Transgenerational Tort Liability for Epigenetic Disease

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INTRODUCTION

First year law students across the country learn about the dangers of falling barrels and the legal theory of res ipsa loquitur. Tort law, developed over centuries in the Anglo-American common law, apportions liability and damages for harms to persons and property. When these harms are brought about by gravity-induced plunges or untimely explosions the law can handle its apportionment role relatively easily; however, twenty-first-century advances in our scientific understanding of the causes of disease and injury are pushing tort law away from the lessons of past experience. How do courts adjudicate liability and apportion damages for harms that come about from damage to the microscopic foundations of life that manifest themselves many years after the tortious act and can affect individuals across generations? Should children of smokers be able to sue their parents for the child’s cancer, even if the parent stopped smoking prior to conception? Could a grandchild sue a grandparent or great-grandparent for an increased risk of disease brought about by that ancestor’s life choices?

This Article explores the mounting scientific evidence concerning the impact of damage to the epigenome and how this damage can be heritable across multiple generations. While myriad articles and cases have discussed the ramifications of manufacturer liability for genetic and epigenetic disease, the issue of parental liability has apparently gone unexplored. The Article will proceed in four parts. Part I explains the epigenome in general, the harms that damage to the epigenome can cause, and issues surrounding how scientists are beginning to unravel the heritability of that damage. Part II surveys existing case and statutory law
concerning tort remedies for transgenerational harms, focusing especially on the DES cases. This Part will also explore existing state laws regarding maternal negligence (such as drug use) that causes harm to a child in utero. Part III identifies the unique problems associated with epigenetic tort liability across one or more generations, and how existing tort law both is and is not able to handle this emerging scientific understanding. Part IV proposes a liability framework for epigenetic disease and harm that allows for parental liability while examining the legal and economic justifications for such a system. Finally, Part V concludes the Article.

PART I: EXPLORING EPIGENETICS AND EPIGENETIC DISEASE

Many are familiar with the basic building blocks of genetics: deoxyribonucleic acid, better known as DNA. While DNA remains the fundamental code of life, advances in our understanding of genetics and genetic disease have led scientists to conclude that DNA alone fails to account for all of the information present within our genes. Another system, known as the epigenome, plays a crucial role in the regulation of genetic activity and phenotypic expression. "The term 'epigenetics' defines all meiotically and mitotically heritable changes in gene expression that are not coded in the DNA sequence itself. Three systems, including DNA methylation, RNA-associated silencing and histone modification, are used to initiate and sustain epigenetic silencing." This 'meta-genome' can have a marked impact on an organism's health and susceptibility to disease, and many of these mutations in humans "can be inherited or somatically acquired."

What makes epigenetic disease and inheritance different from genetics is the method by which mutations or changes occur. In DNA, genes are inherited by the child from their parents; changes to the genetic code come about from the mixing of parental DNA. Epigenetic mutation is different. "DNA methylation patterns fluctuate in response to changes in diet, inherited genetic polymorphisms and exposures to environmental

3. Id. at 458.
These changes to the epigenome may not be restricted to just the individual exposed to the environmental influence. In some animal studies, exposure to certain chemicals resulted in changes to the phenotypic expression of that animal’s offspring, likely indicating “transgenerational phenotypic effects” caused by epigenetic changes.7 Known epigenetic modifiers include metals such as cadmium, arsenic, nickel, and chromium; particulates in air pollution; endocrine disruptors like DES8, Bisphenol-A, and dioxin; and other chemicals like benzene and trichloroethylene.9 Other environmental factors have also been implicated in epigenetic inheritance, including maternal exposure to malnutrition or famine prior to the child’s conception.10 “If environmentally induced heritable epigenetic changes are common and influence disease risk, they would result in transient changes in both average risk and recurrence risk.”11 Notably, assisted reproductive technologies (“ART”) such as in vitro fertilization may be causing epigenetic damage to embryos:

Recent evidence suggests that the manipulation of embryos for the purposes of assisted reproduction or cloning may impose inherent risks to normal development. . . . [A]n apparent association with ARTs was recently found in registries of children with Angelman syndrome and Beckwith-Wiedemann syndrome. . . . Molecular analyses of patients with Angelman syndrome and Beckwith-Wiedemann syndrome conceived by in vitro fertilization or intracytoplasmic sperm injection revealed a loss of maternal-specific DNA methylation at imprinting centres, which indicates that the errors were epigenetic in nature. Although individually rare, as a group, epigenetic errors may impose significant risk for people conceived by ART. . . . Such reports suggest that technologies involving the manipulation of cultured embryos may be the ‘tip of the iceberg’ for a wider spectrum of epigenetic alterations.12

Epigenetic damage could be the cause of many previously

8. See Part IIA, infra.
9. See Baccarelli, supra note 7, at 244-49.
11. Id.
12. Rodenhiser, supra note 6, at 344.
unexplained negative outcomes from ART procedures or could result in longer-term complications for ART children (and perhaps the children of these children) due to epigenetic complications. This is not to say that all epigenetic silencing or modifications are necessarily heritable. Many modifications are wiped out during conception when the epigenome is essentially ‘reprogrammed’ to a fresh epigenetic slate. Moreover, isolating epigenetic risk from larger environmental and genetic factors is incredibly difficult. While the body of knowledge indicating human epigenetic heredity is not extensive, more controlled studies in animals have demonstrated the validity of the underlying theory.

What science has shown is that epigenetics and epigenetic heredity is a plausible answer for several disorders and diseases. The effects of epigenetic modification can run the gamut from nonexistent to deadly. Diseases causing intellectual disability, such as ATR-X syndrome, Fragile X syndrome, Angelman’s syndrome, Prader-Willi syndrome, Rett syndrome, Rubinstein-Taybi syndrome and Coffin-Lowry syndrome have been associated with an epigenetic cause. Likewise, ICF syndrome (chromosomal instability and immunodeficiency), Beckwith-Wiedemann Syndrome (overgrowth of organs), and leukemia have been shown to have epigenetic bases. “Epigenetic changes can also have a major role in the development of human cancer. For example, a high percentage of patients with sporadic colorectal cancers with a microsatellite instability phenotype show ...” epigenetic silencing, indicating that this epigenetic change “can result directly in genetic instability.” Exposure to environmental influences, then, can have lasting impacts beyond the individual who was exposed. Scientists are beginning to reveal the potential for transgenerational harms. Take cancer, for example. In some cases, epigenetic silencing (which scientists believe is a contributing factor to the development of cancer) is found both within the cancerous tumor and in “normal somatic tissue, including spermatozoa. These germline

15. See id. at 396.
16. Id.
17. See Egger, supra note 2, at 459.
18. See id.
19. Id.
'epimutations' predispose individuals carrying aberrant methylation patterns to multiple cancers."\textsuperscript{20} Very recent evidence suggests that affirmative acts of an individual, such as smoking, can lead to epigenetic damage. Researchers in one study found that, in their study group, young women who took up smoking during the study showed increased methylation (epigenetic modification) compared to their non-smoking counterparts.\textsuperscript{21}

The field of epigenetics is undergoing rapid change and development with new insights coming at a regular pace. A definitive, acquired and heritable epigenetic disease has not been discovered in humans. But damage to the epigenome from environmental influences (such as smoking) has been shown, as has the ability to pass epigenetic information transgenerationally.

\textbf{PART II: TRANSGENERATIONAL HARMs: DES AND MATERNAL ABUSE}

There are two areas of existing law that have dealt with what could be called transgenerational harms: products liability, specifically in the DES cases; and maternal drug or alcohol abuse resulting in harm to the fetus. Each provides informative guidance for the policy and practical limitations of transgenerational liability.

\textbf{A. The DES Cases}

In 1980, the California Supreme Court issued its landmark ruling of \textit{Sindell v. Abbott Laboratories}, in which the court held the manufacturers of Diethylstilbestrol (better known as DES) liable in proportion to their share of the market at the time of the injury.\textsuperscript{22} DES was a widely prescribed synthetic estrogen that was intended to reduce the risk of miscarriage in pregnant women.\textsuperscript{23} The drug was used for a period of roughly thirty years before the Food and Drug Administration banned its use in 1971.\textsuperscript{24} As it turns out, exposure to DES \textit{in utero} resulted in a greatly increased risk of certain cancers amongst the daughters of women

\textsuperscript{20} See Egger, supra note 2, at 459.
\textsuperscript{22} Sindell \textit{v. Abbott Laboratories}, 607 P.2d 924, 936-37 (Cal. 1980).
\textsuperscript{23} See id. at 925.
\textsuperscript{24} Id.
Mothers were unable to causally connect the drug they took to a specific manufacturer, and were thus "unable to establish traditional tort liability." The court concluded that "as between an innocent plaintiff and negligent defendants, the latter should bear the cost of the injury." The court reasoned that manufacturers "are better able to bear the cost of injury resulting from the manufacture of a defective product. . . [and are] in the best position to discover and guard against defects in its products and to warn of harmful effects."

Many courts would go on to accept the market share theory for DES cases; however, it should be noted that the California Supreme Court was grappling with a periconception tort (as the harm came about from the child’s exposure in utero) rather than a preconception tort. More recently, courts have begun dealing with so-called third generation DES cases (the grandsons and granddaughters of the women who took DES). The two most prominent appellate decisions grappling with this third generation of cases have both denied relief to the plaintiffs.

In *Enright v. Eli Lilly & Co.*, the Court of Appeals of New York held that DES cases were not uniquely different from general actions in tort and refused "to recognize a cause of action not available in other contexts simply (or at least largely)" because the case involved a DES injury. Citing its decision in *Albala* (which will be discussed, infra), the court argued that the recognition of multi-generational causes of action could have "staggering implications" and held that it was the court’s "duty to confine liability within manageable limits. Limiting liability to those who ingested the drug or were exposed to it in utero serves this purpose."

Similarly, in *Grover v. Eli Lilly & Co.*, the Ohio Supreme Court rejected the plaintiff’s cause of action, holding that “[k]nowledge of a risk to one class of plaintiffs does not necessarily extend an actor’s liability to every potential plaintiff.” While the court conceded that the manufacturer owed a duty to the individual taking a drug and to that individual’s fetus, that duty does not extend to “her daughter’s infant who

25. *Id.*
28. *Id.*
31. *Id.* at 203 (internal citations omitted).
will be conceived twenty-eight years later,” as the injury is ultimately too remote in “time and causation.” These DES grandchildren have thus faced high barriers of entry to the legal system, with courts essentially drawing a line between second- and third-generational damages.

B. Maternal Drug Abuse

From approximately 1977 onward, some states have prosecuted mothers for harm done to their children in utero due to drug and alcohol abuse. “Since 1996, women in Hawaii, Oklahoma, South Carolina, Utah, and Wisconsin have been charged with murder, attempted murder, or manslaughter based on their use of drugs while pregnant or other behavior alleged to have caused stillbirth, death, or other fetal harm.” Most of these prosecutions, however, have been unsuccessful. While criminal liability has been difficult to enforce, many more states have amended their civil child welfare laws to address maternal drug and alcohol abuse. Six state courts have considered whether a child can sue a mother in tort for prenatal harms suffered because of the mother’s drug abuse while pregnant, with three courts endorsing liability and three courts precluding it. Michigan, New Hampshire, and Florida have allowed children to maintain these suits in order to provide compensation for their injuries, holding that the child’s right to recovery outweighed the competing interests of maternal privacy or the intra-family relationship. The Florida appellate court framed the issue syllogistically:

Since a child born alive may maintain a cause of action against a third party for injuries sustained in utero, and a child may sue her mother in tort for negligence, it follows that a child born alive may maintain a cause of action against her mother based upon the negligence of the mother that caused the injury to the child before her birth and that such recovery is available up to the limits of liability insurance coverage. It is the Court’s ruling that a child should not be denied compensation for such injury

33. Id.
34. Linda C. Fentiman, In the Name of Fetal Protection: Why American Prosecutors Pursue Pregnant Drug Users (and Other Countries Don’t), 18 COLUM. J. GENDER & L. 647, 649 (2009).
35. Id.
merely because of the identity of the tortfeasor.\textsuperscript{39}

The court cautioned, however, that its holding was informed by the fact that the case involved a case of simple automobile negligence and that the defendant-mother was insured.\textsuperscript{40} Illinois, Texas and Massachusetts, in contrast, have denied recovery citing the notable problem of determining the duty of care owed by the mother.\textsuperscript{41} This split among courts reflects the uneasiness of the judiciary in imposing liability for prenatal torts—or, for that matter, imposing criminal punishment—because of the difficulty in fitting the parent-child relationship into our understanding of the duty of care. Nevertheless, it is not unreasonable—and courts have ruled as such—to impose this type of civil liability despite the concerns over parental tort immunity or the constraint of maternal action; indeed, courts have trended toward recognizing that “children are almost universally permitted to sue their parent for prenatal injuries. When a fetus is harmed in utero due to the mother’s wrongdoing, the born child can sue the mother for those injuries.”\textsuperscript{42}

\textbf{PART III: EPIGENETICS AND PRECONCEPTION LIABILITY}

“Few issues in tort law are more in need of clarification than those encompassed by the concepts of legal cause and duty,” notes Professor Richard Wright.\textsuperscript{43} This is no less true when attempting to fit the square peg of our modern understanding of the epigenetic causes of disease into the round hole of common law tort, all while balancing the unique problems of the tortious act occurring prior to conception.

\textbf{A. Courts Grapple with Preconception Torts}

Over the past few decades, courts have had to deal with a growing number of cases involving preconception torts, where the negligent act occurred prior to the plaintiff’s conception yet was claimed to be the cause of plaintiff’s injury. Two cases are representative of the divide between

\begin{itemize}
\item \textsuperscript{39} Nat'I Cas. Co., 807 So. 2d at 87.
\item \textsuperscript{40} \textit{Id.; see also infra Part IV} (discussing insurability).
\item \textsuperscript{41} \textit{See Stallman v. Youngquist}, 531 N.E.2d 355 (Ill. 1988); 
\textit{Chenault v. Huie}, 989 S.W.2d 474 (Tex. App. 1999); 
\end{itemize}
the courts endorsing preconception torts and those that are skeptical of the cause of action. One of the first cases to test the viability of preconception torts was *Jorgenson v. Meade Labs.* In that case, the plaintiff alleged that the defendant's birth control pills altered her chromosomal structure ultimately resulting in the birth of twins with Down Syndrome. The court approved of the plaintiff's theory, reasoning that preconception torts are not so different from other torts to warrant blanket disapproval. The court reasoned, hypothetically, that if "the view prevailed that tortious conduct occurring prior to conception is not actionable in [sic] behalf of an infant ultimately injured by the wrong, then an infant suffering personal injury from a defective food product, manufactured before his conception, would be without remedy." In contrast, the New York Court of Appeals rejected preconception liability in *Albala v. City of New York.* In *Albala,* the plaintiff-mother suffered a perforated uterus during an abortion that preceded the birth of the child at issue in the case. Plaintiff alleged that the damage to her uterus ultimately resulted in brain damage to her later-conceived child. The court concluded that the "recognition of a cause of action under these circumstances would require the extension of traditional tort concepts beyond manageable bounds . . . ." The *Albala* court and others that have followed its policy-based reasoning seem to fear that:

> if 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.

Professor Greenberg, among other commentators, disagrees with the rationale put forward by these courts, arguing that they are focusing too

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45. *Id.* at 238-39.
46. See *id.* at 240.
47. *Id.*
49. *Id.* at 787.
50. *Id.*
51. *Id.*
much on the timing of the negligent act vis-à-vis the timing of the plaintiff’s conception, as well as the hypothetical risk of multigenerational liability.\textsuperscript{53}

The Illinois Supreme Court in \textit{Renslow v. Mennonite Hospital} noted the problem of multigenerational liability but felt that it could be addressed through (unnamed) judicially enforced boundaries.\textsuperscript{54} The court reasoned that a duty exists to those who are foreseeably harmed, even if they are “unknown and remote in time and place.” The court further reasoned that it would be illogical to allow recovery when the defendant harmed a person who was conceived prior to the tortious act, though the defendant was unaware, but bar recovery for a person who was not yet conceived even though the defendant’s actions were the same.\textsuperscript{55} It is notable that in all of these cases the defendant was either a medical doctor negligent in the care of the mother or a drug manufacturer who created a drug taken by the mother—in no case were the parents themselves held liable for preconception torts.\textsuperscript{56}

\textbf{B. Preconception Tort Liability and Epigenetics}

Three elements of a negligence claim are particularly problematic when confronting a claim for transgenerational epigenetic harm; however, “[s]imply banning preconception genetic torts by declaring that they are not viable claims is ill-advised.”\textsuperscript{57} Instead, it is necessary to look to the actual elements of negligence that are problematic and identify how those problems could be rectified. First, what duty of care is owed by the mother to the child or grandchild? Second, how can the child show that the parent’s negligence proximately caused their injury? And third, what injuries do we recognize as legally cognizable?

1. \textbf{Duty of Care to the Unborn and Their Progeny}

Perhaps the most problematic element of a preconception genetic tort

\textsuperscript{53} See id. at 351, 355-56.
\textsuperscript{54} Renslow, 367 N.E.2d 1250, 1255 (Ill. 1977).
\textsuperscript{55} Id.
\textsuperscript{57} Daniel S. Goldberg, \textit{Against Genetic Exceptionalism: An Argument In Favor Of The Viability Of Preconception Genetic Torts}, 10 J. HEALTH CARE L. & POL’Y 259, 280 (2007) (continuing “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”).
claim is the duty of the tortfeasor to the unconceived, unborn child. Judge Cardozo cautions jurists that "[p]roof of negligence in the air, so to speak, will not do." The Renslow dissent pointed to the lack of foreseeability inherent in multigenerational torts as necessarily invoking mere "negligence in the air." Indeed, Justice Ryan’s dissent argues that multigenerational liability would preclude insurability due to the potentially endless stream of liability flowing from a single tortious act, and that the consequences of abrogating foreseeability are, themselves, unforeseeable. The Albala court similarly based its rejection of preconception torts on the lack of duty. The court recognized that it was foreseeable that the injury plaintiff suffered prior to conception could harm a later-conceived fetus, but felt that this "foreseeability alone" was insufficient to establish duty. Citing the good public policy of manageable judicial standards, the court reasoned essentially that because duty to the unconceived could have negative implications, no duty should be recognized at all.

These cases dealt with third-party liability for preconception harms. Professor Kirsten Smolensky has concluded that while third parties may owe a duty of care to a fetus preconception or during gestation, courts have been reluctant to expand that liability to a mother, it may violate her bodily integrity. In her survey of the limited jurisprudence on parental liability, Smolensky notes that courts have been unwilling to subject mothers to a prenatal duty of care, citing concerns over the expansion of tort law, maternal freedom of action while pregnant, and the interactions between prenatal liability and abortion rights. Parental liability for pre-implementation genetic diagnosis, in Smolensky’s opinion, does not implicate maternal freedom of action as the decisions are made outside the mother’s body and could not therefore interfere with that mother’s freedoms. “Once the embryos are implanted in the womb, the pregnant woman can still make autonomous decisions about her body, including

60. Id.
61. Albala, 429 N.E.2d at 788.
62. See id. ("We determined long ago in a case involving policy issues as sensitive as the ones at bar that foreseeability alone is not the hallmark of legal duty for if foreseeability were the sole test we could not logically confine the extension of liability").
63. See Smolensky, supra note 37, at 325-28.
64. See id. at 325.
65. Id. at 326.
what substances to ingest, and whether to exercise.”66 Importantly though, bodily integrity has usually been understood as “reflect[ing] an interest to be free from some forced intrusion to the body.”67 Professor Jaime King, in her response to Professor Smolensky's article, sought to expand the duty of care from intentional torts to include negligence as well.68 She argued that “[i]ndividuals should owe a duty to their potential offspring to act as reasonably prudent parents when making choices that do not conflict with the mother's bodily integrity.”

2. Problems with Generational Causation

Even if duty to the unconceived is established, plaintiffs in preconception genetic torts still face the “sometimes insurmountable” problem of showing causation when the tortious act occurred years or decades (or in truly multigenerational claims, perhaps centuries) earlier.69 But the inability or difficulty plaintiffs might face in showing legal causation for their injuries should not be a total bar to their claims prior to trial. Causation is difficult to show in many medical cases and is not uniquely different in the realm of preconception torts.70 In Jorgensen, for example, the court reasoned that causation in preconception torts should be shown by “competent medical proof,” similar to any other claim for personal injury.71

3. Legally Cognizable Injuries

After duty, the most problematic aspect of a preconception genetic tort claim is determining just what harms are legally cognizable. It seems clear that actual birth defects (such as Down Syndrome) are remediable.72 In the realm of toxic torts, courts have had difficulty in drafting a manageable rule for when the increased risk of disease becomes judicially actionable.73

The debate over “wrongful life” as a cause of action is instructive. In a wrongful life claim, a claim is brought on a disabled child’s behalf.

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66. Id.
67. Id.
69. See Greenberg, supra note 52, at 346.
71. Jorgensen, 483 F.2d at 240.
72. See, e.g., id.
alleging that the defendant failed to give the plaintiff's parents sufficient information about the disability, and that the failure to do so lead to the plaintiff's birth with the disability. Most jurisdictions have been skeptical of the wrongful life cause of action. Professor Alan Belsky proposes using strict products liability as a model for wrongful life cases, where "damages would inure to the plaintiff not merely because the provider's negligence caused an otherwise avoidable life, but because the avoidable life will bring with it foreseeable suffering." Belsky is opposed, however, to parental liability for wrongful life claims, arguing that it would have a chilling effect on procreative liberty and is duplicative of the pre-existing obligation that parents have to care for their children.

Likewise, the creation of a cause of action for failing to intervene using advanced reproductive technologies has been criticized by commentators. In contrast to this parental nonfeasance, there is support among commentators and some courts to allow for liability under the previously discussed parental misfeasance. Smolensky argues that "future generations have not only a moral right, but also a legal right to an open future, and limiting a child's future should be considered a legally cognizable harm." However, others argue that we should not constrain ourselves to tort claims limiting an open future.

PART IV: PARENTS SHOULD BE LIABLE IN TORT TO THEIR CHILDREN FOR EPIGENETIC DISEASE

"Any current generation - through design or through negligence in
permitting hazardous exposures - that alters the biological inheritance of its successors has 'pre-ordained' the lives of future generations in meaningful ways," writes Professor Mark Rothstein.81 “The current generation will have weakened future generations, limited their options, and required them to pay with their health or their lives for the environmental misdeeds of their forebears.”82 This Article proposes parental liability for epigenetic diseases brought about by their preconception misfeasance.

While there are myriad concerns associated with this form of liability, the mere risk of problems with judicial manageability should not preclude recovery for the actual harm suffered by a child.83 While some have expressed concern over multigenerational liability,84 or proposed arbitrary limitations on liability to a single generation,85 this Article does not foresee the need for this bright-line rule. Epigenetic torts, even if they implicate multiple generations, are not immune to the standard controls used by courts to limit and restrict causes of action for negligence. The problems with duty, to a lesser degree, and causation, to a greater degree, inherent in proving a case of epigenetic harm serve as natural roadblocks to unlimited liability.86

What this proposal does is hold parents liable for the damage caused to their epigenome by their negligent acts, even if those acts occur well before conception occurs. As was discussed, a growing body of evidence suggests that acts such as smoking can lead to heritable epigenetic silencing leading to an increased risk for cancer in the actor’s offspring.87 Given the rapid progression of scientific discovery it seems only a matter of time before we are able to show a causal link between an action like smoking or heavy drinking and the later acquisition (or risk) of cancer in a child due to the damage caused to the epigenome. Before going further it should be noted that this proposal does not envision liability for either epigenetic damage caused by involuntary exposure to chemicals (e.g., general air pollution or work-place exposures), nor does it intend to hold

81. Mark A. Rothstein, et al., The Ghost in Our Genes: Legal and Ethical Implications of Epigenetics, 19 HEALTH MATRIX 1, 58 (2009). Rothstein quotes C.S. Lewis’s concern about “pre-ordain[ing]” future generations due to genetic enhancement or eugenics, and argues that those same dangers apply to epigenetic disease. See id. at 57-58.
82. Rothstein, supra note 81, at 58.
83. See Goldberg, supra note 57 and associated text.
84. See, Renslow, 367 N.E.2d at 1264 (Ryan, J., dissenting).
85. See, Enright, 570 N.E.2d at 203.
86. See supra, Part III.B.
87. See generally supra, Part I.
parents liable for the genes that they were born with—even if those genes result in disease or handicap.

The goal of tort law is to allocate costs in an efficient and rational manner, allowing “actors [to] make a rational cost-benefit calculus regarding whether the potential tort damages outweigh the expected benefits derived from the wrongful conduct.” Not all harmful conduct results in liability; “defendants avoid liability as long as their conduct is reasonable in the view of the fact-finder.” The Renslow court’s concerns over unlimited liability are not unfounded, nor are the concerns of Professors Smolensky and King. There is an understandable uneasiness associated with holding a mother or father liable for the epigenetic damage they caused thirty years prior to conception, when they were an avid smoker or chronic drinker. But the very essence of tort law is to apportion risk to the nearest actor able to prevent the harm from occurring and actions to recover for preconception acts simply do not implicate the many concerns over procreative liberty that are associated with wrongful life claims.

More importantly, concerns over bodily integrity are unlikely to be seriously implicated in a tort scheme allowing this type of recovery. Allowing an as-yet unconceived child to sue for the harm caused by his mother or father’s smoking habit is not fundamentally different from allowing a child—or the state—to sue for harm caused by that child’s exposure to second-hand smoke in the household; in both situations, parents are being asked to act reasonably to prevent harm to their child without being asked to suffer a “forced intrusion to the body” implicating a violation of bodily integrity. If anything, parents—even prior to conception—are uniquely situated to prevent injury to their children brought about from epigenetic damage; by allocating the risk of this disease to the parent, we recognize that:

the starting point should be that granting an injured party a right of action against the tortfeasor serves the interests of the former more than the denial of such a right. It serves the interests of the specific victim by compensating, at least in part, for the injury.

89. Id. at 32.
90. Id.
91. See Belsky, supra note 74, at 241.
92. See Smolensky, supra note 37, at 326.
And it serves the interests of all potential victims by deterring potential injurers from future misconduct. Combining these two considerations would have similar effects where the injured party is a child and the tortfeasor is a parent. In fact, the child's unique material and emotional dependence on a parent should justify extending - not restricting - the parent's duty of care to the child. The emotional effect of injury caused by a person whose devotion is the cornerstone of the child's life is immeasurably graver than that of equivalent injury that a stranger causes. 93

This is essentially the same reasoning used by the Renslow court and is particularly persuasive when examined in the context of the liability being discussed here; deterring harmful behavior by potential parents serves both individual and social welfare by reallocating risk from the general public or the individual victim to the individual tortfeasor.

Given the ubiquity of having children, it seems unlikely that all potentially tortious acts would have unforeseeable consequences—but this theory of liability leaves intact the underlying requirements for proving a negligence claim, among which are the requirement that the harm have been foreseeable. Parental liability in this context merely recognizes that in a world where scientific knowledge can show causal links between certain actions and the subsequent risk of cancer in children, it is implausible to reject these claims wholesale just because they involve epigenetics. Parents ought not be immunized from liability based solely on the unique relationship between mother and fetus, nor are they held liable for every day actions that might "deprive[] a mother of her right to control her life during pregnancy; rather, she is required to act with the appropriate duty of care, as we have consistently held other persons are required to act, with respect to the fetus." 94

Stripping away the argument for allocative efficiencies with regard to risk, and distributing the economic burden to the tortfeasor, children should be able to hold their parents liable for harms that restrict their ability to have a future unburdened by debilitating illness or the unnecessary risk of future disease. 95 However, we do not want to strip all parental autonomy or impose a burden on every member of society to limit

94. See Bonte, 616 A.2d at 466.
95. See Smolensky, supra note 37, at 312 (Smolensky's argument for a cause of action reflecting the loss of an "open future" is appropriate to the claims discussed in this article as well).
every action they take. Traditional notions of reasonableness in tort provide useful guidance, but what is reasonable in this context? Although discussing parental liability for genetic intervention and not the preconception harms at issue here, Professor Jennifer Rosato argues that a standard of “significant risk of harm” is most appropriate.

Ultimately, the definition of harm in this context must strike a delicate balance by protecting children while respecting the parents’ decision making authority. Existing abuse and neglect laws reflect this balanced approach. Actual harm usually must be shown, unless the parents possess a conflict of interest [between parental autonomy and the child’s potential autonomy]. If a conflict exists, then a lesser showing is permitted: state intervention is justified if the parental decision would pose a significant risk of serious physical or emotional harm.

By shifting the timeframe for state intervention, it becomes plausible to imagine the system proposed by this Article. State intervention, then, is justified when the decisions of a prospective parent (or for that matter, anyone who could have children) would pose a significant risk of serious physical harm to their future offspring. Critics would certainly argue that this could lead to draconian intervention and interference by the state, which is why tort law provides a better alternative than state regulation in preventing future harms.

Finally, some may question the appropriateness of shifting the economic burden away from society for what could be described as essentially innocent acts by this new breed of tortfeasor. But this assumes that it is society’s role to provide the economic backstop for this form of harm solely because the act and the consequence are separated by several decades. “Equality of fortune contends that in a just society the child’s misfortune is, in the first instance, its parents’ obligation to correct.”

Professor Eric Rakowski provocatively suggests that the best solution to the problem of group versus individual responsibility is to require parents “to pay a fixed sum per child into an insurance pool that would be used to

97. Id.
compensate any children that were born genetically disadvantaged." 99 This form of shared insurance is a reasonable alternative to the tort model suggested by this Article; however, such a system fails to address the underlying behavioral modification goals of tort that seek to lower, in this case, society’s overall costs associated with epigenetic disease.

PART V: CONCLUSION

The future of epigenetic research will likely yield a growing understanding of how epigenetics affects disease and how this risk is passed from generation to generation. The concerns implicated by this Article are untested by courts largely because we are just now understanding how acts during one’s lifetime can impact children conceived years later. It would be unreasonable to expect that as our understanding progresses, aggrieved children would never seek to hold their parents liable for the risks and illnesses needlessly suffered because of the parents' tortious acts.

All of that is not to say that there are not significant hurdles in tort law that could preclude some, most or even all claims for preconception epigenetic torts. This is, however, more ‘feature’ than it is ‘bug.’ The concerns expressed by commentators and courts over the potential for limitless liability are now, and will continue to be, undermined by the inherent difficulty in showing causation and foreseeability as the harm becomes more attenuated from the tortious act. Allowing parental liability would require individuals to bear more of the risk of their acts rather than foisting that liability on society. Is the tort system the best method of allocating risk and damages for these types of injuries? This Article suggests that it is, but that some form of compulsory and universal insurance system, such as the one proposed by Professor Rakowski, serves as an alternative means of accomplishing the same goal. Insurance, however, is plagued by the risk of moral hazard. Moreover, this insurance system may be little more than six-of-one and half-a-dozen of another; in both the status quo and the world of universal insurance it is society that bears the risk to a greater or lesser degree. The advantage of parental liability in tort is that the individual becomes responsible. While it is unlikely that a great number of children will choose to sue their parents under this theory of liability, there are few good reasons to simply reject parental liability outright, and these children should have the chance to

99. Id. at 1398.
present their case like any other claim for personal injury.