a toxic tort claim in two ways. First, the plaintiff can provide genetic evidence, through the presence of biomarkers, of exposure to the toxic substance in question. This evidence assists the plaintiff in establishing the first aspect of causation which requires actual exposure. However, the plaintiff must also prove specific causation, thus demonstrating that the plaintiff's exposure to the toxin at issue was more likely than not the cause of the plaintiff's illness.⁴⁵ In the case of many toxic substances, exposure increases the risk of developing a particular illness, but not by the greater than fifty percent standard typically required of the plaintiff's burden of proof by a preponderance of evidence.⁴⁶

Second, the use of genetic susceptibility evidence could be used to support the plaintiff's causation analysis by ruling out other potential causes.⁴⁷ For example, plaintiffs could take a genetic test, which shows they are not genetically predisposed to the disease at issue, in order to strengthen their argument that it was the exposure to the toxic substance that more likely than not doubled their risk of developing the disease and caused their illness.⁴⁸ The use of genetic susceptibility data has been successfully used to strengthen a plaintiff's causation analysis in toxic tort claims through expert genetic testimony to dispute genetic predisposition and point to increased causation as a result of exposure.⁴⁹ An example of such

⁴⁴ See Poulter, *supra* note 41, at 214.

⁴⁵ See *id.* at 217 (stating that the plaintiff must prove causation by a preponderance of evidence, thus proving that the exposure to the toxic substance must have at least doubled the plaintiff's risk of developing the illness at issue with an increased likelihood of greater than 50% resulting from exposure).

⁴⁶ See Marchant, *supra* note 4, at 954; Frederica P. Perera, *Environment and Cancer Who Are Susceptible?*, 278 *SCIENCE* 1068, 1072 (1997) ("In epidemiology, it has been difficult to detect relative risks of 1.5 or even 2.0.").

⁴⁷ See Poulter, *supra* note 41, at 218.

⁴⁸ See *id.*

⁴⁹ See *Landrigan v. Celotex Corp.*, 605 A.2d 1079, 1082 (N.J. Supp. Ct. 1992).

a use can be seen in *Landrigan v. Celotex Corp.*,⁵⁰ where the judge permitted a plaintiff's expert testimony regarding the absence of colon cancer in the plaintiff's family history in order to strengthen the plaintiff's causation analysis.⁵¹ Additionally, plaintiffs could utilize genetic tests which could indicate a possible increased likelihood of being effected by a toxin due to their increased susceptibility to said toxin.⁵²

While genetic susceptibility evidence has been admitted to assist in plaintiffs' causation analysis, this approach has thus far mostly failed.⁵³ The genetic susceptibility argument in support of causation has not failed on its merits, but rather it has failed due to a lack of properly developed legal arguments.⁵⁴ For example, in *Hall v. Baxter Healthcare Corp.*,⁵⁵ the court rejected expert testimony of genetic susceptibility to silicone (from silicone breast implants).⁵⁶ In *Hall*, the plaintiff's susceptibility argument was poorly developed because the research presented to the court was not peer reviewed and the plaintiffs offered no evidence that plaintiffs carried any such gene conferring an increased genetic susceptibility to silicone.⁵⁷ Despite the lack of success that plaintiffs in toxic tort litigation have had in using genetic susceptibility to support causation, it still remains clear that there is a strong potential for this strategy to be a useful tool for plaintiffs.⁵⁸

Another case that illustrates the potential benefits to plaintiffs of genetic susceptibility data is *Easter v. Aventis Pasteur, Inc.*⁵⁹ Although the court in *Easter* refused to admit testimony that some children have genetic susceptibility to the preservative in the defendant's vaccine, the expert testimony was excluded on the basis that the genetic data proved that the plaintiff did *not* have a

⁵⁰ *Id.*

⁵¹ *Id.*

⁵² See Marchant, *supra* note 1, at 8.

⁵³ See *id.* at 11.

⁵⁴ See *id.*

⁵⁵ 947 F. Supp. 1387 (D. Or. 1996).

⁵⁶ *Id.* at 1456.

⁵⁷ See *id.*

⁵⁸ See Marchant, *supra* note 1, at 12.

⁵⁹ 358 F. Supp. 2d 574, 576 (E.D. Tex. 2005).

genetic susceptibility to the preservative.⁶⁰ Thus, the *Easter* decision suggests that the genetic susceptibility evidence may have been admitted to support causation had the plaintiff been able to show not only that susceptibility to the toxic substance was a possibility, but also that the plaintiff had a genetic variation that conferred such susceptibility.⁶¹

Thus, the application of genetic data in toxic tort litigation holds substantial potential to offer additional evidence supporting plaintiff's burden of proof of causation by establishing exposure or increased susceptibility to the toxic substance at issue, or both.⁶² Whether genetic data is admitted to bolster the plaintiff's causation argument or to support the defendant's argument against causation, caution must be used in determining such admissibility.⁶³ For example, while the presence of a biomarker can indicate proof of exposure, Steve Gold contends "to require its presence to support a causation inference is to assume that the absence of the biomarker precludes causation."⁶⁴ Gold further explains the dangers of requiring the presence of a biomarker and highlights two main issues.⁶⁵ First, such a requirement is only effective if "no causal pathway exists between a chemical and a disease other than the pathway that produces the biomarker."⁶⁶ Second, if no other pathway exists, "the science seems to be pointing at a world of numerous risk factors, some genetic and some environmental;"

⁶⁰ *Id.* at 575 ("The plaintiffs have conceded that they cannot prove, in Jordan's case, that his autism was caused by thimerosal. This is because Jordan does not meet the genetic profile for children who, according to the plaintiffs, are at an increased risk for developing autism caused by thimerosal in pediatric vaccines. Because the plaintiffs have conceded they cannot prove that Jordan's autism was caused by thimerosal, they seek to recover on a claim that several co-morbid conditions suffered by Jordan were instead caused by, or contributed to by, Jordan's exposure to the thimerosal contained in the pediatric vaccines.").

⁶¹ *See id.*

⁶² *See id.*; see Marchant, *supra* note 1, at 7.

⁶³ *See* Gold, *supra* note 32, at 403–05.

⁶⁴ *Id.* at 405.

⁶⁵ *See id.*

⁶⁶ *Id.* (emphasis omitted).

thus such evidence as biomarker and genetic susceptibility should not be the sole factors to be considered in causation analysis.⁶⁷

2. *Genetic Data and the Defendant's Argument Against Causation*

As discussed above, there are significant potential benefits of genetic data to the plaintiff's case for causation. However, genetic data could also be employed to support the defendant's argument against causation. Defendants may choose to use genetic susceptibility data to argue an alternative causation other than exposure to the toxic substance.⁶⁸ In fact, defendants have already been successful in requesting genetic information for the purpose of arguing alternative causation.⁶⁹ For example, it is common for defendants to seek information regarding a plaintiff's medical history and family history of disease to argue that genetic susceptibility was the cause of plaintiff's illness rather than exposure to a toxic substance.⁷⁰ In addition to expert testimony regarding family history, the influence of environmental factors, and other potential alternative causes, defendants have also used defenses based on alternative causation using genetic test results for susceptibility.⁷¹ However, thus far the defense of alternative causation due to susceptibility has been mostly unsuccessful due to a lack of credible testimony, a lack of proof that the plaintiff is affected by the particular variation conferring susceptibility or both.⁷² Some courts, on the other hand, have shown a willingness to go as far as to heavily rely on genetic evidence or lack thereof, with regards to proof of causation.⁷³ For

⁶⁷ *Id.* at 421.

⁶⁸ *See* Marchant, *supra* note 1, at 12.

⁶⁹ Poulter, *supra* note 41, at 218.

⁷⁰ *Id.*

⁷¹ *See* Marchant, *supra* note 1, at 12–13.

⁷² *See, e.g.,* Willey v. Ketterer, 869 F.2d 648 (1st Cir. 1989) (finding a lack of evidence of genetic predisposition to cerebral palsy); Dombrowski v. Gould Elecs., 85 F. Supp. 2d 456, 477 (M.D. Pa. 2000) (finding a lack of valid testimony showing alternative causation due to family history or environmental factors).

⁷³ Harris v. Mercy Hosp., 596 N.E.2d 160, 161 (Ill. App. Ct. 1992); *see also* Cord v. City of Los Angeles, No. B167756, 2004 Cal. App. LEXIS 8967, at *2 (Cal App. 2d Dist. Sept. 30, 2004) (excluding plaintiffs' medical expert's

example, in *Cord v. City of Los Angeles*⁷⁴, the defendant argued that where the plaintiff alleged exposure to benzene and other chemicals caused her lymphoma, the plaintiff failed to test for biomarkers and argued that biomarkers are “necessary to prove exposure, absorption and toxicity”⁷⁵ The defense expert in *Cord* further argued that the “absence of biomarkers makes it impossible to establish causation” and the court thereafter found that the plaintiff presented insufficient evidence to establish causation.⁷⁶

While defendants may argue, as did the defendants in *Cord*, that the absence of biomarker evidence precludes plaintiffs from establishing causation, the District Court of New Jersey in *Quickel v. Lorillard, Inc.*,⁷⁷ held to the contrary.⁷⁸ In *Quickel*, the plaintiffs argued that exposure to asbestos from the defendants’ cigarette filters caused the mesothelioma suffered by the plaintiff.⁷⁹ However, the defendants in *Quickel* argued that the absence of an autopsy obtaining biomarkers of asbestos related exposure from the plaintiff’s lung tissue demonstrated a lack of proof of causation.⁸⁰ Contrary to the holding in *Cord*, the court in *Quickel* held that “the fact that no study was done at autopsy also does not lead to the conclusion that such fibers were absent.”⁸¹ The *Quickel* court reasoned that “plaintiffs need not prove beyond a reasonable doubt that particles of asbestos were ingested from defendants’ cigarettes and lodged in Mr. Quickel’s lungs causing his mesothelioma; they must prove causation to a jury by preponderance of the evidence.” Thus, the court in *Quickel* did not consider the lack of biomarker evidence as dispositive, rather the court stated that “the plaintiff does not have the burden, through expert testimony, to rule out all other possible causes of Mr.

testimony because there was no statistically significant link between exposure to the toxin and developing lymphoma).

⁷⁴ See *Cord*, 2004 Cal. App. LEXIS 8967, at *2.

⁷⁵ See *id.* at *5.

⁷⁶ *Id.* at *6.

⁷⁷ No. CIV.A.95-5255, 1999 U.S. Dist. LEXIS 23453 (D.N.J. Mar. 31, 1999).

⁷⁸ See *id.* at *19; *Cord*, 2004 Cal. App. LEXIS 8967, at *2.

⁷⁹ *Quickel* at *1.

⁸⁰ *Id.* at *18–19.

⁸¹ *Id.* at *21.

Quickel's mesothelioma. Indeed, mesothelioma is a rare disease, and its diagnosis (which is undisputed in this case) is associated with an overwhelming 80% of all such events with exposure to crocidolite asbestos beyond background levels.⁸²

While the admission of genetic susceptibility data as part of the defendant's case has been implemented in toxic tort litigation, it is not commonplace.⁸³ However, such evidence has been successfully utilized in medical malpractice cases, especially those involving pregnancy and newborns.⁸⁴ For example, in *Harris v. Mercy Hospital*,⁸⁵ the court allowed the defendants to compel the plaintiff to submit to a blood test in order to determine whether a genetic anomaly was responsible for the plaintiff's condition as an alternative defense to a medical malpractice claim.⁸⁶ The court in *Harris* reasoned that the probative value of the evidence outweighed the potential risk to the plaintiff, focusing on the minimal intrusiveness and the routine nature of the actual blood test compelled.⁸⁷

In cases such as *Harris*, genetic susceptibility data has the ability to quantitatively demonstrate alternative causation by showing a genetic variation that is accepted by the scientific community to be practically synonymous with the afflicted disease.⁸⁸ Genetic variations shown to be highly penetrant (such as the variation in *Harris*) confer a significantly increased susceptibility to the disease associated with that particular variation, whereas less penetrant variations may have little to no

⁸² *Id.*

⁸³ *See, e.g.,* Marchant, *supra* note 1.

⁸⁴ *See* Hoffmann & Rothenberg, *supra* note 22, at 866.

⁸⁵ 596 N.E.2d 160 (Ill. App. 1st Dist. 1992).

⁸⁶ *Id.* at 162.

⁸⁷ *Id.* at 163 (reasoning that where the plaintiff puts his physical condition at issue, pursuant to Illinois Supreme Court Rule 215, the Court has discretion to order plaintiff to submit to a blood test). The court further held that “[b]lood tests are routine procedures in our everyday life” and that the plaintiff presented “no competent evidence that the drawing of blood presents an unreasonable risk to Jennifer [plaintiff]” *Id.* (citing *Breithaupt v. Abram*, 352 U.S. 432, 436 (1957)).

⁸⁸ *See* Hoffmann & Rothenberg, *supra* note 22, at 896–97.

impact on one's health.⁸⁹ In the case of highly penetrant variations, such as Huntington's disease, the variation has been shown to correlate almost one hundred percent with development of the disease, which makes the possibility of causation resulting from exposure highly unlikely.⁹⁰ Therefore, in cases involving highly penetrant susceptibility genes, admission of such data will be extremely probative in a defense of alternative causation by the defendant and there is a very strong argument for their admission.⁹¹

Defendants could alternatively employ genetic data to attempt to disprove plaintiff's exposure to the toxic substance at issue through the absence of a biomarker or signature mutation scientifically linked to such exposure.⁹² For example, in *Tompkin v. Philip Morris USA, Inc.*,⁹³ the defendant argued alternative causation where the plaintiff alleged lung cancer resulting from cigarette smoking and also worked with asbestos.⁹⁴ An expert for the defense in *Tompkin* testified that the plaintiff decedent's lung tissue lacked mutations in the P53 and K-Ras genes known to be caused by smoking and further testified that the plaintiff's lung cancer "most likely resulted from his occupational exposure to silicates and asbestos"⁹⁵ In *Tompkin*, an expert witness for the plaintiff testified that Tompkin's "cancer was due to the combined effect of his cigarette smoking and his exposure to asbestos."⁹⁶ The expert's assertion was based on the reasoning that "asbestos interacts with cigarette smoking by a process we call synergy whereby they have an affect which is beyond an additive effect of each of their potencies."⁹⁷ Even in light of these highly conflicting expert testimonies, the jury in *Tompkin* found that the plaintiff's

⁸⁹ Poulter, *supra* note 41, at 214 ("One type [of genetic variation] involves anomalies in a single gene that are highly likely, sometimes virtually certain, to result in disease or abnormality."). For example, "mutations in [the] BRCA1 and BRCA2 . . . confer[s] a more than 50% lifetime risk of some cancers." *Id.*

⁹⁰ *Id.* at 214.

⁹¹ *See id.* at 231.

⁹² *See* Gold, *supra* note 32, at 402–05.

⁹³ 362 F. 3d 882 (2004).

⁹⁴ *See id.* at 890.

⁹⁵ *Id.*

⁹⁶ *Id.* at 888.

⁹⁷ *Id.*

cancer was caused by his asbestos exposure, clearly giving extreme deference to the absence of biomarkers.⁹⁸

B. *Problems with Genetic Data and Causation*

The application of genetic data to the causation analysis is complicated in several respects. First, the presence or absence of biomarkers must not be viewed as determinative, as Gold explains biomarkers “could of course be relevant to an alleged causal relationship. Relatively rarely, however, is it likely to be absolutely conclusive”.⁹⁹ Second, the interpretation of genetic data can become blurred by cultural assumptions and varying biological arguments such as biological determinism (or genetic determinism).¹⁰⁰

An additional complicating factor is that the nature of genetic susceptibility data is highly complex, which leads to difficulties in interpreting and explaining the implications of such data.¹⁰¹ For example, in *Milward v. Acuity Specialty Product Group, Inc.*,¹⁰² the U.S. Court of Appeals reversed a district court ruling precluding plaintiff’s expert testimony, holding that the expert’s testimony “may give rise to a plausible hypothesis, but not a reliable inference” based on the reasoning that exposure to benzene had not been scientifically linked to the specific disease suffered by the plaintiff.¹⁰³ The First Circuit in *Milward* held that the district court exceeded the scope of its discretion and “misunderstood” the expert testimony regarding weighing the factors which he

⁹⁸ See *id.* at 886.

⁹⁹ See Gold, *supra* note 32, at 406.

¹⁰⁰ Nelkin, *supra* note 18, at 2125 (explaining the concept of biological determinism in society, stating that “[a]s research extends our understanding of the human genome, the media increasingly convey the idea that personhood, behavior, and human destiny can be defined in terms of DNA”). Appropriating genetic concepts, the media use the gene to explain individual differences (“the genes of genius”), race and gender stereotypes (“differences lie in the genes”), family relationships (“the importance of blood ties”), and behavioral problems (“good and bad genes”). *Id.*

¹⁰¹ See *Tompkin v. Philip Morris USA, Inc.*, 362 F. 3d 882, 886 (6th Cir. 2004).

¹⁰² 639 F.3d 11 (1st Cir. 2011).

¹⁰³ See *id.* at 25.

concluded “supported the inference that the association between benzene exposure and APL is genuine and causal.”¹⁰⁴ The *Milward* case highlights the difficulty of interpreting genetic data and its application and admissibility as evidence with regards to establishing causation.¹⁰⁵

1. *The Complex Nature of Genetic Susceptibility Data*

The admissibility and function of genetic susceptibility data become increasingly questionable when the genes involved are less penetrant or have completely unknown penetrance.¹⁰⁶ When the genetic variation at issue involves less penetrant disease susceptibility genes, the evidentiary relationship between the susceptibility and the exposure becomes more difficult to interpret.¹⁰⁷ In this case the genetic susceptibility may be identified as one factor affecting the plaintiff’s likelihood of acquiring the disease.¹⁰⁸ There may be many additional factors, however, which could have an equal or greater impact on the plaintiff’s likelihood of acquiring the disease, such as the exposure to a toxic substance.¹⁰⁹ As a result, when dealing with less penetrant genes, identifying a quantifiable value to place on each factor affecting risk becomes highly complex.

In addition to determining the individual impact of each factor, the scenario becomes increasingly complex as the interactions between multiple factors are taken into account. For example, two factors may combine to react “additively, synergistically, or antagonistically.”¹¹⁰ In other words, when multiple factors are

¹⁰⁴ *See id.* at 26.

¹⁰⁵ *See generally id.*

¹⁰⁶ *See Poulter, supra* note 41, at 232–33.

¹⁰⁷ *Id.*

¹⁰⁸ *See id.* (noting that in addition to genetic susceptibility and exposure to the toxic substance at issue, additional factors can affect plaintiff’s likelihood of acquiring the disease, including environmental factors such as nutrition and lifestyle).

¹⁰⁹ *See id.* at 224.

¹¹⁰ *Id.* at 227–28. An example of a synergistic effect of factors would be smoking cigarettes and exposure to asbestos, which together have a multiplicative effect on risk of lung cancer:

Roughly speaking, significant occupational exposure to asbestos increases the rate of lung cancer by a factor of 5, while a significant

present, such as a genetic predisposition and exposure to a toxic substance, those factors may relate to each other in several different ways yielding several different cumulative effects.¹¹¹ Depending on whether the factors interact in an additive or synergistic manner, and depending on how great the synergistic effect is, the analysis of an alternative causation argument can change drastically.¹¹² It is generally thought that genetic variations acting to predispose individuals to a disease act in combination with exposure to a substance.¹¹³ However, in order to fully understand the impact of each factor there must be clear evidence as to how the factors interact.¹¹⁴ This is yet another complication in assigning a specific predictive weight to a genetic variant that is not highly penetrant. Currently, available genetic tests are capable of determining increased susceptibility to a number of diseases, including breast cancer and Huntington's disease.¹¹⁵ The interpretation of genetic tests for disease susceptibility, however, is not simple and leaves much room for debate as to what value a jury should assign to the predictability of their results.¹¹⁶ In addition to determining how individual factors interact, other separate factors may also complicate the analysis of genetic

smoking history increases the rate of lung cancer by a factor of at least 10. Thus, for a nonsmoking asbestos worker with lung cancer, the likelihood of causation by asbestos exposure is 80% (4/5), and for the smoker, the likelihood that smoking caused the lung cancer is 90% (9/10).” Among smoking asbestos workers, however, the rate of lung cancer is increased by a factor of 50 over background ... rather than the factor of 14 expected by adding the separate effects of asbestos exposure and smoking.

Id.

¹¹¹ *See id.* at 224–28. The combined effect of multiple factors could be additive, in that if both factors have an individual 10 percent increased risk, they will combine additively to have a total increased risk of 20 percent (10+10). *Id.* Alternatively, the same factors could interact synergistically, combining in a multiplicative nature to result in a total risk of 100 percent (10x10), or antagonistically resulting in a lesser-combined effect than either additive or synergistic interactions). *Id.*

¹¹² *See Poulter, supra* note 41, at 229.

¹¹³ *See id.* at 229–30.

¹¹⁴ *See id.*

¹¹⁵ *Id.* at 216.

¹¹⁶ *See id.*

variations.¹¹⁷ For example, the same genetic variation can have a different impact on susceptibility within different populations and ethnic groups.¹¹⁸ Such additional variables make any interpretation more difficult and any conclusion more tenuous.

In addition to the differing impact of genetic variations among different populations or individuals, interactions between genetic variations and toxic substances as well as the additional component of environmental factors combine for a very complex scientific and legal analysis.¹¹⁹ As a result, the ability of genetic susceptibility data to have predictive value with many less penetrant genes is questionable, which therefore leads to questions of accuracy, admissibility, and interpretation in the courtroom.¹²⁰ This article will further explore what effects the accuracy of genetic data combined with the ease of interpretation by the jury have on admissibility.

2. *Impact of Cultural Assumptions and Biological Arguments*

In assessing the implications of new scientific research, such as genetic susceptibility testing, it is important to remember that even scientific developments are the result of human activity and remain “subject to people’s assumptions, preconceptions, and biases.”¹²¹ For example, the way scientific research is structured and analyzed can be influenced by an individual’s race or gender stereotypes.¹²² Additionally, the press plays a role in the way the public perceives and interprets scientific research and can affect the way genetic data will be understood by judges and jurors in the courtroom.¹²³ The concept of biological or genetic determinism, for example, is a popular genetic concept conveyed to the public.¹²⁴ Biological determinism suggests that a person’s characteristics are predictable and genetic traits are “hard-wired” into the “human constitution”

¹¹⁷ *Id.*

¹¹⁸ See Poulter, *supra* note 41, at 216.

¹¹⁹ See *id.* at 216–18.

¹²⁰ See *id.*

¹²¹ Jennifer Wriggins, *Genetics, IQ, Determinisms, and Torts: The Example of Discovery in Lead Exposure Litigation*, 77 B.U.L. REV. 1025, 1068 (1997).

¹²² See Nelkin, *supra* note 18, at 2125.

¹²³ See *id.*

¹²⁴ *Id.*

and are therefore predetermined.¹²⁵ Alternatively, the concept of maternal determinism suggests that a person's characteristics are determined as a result of the mothering they receive.¹²⁶

Applying concepts such as biological and maternal determinism in the courtroom could lead to further complications and bias in understanding the application of genetic data to the causation analysis.¹²⁷ For example, in *Andon v. 302-304 Mott St. Assocs.*,¹²⁸ the defendants in a lead exposure case argued that the infant plaintiff's mental deficit was not the result of exposure to lead, but rather was genetically inherited from the infant's mother.¹²⁹ In *Andon*, the court reversed an order compelling the plaintiff's mother to submit to an IQ test in support of the defendant's alternative causation argument, holding that such an inquiry would only raise more questions and not assist in the determination of causation.¹³⁰ In toxic tort cases, such as *Andon*, it is crucial that requests for genetic testing, such as the IQ test at issue in *Andon*, be limited in order to assure that only evidence that will not further complicate the causation analysis is admitted.¹³¹ The type and quality of mothering an individual receives, the genes an individual possesses, and the environment in which an individual is raised are all factors affecting the characteristics of that individual.¹³² However, no one factor alone is typically deterministic.¹³³ The consideration of these genetic concepts further adds to the complexity of the interpretation of genetic data in the causation analysis and must be carefully censored in order to

¹²⁵ *Id.* (“The assumption pervading popular culture is that genetic traits are hard-wired and immutable, a powerful force in shaping social behavior, and a predictable part of the human constitution.”).

¹²⁶ Wriggins, *supra* note 121, at 1042.

¹²⁷ *See id.*

¹²⁸ 257 A.D.2d 37 (N.Y. App. Div. 1st Dep’t 1999).

¹²⁹ *Id.* at 41.

¹³⁰ *Id.* (stating that the admission of so such maternal IQ data would result in “turning the fact-finding process into a series of mini-trials regarding, at a minimum, the factors contributing to the mother’s IQ, and possibly, that of other family members”).

¹³¹ *See id.*

¹³² *See* Wriggins, *supra* note 121, at 1042–44.

¹³³ *See id.*

prevent the admission of evidence that will further confuse a jury.¹³⁴

III. POTENTIAL HURDLES TO USE IN THE COURTROOM

This section will discuss two primary hurdles to the acceptance of the use of genetic susceptibility data in the courtroom, admissibility and privacy. The analysis of genetic predisposition data can become extremely complex, especially when less penetrant genes are at issue.¹³⁵ The complex nature of this analysis results in decreased accuracy of predictive value as well as possible confusion of jurors.¹³⁶ In addition, the very essence of the genetic data at issue in many toxic tort claims is of a highly personal nature and raises several privacy concerns.¹³⁷

A. Admissibility Standards for Genetic Data

The current standard applicable to the admissibility of genetic testing data has been recently detailed by the United States Supreme Court in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*,¹³⁸ and expanded upon by the Supreme Court in *General Electric v. Joiner*¹³⁹ and *Kumho Tire Co. v. Carmichael*.¹⁴⁰ The Supreme Court in *Daubert* explicitly rejected the previous test for admissibility of genetic data, known as the Frye Test.¹⁴¹ Under the Frye Test, judges were generally deferential to scientific experts, and scientific evidence was generally admissible if it had acquired “general acceptance” in the scientific community.¹⁴² The standard declared in *Daubert* requires federal judges to make a preliminary assessment of scientific testimony by assessing the validity of the

¹³⁴ See generally Nelkin, *supra* note 18, at 2125 (“The presence of a biological condition should not be confused with a specific behavioral trait or even a disease. The gene is not a completely deterministic force, independent of history or environment.”).

¹³⁵ See Poulter, *supra* note 41, at 214.

¹³⁶ See *id.*

¹³⁷ Marchant, *supra* note 1, at 35.

¹³⁸ 509 U.S. 579 (1993).

¹³⁹ 522 U.S. 136 (1997).

¹⁴⁰ 526 U.S. 137 (1998).

¹⁴¹ Hoffmann & Rothenberg, *supra* note 22, at 895.

¹⁴² Frye v. United States, 293 F. 1013, 1014 (D.C. Cir. 1923).

underlying reasoning and methodology and determining whether the scientific knowledge can be properly applied to the facts at issue in order to assist the trier of fact's determination.¹⁴³

The *Daubert* Court placed significant emphasis on the standard of admissibility established by Rule 403 of the Federal Rules of Evidence.¹⁴⁴ According to Rule 403, "evidence may be excluded if its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury, or by considerations of undue delay, waste of time, or needless presentation of cumulative evidence."¹⁴⁵ The majority of respectable, emerging genetic research and tests will likely pass the "scientific acceptance" prong of *Daubert* provided they use the correct scientific method and are published in a peer-reviewed journal.¹⁴⁶ While courts have not been most concerned with the second prong of *Daubert* (relevant to the facts at issue), it seems highly plausible that the complicated analysis of multiple-factor genetic susceptibility data may further confuse jurors rather than assist in their understanding of the causation analysis.¹⁴⁷

The Supreme Court in *Joiner* interpreted *Daubert* and dealt with the question of whether the evidence presented is too far removed or speculative to determine whether exposure was indeed the specific cause of the plaintiff's disease.¹⁴⁸ The Supreme Court held that "a court may conclude that there is simply too great an analytical gap between the data and the opinion proffered."¹⁴⁹ The reasoning of *Joiner* seems to be aimed directly towards the potentially complex situation discussed previously, dealing with less penetrant disease susceptibility genes.¹⁵⁰ Finally, in *Kumho*, the Supreme Court upheld and expanded upon *Daubert* by holding that the *Daubert* standard applies to all experts, not merely

¹⁴³ *Daubert*, 509 U.S. at 589.

¹⁴⁴ *See id.*

¹⁴⁵ FED. R. EVID. 403.

¹⁴⁶ G. Mark Whitehead, *The Use and Abuse of Genetic Testing: Toxicogenomics in the Courtroom*, 17 TOXICS L. REP. (BNA) 963 (2002).

¹⁴⁷ *See id.*; Poulter, *supra* note 41, at 220.

¹⁴⁸ *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 140 (1997).

¹⁴⁹ *Id.* at 146.

¹⁵⁰ *See id.*; Whitehead, *supra* note 146, at 963.

scientific experts.¹⁵¹ Furthermore, the Supreme Court's holding in *Kumho* upheld and strengthened *Joiner*.¹⁵² The Court in *Kumho* held that the evidence at issue was too unreliable to present to a jury and, therefore, inadmissible despite being based on accepted methodology, thus reiterating the analytical gap aspect of *Joiner*.¹⁵³ The combined effect of *Daubert*, *Joiner*, and *Kumho* is to encourage both plaintiffs and defendants to bring forth genetic data evidence which appears to be reliable and not too attenuated from causation.¹⁵⁴ Thus, the most likely result in future toxic tort cases involving genetic susceptibility data is that the more penetrant a gene is and the fewer additional factors which need to come into play, the more likely the evidence will be ruled admissible.¹⁵⁵ Conversely, less penetrant disease-causing genetic data will be less likely to be admissible as too many factors will need to be taken into consideration, thereby increasing the speculative nature of the evidence and decreasing its reliability.¹⁵⁶

B. *Privacy Concerns Regarding Genetic Data*

Genetic information is of a most personal nature and, as a result, is highly sensitive.¹⁵⁷ DNA and genomic data, unlike a fingerprint, yield much more intimate information about a person than just an identity to match to a name.¹⁵⁸ A person's genetic profile details who they are, what they inherited from past generations, and provides insight into what future illnesses may afflict a person.¹⁵⁹ A person's genetic profile may be even too personal and revealing for that individual to handle and, thus, the plaintiff themselves may prefer not to know the results.¹⁶⁰ For example, certain genetic predispositions, such as the predisposition for Huntington's disease, are so strong as to virtually guarantee

¹⁵¹ *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 148 (1998).

¹⁵² Shelton, *supra* note 10, at 316–17.

¹⁵³ See *Kumho*, 526 U.S. at 148; Shelton, *supra* note 10, at 316–17.

¹⁵⁴ See Shelton, *supra* note 10, at 316–18.

¹⁵⁵ See *id.*; Poulter, *supra* note 41, 214–15.

¹⁵⁶ See Shelton, *supra* note 10, at 316–18; see Poulter, *supra* note 41, 214–15.

¹⁵⁷ Marchant, *supra* note 1, at 35.

¹⁵⁸ See *id.*

¹⁵⁹ See *id.*

¹⁶⁰ See *id.*

later onset of a disease.¹⁶¹ For some people, as the American proverb suggests, “ignorance is bliss.”¹⁶²

In the case of genetic information, especially information with a negative implication, some individuals may prefer not to know if they are afflicted, or are likely to be afflicted by a devastating illness.¹⁶³ In addition to an individual’s desire not to choose to explore their own genetic profile, many individuals do not want other people, such as family members and employers, let alone strangers, to gain access to such highly personal information.¹⁶⁴ Despite the prevalent privacy concerns, the toxic tort plaintiff has placed their health at issue, and thus it seems reasonable for courts to request relevant and probative genetic data.¹⁶⁵ Provided that proper safeguards are in place to protect unnecessary access to genetic information, it is permissible for courts to use, and even compel, genetic information.¹⁶⁶ Every individual has an interest in his or her privacy. Yet when a plaintiff places his or her health at issue and the probative value of genetic information outweighs the harm and does not pose an unreasonable risk to the plaintiff, courts have reasonable discretion to compel submission to relevant and minimally invasive genetic testing.¹⁶⁷ Therefore, privacy issues exist when defendants seek to admit evidence, especially evidence that is irrelevant or substantially harmful to plaintiffs, rather than when plaintiffs seek to admit genetic evidence.¹⁶⁸

Given the highly personal and sensitive nature of genetic data, there is a strong need to take precautions to prevent others from gaining access to such information.¹⁶⁹ However, genetic

¹⁶¹ Poulter, *supra* note 41, at 214.

¹⁶² Thomas Gray, *An Ode on a Distant Prospect of Eton College*, UNIVERSITY OF OXFORD <http://www.thomasgray.org/cgi-bin/display.cgi?text=odec> (last visited Dec. 16, 2011).

¹⁶³ *See* Marchant, *supra* note 1, at 35.

¹⁶⁴ *See id.*

¹⁶⁵ *See id.*

¹⁶⁶ *See id.*

¹⁶⁷ *See id.*; Harris v. Mercy Hosp., 596 N.E.2d 160, 161 (Ill. App. Ct. 1992).

¹⁶⁸ *See* Marchant, *supra* note 1, at 35.

¹⁶⁹ Ronald M. Green & A. Mathew Thomas, *DNA: Five Distinguishing Features for Policy Analysis*, 11 Harv. J.L. & Tech. 571, 572–74 (1998) (discussing “informational risks” of genetic information).

information has the potential to be extremely probative to both plaintiffs and defendants.¹⁷⁰ As a result, questions arise as to whether or not judges should be permitted to compel genetic testing of plaintiffs in toxic tort litigation. The answer is two-fold. First, courts have permitted compulsion of genetic tests where the potential probative value outweighs the risk of harm to the plaintiff.¹⁷¹ The reasoning here is that in toxic tort litigation, the plaintiff places their health at issue and if a court is to allow plaintiffs to admit genetic evidence in support of causation, then it would only be fair to allow defendants to use genetic information to argue modes of alternative causation.¹⁷² Second, judges should consider the reasonableness of the request, such as routine and minimally invasive blood samples, which courts typically allow.¹⁷³

IV. POLICY PROPOSALS

This article proposes a method of judicial analysis for admissibility determinations of genetic evidence in toxic tort litigation allowing broad discretion to judges, in addition to the promulgation of formal guidelines for such admissibility determinations. Genetic data has already been utilized in toxic tort litigation and possesses significant potential for future use in this field.¹⁷⁴ Emerging genetic technology may be able to support both the plaintiff's ability to prove causation and the defendant's ability to disprove causation.¹⁷⁵ However the complex nature of genetic data as highlighted herein suggests the importance of clear guidelines for judges faced with admissibility determinations of genetic evidence in toxic tort litigation.¹⁷⁶

A. *Method of Judicial Analysis of Genetic Data*

The foremost consideration of judges confronted with admissibility questions regarding genetic data is to analyze the

¹⁷⁰ See Marchant, *supra* note 1, at 10–14.

¹⁷¹ Kaplowitz v. Borden, Inc., 594 N.Y.S.2d 744, 744 (N.Y. App. Div. 1993).

¹⁷² See Marchant, *supra* note 1, at 35–36.

¹⁷³ See *supra* p. 5 and note 17.

¹⁷⁴ Hoffmann & Rothenberg, *supra* note 22, at 868.

¹⁷⁵ See *id.* at 867–68; see also Poulter, *supra* note 41, at 214.

¹⁷⁶ See Shelton, *supra* note 10, at 310.

information in their “gatekeeper” role under the framework established by the Supreme Court in *Daubert*, *Joiner*, and *Kumho*.¹⁷⁷ While *Daubert*, *Joiner*, and *Kumho* apply to federal courts, this article proposes that state court judges should follow this federal standard in order to ensure universal application of genetic data in toxic tort litigation. Thus, judges should consider the following: (i) whether the data is scientifically valid and generally accepted in the scientific community; (ii) whether the data will assist the trier of fact in their determination of causation; and (iii) whether the gap between the data proffered and the conclusion offered is too attenuated or speculative.¹⁷⁸

The judge’s inquiries, however, should not cease after the *Daubert/Joiner/Kumho* analysis. In their determinations, judges should further consult recent and relevant guidelines and regulatory statements of prominent agencies, such as the Environmental Protection Agency and the Food and Drug Administration.¹⁷⁹ Additionally, if there is a relevant state statute regarding the admissibility of genetic evidence, then the judge should consult this statute and consider any further limitations set forth by the state legislature.¹⁸⁰ An exemplary model of this type of preliminary analysis of genetic evidence by a state court can be seen in *Taylor v. State*.¹⁸¹ In *Taylor*, an Oklahoma state court adopted and effectively applied the *Daubert* standard of analysis, while focusing on the relevance and probative value of the genetic evidence and outlining a detailed framework of analysis.¹⁸² The *Taylor* court completed an exemplary in-depth study of the scientific background regarding the admissibility determination of the DNA profiling evidence at issue and further noted the

¹⁷⁷ *See id.* at 313–16.

¹⁷⁸ *Daubert v. Merrell Dow Pharm.*, 509 U.S. 579, 589–94 (1993); *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 136 (1997); *Kumho Tire Co. v. Carmichael*, 526 U.S.137, 148–49 (1998).

¹⁷⁹ *See* Brian Huseman, *Taylor v. State, Rule 706 and the DNA Database: Future Directions in DNA Evidence*, 22 OKLA. CITY U. L. REV. 397, 442 (1997); Marchant, *supra* note 1, at 33.

¹⁸⁰ *See* Huseman, *supra* note 179, at 442.

¹⁸¹ 889 P.2d 319, 328–32 (Okla. Crim. App. 1995).

¹⁸² *See id.*

following with respect to the reliability determination of scientific or technical evidence:

[f]our factors, neither exclusive nor necessarily dispositive, may aid the reliability determination: first, whether the scientific method at issue has been or can be tested; second, whether the theory has been subjected to peer review; third, whether the scientific procedure has known or potential rate of error; and fourth, whether the scientific evidence at issue has gained acceptance in the relevant scientific community.¹⁸³

Furthermore, judges should consider the totality of circumstances, including the following additional factors, with no one factor being determinative: (i) the relevance of the genetic data to the issue of causation; (ii) the balance of the potential probative value of the data weighted against the potential harm to the plaintiff and the plaintiff's interest in genetic privacy; (iii) the reasonableness of the intrusion posed by any compelled tests; (iv) the penetrance level of the gene(s); (v) the complexity of the application to the causation analysis and the capability of a reasonable juror to clearly understand the implications of the data; (vi) the quantity and quality of additional factors which may also affect the causation analysis; (vii) the length of time since the technology has emerged and became accepted in the scientific community; and (viii) the origin of the technology (i.e., publically versus privately funded, and whether either party has a stake in or financial interest in the research, or has substantially funded the research primarily or through subsidiaries).¹⁸⁴

B. *Promulgation of Universal Guidelines*

While there is Supreme Court case law regarding the application of genetic susceptibility data in toxic tort litigation, additional resources would be a significant asset to judges acting as the gatekeepers in these cases.¹⁸⁵ This article proposes the promulgation of official guidelines by a resource commonly used

¹⁸³ *Id.* at 339 n.88.

¹⁸⁴ See FED. R. EVID. 706; *Daubert v. Merrell Dow Pharm.*, 509 U.S. 579, 589 (1993); *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 136 (1997); *Kumho Tire Co. v. Carmichael*, 526 U.S.137, 148 (1998); Huseman, *supra* note 179, at 442.

¹⁸⁵ See *Daubert*, 509 U.S. at 589; *Joiner*, 522 U.S. at 136; *Kumho*, 526 U.S. at 148.

by judges, such as the American Law Report, the Bureau of National Affairs, the Food and Drug Administration, or the Environmental Protection Agency, to assist judges with these complex determinations. Such guidelines would help ensure consistency throughout the judicial system in the application of genetic susceptibility data in toxic tort litigation, as well as help prevent premature admission of unreliable genetic data.¹⁸⁶

The suggested guidelines should detail the judge's role as gatekeeper and the analytical framework of the *Daubert*, *Joiner*, and *Kumho* case-by-case analysis.¹⁸⁷ In addition, the guidelines should detail the factors listed above to be considered when determining the admissibility of genetic data in toxic tort causation analysis. Example scenarios detailing and explaining the different types of genetic variations and the possible interactions between multiple factors should also be provided within the official guidelines. Similar but more general guidelines for courts have already been promulgated by the National Academy of Sciences regarding the general use of DNA in courts, and provide an excellent resource for judges.¹⁸⁸ Additionally, the proposed guidelines should provide sample jury instructions regarding genetic susceptibility data.¹⁸⁹ Furthermore, this article suggests that the implication of the genetic data and its application and scientific significance should be briefly explained to the judge and jury by an expert in the field who was chosen by the court rather than an expert witness for either party.¹⁹⁰ The Federal Rules of Evidence Rule 706 permits such appointments of expert witnesses by the court; however, this article proposes that it is imperative for all states adopt an equivalent to Rule 706 in order to permit state court judges to appoint expert witnesses.¹⁹¹

¹⁸⁶ See Huseman, *supra* note 179, at 442; Poulter, *supra* note 41, at 224.

¹⁸⁷ See *Daubert*, 509 U.S. at 589; *Joiner*, 522 U.S. at 136; *Kumho*, 526 U.S. at 148; Huseman, *supra* note 179, at 442.

¹⁸⁸ See Huseman, *supra* note 179, at 447; Glennda Chui, *Proposal Aims to Legitimize Use of DNA Evidence in Court*, SEATTLE TIMES, May 4, 1996, at A3.

¹⁸⁹ See Shelton, *supra* note 10, at 376–78.

¹⁹⁰ See Huseman, *supra* note 179, at 441–42.

¹⁹¹ See FED. R. EVID. 706; Huseman, *supra* note 179, at 442.

Finally, this article proposes that proportional recovery should be considered in toxic tort litigation. By allowing proportional recovery, plaintiffs would not have the burden of proving causation attributable to exposure to the toxic substance is greater than fifty percent likelihood.¹⁹² In addition, this type of recovery would also reduce the overcompensation problem which results from forcing defendants to compensate a plaintiff for the entirety of the loss when the defendant was only fifty-one percent responsible.¹⁹³ Allowing proportional recovery would reduce the need for all or nothing causation and allow for a more reasonable burden on the defendants.¹⁹⁴ In addition, this type of recovery system could alleviate the necessity for plaintiffs to rule out any genetic susceptibility and prevent defendants from over exploiting the effects of genetic variations in order to escape liability.¹⁹⁵ For example, if fifty percent of the Caucasian population has a genetic variation predisposing them to lung cancer, should that be considered a rare genetic mutation when so many individuals are affected?¹⁹⁶ In such instances, rather than allowing defendants to escape liability altogether, proportional liability would eliminate the need for all or nothing compensation and allow just compensation of plaintiffs, which jurors are much more likely to accept compared to overcompensation.¹⁹⁷

V. CONCLUSION

The field of genetic susceptibility is an emerging field which holds the potential to revolutionize the way toxic tort cases are analyzed.¹⁹⁸ As with any emerging area of science, however, great caution is required to ensure that technology in this field is not prematurely applied to litigation before an accurate significance of the technology has been established. As Professor Gold insightfully noted, “[a]though there is such a thing as ‘bad

¹⁹² See Poulter, *supra* note 41, at 224.

¹⁹³ *See id.*

¹⁹⁴ *See id.*

¹⁹⁵ *See id.*

¹⁹⁶ See Marchant, *supra* note 4, at 951.

¹⁹⁷ See Poulter, *supra* note 41, at 224.

¹⁹⁸ See Marchant, *supra* note 1, at 9–14.

science,' or more properly, 'non-science,' even among scientists 'good science is not some unanimously-endorsed Platonic ideal at any given time.'¹⁹⁹ Consequently, this area of science and technology presents significant issues, due to the complex nature of the data, which must be carefully considered before the decision to present new genetic data to a jury is made. Given the complex nature of genetic data and its potential to substantially confuse or impact a jury's decision, it is essential that official guidelines be promulgated in order to assist judges in their critical role as gatekeepers and prevent evidence which could taint a jury's analysis from being admitted.

Precautions must be taken to protect any genetic information admitted into evidence and serious considerations of reliability, relevance, and probative value must be taken into account before genetic testing is admitted or compelled by the court. Finally, courts must make strong efforts to stay abreast of the emerging technology and any regulations involving genetic information in order to safeguard against allowing premature information into the courts, as well as to prevent skeptical research and data from being used to prove or disprove a party's case.

¹⁹⁹ Gold, *supra* note 32, at 404.