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AGAINST GENETIC EXCEPTIONALISM: AN ARGUMENT IN FAVOR OF THE VIABILITY OF PRECONCEPTION GENETIC TORTS

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Courts have generally grappled with the issue of prenatal torts for the last fifty years. Prenatal torts are commonly defined as “actions involving injuries that result from tortious acts that occurred before a plaintiff’s birth.”¹ The most well-known instance of these torts being litigated is cases involving diethylstilbestrol (DES) exposure.² DES was a drug administered to pregnant women from 1947 to 1971 to prevent miscarriages.³ The drug caused damage in utero to the fetuses, who later in life developed abnormalities such as cervical cancers or malformations of the uterus.⁴ DES is an example of a teratogen, which is “a chemical or physical agent that produces or raises the incidence of congenital malformations” in utero.⁵

Because the effects of DES did not manifest until the plaintiffs were adult women, these women had possibly already reproduced, producing putative additional plaintiffs.⁶ Many plaintiffs have successfully litigated DES claims,⁷ and

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1. Julie A. Greenberg, *Reconceptualizing Preconception Torts*, 64 TENN. L. REV. 315, 316 n.1 (1997).

2. See *infra* notes 9-12, 79-83 and accompanying text.

3. Greenberg, *supra* note 1, at 322 n.55 (citing *Enright v. Eli Lilly & Co.*, 570 N.E.2d 198, 200 (N.Y. 1991), *cert. denied*, 522 U.S. 868 (1991)).

4. Greenberg, *supra* note 1, at 322 n.55.

5. THOMAS D. GELEHRTER ET AL., *PRINCIPLES OF MEDICAL GENETICS* 349 (2d ed. 1998); Robert Mittendorf, *Teratogen Update: Carcinogenesis and Teratogenesis Associated With Exposure to Diethylstilbestrol (DES) In Utero*, 51 TERATOLOGY 435 (1995).

6. However, courts have generally denied third-generation grandchildren of the original mother who took DES recovery based on a theory of limited duty. Greenberg, *supra* note 1, at 322 n.55, 339-40; see also *Enright*, 570 N.E.2d at 202 (holding that strict products liability of DES manufacturers would not support a “multigenerational cause of action”).

7. See, e.g., *McMahon v. Eli Lilly & Co.*, 774 F.2d 830, 835-36 (7th Cir. 1985) (finding sufficient evidence for a jury to reasonably conclude that Eli Lilly “knew or should have known that DES might cause reproductive abnormalities” in 1955).

generally, courts have not voiced concern with the prospect of multiple generations of plaintiffs litigating in DES cases.⁸ For example, in *McMahon v. Eli Lilly*, the court reversed a directed verdict granted to the defendant drug manufacturer.⁹ The plaintiffs, a woman and her husband representing themselves and their deceased son, were second- and third-generation plaintiffs in the DES-based action.¹⁰ The court ruled that the plaintiff-mother was not barred from recovery either for herself or for her child simply because she was a second-generation victim of injuries caused by the teratogenic effects of prior exposure to DES.¹¹

There is a plethora of literature addressing prenatal torts caused by teratogens such as DES.¹² However, a distinct, but related, issue has yet to be appropriately handled either by the courts or by state legislatures: preconception genetic torts. The DES claims are all based on prenatal, postconception genetic torts, where the chromosomal breakage, if any, occurred *in utero*.¹³ Chromosomal breakage occurs as a result of the addition or deletion of chromosomes or specific parts of chromosomes.¹⁴ In each human cell there are forty-six chromosomes; these chromosomes contain the nearly three billion base pair sequences that comprise each individual's genetic code.¹⁵ A mutation in these chromosomes is "any permanent heritable change in the sequence of genomic DNA."¹⁶ What makes chromosomal breakage due to mutagenic effect unique is that it can occur to pre-existing DNA.¹⁷ This bears special significance for females, who are born with all of the gametes they will ever need in their lifetime, unlike males who continuously produce gametes for most of their lives.¹⁸ Where a particular mutagen alters or breaks the chromosomes of the reproductive cells, including the eggs or oocytes, a

8. See *infra* notes 9-12 and accompanying text. But see *Enright*, 570 N.E.2d at 202 (citing fears of multigenerational liability as a reason to deny relief).

9. 774 F.2d at 830, 832, 838.

10. *Id.* at 831.

11. *Id.* at 835-36.

12. E.g., Marisa L. Mascaro, *Preconception Tort Liability: Recognizing a Strict Liability Cause of Action for DES Grandchildren*, 17 AM. J.L. & MED. 435 (1991); Stephen A. Hurst, *Sindell v. Abbott Laboratories: DES Cases and the Move Toward New Tort Remedies*, 49 U. MO. KAN. CITY L. REV. 245 (1981).

13. See Mascaro, *supra* note 12, at 436; *Enright*, 570 N.E.2d at 199; *McMahon*, 774 F.2d at 832.

14. GELEHRTER ET AL., *supra* note 5, at 3.

15. Andrew Askland, *What, Me Worry? The Multi-Front Assault on Privacy*, 25 ST. LOUIS U. PUB. L. REV. 33, 47 (2006).

16. GELEHRTER ET AL., *supra* note 5, at 346.

17. A mutagen is "a chemical or physical agent that increases the mutation rate by causing changes in DNA." *Id.*

18. See Katharine K. Baker, *Gender, Genes, and Choice: A Comparative Look at Feminism, Evolution, and Economics*, 80 N.C. L. REV. 465, 471 (2002). A gamete is defined as a "haploid germ cell," and a germ cell is defined as "a reproductive cell that fuses with one from the opposite sex in fertilization to form a single-celled zygote." ROBERT C. KING & WILLIAM D. STANSFIELD, A DICTIONARY OF GENETICS 133, 141 (5th ed. 1997).

later-fertilized egg may possess the chromosomal alteration.¹⁹ This mutative process is the phenomenon of chromosomal breakage.

In the past thirty years, courts have seen a slow rise in the number of actions brought based entirely on preconception genetic torts—where the chromosomal damage occurs to the mother before the child is conceived.²⁰ Unlike in the DES cases, courts have generally been unwilling to grant as much freedom to preconception torts plaintiffs.²¹

This article addresses the status of preconception genetic torts. Preconception torts may be defined as “actions involving injuries that result from tortious acts that occurred before a plaintiff’s conception.”²² A “preconception genetic tort” is the shorthand I will use to refer to those injuries that alter the chromosomal structure of the mother rather than cause damage to somatic cells.²³

There are a host of complicated questions that arise in the preconception genetic tort paradigm, including: (1) Is chromosomal breakage a legally compensable injury in and of itself, or must an attendant syndrome or condition manifest to constitute a legally compensable injury? (2) Should the extent of the relief be limited to injuries resulting from developments prior to conception? (3) What relief should be granted where chromosomal breakage occurs during preconception, but where the genetic syndrome is multifactorial and only develops *in utero*? (4) Is increased risk of cancer due to a preconception mutagen a legally compensable injury? (5) Who may sue? The mother? The child? The grandchildren of the mother? The great-grandchildren?²⁴ (6) Will it be impossible to prove that but for a defendant’s negligence, a child would not have been born with chromosomal alteration or subsequent genetic disorders? (7) Should fears of multi-generational liability justify courts in denying preconception tort actions based on an analysis of proximate cause? (8) Should fears of the growing relevance of an individual’s private genetic information to a personal injury action justify courts in denying the viability of preconception genetic torts?

I will examine each of these questions in turn. Part I considers the problems relating to the injury: when does it occur, and is it (such as increased risk of cancer

19. See GELEHRTER ET AL., *supra* note 5, at 163-66.

20. Matthew Browne, *Preconception Tort Law in an Era of Assisted Reproduction: Applying a Nexus Test for Duty*, 69 FORDHAM L. REV. 2555, 2556 (2001).

21. E.g., *Taylor v. Cutler*, 703 A.2d 294, 295 (N.J. Super. Ct. App. Div. 1997) (declining to recognize “preconception negligence” as a tort); *Albala v. City of N.Y.*, 429 N.E.2d 786, 787-88 (N.Y. 1981) (denying recovery to a plaintiff child with severe brain damage resulting from negligent perforation to the mother’s uterus).

22. Greenberg, *supra* note 1, at 316 n.2 (citing *Grover v. Eli Lilly & Co.*, 591 N.E.2d 696, 698 (Ohio 1992)).

23. A somatic cell is “any cell of an organism not involved in the germline.” GELEHRTER ET AL., *supra* note 5, at 349.

24. This is known as the problem of multiple-generation liability. See *infra* notes 111-115 and accompanying text.

due to chromosomal breakage) cognizable. Part II surveys the intricate problems of causation that attend preconception genetic torts, including the policy considerations that animate proximate cause analyses, as well as difficulties in adducing adequate proof of causation. Finally, Part III considers the problems of preconception genetic torts from a broad-based, policy-oriented view, and concludes that there is little justification for denying the viability of preconception genetic torts, or for treating them altogether differently from preconception torts in general. The judicial system is well-equipped to handle any of the difficulties and problems presented by claims for preconception genetic torts. There is little policy justification for rejecting such claims as non-cognizable.

I. THE INJURY

A. *When Does the Injury Occur?*

The first notable case involving preconception genetic torts was *Jorgensen v. Meade Johnson Laboratories, Inc.*²⁵ In this case, a father brought an action based on theories of negligence, strict liability, and breach of express and implied warranties on behalf of his daughters afflicted with Down syndrome.²⁶ The plaintiff's wife took birth control pills manufactured by the defendant for a period of approximately six months.²⁷ Nine months after she ceased taking the birth control pills, she gave birth to twin daughters.²⁸ The plaintiff contended that the birth control pills altered the chromosomal structure of his wife, which directly and proximately caused his twin daughters to develop the trisomy 21 mutation responsible for Down syndrome *in utero*.²⁹ The district court dismissed the action for failing to state a claim because it concluded that no cause of action existed in Oklahoma for preconception injury to the chromosomal structure of the mother.³⁰

The United States Court of Appeals for the Tenth Circuit reversed and held that the plaintiff had stated an actionable claim for two reasons. First, broadly reading the complaint, the court reasoned that because the plaintiff pled that the genetic disorder developed *in utero* due to the mother's altered chromosome structure, "the pleading should not be construed as being limited to effects or developments prior to conception."³¹ Second, because the twin daughters, rather than the mother, were the plaintiffs, the only relevant injuries were those suffered

25. 483 F.2d 237 (10th Cir. 1973).

26. *Id.* at 238.

27. *Id.*

28. *Id.*

29. *Id.* at 239.

30. *Jorgensen v. Meade Johnson Labs., Inc.*, 336 F. Supp. 961, 963 (W.D. Okla. 1972), *rev'd*, 483 F.2d 237 (10th Cir. 1973).

31. *Jorgensen*, 483 F.2d at 239.

by the twin daughters.³² The court then drew an analogy to several cases where defendant manufacturers were held liable for manufacturing a defective food product prior to conception that ultimately caused injury to the infant child upon consumption.³³ The court reasoned that ample precedent supports the notion that tortious conduct occurring prior to conception is actionable if it ultimately causes injury to the infant.³⁴

The court's analysis is noteworthy for several reasons. First, the court avoids the difficult question of whether the mother's altered chromosome structure is in and of itself an injury by referring to the fact that the plaintiff's pleading contained references to injuries from teratogenic phenomena rather than injuries that occurred prior to conception (in which case the causes would be mutagenic in nature).³⁵ *Jorgensen* says nothing about whether there are two distinct injuries in this fact pattern (those of the twin daughters and those of the mother). Second, the analogy to the defective food product line of cases is inapposite, largely because in all of these cases, it was only the tortious conduct in manufacturing the defective product that occurred prior to conception.³⁶ By the court's analogy, the injury occurred only when the infant child consumed the defective food product.³⁷ The court thus begs the question by assuming or at least implying that the only relevant injury to the twin daughters occurred postconception, as did all of the injuries from the defective food products.³⁸ If this were not the assumption and the possibility that the daughter's injury occurred preconception was left open, then the defective food product cases where the injuries unquestionably occurred postconception would not be analogous.

The contention that by altering the chromosomal structure of the mother, the injury to the twin daughters themselves occurred preconception is not implausible. Naturally, this claim could only be brought by children who were subsequently born,³⁹ but the science supporting this chain of reasoning is sound. Once the chromosomal structure of the putative mother is altered, any subsequent fetus may have its chromosome structure altered as well.⁴⁰

32. *Id.* at 240.

33. *Id.*

34. *Id.*

35. See *supra* notes 5-19 and accompanying text for an explanation of teratogenic phenomena.

36. *Jorgenson*, 483 F.2d at 240.

37. *Id.*

38. *Id.* at 237 (demonstrating that the injuries at issue are those that occurred to the twins postconception, and not the "alleged effects on the mother" which occurred preconception).

39. In some jurisdictions, actions may be brought on behalf of the fetuses even if they are not actually born. See, e.g., *Volk v. Baldazo*, 651 P.2d 11, 12 (Idaho 1982) (holding that a wrongful death action for an unborn fetus is a viable claim).

40. See *supra* text accompanying note 19.

The core of the problem lies in the nature of genetics. Because any alteration in the germline chromosome structure of a mother's eggs may be passed on to descendants,⁴¹ it is difficult to pinpoint exactly when the injury occurs. Perhaps the injury to later-born children occurs when the mother's chromosome structure is altered. Perhaps it occurs at the moment of conception, in which case it is even more unclear whether the injury is preconception, postconception, or neither. Or perhaps the injury in the form of a genetic disorder only occurs *in utero*, in which case the injury is postconception. Yet another possibility is that the genetic disorder is multifactorial, requiring both inheritance of the mother's altered chromosomal structure as well as environmental influences *in utero*.⁴² The boundaries between these options are hard to discern.⁴³ Perhaps the best solution is to act as the *Jorgensen* court did by implying that the boundaries between preconception and postconception injury are unclear, and that where a plaintiff pleads both, he ought not be barred from recovery.⁴⁴

*B. Is Increased Risk of Cancer Due to Chromosomal Breakage
a Viable Cause of Action?*

At least one court has held that a plaintiff who presents evidence of increased risk of cancer due to chromosomal breakage ought to be able to submit the evidence to the trier of fact.⁴⁵ In *Bryson v. Pillsbury Co.*, the plaintiff was exposed to the chemical Captan.⁴⁶ The plaintiff presented evidence of extensive chromosomal breakage and claimed that she had an increased risk of cancer as a result of the exposure to Captan.⁴⁷ The defendant moved for summary judgment, claiming that the plaintiff assumed the risk of harm and that, in any case, the plaintiff's claimed damages were too speculative as a matter of law.⁴⁸ Agreeing with the defendant, the trial court granted the motion.⁴⁹

41. *Id.*

42. GELEHRTER ET AL., *supra* note 5, at 346. Multifactorial inheritance is defined as "traits resulting from interplay of multiple environmental factors with multiple genes." *Id.*

43. Some of the new genetic research on biomarkers may aid in pinpointing with precision when the injury occurs. For a discussion of this new research, see *infra* notes 188-92 and accompanying text. Nevertheless, difficulties in pinpointing the time the injury occurred has likely played a role in courts' unwillingness to recognize the viability of preconception genetic torts.

44. *Jorgensen v. Meade Johnson Labs., Inc.*, 483 F.2d 237, 239 (10th Cir. 1973). Of course, there are other ways to read the *Jorgensen* court's opinion. Among them is the notion that the court agreed with the district court's assessment that preconception genetic torts are not actionable. Because in this case the plaintiff pled post-conception injury, the claim is actionable; whereas, if the plaintiff had pled preconception injury alone, it would not be. Nonetheless, as noted above, this necessarily begs at least the scientific question by implying that the injuries all occurred after conception.

45. *Bryson v. Pillsbury Co.*, 573 N.W.2d 718, 721 (Minn. Ct. App. 1998).

46. *Id.* at 720.

47. *Id.*

48. *Id.*

49. *Id.* at 720.

The Court of Appeals of Minnesota reversed the entry of summary judgment for not proving a present injury,⁵⁰ reasoning that there was precedent supporting the idea that whether chromosomal breakage constitutes a present injury is a question of fact.⁵¹ So long as the plaintiff proffers expert testimony alleging that chromosomal breakage is a present injury, the court ruled that whether the chromosomal alteration rises to the level of a legally compensable injury is an issue for the trier of fact.⁵² The court also reversed the grant of summary judgment as to the plaintiff's claims for present damages, which amounted to claims of damages for emotional distress and medical monitoring expenses.⁵³ The court found that since there were genuine issues of material fact as to the existence of a present injury, there were similar issues of fact as to the damages caused by those injuries.⁵⁴

However, the court of appeals affirmed the grant of summary judgment as to the plaintiff's claim for future damages, finding that the plaintiff's expert witness "admitted that [the plaintiff's] increased risk of cancer could not be measured or quantified."⁵⁵ Thus, the court held that the claim of future damages was too speculative and not capable of being established with reasonable certainty.⁵⁶ Though increased risk of cancer has generally been perceived as a viable cause of action in the sense that plaintiffs usually survive motions to dismiss and or summary judgment motions,⁵⁷ many jurisdictions accept increased risk of cancer cases only reluctantly, or dismiss the damages as too speculative.⁵⁸

Furthermore, the scope of the injury affects who has standing to bring an action. If chromosomal alteration is not an actionable injury in and of itself, then, for example, the mother of the plaintiffs in *Jorgensen* could not recover. Her only injury was the alteration in her chromosome structure; it was her twin daughters who developed Down syndrome.⁵⁹ In addition, by denying chromosomal breakage status as a *sui generis* injury, the problem of multi-generational liability may diminish. This is because subsequent generations may have chromosomal

50. *Id.* at 722.

51. *Id.* at 721 (citing *Werlein v. United States*, 746 F. Supp. 887, 901 (D. Minn. 1990)).

52. *Id.*

53. *Id.*

54. *Id.*

55. *Id.*

56. *Id.*

57. See Fournier J. Gale, III & James L. Goyer, III, *Recovery for Cancerphobia and Increased Risk of Cancer*, 15 CUMB. L. REV. 723, 743 (1984-85) (discussing how numerous courts recognize statistical evidence of increased risk of cancer as admissible and sufficient to present a question for the jury).

58. See, e.g., *Klein v. Weisberg*, 694 A.2d 644, 646 (Pa. Super. Ct. 1997) (denying plaintiff recovery for increased risk of liver cancer claim and holding that damages are only awarded "for [] non-speculative injuries"); *DeStories v. City of Phoenix*, 744 P.2d 705, 706-08 (Ariz. Ct. App. 1987) (refusing to award plaintiff damages for claim of increased risk for asbestosis or lung cancer).

59. See *supra* notes 31-32 and accompanying text.

alterations. However, unlike first-generation children, who have both chromosomal breakage and a concomitant genetic disorder, the subsequent generations may have no attendant disorder. Thus, the later-generation children will have no actionable injury if no genetic disorder manifests. In these scenarios, the question of whether increased risk of cancer is an actionable injury is particularly salient, because this may be the only avenue for the later-generation children to obtain relief. As knowledge of the link between specific mutations and disease increases, it is not difficult to see that if increased risk of cancer is an actionable injury, so too might increased risk of Alzheimer's disease, increased risk of osteoporosis, and so on and so forth. The dangers of the "geneticization of lawsuits" are all too real, where plaintiffs may simply run down the line of genetic disorders until they find one that may have some link to the particular mutation they possess.⁶⁰ This factor may bear on courts' general unwillingness to recognize preconception genetic tort claims as viable.

II. THE CAUSE

A. Proximate Causation & Policy Concerns

The majority of courts, however, have disagreed with the Tenth Circuit's analysis in *Jorgensen* on the grounds of proximate causation rather than on the nature of the injury.⁶¹ The requirement that a plaintiff prove proximate cause is longstanding in the law of negligence.⁶² Generally, to show proximate cause, a plaintiff must prove that he or she was in the foreseeable zone of risk of a defendant's negligence.⁶³ The idea is that actionable negligence requires a significant relation in time and place between the parties.⁶⁴ As acknowledged by Chief Judge Cardozo, "[p]roof of negligence in the air . . . will not do."⁶⁵ The notion of foreseeability embodies the requirement that the causal connection be proximate, or related in time and space. Moreover, proximate cause enmeshes the concepts of duty and foreseeability. *Palsgraf v. Long Island Railroad Co.* stands for the proposition that one does not owe a duty to anyone not in the foreseeable zone

60. See Bob Van Voris, *Tort Lawyers Discover the Power of Genetics*, NAT'L L.J., Sept. 14, 1998, at A16.

61. Of course, courts will often deny proximate cause for policy reasons relating to problems arising from the imposition of liability for a particular injury. See *supra* notes 25-60 and accompanying text.

62. See *Palsgraf v. Long Island R.R. Co.*, 162 N.E. 99, 101 (N.Y. 1928); MORTON BARROWS, HANDBOOK ON THE LAW OF NEGLIGENCE 17 (1899).

63. DAN B. DOBBS, THE LAW OF TORTS 77 (West Group 2000).

64. See *Palsgraf*, 162 N.E. at 101 ("Negligence, like risk, is a term of relation.").

65. *Id.* at 99 (quoting FREDERICK POLLOCK, TORTS 455 (11th ed.)).

of risk.⁶⁶ In addition, where a court finds a lack of proximate cause, the plaintiff is cut off from recovery largely for policy reasons.⁶⁷

A prime example of a court denying recovery in a preconception tort case based on a proximate cause analysis is *Albala v. City of New York*.⁶⁸ In this case, New York's highest court denied relief to a child born with severe brain damage.⁶⁹ Four years prior to the plaintiff's conception, the mother's uterus had been perforated due to medical malpractice.⁷⁰ In *Albala*, the injury was to the uterus, an organ, rather than to a chromosome.⁷¹ Because the mother's genetic code was not altered, there was no question that the child's injury occurred postconception and *in utero*.⁷² The court, thus, did not focus on the injury, but rather denied recovery based on a lack of proximate cause.⁷³ Moreover, the court was alarmed at the prospect of endless liability where a physician's negligence caused foreseeable harm to a later-conceived child.⁷⁴ It seems difficult to comprehend how, based on the facts of *Albala*, the defendant in this case could possibly be exposed to multi-generational liability. Where the structural harm was not genetic, the structural defect is extremely unlikely to be passed on from parent to offspring past the first generation.⁷⁵

66. See *id.* at 101; see also William L. Prosser, *Palsgraf Revisited*, 52 MICH. L. REV. 1, 16 (1953) ("[C]ardoza contended, that the risk may be an outer boundary beyond which duty cannot extend, and that there is never any duty as to the unforeseeable plaintiff or the unforeseeable damage."). Dean Prosser demonstrated his prescience in this 1953 article when he noted, "[f]or more than two generations it has been repeated that there can be no duty toward an unborn child; now all of a sudden the cases on prenatal injury are going the other way." *Id.* at 14. It appears that as far as preconception genetic torts are concerned, the pendulum has swung back in the direction of limited or non-existent duties owed from defendants to plaintiffs.

67. See *Palsgraf*, 162 N.E. at 103-104 (Andrews, J., dissenting).

The damages must be so connected with the negligence that the latter may be said to be the proximate cause of the former. . . . What we do mean by the word 'proximate' is that because of convenience, of public policy, of a rough sense of justice, the law arbitrarily declines to trace a series of events beyond a certain point.

Id. at 103; see also *Atl. Coast Line R.R. Co. v. Daniels*, 70 S.E. 203, 205 (Ga. Ct. App. 1911) (reiterating that limitations on proximate cause are arbitrary lines drawn by the court for practical purposes).

68. 429 N.E.2d 786, 786-87 (N.Y. 1981). As Professor Greenberg notes, some scholars have contended that this case is "thinly reasoned." Greenberg, *supra* note 1, at 328 n.120 (citing W. PAGE KEETON ET AL., PROSSER & KEETON ON THE LAW OF TORTS § 55, at 367 (5th ed. 1984); see also Case Comment, *Preconception Tort as a Basis for Recovery*, 60 WASH. U. L.Q. 275, 291-92 (1982) (criticizing *Albala* as outmoded because its reasoning "unnecessarily produce[s] an unjust result.>").

69. *Albala*, 429 N.E.2d at 787.

70. *Id.*

71. See *id.*

72. See *id.*

73. *Id.* at 788.

74. *Id.*

75. See *Renslow v. Mennonite Hosp.*, 367 N.E.2d 1250, 1255 (Ill. 1977) (reasoning that a physician's negligence in misdiagnosing Rh-compatibility when transfusing blood causes birth defects

The New York court's analysis seems more apposite to the facts in *Enright v. Eli Lilly & Co.*⁷⁶ There, the New York court refused to impose liability in a strict products liability claim brought by a third-generation plaintiff against a manufacturer of DES.⁷⁷ In coming to its decision, "[t]he *Enright* court was concerned that imposing a duty in one case could lead to an expansion of liability beyond manageable boundaries."⁷⁸ The source of the court's fear of boundless liability was the prospect of multiple-generation liability.⁷⁹ The specter of multiple-generation liability was enough to persuade the court to limit liability to those who ingested the drug or were directly exposed to its teratogenic effects *in utero*.⁸⁰

In 1986, the Supreme Court of New York in Erie County extended the ruling of *Albala* to preconception genetic torts in *Catherwood v. American Sterilizer Co.*⁸¹ In this case, an action was brought on behalf of a child decedent who was conceived subsequent to the mother's exposure to the mutagen ethylene oxide.⁸² The plaintiff claimed that this mutagen caused the child's chromosomal damage.⁸³ The court acknowledged that the issue of whether a preconception genetic tort gave rise to a cause of action was one of first impression in New York.⁸⁴ The court characterized the court of appeals' decision in *Albala* as based purely on policy considerations,⁸⁵ and framed the issue of liability in *Catherwood* solely in terms of proximate cause and policy analyses.⁸⁶

The court granted the defendants' motion to dismiss for policy reasons. First, the court distinguished *Jorgensen* by noting that the plaintiff in *Catherwood* brought a negligence action, as opposed to the strict liability action at issue in *Jorgensen*.⁸⁷ Furthermore, the court reasoned that limitations on liability are less necessary in strict liability actions than they are in negligence actions brought on the basis of exposure to a toxin or mutagen.⁸⁸ In addition, the court stated that

to a later-conceived child, fears of multiple-generation liability are groundless where the damage is not "by its nature, self-perpetuating."). Doubtless the *Renslow* court is relying on the fact that the preconception tort in the case is a structural defect in the blood, rather than any alteration to the germline chromosomal structure.

76. 570 N.E.2d 198 (N.Y. 1991).

77. *Id.* at 199-200, 204.

78. Greenberg, *supra* note 1, at 328 (citing *Enright*, 570 N.E.2d at 203).

79. *Enright*, 570 N.E.2d at 203.

80. *Id.*; but see *infra* Part III.C.

81. 498 N.Y.S.2d 703, 705-06 (N.Y. App. Div. 1986).

82. *Id.* at 704.

83. *Id.*

84. *Id.* at 705.

85. *Id.*

86. *Id.* at 705-06.

87. *Id.* at 705.

88. *Id.* at 705-06.

imposing liability in a preconception tort case is untenable because the plaintiff would garner a cause of action before he or she actually came into existence.⁸⁹

This last point, of course, is a policy consideration based not on an analysis of causation, but on the difficulty of determining the specific point at which the injury occurs. The Supreme Court of Erie County inverts the assumption made by the *Jorgensen* court in identifying the point in time when the injury occurred. Where the court in *Jorgensen* implied that the only actionable injury occurred postconception,⁹⁰ the court in *Catherwood*, in denying the viability of the preconception genetic tort, assumes that the injury claimed by the plaintiff occurred entirely preconception.⁹¹ If it did not, the perilous policy the court is afraid of endorsing vanishes. The court very easily could have narrowed its decision so as to hold that claims brought where the injury is entirely preconception will be disallowed. It is entirely plausible to reason that though the exposure in *Catherwood* occurred preconception, the injury to the later-conceived child occurred only after conception.⁹²

Under the latter analysis, preconception genetic torts are conflated with the more well-settled postconception torts, with the caveat that the exposure itself occurs prior to conception. The consequence of this perspective, however, is that chromosomal damage with no attendant genetic disorder or syndrome may no longer be viewed as an injury *sui generis*, because, by definition, the only compensable injury would occur after conception. If the injury is perceived as occurring preconception, then the *Catherwood* court's concern of a cause of action accruing to a person not yet in existence is relevant. This is because the chromosomal breakage that affected the later-conceived child occurred prior to conception.

Whether or not chromosomal damage is seen as an actionable injury has many implications for the viability of claims such as increased risk of cancer. Perhaps much of this may turn upon the pleadings themselves. If the plaintiff in *Catherwood* had pled that the exposure occurred before conception and the injuries occurred subsequent to conception, as did the plaintiffs in *Jorgensen*, perhaps the New York court would have decided differently.⁹³ At the very least, its predilection

89. *Id.* at 706.

90. *See Jorgensen v. Meade Johnson Labs., Inc.*, 483 F.2d 237, 239-40 (10th Cir. 1972).

91. *Catherwood*, 498 N.Y.S.2d at 706.

92. *See supra* notes 39-44 and accompanying text. Of course, this legal plausibility of this claim will rest on the sufficiency of the proof presented. For a discussion of the difficult proof issues attendant in preconception genetic torts, see *infra* notes 121-147 and accompanying text. The court in *Catherwood* takes the analysis of proof of preconception injury out of the hands of the jury, and possibly, of any jury in Erie County in the event that other courts follow its lead in rejecting the viability of preconception genetic torts.

93. *See Catherwood*, 498 N.Y.S.2d at 706 (reasoning that a cause of action for a "plaintiff not in being" is "untenable").

against animating a cause of action where the plaintiff is not conceived at the point of injury might not be implicated if a postconception injury is pled.

The court in *Catherwood* also clothed its decision in terms of duty, stating that "[i]n order to allow a cause of action for pre-conception tort there requires the finding of a duty to the unconceived. Such a duty can only be couched in terms of a duty to protect the potentiality of life New York has not recognized any such duty."⁹⁴ Of course, the court's reliance on the concept of duty converges perfectly with its analysis of proximate cause because the two concepts are intertwined.⁹⁵ As Dean Prosser explicitly noted, cases involving prenatal torts are a common locus for issues of limited duty due to the plaintiff not being in the foreseeable zone of risk.⁹⁶ However, as Dean Prosser also noted,

There is a duty if the court says there is a duty; the law, like the Constitution, is what we make it. Duty is only a word with which we state our conclusion that there is or is not to be liability; it necessarily begs the essential question. When we find a duty, breach and damage, everything has been said.⁹⁷

Dean Prosser's analysis explains that the divergence between courts in extending or restraining the boundaries of a particular duty rests on conflicting policy analyses, rather than any disagreements about whether a legal mandate compels a particular boundary. Whereas the court in *Catherwood* was concerned with extending a duty from defendants to unconceived plaintiffs, other courts have not felt similarly constrained. For example, the Supreme Court of Indiana expressed no such qualms in *Walker v. Rinck*.⁹⁸ In that case, the plaintiffs were children who brought an action against a physician for misdiagnosing their mother's Rh-compatibility during a previous pregnancy.⁹⁹ The resultant Rh-sensitivity in the mother's blood allegedly caused injuries to later-conceived children.¹⁰⁰ The court reversed a lower court's grant of summary judgment to the defendant physician, reasoning that where the plaintiffs were foreseeable, the physician had a duty to use reasonable care in administering care to the mother.¹⁰¹ The facts of the case, according to the court, sufficiently illustrated that it was foreseeable when the defendant physician initially treated the mother knowing that she might conceive again.¹⁰² Accordingly, the court held that the defendant

94. *Id.*

95. See *supra* notes 65-66 and accompanying text.

96. See Prosser, *supra* note 66, at 14-15.

97. *Id.* at 15.

98. 604 N.E.2d 591, 592 (Ind. 1992).

99. *Id.* at 592-93.

100. *Id.*

101. *Id.* at 595, 597.

102. *Id.*

physician did owe a duty to the later-conceived children of the mother because they were foreseeable plaintiffs.¹⁰³

However, in reiterating his point that a court's refusal to extend duty or proximate cause *follows* rather than *precedes* the court's conclusion that liability ought not attach to a defendant based on the given fact pattern, Dean Prosser cautions that duty and foreseeability are not interchangeable concepts.¹⁰⁴ That is, courts do not always impose liability where a plaintiff was foreseeable.¹⁰⁵ The *Albala* court's decision evinces this notion. The court granted that it was foreseeable that when the mother underwent an abortion, she might at some point wish to conceive again, and thus any later-conceived children were in the foreseeable zone of risk of the defendant's negligence in performing the prior abortion.¹⁰⁶ Nevertheless, the court found that the presence of foreseeability alone was not enough to establish a duty from the defendant to the plaintiff in this case.¹⁰⁷ Whereas foreseeability was enough to establish a duty from the defendant to the plaintiff in *Walker*, it was not sufficient to establish a duty in *Albala*. If issues of proximate cause and duty turn on policy considerations, the logical question becomes what policy considerations have prompted the majority of courts to deny proximate cause and duty to plaintiffs in preconception genetic torts?

The major reason courts deny recovery for preconception genetic torts is fear of multiple-generation liability. Courts are afraid that no practical limit on liability may exist.¹⁰⁸ A commentator described the rationale of courts in this position:

[I]f courts were to allow the first generation of plaintiffs to recover, they would also be required to allow later generations to recover. Subjecting a tortfeasor to these claims by multiple generations would impose a burden disproportionate to the risk created. To avoid such a result, and to keep from drawing unprincipled distinctions between first generation and later generation plaintiffs, it is better to deny recovery to all preconception injury plaintiffs.¹⁰⁹

Thus, multiple-generation liability is disfavored not only because it is inherently unfair to the defendant.¹¹⁰ Courts also fear that if they allowed first

103. *Id.* at 595. In the language of *Palsgraf*, the court's findings rested upon the notion that the children were in the foreseeable zone of risk. *See supra* notes 61-67 and accompanying text.

104. Prosser, *supra* note 66, at 16 ("[D]uty does not always coincide with the foreseeable risk.").

105. *See id.* (explaining by example that an observer owes no duty to a drowning swimmer).

106. *Albala v. City of N.Y.*, 429 N.E.2d 786, 788 (N.Y. 1981).

107. *Id.*

108. *See, e.g., Enright v. Eli Lilly & Co.*, 570 N.E.2d 198, 203 (N.Y. 1991) (explaining that liability for DES manufacturers should be limited "to those who ingested the drug or were exposed to it in utero").

109. Greenberg, *supra* note 1, at 345.

110. Perhaps the unfairness to the defendant changes depending on whether the plaintiff brings a claim for negligence or strict liability. The court in *Catherwood* may have been implying this when it reasoned that cutting off liability in a preconception tort case might be better justified when the plaintiff

generation plaintiffs to recover, in order to avoid imposing limitless liability on a defendant, they might have to cut off second and third generation plaintiffs for no reason other than that they are second and third generation plaintiffs.¹¹¹ This, according to Professor Greenberg, is too arbitrary for the courts, and so they deny recovery to any plaintiff for preconception genetic torts.¹¹²

The Supreme Court of Illinois may have been the first to address the issue of multiple-generation liability for preconception torts in *Renslow v. Mennonite Hospital*.¹¹³ In this case, a physician negligently transfused the plaintiff's mother with 500 cubic centimeters of Rh-positive blood.¹¹⁴ The transfusion caused sensitization of the mother's Rh-negative blood.¹¹⁵ Nine years later, she gave birth to the plaintiff, who suffered from severe brain and organ damage, allegedly caused by the Rh-sensitivity.¹¹⁶ Though the majority found that the plaintiff had stated a viable cause of action, Justice Ryan dissented.¹¹⁷ Among the reasons for his dissent were fears of multiple-generation liability.¹¹⁸ Furthermore, Justice Ryan introduced the issue of insurance into the equation. He opined that "[u]nder these circumstances, it is difficult to perceive how an individual or institution could adequately provide insurance coverage, or how an insurer could establish reserves to cover a potential loss."¹¹⁹

According to this line of reasoning, the difficulty in obtaining insurance would increase the unfairness of exposing a defendant to multiple-generation liability, not to mention the burden that the insurer of the putative defendant would have to carry. Should the burden become unmanageable, it "may lead manufacturers and service providers to stop providing potentially useful

brings an action in negligence rather than in strict liability. See 498 N.Y.S.2d 703, 705-06 (N.Y. App. Div. 1986). Issues of duty and foreseeability do not arise under strict liability. Vanessa Merton, *The Exclusion of Pregnant, Pregnable, and Once-Pregnable People (a.k.a. Women) from Biomedical Research*, 19 AM. J.L. & MED. 369, 410 (1993) (noting how the *Jorgensen* court "did not need to reach issues of duty or foreseeability" because claims were brought under a theory of strict products liability).

111. See, e.g., *Enright*, 570 N.E.2d at 203 (explaining that a plaintiff's cause of action "could not be confined without the drawing of artificial and arbitrary boundaries" since "the rippling effect of DES exposure may extend for generations").

112. Greenberg, *supra* note 1, at 344-45.

113. 367 N.E.2d 1250, 1251 (Ill. 1977).

114. *Id.*

115. *Id.*

116. *Id.*

117. *Id.* at 1262 (Ryan, J., dissenting).

118. *Id.* at 1264.

119. *Id.* In *Renslow*, the birth defects were not caused by alterations in the chromosomal structure of either the mother or the later-conceived child, but rather by Rh-sensitivity in the mother's blood. *Id.* at 1251. Nevertheless, Justice Ryan was alarmed at the creation of a general precedent for preconception torts, which, he reasoned, could very well sustain the viability of a preconception genetically transmitted tort. *Id.* at 1264.

commodities and services.”¹²⁰ In addition, second and third generation plaintiffs might be unable to recover anything from a defendant that has either gone bankrupt due to litigation or that has been unable to obtain insurance. In this case, the fear of the arbitrary cut-off between first and later generation plaintiffs that motivated the court in *Enright* and Justice Ryan in *Renslow* may be drawn by the market, rather than the judicial system.

B. Difficulties of Proof and Consequences of Proving Causation

Another policy consideration that has motivated courts to deny the viability of preconception genetic torts are the extremely difficult issues of proof,¹²¹ which revolve around “the difficulty of establishing the causal link between a child’s health problem and the defendant’s preconceptual conduct.”¹²² For example, the mother in the *Jorgensen* case was apparently told that the pills she took were completely safe to use, and was given no warning of any possible harm.¹²³ Nonetheless, “[w]hether she could have proved this warranty and lack of warning, and then proven that the pills caused an alteration in her chromosomes, is not at all clear.”¹²⁴

The case of *Wintz v. Northrop Corp.* demonstrates the significant burden the plaintiff has in mustering enough evidence of causation.¹²⁵ In this case, a husband and wife brought an action both individually and on behalf of their daughter, Jessica, against Eastman Kodak Company and Northrop Corporation, the latter of which was the mother’s employer.¹²⁶ The mother worked as an industrial engineer both prior to and during her pregnancy.¹²⁷ Her line of work involved mixing various chemicals, one of which was bromide, to develop photographic film.¹²⁸ She alleged that she had inhaled bromide dust both prior to and during her pregnancy with her daughter.¹²⁹ After Jessica was born, she exhibited several abnormalities, including poor muscle tone, a weak sucking reflex, infrequent cry, and anomalous facial features.¹³⁰

120. Greenberg, *supra* note 1, at 345.

121. See, e.g., *Catherwood v. Am. Sterilizer Co.*, 498 N.Y.S.2d 703, 706 (N.Y. App. Div. 1986) (“If the cause of action accrues long before conception, how can a plaintiff not in being at date of accrual have a cause of action.”).

122. Merton, *supra* note 110, at 406.

123. *Jorgensen v. Meade Johnson Labs., Inc.*, 483 F.2d 237, 238-39 (10th Cir. 1973).

124. Merton, *supra* note 110, at 411; *Jorgensen*, 483 F.2d at 241.

125. 110 F.3d 508, 516 (7th Cir. 1997).

126. *Id.* at 510.

127. *Id.*

128. *Id.*

129. *Id.*

130. *Id.*

The treating physician suspected elevated bromide levels were the cause of the baby's behavioral abnormalities, and ordered bromide tests for both the child and the mother.¹³¹ Four years later, Jessica was examined by a different physician, Dr. Barbara Burton, an expert in genetic disorders.¹³² After examining Jessica's symptoms, which then included myopia, problems with the enamel in her teeth, frequent respiratory infections, and abnormal mental development, Dr. Burton diagnosed Jessica with a genetic disorder known as Prader-Willi syndrome.¹³³ Subsequent tests confirmed this diagnosis.¹³⁴ According to the court's findings of fact, Prader-Willi syndrome "is caused by a deletion of genetic material from the father's chromosomes. It is a purely genetic disorder which occurs prior to conception, and it cannot be caused by environmental exposure."¹³⁵

The plaintiffs retained a toxicologist as an expert witness.¹³⁶ The toxicologist was going to testify that exposure to bromide, rather than Prader-Willi syndrome, caused Jessica's developmental anomalies.¹³⁷ The defendants filed a motion for summary judgment.¹³⁸ They claimed that a toxicologist was not properly qualified under Federal Rule of Evidence 702 to testify as to the comparative effects of bromide and Prader-Willi syndrome on a child because the toxicologist was not a licensed physician.¹³⁹ The federal district judge granted the motion,¹⁴⁰ and the Court of Appeals for the Seventh Circuit affirmed.¹⁴¹

The Seventh Circuit agreed with the defendants that the toxicologist simply did not have the requisite knowledge and experience to enable him to offer expert testimony on the principles of toxicology as applied to a human.¹⁴² As the court noted, he had no knowledge of the frequency or quantity of the bromide to which the mother was exposed, nor the extent to which her work environment was ventilated, nor whether she wore a mask at work.¹⁴³ The court reasoned that at best the toxicologist professed expertise as to a "general understanding of bromide, with only unsupported speculation having been used to relate the general knowledge to the facts surrounding [the mother's] exposure."¹⁴⁴ Moreover, the original treating

131. *Id.*

132. *Id.* at 511.

133. *Id.*

134. *Id.*

135. *Id.*

136. *Id.*

137. *Id.*

138. *Id.*

139. *Id.*

140. *Id.* at 511-12.

141. *Id.* at 516.

142. *Id.* at 513.

143. *Id.*

144. *Id.* at 514.

physician testified that Jessica's physical and mental abnormalities were very likely to have been caused by Prader-Willi syndrome.¹⁴⁵ The court found that under these circumstances, the plaintiffs had not met their burden of showing proximate cause under Illinois law, which required a showing to a reasonable medical certainty that the defendant's acts caused the injury.¹⁴⁶

It is not clear what the plaintiffs could have done to survive summary judgment. Arguably, the defendants could have attacked the qualifications of any physician proffered as an expert witness by the plaintiffs on the grounds that he or she did not possess the requisite knowledge on the toxic effects of bromide.¹⁴⁷ Perhaps both a toxicologist and a physician would have to be called, although the defendant might object on the grounds that an expert in genetics would be needed. It is simply not clear what expert testimony would have been required to sustain the plaintiff's claim. The question of how to present expert testimony on genetics moves this inquiry into a different constellation of issues—the role that genetic information may play in personal injury litigation.

C. The "Geneticization" of Personal Injury Litigation

A related policy concern is the fear of the so-called "geneticization" of civil litigation.¹⁴⁸ Consider *Severson v. Markem Corp.*,¹⁴⁹ an unreported 1990 California case. In that case, a plaintiff claimed that her son's severe birth defects were caused by *in utero* exposure to methyl ethyl ketone.¹⁵⁰ The defendant claimed that the plaintiff's injuries were caused by a genetic disorder called fragile X syndrome.¹⁵¹ The defendant filed a motion asking the court to require the plaintiff to undergo genetic testing to ascertain whether the plaintiff was afflicted with fragile X

145. *Id.* at 514-15.

146. *Id.* ("Proximate cause can only be established when there is a reasonable certainty that the defendant's acts caused the injury.") (quoting *Schultz v. Hennessy Indus.*, 584 N.E.2d 235, 241 (Ill. App. Ct. 1991)).

147. This, of course, should not be taken as an argument that the plaintiffs proffered no witness who could have been qualified as an expert. It is only meant to show the intricacy of proof of causation issues that arises in preconception genetic tort cases.

148. Bob Van Voris, *Tort Lawyers Discover the Power of Genetics*, NAT'L L.J., September 14, 1998, at A16.

149. See generally Mark A. Rothstein, *Preventing the Discovery of Plaintiff Genetic Profiles by Defendants Seeking to Limit Damages in Personal Injury Litigation*, 71 IND. L.J. 877, 899, 899 n.170 (1996) (using *Severson* as an example of the negative consequences of court ordered genetic profiles); Sally Lehrman, *Pushing Limits of DNA Testing: Suit Prompts Study into Whether a Birth Defect Was Inherited or Caused by Toxics*, S.F. EXAM'R, June 5, 1994, at A1 (discussing the facts of the case).

150. Rothstein, *supra* note 149, at 899.

151. *Id.*

syndrome.¹⁵² The court granted the motion, “rejecting the argument that this was an invasive procedure which would cause severe distress.”¹⁵³

The facts in *Severson* are analogous to those in *Wintz*, but there are two significant differences. However, these differences only render the analogy imperfect. First, the plaintiff in *Severson* unquestionably suffered a postconception injury in that the exposure occurred *in utero*, whereas there was a question as to when the daughter’s injuries occurred in *Wintz*. Second, the plaintiff daughter in *Wintz* unquestionably was afflicted with a genetic disorder, while this was a crucial question in *Severson*. It does not tax the imagination, however, to envision an amalgam of these two cases: where a plaintiff brings a claim for a preconception genetic tort and where there is substantial dispute as to whether the plaintiff suffers from a genetic disorder unrelated to the defendant’s negligence. In this scenario, a defendant would in all likelihood make a motion to compel the plaintiff to undergo genetic testing to confirm or disconfirm whether the plaintiff is afflicted with the relevant genetic disorder.

Gary Marchant argues that because “[t]here are major data gaps and uncertainties about the health risks of most potentially toxic substances,” new “genetic developments” have the potential to fill some of these conceptual gaps take on increased importance in toxic tort litigation.¹⁵⁴ On the other hand, he notes that use of genetic data may render toxic tort litigation “even more complex, contentious, and ethically problematic.”¹⁵⁵ He notes two kinds of genetic data most likely to be used in toxic tort litigation: “data on genetic susceptibility of individual plaintiffs” and “genetic biomarkers of exposure and effect.”¹⁵⁶ As to the former, he acknowledges the likelihood that toxic tort claims that turn on issues of genetic susceptibility will require “genetic test data from the individual plaintiff showing the presence or absence of the genetic trait at issue” to survive summary judgment on the issue of causation.¹⁵⁷ He even cites an expert who has argued that it should

152. *See id.*

153. *Id.* at 899 n.172 (chronicling that the plaintiff in *Severson* “was terrified of needles and had to be held down by his mother” during a court ordered blood testing and suffered emotional trauma as a result).

154. Gary E. Marchant, *Genetic Data in Toxic Tort Litigation*, 14 J.L. & POL’Y 7, 7-8 (2006) [hereinafter Marchant, *Toxic Tort Litigation*]. For further discussion of the use of genetic data in the courtroom from Marchant, see Gary E. Marchant, *Genetics and Toxic Torts*, 31 SETON HALL L. REV. 949, 980 (2001) (discussing how different types of genetic information could be applied in toxic injury litigation); Gary E. Marchant, *Genetic Susceptibility and Biomarkers in Toxic Injury Litigation*, 41 JURIMETRICS 67, 102-03 (2000) (exploring the potential and existing uses of biomarkers in toxic tort litigation and evaluating the issues they raise).

155. Marchant, *Toxic Tort Litigation*, *supra* note 154, at 8.

156. *Id.*

157. *Id.* at 13.

be common practice "for defendants to seek genetic testing of plaintiffs in order to identify potential alternative causes."¹⁵⁸

In a 1996 article, Professor Mark Rothstein canvassed the public policy reasons militating against permitting coerced genetic testing of the kind in *Severson*.¹⁵⁹ He argues that forcing a plaintiff to undergo genetic testing is a serious infringement on the plaintiff's right to privacy.¹⁶⁰ Rothstein identifies three different forms of privacy threatened by the sort of compelled genetic ordered in *Severson*: (1) physical privacy, (2) informational privacy, and (3) decisional privacy.¹⁶¹ By forcing the plaintiff to have blood drawn or even a cotton swab deployed in his or her mouth, the plaintiff's physical privacy is violated.¹⁶² The results of the genetic test constitute a compelled disclosure of information and thereby are a violation of the right not to disseminate information about oneself, constituting a violation of informational privacy.¹⁶³ Finally, forcing the plaintiff to submit to the genetic test without his or her consent constitutes a transgression on decisional privacy.¹⁶⁴ Rothstein argues that the decision of whether or not to undergo genetic testing can be extremely difficult, and that forcing a plaintiff to undergo this procedure violates the plaintiff's autonomy by taking the decision out of his or her hands.¹⁶⁵

Second, compelled genetic testing raises concerns regarding confidentiality.¹⁶⁶ In contrast to privacy, which relates to the right to prevent anyone from acquiring personal information, "confidentiality refers to an individual's reasonable expectation that certain sensitive information revealed within a confidential relationship will not be redisclosed to a third party without the individual's consent."¹⁶⁷ Although the view prevails that in personal injury litigation the patient impliedly consents to the disclosure of medical information by making his or her medical status an issue in the case,¹⁶⁸ the unique nature of genetic information makes the consequences of compelled disclosure all the more stark.¹⁶⁹

158. *Id.* at 13-14 (citing Diane E. Lewis, *Under a Genetic Cloud: The Benefits of DNA Testing Come with a Potential for Abuse*, BOSTON GLOBE, Aug. 14, 1994, at A1).

159. Rothstein, *supra* note 149, at 894-99.

160. *Id.* at 894-95.

161. *Id.* at 894.

162. *Id.* at 895.

163. *Id.*

164. *Id.*

165. *Id.*

166. *Id.* at 896.

167. *Id.* (citing William J. Winslade, *Confidentiality*, in *ENCYCLOPEDIA OF BIOETHICS* 451-52 (Warran T. Reich, ed., 1995)).

168. Rothstein, *supra* note 149, at 896.

169. *Id.* at 894-98.

Disclosure of genetic information subjects an individual to risks of public stigma and humiliation.¹⁷⁰ Such information has been referred to as a future diary, inasmuch as it reveals information about an individual's past, present, and future relatives.¹⁷¹ Furthermore, genetic testing may reveal information about paternity, which is somewhat prone to error.¹⁷² Fears of later disclosure of such information may have a chilling effect on genetic research, in that individuals may refuse to participate in genetic research because of concerns that genetic test results could conceivably be discovered by employers, insurers, or other third parties.¹⁷³

These scenarios by their nature are particularly likely to arise in preconception genetic tort cases. This is because the injury in a preconception genetic tort case will either simply be a chromosomal alteration or a concomitant (and perhaps resultant) genetic disorder. Where a particular genetic disorder could not be caused by exposure to any kind of mutagen or toxin, a defendant will in all likelihood desire to show that the plaintiff's injury was a consequence of the genetic disorder rather than the defendant's negligence. The fact that this problem has not yet arisen—or, at least, that no court has mentioned it—in a preconception genetic tort case can be explained first by the paucity of such cases in general, and second by courts' general unwillingness to recognize preconception genetic torts as a viable cause of action. Nonetheless, the inherent problems raised by compelled genetic testing may well factor into a future court's analysis regarding whether to recognize a preconception genetic tort claim as viable or to dismiss the claim for policy reasons.

In short, Rothstein concludes that concerns of privacy and confidentiality merit attempts to prohibit defendants from seeking to compel plaintiffs in personal injury litigation to undergo genetic testing. While I agree with Rothstein that the privacy and confidentiality concerns are serious and merit careful attention, the significance of his recommendations is to urge that genetic information be treated exceptionally. I think genetic exceptionalism in the realm of preconception genetic torts is exceedingly unwise for a variety of reasons, a topic to which I turn now.

170. *Id.* at 896.

171. *Id.*; George J. Annas, *Privacy Rules for DNA Databanks: Protecting Coded 'Future Diaries'*, 270 JAMA 2346, 2346 (1993).

172. Rothstein, *supra* note 149, at 896-97.

173. *Id.* at 897.

III. THE RECOMMENDATION

A. *The Pitfalls of Genetic Exceptionalism*

Preconception torts are more widely accepted as viable causes of action than are preconception genetic torts, evidenced by the DES cases.¹⁷⁴ The fact that preconception genetic torts are not generally recognized necessitates some distinguishing factor between preconception torts where chromosomal breakage to the mother is not involved, and where it is. This article has attempted to suggest some possible reasons for the divergent treatment by the courts, including multiple-generation liability, difficult proof issues, and concerns about the geneticization of personal injury litigation.

The question is, are these reasons enough to justify the distinction between preconception torts and preconception genetic torts? More broadly, several scholars are alarmed at the culture of “genetic exceptionalism” that permeates the legislative and judicial landscape.¹⁷⁵ For example, Gostin and Hodge have expressed concern at the number of laws protecting genetic privacy, compared to the dearth of statutes comprehensively addressing privacy in general.¹⁷⁶

Suter argues that “genetic information is not unique and that concerns about abuses of information should not be limited to genetic information, but should extend to other medical information.”¹⁷⁷ She reasons that “[t]he presumption that genetic information is unique is severely tested by the fact that no sharp line divides genetic from nongenetic information. Instead, there is a great deal of overlap between these categories, making line-drawing exceedingly difficult.”¹⁷⁸

Even assuming that unique concerns are raised in a genetic paradigm, it does not necessarily follow that preconception genetic torts ought to be treated in

174. See Lars Noah, *Assisted Reproductive Technologies and the Pitfalls of Unregulated Biomedical Innovation*, 55 FLA. L. REV. 603, 637-39 (2003); but see, e.g., *Enright v. Eli Lilly & Co.*, 570 N.E.2d 198, 204 (N.Y. 1991), *cert denied*, 522 U.S. 868 (1991).

175. Lawrence O. Gostin & James G. Hodge, Jr., *Genetic Privacy and the Law: An End to Genetics Exceptionalism*, 40 JURIMETRICS 21, 23 (1999). “Genetics exceptionalism” is the term the authors use to describe the belief that genetic information by virtue of its uniqueness is entitled to greater privacy protections than other kinds of information. *Id.* at 23, 31. Of course, fears of the “geneticization” of civil litigation rely to some extent on the unique nature of genetic information. *Id.* at 31. Nevertheless, Gostin and Hodge raise important questions about the dangers of genetics exceptionalism, which ought to be considered in any analysis of genetic information and policy. *Id.* at 31-36. Such an analysis is somewhat beyond the scope of this inquiry (which is focused on the viability of preconception genetic torts), but concerns about genetics exceptionalism are relevant to the extent they cast doubt on the justification and efficacy of treating preconception genetic torts differently from other preconception or even postconception torts.

176. *Id.* at 41-53.

177. Sonia M. Suter, *The Allure and Peril of Genetics Exceptionalism: Do We Need Special Genetics Legislation?*, 79 WASH. U. L.Q. 669, 671 (2001).

178. *Id.* at 701.

drastically different ways from preconception torts not involving chromosomal breakage. Professors Gostin and Hodge frame the inquiry in terms of the adverse consequences that arise out of legislatures creating law that treats genetic policy issues differently from other kinds of policy issues.¹⁷⁹

The point is that the common law ought to grapple with these issues on a case-by-case basis. Simply banning preconception genetic torts by declaring that they are not viable claims is ill-advised. As nearly all courts specifically addressing the problem have noted explicitly or implicitly, preconception genetic torts pose novel and challenging issues. However, setting precedent denying the cause of action in and of itself is a knee-jerk response to a problem that is unlikely to vanish altogether. Moreover, upon examination of court's cited fears regarding injury and causation in a preconception genetic tort paradigm, the knee-jerk response is not justified.

B. Difficulties in Pinpointing the Injury Do Not Justify Denial of Relief

In many cases, a genetic disorder will manifest itself in conjunction with a chromosomal alteration. In *Jorgensen*, the plaintiffs were born with Down syndrome.¹⁸⁰ In *Wintz*, the daughter Jessica was diagnosed with Prader-Willi syndrome.¹⁸¹ The fact that a plaintiff has undoubtedly suffered some harm does not relieve the plaintiff from any difficulties in proving causation, but where a genetic disorder or condition is present, it is clear that some kind of harm has befallen the later-conceived person.

Moreover, new research on biomarkers, or biologic markers,¹⁸² has dramatically increased geneticists' ability to "delineate more precisely how a given ambient toxic exposure causes disease by tracing the 'molecular footprints' as the toxin passes through the body, interacts with critical target molecules in the body, and produces the molecular and cellular effects that eventually manifest as pathology."¹⁸³ This new research has particular importance for occupational or environmental exposure cases.¹⁸⁴ In other words, biomarkers may help in determining the specific steps in the causal pathway that a toxin takes in producing a given injury.¹⁸⁵ In effect, biomarkers may help to pinpoint the point at which a particular genetic disorder manifests by identifying the sequence of causal effects

179. Gostin & Hodge, *supra* note 175, at 23-24.

180. *Jorgensen v. Meade Johnson Labs., Inc.*, 483 F.2d 237, 238 (10th Cir. 1973).

181. *Wintz v. Northrop Corp.*, 110 F.3d 508, 511 (7th Cir. 1997).

182. A biomarker is a "biochemical, molecular, genetic, immunologic, physiologic, or other signal of events in biologic systems." Paul W. Brandt-Rauf & Sherry I. Brandt-Rauf, *Biomarkers—Scientific Advances and Societal Implications*, in GENETIC SECRETS: PROTECTING PRIVACY AND CONFIDENTIALITY IN THE GENETIC ERA 184, 184 (Mark A. Rothstein ed., 1997).

183. *Id.* at 184-85.

184. *Id.* at 185.

185. *Id.*

on the genetic level. As such, “[b]iomarkers may be important in . . . establishing the subclinical or preclinical harms upon which damages for future harms may be based.”¹⁸⁶ The establishment of these types of harms is absolutely crucial in preconception genetic tort claims, and the biomarker research may indicate whether the injury occurs preconception, at the moment of conception, or postconception based on the particular step in the causal pathway that the disorder manifests.

However, even assuming the difficulties in pinpointing when the precise injury occurs are not overcome, these difficulties do not justify barring relief for every preconception genetic tort. The distinction between permitting recovery for postconception torts and preconception torts seems somewhat arbitrary, especially in light of the fact that increased risk of cancer due to chromosomal alteration is generally an actionable, albeit controversial tort.¹⁸⁷ Where a plaintiff cannot adequately prove that an injury occurred, or cannot prove the causal link between the negligence and the injury, that plaintiff ought to be barred from recovery. However, the difficulty in pinpointing when the injury has occurred is a weak justification for barring any recovery whatsoever for plaintiffs who have undoubtedly suffered harm in most cases. Furthermore, the most controversial aspect of the injury issue in preconception genetic tort cases is often the mother’s individual claim for increased risk of cancer due to chromosomal alteration. Where many courts now, at a minimum, refuse to dismiss such claims or refuse to grant summary judgment,¹⁸⁸ the reasons for denying the viability of preconception genetic tort claims because of the questions surrounding the claimed injuries are even less persuasive.

C. Proximate Cause Policy Considerations Do Not Justify Barring Recovery

In spite of many courts’ doubts about the existence of a duty owed to persons not yet in existence, “courts throughout the country [have] abandoned the no-duty rule, and virtually all jurisdictions presently recognize a child’s cause of action for the consequences of prenatal injuries.”¹⁸⁹ Part of the reason for this is that the concept of foreseeability can be used by courts to limit preconception genetic tort liability.¹⁹⁰ Where a potential injury to a later-conceived person is foreseeable, it is much easier for courts to impose a duty.¹⁹¹ Again, the link between foreseeability

186. *Id.* at 192.

187. See Greenberg, *supra* note 1, at 353-54 (contending that there is “no principled reason” for the distinction between an action where the injury occurs shortly after birth and an action where the injury occurs at the moment of birth).

188. *E.g.*, Cudone v. Gehret, 821 F. Supp. 266, 271 (D. Del. 1993); Karol v. Berkow, 603 A.2d 547, 550-51 (N.J. Super. Ct. App. Div. 1992); D’Augustino v. Bristol-Myers Squibb Co., 980 F. Supp. 1452, 1455 (M.D. Fla. 1997).

189. Lynch v. Scheininger, 744 A.2d 113, 120 (N.J. 2000).

190. Greenberg, *supra* note 1, at 355.

191. *Id.*

and duty reflects the intertwining of the concepts of proximate cause and duty. As Chief Judge Cardozo noted in *Palsgraf*, a duty is owed to someone in the foreseeable zone of risk.¹⁹² If the injury to the later-conceived person is deemed foreseeable, then a proper *Palsgraf* analysis dictates that the court ought to impose a duty on the defendant charged with negligence who caused the injury. If the injury is not foreseeable, then the plaintiff is not within the zone of risk, and no duty should be imposed.¹⁹³ As Professor Greenberg explained, "[a]ny action that would foreseeably harm a woman's reproductive system and her ability to carry a child to term would presumably also foreseeably harm a child she conceives in the future."¹⁹⁴

The court in *Albala*, for example, recognized that where a doctor's negligence resulted in a woman's perforated uterus, it was foreseeable that this negligence could deleteriously affect the health of later-conceived children.¹⁹⁵ However, this did not stop the *Albala* court from denying the viability of a preconception tort.¹⁹⁶ Nonetheless, the fact that a proper *Palsgraf* analysis of foreseeability may be used to set boundaries of reasonable recovery for preconception genetic torts is reason for affirming the viability of preconception genetic torts. In addition, the applicability of a *Palsgraf* foreseeability analysis underscores the notion that genetic torts need not be treated differently from other kinds of torts. This follows inasmuch as modern courts frequently apply analyses of foreseeability to determine whether to deny proximate cause; there is no reason that such an analysis cannot be applied to preconception genetic torts.

Moreover, the problem of multi-generational liability is the proverbial storm in a teacup. First, few claims involving multi-generational liability in preconception tort cases have been reported or even cited in any case where a court raises the issue.¹⁹⁷ Second, where a later-generation plaintiff brings such a claim,

[T]he injury suffered by the first generation must be significant enough to constitute a compensable injury, but not so significant that it alters the first generation's ability to conceive. Although toxic agents may exist that are capable of causing such harm, no such claims have been brought to date.¹⁹⁸

DES is one such agent, as demonstrated by the third generation plaintiff in *Enright*.¹⁹⁹ Judge Hancock explained in his dissent in *Enright* the somewhat

192. *Palsgraf v. Long Island R.R. Co.*, 162 N.E. 99, 100 (N.Y. 1928).

193. Greenberg, *supra* note 1, at 355.

194. *Id.*

195. *Albala v. City of N.Y.*, 429 N.E.2d 786, 788 (N.Y. 1981). For further discussion of *Albala*, see *supra* notes 68-75 and accompanying text.

196. *Id.*

197. Greenberg, *supra* note 1, at 345, 356.

198. *Id.* at 345.

199. *Enright v. Eli Lilly & Co.*, 570 N.E.2d 198, 199 (N.Y. 1991).

grotesque irony of denying relief to later-generation victims because of fears of boundless liability:

[W]hen defendants' arguments are applied here to urge that although claims of DES daughters should be allowed the claims of granddaughters should not be, their forebodings strike a particularly ironic note: i.e., the very fact of the "insidious nature" of DES which may make the defendants liable for injuries to a future generation is advanced as the reason why they should not be liable for injuries to that generation.²⁰⁰

The irony of this argument ought not mask its injustice. There seems something intuitively unfair about denying recovery to later-generation plaintiffs solely because the particular agent responsible for their injuries has multi-generational effects. This unfairness is compounded by the dearth of such later-generation preconception tort claims. Perhaps if such claims were inundating the courts, concerns of boundless liability might be relevant, but such cases are rare.²⁰¹ Moreover, there are dangerous policy implications arising from signaling to drug and chemical manufacturers that any liability they may accrue from marketing a defective product will automatically cut off after one generation of plaintiffs.²⁰²

Third, if and when multiple-generation liability claims begin to frequent, or occur at all, in the judicial system, "the courts or legislatures will need to establish reasonable boundaries for recovery."²⁰³ Furthermore, to deny recovery by dismissing cases where obvious harm has been done to a plaintiff before it reaches the trier of fact, and where a defendant has been negligent, is irrational when justified on the basis of a fear that has yet to materialize.²⁰⁴

Finally, the fears of the possible geneticization of civil litigation are reasonable and worrisome. However, these concerns extend beyond the parameters of preconception genetic tort claims into all types of personal injury claims. In any personal injury action, a defendant may seek to compel a plaintiff to submit to genetic testing, on the theory that if the plaintiff has a reduced life expectancy due to a genetic disorder, the plaintiff will be entitled to a lower damage amount.²⁰⁵ Thus, these concerns are relevant in the entire arena of personal injury litigation. Using these fears as a justification for rejecting the viability of preconception genetic tort claims will do little to solve the systemic problem that may grow into

200. *Id.* at 207 (Hancock, J., dissenting).

201. Of course, one could argue the reason for the paucity is because putative plaintiffs know that courts generally disfavor them, and that if courts permitted them, they would rapidly grow in number. However, the dearth of these claims is apparent even in jurisdictions which permit preconception genetic torts.

202. *See Enright*, 570 N.E.2d at 207 (Hancock, J., dissenting).

203. Greenberg, *supra* note 1, at 356.

204. *Id.*

205. Rothstein, *supra* note 149, at 878.

the geneticization of personal injury litigation. The problem will still exist, regardless of whether preconception genetic torts are viable causes of action.

Moreover, there seems little justification for “exceptionalizing” the geneticization of litigation by prohibiting compelled genetic testing. Marchant seems to agree, arguing that a “blanket prohibition on any use of genomic data in order to protect plaintiffs’ confidentiality would be unwise, because both plaintiffs and defendants can benefit from such data in appropriate cases.”²⁰⁶ He also contends that “plaintiffs who put their health status at issue by bringing the litigation cannot expect such a blanket prohibition.”²⁰⁷ This point is directly analogous to the generally required disclosure of health records of plaintiffs bringing medical malpractice suits. The argument in the latter scenario, which is not widely disputed, is simply that if the plaintiff is uncomfortable sharing details of their health information, then they should not bring a suit that places the contents of that health information directly at issue.²⁰⁸ Why litigation implicating genetic information should be treated so differently is unclear.

Of course, the argument here is simply that exceptionalizing genomic data by prohibiting its usage or even prohibiting compelled genetic testing if a court finds that genetic information is vital to the litigation is unwise. It does not follow that courts ought to admit such evidence with no hesitation or freely order plaintiffs to undergo genetic testing. The argument that genetic information does not seem to merit exceptional treatment does not imply that there are no material differences between genetic information and other kinds of individually identifiable health information. Requests for the disclosure of genetic information or requests to compel the plaintiff to undergo genetic testing should, like many other evidentiary matters in toxic tort litigation, be handled on an individual basis.

206. Marchant, *Toxic Tort Litigation*, *supra* note 154, at 35.

207. *Id.*

208. See, e.g., Scott D. Stein, *What Litigators Need to Know About HIPAA*, 36 J. HEALTH L. 433, 438 (2003) (“[A] plaintiff who puts his medical condition at issue in a lawsuit, such as by filing a medical malpractice suit, traditionally has been deemed to have waived any right to object to the disclosure of personal medical information. The rationale is that when a party puts his medical condition at issue, that party cannot deprive the opposing party of relevant evidence on that issue.”); Marian E. Silber & Maria Elyse Rabar, *Access to Medical Records*, 8 HEALTH LAW. 10, 10 (1996) (“It is axiomatic that by initiating a medical malpractice action, a plaintiff puts his or her physical condition at issue and waives the physician-patient privilege and any right to confidentiality accruing therefrom.”). Silber & Rabar explain:

Part and parcel of such a waiver is the requirement that a plaintiff provide access to records of all health care providers with information relating to medical history, diagnosis, and treatment. Those records are frequently as critical to the preparation of the defense of the litigation as they are to its prosecution, and the importance of equal access to the materials has been recognized.

Id.

Marchant points out that “[f]ocused and scientifically-justified genetic inquiries and tests can help to resolve some lawsuits.”²⁰⁹ However, he acknowledges, that “broader and more intrusive ‘fishing expeditions’ into the plaintiff’s genome that lack any probable cause in terms of having a reasonable basis for investigating a specific gene or trait are likely to create more mischief than insight needed to resolve a case.”²¹⁰ Rather than prohibiting the use of such information, or even the compelled genetic testing, courts have a variety of tools in their judicial armamentarium for assessing the merits of such requests in the course of discovery. Indeed, determining whether to compel disclosure or deny it on the basis that it is a “fishing expedition” is one of a trial judge’s more common activities in the discovery process. Similarly common is the use of protective orders that circumscribe and narrow the disclosure of sensitive and personal information.²¹¹

Thus, there seems little basis for arguing that genetic information is so different that the judge’s diverse set of instruments in this regard should be ignored by simply stating that no plaintiff ever may be compelled to undergo genetic testing even where the basis of their case turns on the plaintiff’s genome.²¹² Garrison argues that “the court system already deals with highly personal and protected information on a daily basis, and there is no reason why such safeguards cannot be successfully applied to genetic information.”²¹³ *A fortiori*, there is even less merit in using the concerns of the geneticization of litigation as a reason for denying altogether the viability of preconception genetic torts.

CONCLUSION

There is little reason, neither for “pure” legal considerations, nor for policy concerns, to prevent any preconception genetic tort claim from being brought before a trier of fact. To be sure, difficult issues of proof, especially of causation, may prove daunting to the success of these claims in obtaining compensation. However, this is hardly a reason to deny all such claims before a plaintiff has an

209. Marchant, *Toxic Tort Litigation*, *supra* note 154, at 35.

210. *Id.* at 35-36.

211. Jordan K. Garrison, Note, *Courts Face the Exciting and the Inevitable: DNA in Civil Trials*, 23 REV. LITIG. 435, 458 (2004).

212. See *id.* at 446 (citing Thomas H. Murray, *Genetic Exceptionalism and “Future Diaries”: Is Genetic Information Different from Other Medical Information?*, in GENETIC SECRETS: PROTECTING PRIVACY AND CONFIDENTIALITY IN THE GENETIC ERA 60, 61 (Mark A. Rothstein ed., 1997)) (highlighting the conclusion of a task force working under the Ethical, Legal, and Social Implications arm of the Human Genome Project that genetic information is not so different from other kinds of health information as to merit exceptional protections).

213. Garrison, *supra* note 211, at 458; accord Marchant, *Toxic Tort Litigation*, *supra* note 154, at 36 (“Courts must use their discretion . . . to determine which genetic tests and data are justified, and also to provide for protective orders in appropriate cases to prevent disclosure of a plaintiff’s genetic information to non-parties.”).

opportunity to bring forth such proof. Moreover, the *Albala* court's concern over the proliferation of frivolous lawsuits that could occur if preconception genetic torts are ruled to be viable causes of action seems to be much ado about nothing, particularly where so few preconception genetic tort claims have been brought in the last twenty years. In any case, even if such cases do begin to proliferate, courts have a ready armamentarium of implements at their collective disposal that may be used to fashion reasonable boundaries on liability and recovery.