Fordham Law Review

Volume 60 | Issue 5 Article 3

1992

Toxic Reproductive and Genetic Hazards in the Workplace: Challenging the Myths of the Tort and Workers' Compensation Systems

Jean Macchiaroli Eggen

Toxic Reproductive and Genetic Hazards in the Workplace: Challenging the Myths of the Tort and Workers' Compensation Systems

Cover Page Footnote

The author wishes to thank John Culhane, Barry Furrow, and Martin Kotler for reviewing and commenting upon drafts of this article. The author also gratefully acknowledges the research assistance of Cynthia Funaro, Lisa Hunn, and David Schlechter.q

TOXIC REPRODUCTIVE AND GENETIC HAZARDS IN THE WORKPLACE: CHALLENGING THE MYTHS OF THE TORT AND WORKERS' COMPENSATION SYSTEMS

*JEAN MACCHIAROLI EGGEN**

In this Article, Professor Eggen discusses how various scientific studies suggest a causal connection between workers' reproductive and genetic injuries and their exposure to toxins in the workplace. Because of conflicts between scientific and legal causation standards, workers and affected family members often cannot prove a sufficient causal connection between toxic exposure and ensuing injury to recover under existing workers' compensation and tort laws. Thus, Professor Eggen proposes several specific reforms to both the workers' compensation and tort law systems to improve the availability of these relief mechanisms for toxic exposure victims.

INTRODUCTION

IN the nineteenth century, public health analysts acknowledged the social and economic significance of reproductive health by recording data pertaining to reproductive outcomes.[1] Despite this initial interest, reproductive health in the workplace has received little attention until recent years. Extraordinarily few studies have been conducted on workplace toxins to examine their potential reproductive and genetic effects.[2] While the Occupational Safety and Health Act[3] ("OSH Act") provides some regulation of toxic substances in the workplace,[4] the Act offers no

* Associate Professor, Widener University School of Law; J.D. 1982, Syracuse University College of Law; M.A. 1974, Michigan State University; A.B. 1971, Connecticut College. The author wishes to thank John Culhane, Barry Furrow, and Martin Kotler for reviewing and commenting upon drafts of this article. The author also gratefully acknowledges the research assistance of Cynthia Funaro, Lisa Hunn, and David Schlechter.

1. *See* Bregman, Anderson, Buffler & Salg, *Surveillance for Work-Related Adverse Reproductive Outcomes*, 79 Am. J. Pub. Health 53, 54 (Supp. 1989).

2. *See* United States Congress, Office of Technology Assessment, Reproductive Health Hazards in the Workplace 3 (1985) (stating that "most commercial chemicals have not been thoroughly evaluated for their possible toxic effects on reproduction and development") [hereinafter Office of Technology Assessment, Reproductive Health Hazards]. In general, very few states keep any form of centralized records on occupational hazards to the reproductive and genetic systems of workers. Although thousands of chemicals appear on the Toxic Substances Control Act inventory, toxicity statistics are unavailable for the vast majority. *See* M. Rothstein, Medical Screening and the Employee Health Cost Crisis 48 (1989).

3. Occupational Safety and Health Act of 1970, Pub. L. No. 91-596, § 2, 84 Stat. 1590 (codified at 29 U.S.C. §§ 651-78 (1985) [hereinafter OSH Act]).

4. Congress set forth the following means of achieving the goals of the OSH Act:

> (1) by encouraging employers and employees in their efforts to reduce the number of occupational safety and health hazards . . . and to stimulate [them]

avenue of relief for occupational illness generally or for genetic injury specifically. The remedial mechanisms of workers' compensation and tort law are similarly inadequate to address the growing number of reproductive and genetic health problems arising from the workplace.

The insidious nature of occupational reproductive and genetic injury lies in its causal indeterminacy. Reproductive injuries, such as infertility and spontaneous abortion,[5] can often be virtually indistinguishable from background levels of these conditions in the general population, rendering medical identification and analysis nearly impossible.[6] In addition, although the worker experiences the most direct exposure to a toxic substance, third parties, such as the worker's spouse or offspring, often suffer

---

to institute new and to perfect existing programs for providing safe and healthful working conditions;

(2) by providing that employers and employees have separate but independent responsibilities and rights with respect to achieving safe and healthful working conditions;

. . .

(5) by providing for research in the field of occupational safety and health . . . and by developing innovative methods, techniques, and approaches for dealing with occupational safety and health problems;

(6) by exploring ways to discover latent diseases, establishing causal connections between diseases and work in environmental conditions. . . . ;

(7) by providing medical criteria which will assure . . . that no employee will suffer diminished health, functional capacity, or life expectancy as a result of his work experience.

OSH Act, *supra* note 3, § 651(b)(1)-(7). *See generally* Holzman, *The Occupational Safety & Health Act: Is It Time for Change?*, 17 N. Ky. L. Rev. 177, 178-81 (1989) (presenting review of legislative history and public policy underlying OSH Act). The general duty clause of the OSH Act provides that an employer must furnish a workplace free from "recognized hazards" that are causing or are likely to cause death or serious physical harm to its employees. OSH Act, *supra* note 3, § 654(a). The OSH Act, however, does not grant the Occupational Safety and Health Administration ("OSHA") or any other agency the authority to compensate victims of occupational accidents or illness. *See* Viscusi, *Structuring an Effective Occupational Disease Policy: Victim Compensation and Risk Regulation*, 2 Yale J. on Reg. 53, 61-62 (1984). The Act expressly preserves existing remedies for employees under state statutory and common law. Nothing in the Act

shall be construed to supersede or in any manner affect any workmen's compensation law or . . . affect in any . . . manner the common law or statutory rights, duties, or liabilities of employers and employees under any law with respect to injuries, diseases, or death of employees arising out of, or in the course of, employment.

OSH Act, *supra* note 3, § 653(4). Thus, the state-law mechanisms of workers' compensation and tort law provide the sole means of remedying injuries resulting from toxic exposures in the occupational setting. While this Article addresses the problems raised by judicial efforts to apply these remedial mechanisms to toxic reproductive and genetic injuries in the workplace, an analysis of the regulatory problems presented by such injuries is beyond its scope and is more appropriately the topic of a separate article.

5. "Spontaneous abortion" is defined as "[g]iving birth to an embryo or fetus prior to the stage of viability" in a manner "that has not been artificially induced." Stedman's Medical Dictionary 3 (24th ed. 1982).

6. *See* Paul & Himmelstein, *Reproductive Hazards in the Workplace: What the Practitioner Needs to Know About Chemical Exposures*, 71 Obstetrics & Gynecology 921, 921 (1988).

the full force of the injury.[7] The expressions of many chemically induced genetic mutations[8] in the hereditary line may not occur for one or more generations.[9] Not only do these characteristics permit occupational injuries to masquerade in the population as arbitrary background events, but they also prevent victim compensation for legitimate work-related injuries.

For several decades, the law of toxic exposures has evolved in response to increasing public awareness of the vast array of problems generated by the use and disposal of toxic substances. The workplace setting has given rise to a large portion of the early toxic tort case law, most notably in the multitude of lawsuits brought by injured asbestos insulation workers against the asbestos industry's manufacturing giants.[10] Yet, courts have struggled with the application of the traditional modes and presumptions of the legal doctrines to these unconventional injuries.

Recently, the issue of reproductive toxins in the workplace moved to a central position in the public consciousness with the United States Supreme Court's decision in *United Auto Workers v. Johnson Controls, Inc.*.[11] The Court in this case held that an employer's fetal protection policy, which excluded all fertile women from jobs involving exposure to lead, violated federal law by unjustifiably discriminating against women.[12] Although the majority denied concern for any incremental expansion of employer liability that might accompany widespread compliance with the *Johnson Controls* decision,[13] Justice White's concur-

7. *Cf.* Enright v. Eli Lilly & Co., 77 N.Y.2d 377, 381, 570 N.E.2d 198, 199, 568 N.Y.S.2d 550, 551 (1991) (denying cause of action for injured granddaughter of woman who ingested drug DES while pregnant), *cert. denied*, 112 S. Ct. 197 (1991).

8. A mutation is a "change in the character of a gene that is perpetuated in subsequent divisions of the cell in which it occurs." Stedman's Medical Dictionary 912 (24th ed. 1982). For further discussion of mutations, see *infra* note 49 and accompanying text.

9. *See* United States Congress, Office of Technology Assessment, Genetic Monitoring and Screening in the Workplace app. A, at 191-96 (1990) [hereinafter Office of Technology Assessment, Genetic Monitoring].

10. *See, e.g.*, Karjala v. Johns-Manville Prods. Corp., 523 F.2d 155, 158 (8th Cir. 1975) (holding manufacturer liable under strict products liability theory for failure to warn workers of asbestos dangers); Borel v. Fibreboard Paper Prods. Corp., 493 F.2d 1076, 1089-90 (5th Cir. 1973) (same), *cert. denied*, 419 U.S. 869 (1974).

11. United Auto Workers v. Johnson Controls, Inc., 111 S. Ct. 1196 (1991).

12. *See id.* at 1207. The Court noted that the bias inherent in the employee protection policy at issue in the *Johnson Controls* case affected women disproportionately to men: "Fertile men, but not fertile women, are given a choice as to whether they wish to risk their reproductive health for a particular job." *Id.* at 1202. The Court found this to be constitutionally untenable, finding that the true classification represented by the policy was not the gender-neutral concept of fertility, but the gender-specific fact of child-bearing capacity. *See id.* at 1203.

13. *See id.* at 1208-09. In dicta, the Court acknowledged the collateral issue of potential employer tort liability for prenatal injuries suffered by offspring whose mothers now may choose to work in a lead-exposed environment rather than accept a lesser paying position. *See id.* at 1208-09. The Court observed that a future issue for decision in an appropriate case would be whether compliance by an employer with relevant OSHA standards for exposure to lead in the workplace would pre-empt state common-law tort liability for offspring injuries arising out of maternal occupational lead exposure. *See id.* In

rence suggested that "the possibility of tort liability is not hypothetical," particularly in light of the potential harm to the offspring of exposed workers.[14]

Johnson Controls portends critical issues that will confront the legal system well into the next century. First, the etiology of reproductive and genetic injury and the relationship of occupational exposure to hazardous substances currently are poorly understood.[15] Accordingly, these injuries have been subject to ambiguous treatment by the legal system. This is particularly evident in the often preclusive operation of legal causation standards within the contexts of both the workers' compensation system and the tort system. Second, toxic exposures in the workplace may have significant ramifications beyond the immediate employer-employee relationship. Actions by the affected spouses and offspring of workers will put the system to the test. The law, in its current state of evolution, is ill-equipped to address the myriad of complicated questions raised by this type of injury.

The evolution of the law of toxic exposures has been tortuous, and analysis reveals a disconcerting ambivalence toward reproductive and genetic injuries. Standards that arose within the narrow confines of traditional accident law no longer suit the more complex and convoluted medical and legal issues presented by these injuries. The time is appropriate to reexamine the operation of the fundamental policies of compensation, accident avoidance, and loss-spreading in light of this new and problematic class of injuries. Novel approaches and reformulations of traditional rules are warranted to revitalize the legal system's alleged commitment to provide reasonable victim compensation and to assign appropriate industry accountability.

Part I of this Article surveys the state of medical knowledge regarding reproductive and genetic injuries caused by exposure to toxic substances in the workplace. Part II analyzes the current treatment of these injuries under the existing dual remedial scheme of workers' compensation and common-law tort liability, concluding that these regimes provide inadequate relief to legitimately injured workers and their families. Part III examines the ways in which reproductive and genetic injuries challenge the traditional presumptions and policies of the tort and workers' compensation systems, emphasizing the need for a new jurisprudence of toxic

---

his concurrence, Justice White noted that "it is far from clear that compliance with Title VII will pre-empt state tort liability," speculating that under the rule of *Johnson Controls*, employers will face increased difficulty in determining appropriate workplace safety practices so as to avoid tort liability. *Id.* at 1211 (White, J., concurring).

14. *Id.* at 1210-11 (White, J., concurring). Justice White observed that while barring claims by injured employees who have been adequately warned of potential workplace exposures is reasonable, preventing claims by the employees' children is far less likely under traditional tort law: "[T]he general rule is that parents cannot waive causes of action on behalf of their children, and the parents' negligence will not be imputed to the children." *Id.* at 1211 (White, J., concurring) (footnote omitted).

15. *See* Paul & Himmelstein, *supra* note 6, at 921.

liability. Finally, Part IV proposes several judicial and legislative reforms including realignment of the burden of proof of causation, expansion of exceptions to the exclusivity of workers' compensation, establishment of a legislative compensation scheme for subsequent generations of persons injured as a result of an initial reproductive or genetic occupational injury, availability of medical surveillance compensation, and encouragement of scientific research and information dissemination regarding reproductive and genetic hazards.

I. THE SCIENTIFIC CHALLENGE OF REPRODUCTIVE AND GENETIC HAZARDS IN THE WORKPLACE

For almost a century, studies have suggested an association between certain chemical exposures in the workplace environment and reproduction-related injuries in both female and male workers.[16] The earliest conclusions connected workplace lead exposure to infertility, pregnancy loss, and a host of injuries to offspring.[17] Notwithstanding these initial discoveries, the problem of reproductive hazards in the workplace was largely ignored by researchers until the 1970s.[18] During that decade, scientists observed relationships between workplace toxins and both reproductive and genetic injuries.[19] In addition, public awareness of the events surrounding Love Canal increased concern for the effects of exposure to all environmental toxins and spurred further study of toxic hazards in the workplace.[20] Nevertheless, reproductive and genetic workplace hazards have not yet received a level of attention commensurate with the threat they pose.

A. *The Demography of Reproductive and Genetic Injuries in the Workplace*

Studies estimate that up to fourteen million American workers may be

16. *See* S. Barlow & F. Sullivan, Reproductive Hazards of Industrial Chemicals 3 (1982) [hereinafter Barlow & Sullivan].

17. *See id.* (citing Rom, *Effects of Lead on the Female and Reproduction: A Review*, 43 Mt. Sinai J. Med. 542 (1976)).

18. Interest in reproductive hazards generally was piqued by Japanese studies on the toxicity of mercury found in fish, which issued in the 1970s. *See* Barlow & Sullivan, *supra* note 16, at 3-4. In 1976, scientific and public attention focused on an industrial accident in Seveso, Italy, in which a toxic cloud of dioxin was released into the environment with subsequent teratogenic effects. *See id.* at 4. *See generally* T. Whiteside, The Pendulum and the Toxic Cloud 31-62 (1979) (discussing the 1976 explosion at a chemical factory in the Lombardy region of Italy). At approximately the same time, studies began to appear regarding reproductive hazards to men who were exposed to pesticides or vinyl chloride in the course of their employment. *See* Barlow & Sullivan, *supra* note 16, at 4.

19. The workplace toxins studied included various industrial chemicals, biological agents, and ionizing radiation. *See* Barlow & Sullivan, *supra* note 16, at 4.

20. *See generally* Guidelines for Studies of Human Populations Exposed to Mutagenic and Reproductive Hazards (A. Bloom ed. 1981) (this report was compiled in response to the controversy arising from cytogenetic studies of residents of contaminated communities).

exposed each year to potential reproductive hazards in the workplace.[21] Indeed, the Centers for Disease Control have included reproductive disorders among the ten most common work-related complaints in the United States.[22] Notwithstanding preliminary concerns, comparatively few workplace substances have been studied for their potential effect on human reproduction or genetic make-up.[23] Existing studies, however, show that both men and women workers suffer genetic and reproductive injuries from exposure to workplace toxins. These toxic exposure injuries include infertility, various pregnancy-related injuries such as spontaneous abortion and stillbirth, cancer in children of exposed workers, and various genetically-related disorders.

1. Infertility

In general, statistics indicate that involuntary infertility affects anywhere from eight and one-half percent to fifteen percent of all couples.[24] Infertility diagnoses are about evenly divided between female-based infertility, male-based infertility, and infertility attributable to a combination of female and male factors.[25]

With few exceptions, scientific research on reproductive workplace injuries has emphasized female exposure to toxins.[26] Studies suggest that toxic exposure in the workplace may cause a direct defect in the ovum, prevent implantation of a fertilized ovum in the uterus, and interfere with the process by which the pituitary or the ovaries direct the release of hormones during the menstrual cycle, thus causing a variety of disturbances in fertility.[27] Male exposure to workplace toxins may result in diminished sperm count or motility or in an increased number of abnormal or damaged sperm.[28] Recent studies implicating male occupational

21. *See* Paul, Daniels & Rosofsky, *Corporate Response to Hazards in the Workplace: Results of the Family, Work and Health Survey*, 16 Am. J. Indus. Med. 267, 267 (1989).

22. *See Leading Work-Related Diseases and Injuries—United States*, 34 Morbidity & Mortality Weekly Rep. 537, 537 (Sept. 6, 1985). The National Institute for Occupational Safety and Health ("NIOSH") ranked reproductive disorders as the sixth highest work-related disease or injury in the United States. *See id.* An editorial note in the CDC report states that the preliminary observations reported demonstrate that the problem of occupationally-induced reproductive disorders is "widespread and serious," but notes that scientific research in the area is "in its infancy." *Id.* at 539.

23. *See* Office of Technology Assessment, Genetic Monitoring, *supra* note 9, at 67.

24. *See* Commonwealth of Massachusetts, Family, Work & Health 8 (1988); Hirsch & Mosher, *Characteristics of Infertile Women in the United States and Their Use of Infertility Services*, 47 Fertility & Sterility 618, 618 (1987). Typically, infertility is defined as "the inability of a couple to conceive after 12 months of intercourse without contraception." United States Congress, Office of Technology Assessment, Infertility: Medical and Social Choices 35 (1988) [hereinafter Office of Technology Assessment, Infertility].

25. *See* Office of Technology Assessment, Infertility, *supra* note 24, at 36.

26. For a discussion of the historically disparate impact of workplace regulation on females as a result of their gestational role, see Comment, *Birth Defects Caused By Parental Exposure to Workplace Hazards: The Interface of Title VII with OSHA and Tort Law*, 12 U. Mich. J.L. Ref. 237, 238-48 (1979).

27. *See* Barlow & Sullivan, *supra* note 16, at 5.

28. *See id.* at 5-6.

exposure in couple infertility[29] may stimulate more systematic research on the association between reproductive injuries and men's workplace environment.

Impaired fertility has been associated with exposure to a variety of workplace toxins,[30] including metals such as lead and mercury,[31] agricultural chemicals such as dichlorodiphenyldichloroethane ("DDT"),[32] several classes of industrial chemicals,[33] and physical agents such as ionizing radiation.[34] Associations between workplace substances and decreased fertility occasionally have been discovered by workers themselves, as in the case of dibromochloropropane ("DBCP"), a pesticide eventually confirmed to be spermatotoxic.[35]

A natural limitation on the study of the relationship between infertility and workplace exposures is that multiple biological processes are involved in human conception. A substance may have an adverse impact on a single isolated step in the reproductive process, such as the ability of a fertilized egg to implant in the uterus, or it may have a more complex effect on several stages in the process. Identification of both the specific injury and its cause often can be difficult.[36]

---

29. *See generally Occupational Exposure to Lead, Other Toxins May Be Missed Source of Male Infertility*, Reproductive Tech. Update, July 1991, at 78 (stating that several occupational toxins are known to harm the male reproductive system including lead, ethylene glycol ethers, and carbon disulfide).

30. This Article focuses on toxic exposures in the workplace affecting the reproductive systems of males and females and human genetic structure. The injuries discussed can be direct—such as damage to human eggs or sperm—or indirect—such as carcinogenesis affecting these systems. This Article does not address non-toxic workplace stimuli or conditions, such as physical and psychological stress.

31. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 69-74.

32. *See id.* at 74-78.

33. Examples of industrial chemicals that have been associated with disruption of fertility are polybrominated biphenyls ("PBBs"), polychlorinated biphenyls ("PCBs"), organic solvents, formaldehyde, and chloroprene, which is used in the production of rubber. *See id.* at 78-91.

34. *See id.* at 94-96. Ionizing radiation may be defined as "energy that is transmitted in wave or particle form and is capable of causing ionization (ejecting orbital electrons) of atoms or molecules in the irradiated tissue." *Id.* at 94. Ionizing radiation is distinguishable from nonionizing radiation which is produced by ultraviolet and infrared light, lasers, electromagnetic fields such as shortwave radio and microwave sources and some appliances, and video display terminals ("VDTs"). *See id.* at 96-102.

35. *See* Whorton, *Environmental and Occupational Reproductive Hazards*, in Men's Reproductive Health 193-95 (J. Swanson & K. Forrest eds. 1984). In seven follow-up studies performed in the 1970s on males exposed to DBCP in the workplace, results demonstrated that 14.5% of the men were azoospermic, while 21% were oligospermic. *See id.* at 194. The DBCP case studies represented a turning point that lent credence to the role of workplace exposures in male infertility and encouraged speculation into the role of male reproductive injuries in adverse pregnancy outcomes. *See id.* at 195.

36. *See generally* Baird & Wilcox, *Effects of Occupational Exposures on the Fertility of Couples*, 1 Occupational Medicine: State of the Art Reviews 361, 362-63 (1986) (discussing reasons for studying the fertility of couples as basis for determining toxic reproductive injuries).

2. Pregnancy-Related Injuries

Toxic exposures to a developing fetus can occur when a pregnant woman is exposed to a harmful substance in the course of employment. Damage can result not only in spontaneous abortion, stillbirth, or infant death, but also in physical, mental, or behavioral abnormalities in offspring. Furthermore, such toxic exposures can account for cancer developed by the offspring.[37] Typically, structural teratogens[38] do their greatest damage after implantation of the embryo in the uterus and through the embryonic period.[39] Functional teratogens may cause adverse effects throughout the embryonic and fetal periods.[40] The nature and extent of injury to the embryo, fetus, or offspring will depend on the timing and degree of the exposure as well as on the characteristics of the toxic substance.[41]

Workplace toxins affecting pregnancy outcome and offspring health often are the same substances that affect fertility. These substances range from chemicals and metals to physical agents such as ionizing radiation.[42] Moreover, certain maternal changes during the course of pregnancy may render both the mother and the developing fetus more susceptible to toxic exposures in the workplace. For example, normal changes in digestion and blood transport during pregnancy could enhance the detrimental effect of exposure to some toxins through ingestion or inhalation.[43]

Paternal exposures also can affect a developing fetus. For example, clothing contaminated by workplace toxins could expose a pregnant woman and the fetus she carries to the hazardous substances if that clothing is introduced into the home, similarly increasing the risk of spontaneous abortion, stillbirth, and adverse outcomes in the offspring.[44]

3. Carcinogenicity

Transplacental carcinogens[45] can trigger a carcinogenic process in the

37. *See* Barlow & Sullivan, *supra* note 16, at 6.

38. In general, a teratogen is "a drug or other agent that causes abnormal development" in a fetus. Stedman's Medical Dictionary 1418 (24th ed. 1982).

39. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 56. The embryonic period lasts from approximately the third through the eighth or ninth week of pregnancy. *See id.* at 49.

40. *See id.* at 56. The fetal period begins at approximately the eighth or ninth week of pregnancy and continues until birth. *See id.* at 49.

41. *See* Barlow & Sullivan, *supra* note 16, at 6-7; Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 57.

42. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 69-96.

43. *See* Paul & Himmelstein, *supra* note 6, at 926.

44. *See id.* The authors also note that some pharmacological agents can be present in the seminal fluid, thus creating a risk of injury to an exposed fetus. *See id.*

45. "[T]ransplacental carcinogens" are "[c]hemicals that induce cancer with gestational exposure." Barlow & Sullivan, *supra* note 16, at 7. The discussion in this Article of workplace carcinogens will be limited to gestational exposures of the embryo or fetus

fetus resulting in the infant being born with a tumor.[46] In some cases, older children of exposed workers develop cancer[47] which can be traced to their exposure to toxins in utero. For example, gestational exposure to ionizing radiation has been associated with childhood cancer.[48]

4. Genetic Hazards

Genetic mutations caused by exposure to workplace toxins have been associated with injuries such as pregnancy loss and infertility in the worker, as well as with cancer and other disease in offspring.[49] Exposure to a genotoxic substance may cause genetic damage to both the reproductive cells and non-reproductive system cells.[50] With respect to the former, a workplace toxin may cause chromosomal abnormalities during the development of the egg or sperm, preventing fertilization;[51] in cases in which fertilization does occur, the embryos often spontaneously abort.[52] As to the latter, exposure of a developing embryo or fetus to a

---

and their effect on resulting offspring. Accordingly, this Article will not address the effects of workplace exposures to carcinogenic substances on men and women of childbearing age.

46. *See id.*

47. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 96.

48. *See* M. Whorton, *Reproductive Disorders*, in Occupational Health: Recognizing and Preventing Work-Related Disease 307, 309 (B. Levy & D. Wegman 1st ed. 1983).

49. Genetic mutations have been succinctly described as

> microlesions in DNA involving the addition, deletion, or substitution of one or more nucleotides in the DNA sequence. Mutations can be inherited through the male or female germ cell or can occur in somatic cells during embryogenesis. Genotoxic damage to the germ cell, if unrepaired, can lead to sterility or can be passed on to offspring, resulting in embryolethality. . . . Mutagenic insult to somatic cells may be associated with carcinogenesis.

Paul & Himmelstein, *supra* note 6, at 924.

50. *See* D. Brusick, Principles of Genetic Toxicology 45 (1980). Research on the genetic effects of toxic substances on non-reproductive system cells, known as somatic cells, has focused on carcinogenesis. *See id.* at 45-47. Because mutations of the somatic cells have been associated with the development and growth of tumors in the immediately exposed individual, scientists have concentrated on genotoxicity within this context. *See id.*; Office of Technology Assessment, Genetic Monitoring, *supra* note 9, at 55. This Article addresses somatic mutations only insofar as they occurred as the result of exposure of a developing embryo or fetus to the occupational genotoxin. This Article also encompasses the issue of mutations in the reproductive system cells and their broader implications. For example, mutations could impact not only the immediately exposed individual, but also the descendants of that person for generations to come. *See* D. Brusick, *supra*, at 45-46.

51. Because the female's complete supply of eggs exists at the time of her birth, mutagenic exposures in adulthood may not affect the eggs. A mutagenic exposure to a female fetus in utero, however, could affect her eggs when she develops into adulthood. *See* Barlow & Sullivan, *supra* note 16, at 7. In contrast, males produce sperm continuously during adulthood and thus are more susceptible to mutagenic damage. *See id.* Studies have shown that over 90% of fetuses with chromosomal abnormalities are spontaneously aborted, *see* Paul & Himmelstein, *supra* note 6, at 924, and that over 50% of spontaneously aborted fetuses show evidence of chromosomal abnormalities, *see* Barlow & Sullivan, *supra* note 16, at 6.

52. *See* Barlow & Sullivan, *supra* note 16, at 6.

genotoxic substance could result in teratogenesis[53] or other abnormalities within that developing entity.[54]

Some genetic abnormalities are recessive.[55] Thus, an occupational exposure of either the sperm or egg to a genotoxic substance could result in an alteration that is transmitted to subsequent generations.

Only sparse research has been conducted on the relationship between genetic abnormalities and workplace chemicals or other toxins. The possibility that the mutagenic effects of a substance may not be expressed for one or more generations has hindered research efforts by complicating data collection.[56] Moreover, prediction of mutagenic effects resulting from exposures in the occupational setting is complicated by the difficulty scientists experience in distinguishing natural, spontaneous mutations from environmentally induced mutations in certain instances.[57]

B. *Epidemiological and Toxicological Considerations*

The full ramifications of toxins in the workplace are not yet known. Potentially, they can affect reproduction in both the genetic and developmental stages, from maturation of the egg or sperm to health of the fetus, child, or even subsequent generations.[58] One substance might cause different types of injuries, whereas several different substances may cause identical harms.[59] A substance's effect might manifest itself immediately or may require cumulative exposures to become apparent. In addition, some injuries may be the result of the interaction between more than one workplace toxin and the system of the worker.[60]

In the traditional personal injury claim, clinical test results are the pri-

53. Teratogenesis may be defined as "the origin or mode of production of a malformed fetus; the disturbed growth processes involved in the production of a malformed fetus." Stedman's Medical Dictionary 1418 (24th ed. 1982).

54. *See* D. Brusick, *supra* note 50, at 50-51.

55. Recessive mutations may be defined as "those events which are not expressed unless the affected individual receives mutant genes from both parents." *Id.* at 54. Because several generations may pass for the incidence of a particular mutation to increase in the human population, the expression of the mutation may not appear until such late date. *See id.* at 55; *see also* Barlow and Sullivan, *supra* note 16, at 6. For a discussion of recessive mutations, see D. Brusick, *supra* note 50, at 54-56.

56. In the words of one genetic toxicologist, "[c]oncern over genotoxicity must be developed through an awareness of the serious consequences likely to occur 100 years or more from today if human exposure to significant levels of mutagenic agents is not prevented." D. Brusick, *supra* note 50, at 46.

57. *See id.* at 109.

58. *See supra* notes 21-57 and accompanying text.

59. For example, exposure to the organic solvent styrene has been linked to spontaneous abortion as well as congenital hydrocephalus and hepatic angiosarcomas. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 130. Both DBCP and lead have been linked to low or subnormal sperm counts in men exposed to the substances. *See id.* at 69, 75.

60. *See* Whorton, *supra* note 48, at 312. "Theoretically, a single agent could cause several different outcomes: infertility, mutation, and teratogenic effect. For example, a congenital malformation, low birth weight, or a childhood behavior problem may all result from teratogens or toxins that slow the growth of crucial cell groups." *Id.*

mary means of determining both the cause of the claimant's injury and the extent of damage. With reproductive and genetic injuries, however, scientific uncertainty regarding toxicity and mutagenicity—along with insufficient insight into the disease processes—render clinical studies inadequate. This may be true even where clinical studies demonstrate the presence of a known or suspected hazardous substance in the claimant's body. Studies of sperm count and morphology, for example, have revealed strong associations between occupational exposure to certain substances and abnormal profiles.[61] Even such results, however, may be inconclusive in light of the many possible confounding factors.

Accordingly, epidemiological and toxicological studies have become the principal scientific methods of more precisely ascertaining the relationship between the occupational setting and reproductive and genetic injuries. While these studies are pervasively used, they are imperfect for a variety of reasons.

1. Epidemiological Studies

Epidemiology is a statistical methodology that employs surveys and sampling to reveal probabilities of relationships between exposures and clusters of disease or other conditions.[62] Because direct human experimentation with workplace toxins is not acceptable on ethical grounds, epidemiology is the method of choice for studying human populations. By studying groups of humans, epidemiologists draw inferences regarding the causes of individual occurrences about which clinical data alone may be insufficient.[63] By its very nature, however, epidemiology is imperfect, as it can only express probabilities and cannot identify actual causes.[64] The principal value of epidemiological studies is in determining

61. *See* Office of Technology Assessment, Genetic Monitoring, *supra* note 9, at 62.

62. "Epidemiology" is the "study of relationships between the frequency and distribution, and the factors that may influence frequency and distribution, of diseases and injuries in human populations." Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 163.

63. *See* Dore, *A Commentary on the Use of Epidemiological Evidence in Demonstrating Cause-In-Fact*, 7 Harv. Envtl. L. Rev. 429, 431-32 (1983).

64. The uncertainty inherent in epidemiological study derives from a variety of factors, which are associated with either statistical limitations or limitations in drawing a causal connection. For example, low risks may not be statistically distinguishable from background levels of the condition studied. *See* McElveen & Eddy, *Cancer and Toxic Substances: The Problem of Causation and the Use of Epidemiology*, 33 Clev. St. L. Rev. 29, 39 (1984-85). Thus, demonstrating a significant statistical risk to warrant the inference of a causal connection may be particularly problematic for early claimants who enter the legal system seeking relief prior to the establishment of a sufficient epidemiological data base on their injury. *See* Black & Lilienfeld, *Epidemiologic Proof in Toxic Tort Litigation*, 52 Fordham L. Rev. 732, 776-78 (1984) (discussing "first-case" phenomenon). Low levels of risk in some instances may be associated with low doses of a suspected toxin. Extrapolation of the effects from high doses to low doses will not necessarily result in an accurate estimate of risk. For a discussion of this problem within the context of ionizing radiation in the workplace, see McElveen & Eddy, *supra*, at 41 (noting that complicating factors may be ability of DNA to repair itself at low doses and uncertainty as to extent dividing dose may reduce carcinogenic effect of substance). With respect to

the level of risk that certain segments of the population face for contracting a particular disease or other disorder.[65]

Epidemiological studies may survey large segments of the general population to measure the occurrence rates of certain specific illnesses or conditions. The results of such studies often can establish baseline levels of the targeted conditions in the general population.[66] Occasionally, such studies may uncover statistically significant clusters of illness that warrant further study. In another variant, researchers may elect to examine a narrow subdivision of the population, such as workers exposed to ionizing radiation.[67] Epidemiological studies can be either prospective or retrospective,[68] with the former focusing on ongoing surveillance of a given exposed population and the latter focusing on persons who have developed a particular injury.[69]

Use of epidemiological studies to determine medical causation, however, can be problematic for several reasons. First, bias in the design of

---

causation, epidemiologists impose an element of subjective personal judgment on the statistical data in reaching a conclusion regarding the existence or nonexistence of a causative factor. *See* J. Mausner & S. Kramer, Epidemiology—An Introductory Text 180-87 (2d ed. 1985) (explaining epidemiologist's analysis of statistical association between factor and disease to determine whether relationship is causal, spurious, or indirect). Because the causal determination can be dependent upon evaluation of a multitude of factors, the determination often may be open to various interpretations.

65. *See* Wegman & Giusti, *Epidemiology*, in Occupational Health: Recognizing and Preventing Work-Related Disease 51, 51 (B. Levy & D. Wegman 1st ed. 1983).

66. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 163.

67. *See id.* at 163-64.

68. Prospective studies, often called cohort studies, typically survey populations of exposed individuals to determine whether those individuals have an increased risk over the general nonexposed population of developing disease. *See* Gordis, *Epidemiologic Approaches for Studying Human Diseases in Relation to Hazardous Waste Disposal Sites*, 25 Hous. L. Rev. 837, 839 (1988). Assuming that the nonexposed population will manifest disease levels, the risk associated with the exposure surveyed is determined by comparing the rate of particular diseases in the exposed population to the background rates of the same diseases in the nonexposed population. If this comparison yields a "relative risk" rate that is higher in the exposed population, the degree to which the incidence of disease is greater in the exposed population is examined to determine whether a causal association can be drawn between the exposure and the elevated incidence of disease. *See id.* at 840. Epidemiologists first determine the "attributable risk," which reflects the difference between the background levels of the disease in the nonexposed population and the level of the disease in the exposed population. They then undertake a more subjective analysis to test the plausibility of the proposed causal connection and either discount or incorporate confounding factors. *See id.* at 840-43.

Unlike prospective studies, retrospective, or case-control studies, typically begin by identifying individuals who have contracted a particular disease and compare the characteristics of the group identified with a group of individuals who have not contracted the disease. *See id.* at 838. Case-control studies are particularly susceptible to bias in design of the study, as where the researcher has in mind an association between a particular exposure and the disease and selects a disease group and control group reflecting that bias. *See id.* at 838-39.

69. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 164-65.

the study can impair researchers' ability to obtain accurate results.[70] Second, epidemiological studies are dependent upon a sufficiently large sample size to reveal subtle, yet significant, statistical variations that may identify a causal relationship.[71] Results must be distinguishable from background levels of the disease or disorder in the general population. Studies of worker populations are necessarily small, frequently masking relationships that might otherwise be apparent had the sample size been larger.[72] Furthermore, the effect of a long latency period—particularly problematic in the case of genetic injury—may produce false negatives if the study has not accurately estimated an appropriate time period for manifestation of the condition.

Third, accurate analysis of reproductive and genetic injuries may be difficult because infertility can result from a variety of dysfunctions involving the male, the female, or the couple combined.[73] Even where a single source of infertility can be identified, such as male infertility, accurate assessment of the injury may be obscured by inconsistent laboratory procedures and disagreement among scientists as to the optimum parameters for study.[74] A single toxic substance might affect reproduction at multiple points, from conception through birth and development of the offspring.[75] Often, reproductive or genetic injuries are so rare in the pop-

70. *See* Wegman & Giusti, *supra* note 65, at 63.

71. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 166-67.

> For example, in order to detect a twofold increase in the spontaneous abortion rate (during the period from the point at which a pregnancy is recognized to 20 weeks gestation), 161 pregnancies are needed in both the exposure group and the control group. In order to study this many pregnancies, the investigator must draw . . . from a population of more than 11,000 workers to find a sufficient number of pregnancies to study.

Id. (citing Rosenberg, Feldblum, Shy & Marshall, *Epidemiologic Surveillance of Occupational Effects on Reproduction*, Office of Technology Assessment, Contract Rep. (1984)).

72. *See id.* at 166.

73. *See* Office of Technology Assessment, Infertility, *supra* note 24, at 36: Conkling, *The Long Road from Infertility to Fatherhood*, L.A. Times, Mar. 14, 1991, at E20, col. 1; Ellis, *New Infertility Treatment Returns Patients to Work Faster*, Reuter Libr. Rep., June 4, 1991. *See generally supra* notes 24-36 and accompanying text (discussing infertility).

74. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 165. Other difficulties arise in the study of spontaneous abortions:

> It has been estimated that only about 31 percent of all fertilized eggs survive to term: about 16 percent do not make the first cell division, another 15 percent are lost during the first week, and a further 27 percent during implantation. By the time of the first missed menstrual period, only about 42 percent of the fertilized eggs have survived. Many women thus spontaneously abort without realizing that they have been pregnant.

Id. (citations omitted). A survey based upon the number of reported spontaneous abortions would not accurately reflect the total number of actual spontaneous abortions. Thus, accurate analysis of the relationship between a particular exposure and spontaneous abortion could be obscured, depending upon the parameters selected for study and the limitations of medical capabilities.

75. For example, mercury may cause spontaneous abortion, a low birth weight, congenital malformation, and the abnormal development of the nervous system. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 72. Similarly,

ulation that epidemiological study would require a prohibitively large population sample to determine the existence of a causal relationship.[76] These problems are exacerbated in cases of genetic injury, which can be expressed randomly in a variety of manifestations.[77]

Fourth, confounding factors[78] can impede accurate analysis of statistical information obtained through epidemiological studies. Cigarette smoking is the classic confounding factor in many epidemiological studies generally. In studies of human reproductive function in particular, male factors and age of the couple are common confounding factors.[79]

Finally, epidemiological studies generally are not favored for the study of genetic workplace injuries. Occupationally induced genetic disorders often are indistinguishable from spontaneous mutations in the general population.[80] In addition, epidemiological surveillance of the offspring of exposed workers would entail prohibitively long periods of time and would incur great expense.[81]

2. Toxicological Studies

Toxicological studies may offer some advantages over epidemiological studies. These studies are conducted both in vitro and in vivo on animals, resulting in correlative associations between human disease and exposure to certain substances.[82] The ability of the researcher to control the exposure dose and route gives toxicological studies an advantage over epidemiological studies.[83] Any conclusions regarding human exposure and response, however, must derive from statistical extrapolation from the animal or in vitro data to the projected human responses.

Critics of animal testing discount the value of such tests by noting significant differences between the human reproductive system and the reproductive systems of frequently used test animals.[84] Moreover, these critics have challenged the underlying assumption that dose-disease relationships observed in animal studies are predictive of similar relationships between humans and their exposure to certain substances. The preponderance of variables—such as genetic predisposition to disease or

---

alcohol consumption also has been associated with spontaneous abortion, as well as with physical and developmental abnormalities in offspring. *See id.*

76. *See* Wegman & Giusti, *supra* note 65, at 64; Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 166-67 & table 6.2.

77. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 165.

78. A "confounding factor" is a "variable that is correlated with both exposure and outcome. It can therefore partially or wholly account for an apparent effect of the exposure levels under study or mask an underlying true association." *Id.* at 167.

79. *See id.*

80. *See* Brusick, *supra* note 50, at 109.

81. *See id.*

82. *See* Office of Technology Assessment, Genetic Monitoring, *supra* note 9, at 60.

83. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 167.

84. *See id.* at 168.

dysfunction[85]—that could enter into the equation dilutes the merit of strict mathematical extrapolation.[86] Nevertheless, animal studies may prove useful in suggesting a causal relationship between certain environmental substances and reproductive and genetic diseases and disorders.[87]

The short-term bioassay, commonly known as the Ames test, examines the effects of suspected mutagenic agents[88] on rapidly reproducing bacteria in vitro.[89] Some analysts have expressed concern that the test is underinclusive because mutagenic substances may kill some cells and because the test cannot measure mutations unless the cell is viable.[90] Moreover, due to the large amounts of the suspected mutagen that must be used in the test, critics have complained that the test yields a high percentage of false positives.[91] Finally, extrapolation down to achieve prediction of low-dose hazards has been subject to conflicting standards.[92]

Both animal toxicological studies and short-term bioassays are commonly used by researchers studying environmental genetic mutations, as epidemiological studies can prove overly protracted and costly.[93] The processes by which the various toxicological testing methods are used to predict genetic risk to either human somatic cells or germ cells are undergoing constant reevaluation and improvement.[94] Toxicological studies have suggested an association between reproductive injuries and exposure to toxins in the workplace, and many scientists believe in the reliability of such studies.[95] Thus, despite their flaws, such studies may

85. *See id.* at 170.

86. *See generally id.* at 168-70 (discussing the general considerations of toxicological studies). An associated concept is the belief among the scientific community that certain threshold, or resistance, levels exist for toxic substances. *See id.* at 170.

87. For example, rodent studies have proven to be particularly useful in determining the causes of some genetic mutations in humans. *See* D. Brusick, *supra* note 50, at 110.

88. The Ames test can demonstrate direct mutagenic effects. To a great extent, however, mutagenic results correlate with carcinogenicity. *See* Office of Technology Assessment, Genetic Monitoring, *supra* note 9, at 61 (reporting that 85% of substances carcinogenic to rodents are mutagenic in Ames test).

89. Typically, the test

> involves treating a bacterial cell population containing a designated genetic marker with a mutagen. The mutagen kills off a fraction of the cell population with survivors growing back into a larger population. Within this survivor population, a fraction of the cells will have lost the marker. This fraction, expressed as a percentage, is taken as a measure of the mutagenic action suffered by the original population.

Id.

90. *See id.*

91. *See id.*

92. *See id.*

93. *See* D. Brusick, *supra* note 50, at 109-10.

94. *See generally id.* at 110-22 (presenting approaches to definition of genetic risk).

95. *See generally* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 67-104 (reporting studies that demonstrate the effects of specific chemicals on the reproductive function).

prove valuable in establishing the risk of injury presented by some workplace toxins.

D. *Scientific Uncertainty Breeds Legal Obstacles*

The nature of reproductive and genetic injuries, coupled with the technological limitations of scientific inquiry, have created an almost unprecedented level of uncertainty for the legal system. Accordingly, application of traditional legal doctrines to these issues may prove fruitless for claimants and may operate as a windfall for employers. Perhaps because these workplace injuries mimic injuries to the population at large, the legal community has been slow to recognize them as a discrete category of injuries compensable under either the tort or workers' compensation regimes.[96]

The problems inherent in the scientific processes of investigation render a legal determination of a causal relationship between an occupational exposure and a reproductive or genetic injury difficult.[97] The level of certainty required to pronounce a causal relationship in scientific endeavors is much higher, however, than the preponderance of the evidence standard employed to establish legal causation in civil jurisprudence in this country. Even so, the legal system has been reluctant to recognize a causal relationship in the absence of scientific concurrence. Courts are sharply divided on the value of permitting epidemiological and toxicological studies to be introduced into evidence on proof of general causation, absent significant corroborating evidence of specific causation.[98]

The conflict between medical and legal causation is being played out in both the workers' compensation and tort liability forums. The inherent natures of the workers' compensation and tort systems have deterred efforts to establish a niche for reproductive and genetic workplace injuries in the remedial structure of the American legal system. As will be discussed below, both the tort and compensation systems carry with them

96. The scientific community has manifested a similar reluctance in its pervasive disinterest in the scientific study of the effects of industrial toxins on the reproductive systems of employees. Moreover, public misperception of reproductive and genetic injuries may indirectly inhibit research by directing funding initiatives away from these endeavors. Certain reproductive harms—particularly infertility and spontaneous abortion—traditionally have been viewed as a lesser concern to society, possibly because these harms are erroneously considered neither life-threatening nor disabling to the worker. Genetic injuries are often viewed as abstract and theoretical because they may not be manifested until future generations, yet the danger to workers and their progeny is concrete.

97. Commentators have observed the difficulties in attempting to define such terms as "adverse health effects," "early health impairment," and even "death" within the context of epidemiological studies: "Is a lung function test abnormal when it is less than 80 percent of predicted or when it is less than 75 percent of predicted? . . . Is cause of death as listed on a death certificate accurate?" Wegman & Giusti, *supra* note 65, at 65.

98. *Compare* Ferebee v. Chevron Chem. Co., 736 F.2d 1529 (D.C. Cir.) (allowing case to go to jury on epidemiological evidence and testimony of treating physicians), *cert. denied*, 469 U.S. 1062 (1984) *with* Cook v. United States, 545 F. Supp. 306 (N.D. Cal. 1982) (disallowing claim based upon epidemiological evidence).

archaic baggage and are characterized by overmythologized presumptions. Any effort to carve a remedial niche for reproductive and genetic workplace injuries must necessarily confront these myths and presumptions.

II. A CRITIQUE OF THE EXISTING REMEDIAL SCHEME FOR TOXIC WORKPLACE INJURIES

Although the OSH Act[99] vests primary regulatory authority for rulemaking and enforcement related to workplace hazards in the Occupational Safety and Health Administration ("OSHA"), it does not grant OSHA or any other agency the authority to compensate victims of occupational injury or disease.[100] Instead, it expressly reserves any existing remedial schemes available under state or federal law.[101] With rare exceptions,[102] state workers' compensation programs and tort liability schemes are the primary, and typically the exclusive, avenues of recovery for workers injured in the workplace. Trapped in ancient modes and presumptions, these remedial schemes are inadequate to resolve the legal issues presented by toxic reproductive and genetic injuries.[103]

A. *The Paradox of Workers' Compensation*

Workers' compensation was designed to grant expedited—if not complete—compensation to workers injured during the course of employment without requiring employees to demonstrate employer fault.[104] In theory, the compensation system treats the costs of worker injuries as a cost of production to be passed to the consumer.[105] At its inception, workers' compensation contemplated remuneration for accidental inju-

99. OSH Act, *supra* note 3, §§ 651-78.

100. *See* Viscusi, *supra* note 4, at 61-62.

101. *See* OSH Act, *supra* note 3, § 653(4).

102. State law does not always allow recovery for individuals injured in the workplace. For example, coal miners suffering from pneumoconiosis were often unable to collect benefits for death or disability under state compensation plans. Congress specifically addressed the coal miners' inability to recover benefits under state law when it enacted legislation to allow recovery for death or disability due to pneumoconiosis. *See* 30 U.S.C. § 901(a) (1988).

103. *See generally* OSH Act, *supra* note 3, § 651 (addressing need to assure safe and healthful working conditions).

104. *See* Haas, *On Reintegrating Workers' Compensation and Employers' Liability*, 21 Ga. L. Rev. 843, 846-48 (1987).

105. 1 A. Larson, Workmen's Compensation Law § 2.20, at 5 (1990). One commentator has stated that "[i]n a very real sense, those who work in a contaminated environment sacrifice their health to the profit of employers who, for whatever reason, do not provide a healthy workplace," thus rendering it appropriate to charge the employer and the employer's product's consumers with the costs of compensation of work-related injuries. Robblee, *The Dark Side of Workers' Compensation: Burdens and Benefits in Occupational Disease Coverage*, 2 Indus. Rel. L.J. 596, 623 (1978).

ries only.[106] Eventually, however, occupational disease,[107] to one degree or another, came to fall within the rubric of compensable injuries under the state workers' compensation statutes.[108]

Workers' compensation was aimed at offering a compromise to both employers and employees as a means of avoiding the perceived failures of tort law. No-fault liability and abrogation of the common-law defenses of contributory negligence, assumption of the risk, and the fellow-servant rule benefitted the employee,[109] as did an overall speedier resolution of the claim than occurs in the civil litigation process. The employer benefitted from a reduction in overall accident costs by receiving both statutory limitations on employee recovery as well as immunity from tort liability.[110] A related but broader social aim of workers' compensation was industrial safety. Employers faced with the high expense of multiple compensation claims presumably would elect to invest in workplace safety measures rather than continue to risk the financial uncertainty and unprofitability of industrial accidents.[111]

These underlying presumptions of the workers' compensation system, however, break down when applied to toxic reproductive and genetic injuries in the workplace. Although workers' compensation was created to help workers recover for injuries suffered on the job, the requirements of these statutes hinder recovery for toxic reproductive and genetic injuries sustained in the workplace. First, because of the nature of these injuries, claimants may not be able to satisfy the prerequisites for coverage.[112] Second, an injured worker may have difficulty demonstrating the neces-

106. Larson, *Occupational Diseases Under Workmen's Compensation Laws*, 9 U. Rich. L. Rev. 87, 87 (1974).

107. Initially, interest in occupational disease in this country arose in the 1930s with public awareness of the relationship of silicosis to employment. Legislation designed to provide compensation for silicosis victims proved unsatisfactory for claimants because it catered to industry. *See* Robblee, *supra* note 105, at 599-600. The silicosis situation was representative of the general climate of limitation and suspicion that other work-related diseases encountered. *See id.* at 600.

108. Professor Larson has noted that in 1914, one court stated its inability to identify any cases in which the common law allowed recovery for occupational disease. *See* Larson, *supra* note 106, at 87 (quoting Adams v. Acme White Lead & Color Works, 182 Mich. 157, 159, 148 N.W. 485, 486 (1914)). The bias of the workers' compensation system, therefore, reflected this bias at common law. The bias derived from several sources. Originally, occupational disease was deemed to be associated with the standard conditions of employment, rather than employer misbehavior. *See id.* Second, the potential scope of occupational disease claims raised the question whether their inclusion within workers' compensation would impose a financial drain on the system. *See id.* at 88.

109. *See* Note, *Compensating Victims of Occupational Disease*, 93 Harv. L. Rev. 916, 918 (1980).

110. *See* Maakestad & Helm, *Promoting Workplace Safety and Health in the Post-Regulatory Era: A Primer on Non-OSHA Legal Incentives That Influence Employer Decisions To Control Occupational Hazards*, 17 N. Ky. L.J. 9, 18 (1989).

111. Peirce & Dworkin, *Workers' Compensation and Occupational Disease: A Return to Original Intent*, 67 Or. L. Rev. 649, 653 (1988).

112. *See infra* notes 117-25 and accompanying text.

sary showing of work-relatedness of the illness.[113] Third, the availability of compensation benefits is drastically reduced because many employees who develop reproductive or genetic injuries are not physically disabled from work.[114] Fourth, procedural obstacles may block recovery for these injuries.[115] Finally, the exclusivity rules of workers' compensation statutes may bar workers from bringing common-law tort actions against their employers even if the injuries involved are not compensable under the relevant statute.[116] The inequalities that exist for victims of reproductive and genetic workplace injuries undermine the compensation and deterrence goals of workers' compensation.

1. Impediments to Coverage

Victims of reproductive and genetic injuries encounter several coverage problems under existing workers' compensation statutes. First, these statutes only compensate injuries that occur in the course of the employment relationship.[117] With some reproductive and genetic injuries, the latency period from the time of the worker's exposure to the manifestation of the injury can be of considerable length, often going beyond the period of employment. This characteristic, coupled with the background frequency of reproductive and genetic dysfunction in the general population, can mask a true workplace injury, thus preventing coverage under the workers' compensation scheme.

The principal difficulty faced by reproductively or genetically injured workers is proving that exposure to workplace toxins caused their injuries. Because such injuries frequently masquerade as "ordinary diseases of life," an employer may have little or no basis for agreeing that the injury could be characteristic of or peculiar to the workplace environment. This dilemma is complicated by the possibility that some reproductive or genetic injuries could have multiple causes, some within the workplace and some outside it.[118] Some workers' compensation statutes have attempted to address this problem by expressly allowing recovery if the worker has been exposed to a substance in the workplace for a certain amount of time.[119] Such provisions, while solving problems on one end

113. *See infra* notes 126-33 and accompanying text.
114. *See infra* notes 134-40 and accompanying text.
115. *See infra* notes 141-46 and accompanying text.
116. *See infra* notes 147-76 and accompanying text.
117. *See* 2A A. Larson, Workmen's Compensation Law § 65.13, at 12-13 (1990). If a disease is manifested after the employment relationship terminates, but was caused by an exposure during the course of employment, workers' compensation should be the exclusive remedy. *See* Gideon v. Johns-Manville Sales Corp., 761 F.2d 1129, 1145 (5th Cir. 1985). In *Gideon*, the Fifth Circuit held that the state workers' compensation statute provided the exclusive remedy for an asbestos insulation worker against an employer for whom he had worked from 1944 to 1969. *See id.* The plaintiff developed asbestosis subsequent to termination of the employment relationship, but was nevertheless held to the exclusivity requirement of the workers' compensation statute. *See id.*
118. *See supra* note 75 and accompanying text.
119. *See* Cal. Lab. Code § 5500.5 (West 1989); Ky. Rev. Stat. Ann. § 342.316(10)(a)

of the spectrum, may create problems on the other end. Thus, some more acute injuries that may be directly employment-related may not be covered, whereas those with a longer latency period would fall within the coverage requirements.[120]

Second, many state workers' compensation statutes require that for an occupational disease to be compensable, it must be of the type that is associated specifically with the occupation, rather than with everyday life more generally. To this end, occupational diseases may be expressly scheduled in the compensation statute.[121] The appearance of a disease in a schedule creates a presumption that the disease is adequately associated with a particular workplace so as to merit compensation within the statutory scheme. Nonscheduled occupational diseases, however, also may fall within the compensation scheme if the particular disease is "peculiar to" the worker's occupation.[122] For example, the Michigan workers' compensation statute provides coverage for illnesses that "are characteristic of and peculiar to the business of the employer . . . [;] [O]rdinary disease[s] of life to which the public is generally exposed outside of the employment [are] not compensable."[123]

Third, some workers' compensation statutes require the claimant to have worked for a specified minimum period of time before the occupational disease claim will be covered.[124] Victims of many reproductive and genetic injuries, therefore, may be effectively foreclosed from compensation if their injuries manifest themselves before the statutory time has elapsed.

Finally, state workers' compensation coverage is expressly directed to-

(Michie Bobbs-Merrill 1983); La. Rev. Stat. Ann. § 23:1031.1(D) (West 1985); Mont. Code Ann. § 39.72.102(10) (1991); Nev. Rev. Stat. Ann. § 617.430 (Michie 1992). The Idaho workers' compensation statute relieves an employer of compensation liability for any "nonacute" occupational disease if the employee was exposed to the hazard in the employer's workplace for less than 60 days. Idaho Code § 72-439 (1989). The term "nonacute" is not defined in the statute.

120. Very long latency periods from time of exposure until manifestation of the injury create special problems for claimants, particularly in the area of proof. *See infra* notes 233-65 and accompanying text.

121. *See, e.g.*, Pa. Stat. Ann. tit. 77, § 1208 (Purdon 1952 & Supp. 1991) (listing diseases within the scope of the term "occupational disease" as used in the statute).

122. Many statutes exclude any diseases of ordinary life. *See* Ark. Stat. Ann. § 11-9-601(e)(1) (1987); Colo. Rev. Stat. § 8-41-108(3) (1986); Del. Code Ann. tit. 19, § 2301(4) (1985); Fla. Stat. Ann. § 440.151(f)(2) (West 1981); Ga. Code Ann. § 34-9-280(2) (1988); Idaho Code § 72-438 (1989); Ill. Ann. Stat. ch. 48, para. 172.36(d) (Smith-Hurd 1986); Ind. Code Ann. § 22-3-7-10(a) (Burns 1992); Kan. Stat. Ann. § 44-5a01(b) (1986); Md. Lab. & Empl. Code Ann. § 9-101(g) (1991); Minn. Stat. Ann. § 176.66 (West 1966); Mo. Ann. Stat. § 287.067 (Vernon 1965); Neb. Rev. Stat. § 48-151(3) (1988); N.H. Rev. Stat. Ann. § 281:2(V) (1987); N.D. Cent. Code § 65-01-02(7)(a) (1985); S.C. Code Ann. § 42-11-10(4) (Law. Co-op. 1985); Vt. Stat. Ann. tit. 21, § 1002(5) (1987); Va. Code Ann. § 65.2-400 (1991); W. Va. Code § 23-4-1 (1985).

123. Mich. Comp. Laws Ann. § 418.401(2)(b) (1985 & West Supp. 1991-92).

124. *See* Edwards, *Worker Right-To-Know Laws: Ineffectiveness of Current Policy-Making and a Proposed Legislative Solution*, 15 B.C. Envtl. Aff. L. Rev. 1, 32-33 (1987) (discussing various state laws).

ward employees, while reproductive and genetic injuries may affect spouses and offspring of exposed workers. Typically, independent contractors and persons present in the workplace, but not in an employment capacity, are excluded from coverage under workers' compensation statutes.[125] Similarly, spouses, children, and others with illnesses or disorders resulting from a worker's exposure to workplace toxins generally will be limited to the tort system for redress of their individual injuries.

2. Problems of Proof

Even when a worker meets the technical coverage requirements of the relevant workers' compensation statute, the worker may be barred from compensation by serious problems of proof. While a claimant may be able to adequately demonstrate that exposure to a particular substance in the workplace is toxic to the human reproductive system, the claimant may be unsuccessful in proving that the specific injury was in fact related to that occupational exposure. This work-relatedness issue is the compensation system's analog to proof of proximate cause in a civil negligence action. Many of the problems are identical within both systems for persons with occupational reproductive or genetic injuries.

Most states impose the burden of proving work-related causation upon the claimant.[126] In occupational disease cases, the claimant must introduce clinical medical evidence to support the claim of specific causation of the disease or disorder.[127] Thus, the statistical probabilities offered by epidemiological studies may not be sufficient to prove the necessary work-relatedness of the claimant's reproductive or genetic injury. In other toxic-exposure cases, in which the injury claimed was cancer, workers' compensation boards and courts often have denied coverage based upon insufficient proof of a causal connection.[128] The rare excep-

125. *See* 1C A. Larson, The Law of Workmen's Compensation § 44.33(b), at 8-119-30 (1991).

126. *See* McCreary v. Industrial Comm'n, 104 Ariz. Adv. Rep. 22, 24 (Jan. 28, 1992); Board of Educ. of Chicago v. Industrial Comm'n, 83 Ill. 2d 475, 479, 416 N.E.2d 237, 238 (1981); Sondag v. Ferris Hardware, 220 N.W.2d 903, 905 (Iowa 1974); Peirce & Dworkin, *supra* note 111, at 662.

127. *See* Haynes v. Industrial Comm'n, 19 Ariz. App. 559, 562, 509 P.2d 631, 633 (1973); Neas v. Snapp, 221 Tenn. 325, 331, 426 S.W.2d 498 (1968); General Chem. Div., Allied Chem. & Dye Corp. v. Fasano, 47 Del. 546, 549, 94 A.2d 600, 602 (1953); Robblee, *supra* note 105, at 602.

128. *See* Miller v. National Cabinet Co., 8 N.Y.2d 277, 283, 168 N.E.2d 811, 814, 204 N.Y.S.2d 129, 133 (1960) (benzene). A related problem in toxic-exposure cases is that of dual causation and pre-existing conditions. This issue arises when the claimed injury may have arisen from both a workplace exposure and another, non-work related source. For example, when a worker develops lung cancer or other respiratory illness from an exposure to air-borne fumes in the workplace, but was a smoker, the question arises as to whether the illness is compensable under the state workers' compensation statute as work-related. Similarly, a spontaneous abortion could have several causes, including the workplace exposure of the mother to a hazardous substance. Due to the general high incidence of background mortality of embryos, the exposed worker may have considerable difficulty proving that the workplace exposure contributed to any degree to the injury.

tions to the specific-causation rule have been where a traumatic workplace event is followed by an almost immediate onset of disease,[129] an improbable, albeit not impossible, situation within the realm of toxic reproductive and genetic injuries. Moreover, the claimant's burden of proof is not mitigated by a lack of existing scientific knowledge as to the causal connection between a toxic substance and the injury.[130]

These proof problems are especially onerous when the disease is characterized by a long latency period. One unique characteristic of latent reproductive injuries is that some potential claimants may not discover infertility problems until they become involved in reproductive efforts many years after exposure. To meet proof requirements, the claimant must produce evidence showing the extent of exposure, the absence of the problem prior to the occupational exposure, and the elimination of other potential contributing causes outside of the employment environment.[131] For example, such factors as age and heredity can inhibit a finding of coverage for a reproductive or genetic injury.[132] The difficulty of such proof requirements is exacerbated by the frequent unavailability of employer records regarding the extent of occupational exposure.[133]

3. Limitations on Benefits

Once a worker overcomes the coverage and proof hurdles of the relevant workers' compensation statute, the worker still may have inade-

---

The employer, on the other hand, may be unable to identify other contributing causes of the injury because current medical knowledge cannot identify with certainty most causes of early spontaneous abortions. How should the law treat this situation? If the claimant can come forth with proof of general causation—that is, that the workplace hazard has been statistically linked to spontaneous abortions—the burden should be placed upon the employer to disprove work-relatedness or to identify multiple-causation factors or a pre-existing condition. In this manner, the safety goals of the workers' compensation system can remain intact. Depending upon the jurisdiction, the employer may then argue that apportionment should be applied to the claim. For the argument that existing judicial treatment of dual causation and pre-existing condition claims results in overinclusive payment of workers' claims and that current state apportionment statutes are improperly utilized, see Peirce & Dworkin, *supra* note 111, at 667-78.

129. *See* Robblee, *supra* note 105, at 603 (discussing Volk v. Birdseye Div., 16 Or. App. 349, 518 P.2d 672 (1974) (broccoli dusted within unknown substance in eye) and Valente v. Bourne Mills, 77 R.I. 274, 75 A.2d 191 (1950) (breast injury from flying object in which no medical evidence had been submitted to prove causation, but in which compensation was allowed)).

130. *See* Robblee, *supra* note 105, at 605.

131. *See supra* notes 97-98 and accompanying text.

132. *See* Peirce & Dworkin, *supra* note 111, at 663.

133. Current federal regulations concerning employer recordkeeping requirements may provide assistance for employees seeking their personal exposure records. The federal regulations provide that employers must transfer all employee exposure and medical records to successor employers. In the event of company dissolution, the employer must notify the affected employees of their right of access to the records. *See* 29 C.F.R. § 19.20(h) (1990). While such regulations are useful prospectively, they are of very limited use retrospectively, particularly for persons exposed many years ago. For a discussion of the merits of these regulations, see Gelman, *Compensable Industrial Disease a Catching Idea*, 125 N.J.L.J. 748 (1990).

quate relief. This is because the benefits available to an injured worker under state workers' compensation schemes generally are limited to those relating to economic loss and disability.[134] By the very nature of the workers' compensation compromise, compensation is not available for pain and suffering. While deemed appropriate for the accidental or traumatic injury contemplated by the original drafters of workers' compensation legislation, this compensation limitation is unsuitable for reproductive and genetic injuries.

Indeed, the typical benefits schemes fail to provide adequate compensation to the victim of reproductive and genetic occupational injuries for several reasons. First, the concept of a disability contemplates an inability to perform work activities. A worker's infertility, sexual dysfunction, or spontaneous abortion, for example, may result in no disability from work, even though the injury may be severe. Second, medical expenses contemplate nothing more than reimbursement or payment for actual out-of-pocket expenses incurred. Thus, the worker may be denied compensation for the true losses associated with reproductive injury.

Furthermore, state workers' compensation statutes typically limit medical benefits to those that are reasonably required or necessary.[135] Thus, payment for medical treatment that directly relates to the health of the worker will be covered, provided that the treatment has a reasonable expectation of success. Workers suffering from occupational reproductive and genetic injuries will confront considerable obstacles to treatment for their injuries. If the jurisdiction defines "necessary" as required to protect the life and health of the worker, tribunals could refuse to compensate claimants for most infertility and pregnancy-related treatments, many of which are prohibitively expensive and which may not be covered by any health insurance carried by the claimant.[136] Moreover, many pregnancy-related treatments are intended to be therapeutic to the fetus. Employers may attempt to argue that noncoverage is justifiable on the ground that the treatment was for a noncovered third party rather than for the mother's own health.[137]

134. Typically, such benefits include loss of earnings, medical expenses, death, and disability maintenance. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 289-91. In Michigan " 'disability' means a limitation of an employee's wage earning capacity resulting from a personal injury or work-related disease. The establishment of disability does not create a presumption of wage loss." Mich. Stat. Ann. § 17.237(401) (Callaghan 1988).

135. *See* 2 A. Larson, The Law of Workmen's Compensation § 61.00 (1989).

136. *See* Office of Technology Assessment, Infertility, *supra* note 24, at 148-57.

137. Whether a jurisdiction would find in favor of the employer on this argument would necessarily depend upon the state's definition of the commencement of human life. This might vary from state to state. In Webster v. Reproductive Health Services, 492 U.S. 490, 506-07 (1989), the United States Supreme Court declined to rule on the constitutionality of the preamble of a Missouri statute regulating abortion. *See id.* at 501 (construing Mo. Rev. Stat. §§ 1.205.1(1),(2) (1990)). The preamble proclaims that "[t]he life of each human being begins at conception" and provides that "unborn children have protectable interests in life, health, and well-being." Mo. Rev. Stat. §§ 1.205.1(1),(2) (1990). If such statutory proclamations withstand appropriate constitutional challenge,

Treatments considered experimental in the medical profession are not covered under workers' compensation.[138] Some infertility treatments,[139] and virtually all gene therapy treatments, currently are experimental. Even where not experimental, many infertility treatments, such as in vitro fertilization, have statistically low success rates. Thus, the nature of the treatments available for reproductive and genetic injuries could cause tribunals to deny payment of such expensive treatments on the ground that they are unlikely to succeed.[140]

4. Procedural Issues

Workers with reproductive and genetic injuries also encounter many procedural problems under existing workers' compensation laws. For example, some state laws require that an occupational disease must manifest itself within a specified period of time following either the last exposure to the toxic substance or the employee's last day of work.[141] A long latency period from time of exposure to manifestation of the disease would preclude many claimants from obtaining compensation for reproductive or genetic injuries. Indeed, genetic impairments can remain latent for generations,[142] thus preventing the exposed worker from ever learning of the primary personal injury. By the time the defect does appear, it may be untraceable to the original work environment. Even if the disorder is traceable, the likelihood is high that any statutory manifestation requirements will have expired by the time the disorder is discovered.

Similarly, statutory limitation periods for the filing of compensation claims can adversely and disparately impact claimants of reproductive or genetic injuries. Initially, most states adopted strict limitation periods that ran from the time of exposure.[143] Today, however, most states have

---

employers in the affected states could argue that embryos and fetuses are human beings with rights distinct from—and potentially in conflict with—the women who carry them. Such an argument could be counterproductive to the goals of the workers' compensation system, if employers are permitted to refuse to compensate pregnant workers for pregnancy-related treatment necessitated by workplace exposures.

138. *See* 2 A. Larson, The Law of Workmen's Compensation § 61.13 (1989) (use of "necessary" in statutory language impedes use of experimental techniques).

139. For example, the medical profession considers zygote intrafallopian transfer ("ZIFT") and peritoneal ovum and sperm transfer to be experimental. *See* Ethics Comm. of Am. Fertility Soc'y, *Ethical Considerations of the New Reproductive Technologies*, 53 Fertility & Sterility 55S (Supp. 2 1990).

140. *See, e.g.*, Marks, Bressler & Marks, *Recovery for Sexual Impairment Under Workers' Compensation*, 35 Fed'n of Ins. Couns. Q. 107, 114-17 (1985) (discussing case involving impairment of procreative ability in which authors were defense counsel).

141. *See, e.g.*, Ark. Stat. Ann. § 11-9-702(2)(A) (1987) (disablement must occur within three years of last exposure for silicosis or asbestosis); Ill. Ann. Stat. ch. 48, para. 172.36(f) (Smith-Hurd 1986) (disablement must occur within two years of last exposure to most substances); S.C. Code Ann. § 42-11-70 (Law. Co-op. 1985) (disease must be contracted within one year of last exposure to most substances).

142. *See supra* notes 55-56 and accompanying text.

143. *See* Gelman, *supra* note 133, at 756.

amended their statutes to provide that a compensation claim for occupational disease accrues upon discovery of the disease or when the disease could reasonably have been discovered, whichever is earlier.[144] The states vary as to when sufficient medical knowledge is imputed to the claimant for accrual of the claim. In some states, mere diagnosis of the disease may suffice;[145] in other states, the statute may not be triggered until the first medical opinion is rendered tending to establish a causal connection between the disease and the occupational setting.[146]

These liberal approaches to the discovery of disease, however, do not offer much protection to victims of occupational reproductive and genetic injuries. Because of the prevalence of infertility and spontaneous abortion in the general population, a worker initially may have no reason to believe that such a condition would be related to employment. Indeed, it may not be until the worker suffers multiple spontaneous abortions, for example, that she would begin to suspect an occupational exposure as the source of the problem. Yet, employers in some jurisdictions could challenge the worker's claim as dilatory, arguing that an earlier date—perhaps the time of the first spontaneous abortion—triggered the running of the statutory period.

5. The Perils of Exclusivity

A major hurdle to relief that victims of reproductive and genetic injuries face under existing workers' compensation laws is the exclusivity doctrine. The foundation of the workers' compensation system, this doctrine provides that workers' compensation is the exclusive remedy for an

144. *See, e.g.*, Ga. Code Ann. § 34-9-281 (1988 & Supp. 1991) (but not more than seven years from last exposure); N.Y. Work. Comp. Law § 28 (McKinney Supp. 1992) (within two years after disablement and after the claimant knew or should have known that disease was due to nature of employment); Vt. Stat. Ann. tit. 21, § 1006 (1987) (one year from discovery for radiation, but all else five years from last exposure).

145. *See* N.D. Cent. Code § 65-05-01 (1985 & Supp. 1991); *see also* Ratliff v. Dominion Coal Co., 3 Va. App. 175, 177, 349 S.E.2d 147, 149-50 (1986) (letter to coal miner from Department of Labor that miner qualified under federal disability standards held to be notice to miner to trigger running of statute of limitations); Carter v. Continental Telephone Co., 373 N.W.2d 524, 526 (Iowa App. 1985) (employee's claim time barred because company physician diagnosed disease earlier and informed claimant by letter that it might be job related).

146. *See* N.Y. Work. Comp. Law § 28 (McKinney Supp. 1992); *see also* Henry v. Industrial Comm'n of Ariz., 754 P.2d 1342, 1344-45, 157 Ariz. 67, 69-70 (1987) (statute of limitations did not begin to run for police officer suffering post-traumatic stress until after medical diagnosis, despite fact that incident occurred twenty-four years earlier and claimant manifested symptoms and sought medical help two years after initial incident); Dennis v. Department of Labor & Indus., 745 P.2d 1295, 1301, 109 Wash. 2d 467, 473 (1987) ("The causal connection between a claimant's physical condition and his or her employment must be established by competent medical testimony which shows that the disease is probably, as opposed to possibly, caused by employment."); Craddock v. Eagle Picher Indus., 457 N.E.2d 338, 340 (Ohio 1982) (court allowed claim filed four years after employee left work due to a disability, holding that "[d]isability due to an occupational disease shall be deemed to have begun as of the date [of] . . . medical diagnosis . . . [or other scenarios], . . . whichever date is latest.").

injured worker suffering from a covered, although not necessarily compensable, occupational disease or injury.[147] Exclusivity continues to shield the employer even after termination of the employment relationship, as in the case of latent onset of disease. The most severe effects of the exclusivity doctrine occur in cases where the injury falls within a compensation statute's coverage, but compensation is denied for lack of proof of work-relatedness or for failure to satisfy a statute of limitations.

The inequitable operation of the exclusivity doctrine is exemplified by *Cole v. Dow Chemical Co.*,[148] in which the Michigan Court of Appeals held that persons rendered sterile, allegedly as a result of occupational exposure to the chemical DBCP, were barred from bringing a tort action against their employer. The court found that the injured worker's claim was covered under the state workers' compensation statute, even though the injury was deemed to be noncompensable under the statute.[149] The worker was not physically disabled from employment and sought damages primarily for the emotional distress associated with the injury.[150]

Employees seeking to avoid the strictures of the workers' compensation system by bringing independent civil actions against their employers may have available a limited—but generally narrowly construed—set of exceptions to the doctrine of exclusivity. These may include the exception for employer misconduct, the aggravation of injury exception, and the dual-capacity employer exception.[151] These exceptions may arise legislatively or judicially. The legal community, in considering the inequitable impact of the workers' compensation laws for toxic reproductive and genetic injuries, should examine the role that these exceptions can play in any reform efforts.

a. *Employer Misconduct*

Traditionally, the employer misconduct exception to exclusivity has

147. *See* 2A A. Larson, Workmen's Compensation Law § 65.14, at 12-15 (1990). Exclusivity applies regardless of whether the worker made a claim for compensation as a result of the injury. *See id.*

148. Cole v. Dow Chemical Co., 112 Mich. App. 198, 315 N.W.2d 565 (1982).

149. *See id.* at 206, 315 N.W.2d at 569.

150. *See id.* at 201, 315 N.W.2d at 567. In *Cole*, the plaintiffs argued that the exclusivity provision of the Michigan workers' compensation statute did not bar their tort actions against the employer because their claims were not compensable under the workers' compensation act. *See id.* at 204, 315 N.W.2d at 568. The court ruled that because the plaintiffs' sterility is a physical injury, any damages claimed, including emotional distress, arising out of the physical injury fall squarely within the workers' compensation coverage. *See id.* at 206, 315 N.W.2d at 569. The court distinguished other Michigan cases in which the plaintiffs were allowed to maintain actions outside the workers' compensation system on the ground that those actions did not involve physical or mental injuries. *See id.* at 205-06, 315 N.W.2d at 568-69 (citations omitted). The dissent argued in favor of limiting workers' compensation coverage to those disabilities resulting in the "inability to perform labor, not inability to procreate." *Id.* at 207, 315 N.W.2d at 570 (Kelley, J., dissenting). Thus, the dissent placed this case within the category of actions distinguished by the majority as falling outside of the workers' compensation scheme. *Id.*

151. *See infra* notes 153-76 and accompanying text.

been restrictively applied to injuries resulting from intentional acts of the employer specifically directed toward the worker or from acts that the employer knew with substantial certainty would result in injury to the worker.[152] Neither gross negligence nor reckless conduct typically has fallen within the exception.[153]

In a radical departure from traditional law, the West Virginia Supreme Court in *Mandolidis v. Elkins Industrial, Inc.*[154] expanded this exception in 1978 by holding that willful, wanton, or reckless misconduct could constitute deliberate intentional conduct under the state workers' compensation act.[155] The plaintiff in *Mandolidis* was injured while operating a machine that was not equipped with a safety guard, allegedly in violation of state and federal safety laws and industry standards.[156] The complaint alleged that the employer's conduct was "wilful, wanton, malicious, and [in] deliberate disregard for the well-being of the plaintiff with a deliberate intention to injure or kill him."[157] The court interpreted the intentional conduct exception in the West Virginia workers' compensation law[158] to include the conduct alleged by the plaintiff: "In our view when death or injury results from wilful, wanton or reckless misconduct such death or injury is no longer accidental in any meaningful sense of the word, and must be taken as having been inflicted with deliberate intention for the purposes of the workmen's compensation act."[159]

Not long after the *Mandolidis* decision, however, the West Virginia state legislature rejected the court's interpretation that any non-acciden-

152. *See* 2A A. Larson, Workmen's Compensation Law § 68.15 (1990). For a discussion of the various judicial theories employed to justify the intentional conduct exception to the doctrine of exclusivity, see Note, *Employer Intentional Torts in Virginia: Proposal For an Exception To the Exclusive Workers' Compensation Remedy*, 25 U. Rich. L. Rev. 339, 343-49 (1991). *See also* Webb, *Intentional Act Exception to Workers' Compensation*, 32 For the Defense 2 (June 1990) (providing comparison of judicial interpretations of intent under state workers' compensation laws).

153. *See* Comment, *Workers' Compensation - A Proposal to Protect Injured Workers From Employers' Shield of Immunity*, 20 St. Mary's L.J. 933, 942-43 (1989).

154. Mandolidis v. Elkins Indus., Inc., 161 W.Va. 695, 705, 246 S.E.2d 907, 914 (1978).

155. *See id.* at 705, 246 S.E.2d at 914.

156. *See id.* at 707-08, 246 S.E.2d at 914-15. The plaintiff alleged a long list of complaints regarding his employer's conduct with respect to the safety of the machines in the workplace, including the allegation that the employer forced employees to operate the machines without safety guards so that the production rate would increase, leading to increased profits. *See id.* at 707-08, 246 S.E.2d at 915.

157. *Id.* at 707, 246 S.E.2d at 915.

158. At the time of the *Mandolidis* decision, the relevant section of the West Virginia workers' compensation statute provided:

> If injury or death result to any employee from the deliberate intention of his employer to produce such injury or death, the employee . . . shall . . . have cause of action against the employer, as if this chapter had not been enacted, for any excess of damages over the amount received or receivable under this chapter.

W. Va. Code § 23-4-2 (1913), *as amended by* W. Va. Code § 23-4-2 (1985 & Supp. 1991).

159. *Mandolidis*, 161 W. Va. at 705, 246 S.E.2d at 914.

tal conduct could satisfy the statutory exception for deliberate intentional conduct. The legislature amended the workers' compensation act to provide that intent to injure the worker must be demonstrated for the claim to fall outside of the exclusivity of workers' compensation.[160]

Judicial interpretation of the exception in other states generally has not been as bold as the failed attempt in West Virginia. Nevertheless, some courts have found the requisite intent in allegations of employer deceit. An expansive interpretation is particularly suited to cases arising from toxic workplace exposures, due to the latency period and the difficulty in determining work-relatedness of the injury. Indeed, unscrupulous or lax employers could benefit from the traditional narrow construction because a former employee would be unable to determine the precise cause of an injury or to reconstruct the precise working conditions many years after the exposure.

Thus, in *O'Brien v. Ottawa Silica Co.*,[161] the United States District Court for the Eastern District of Michigan relieved a worker from the operation of the exclusivity doctrine when his employer was aware that its employees were suffering from asbestos-related disease but concealed that information. This withholding of specific medical information regarding the worker's personal health condition constituted sufficient intentional fraud to fall outside of the exclusivity doctrine.[162]

Recently, a Florida appellate court held that an employer's alleged deceit in exposing employees to toxic substances without warnings or appropriate safety measures constituted intentional conduct that brought the action outside the state workers' compensation scheme. In *Cunningham v. Anchor Hocking Corp.*,[163] the court gave strict attention to the specific wording of the plaintiffs' complaint and determined that the allegations could reasonably be construed as stating intentional claims, thus permitting a tort action against the employer. The court concluded:

> [T]he complaint alleges that appellees diverted the smokestack so that fumes would flow into, rather than outside of, the plant, and that they periodically turned off the plant ventilation system, thereby intensifying the level of exposure. The complaint further alleges that appellees removed manufacturers' warning labels on toxic substance containers, misrepresented the toxic nature of substances, and knowingly provided inadequate safety equipment, while misrepresenting the danger or extent of toxicity in the plant and the need for proper safety equipment. . . .
>
> [T]he allegations are that injury was "a substantial certainty" and that there was repeated, continued exposure that was intentionally in-

160. *See* W. Va. Code § 23-4-2 (1985 & Supp. 1991).

161. *See* O'Brien v. Ottawa Silica Co., 656 F. Supp. 610, 611-12 (E.D. Mich. 1987).

162. *Accord* Koslop v. Cabot Corp., 631 F. Supp. 1494 (M.D. Pa. 1986) (holding that worker could maintain tort action where employer concealed results of plaintiff's X-ray indicating medical condition associated with workplace exposure to beryllium).

163. Cunningham v. Anchor Hocking Corp., 558 So.2d 93, 95-97 (Fla. Dist. Ct. App. 1990).

> creased and worsened by appellees' deliberate and malicious conduct. Accordingly, the counts sounding in battery, fraud and deceit . . . were sufficient to state a cause of action outside the scope of workers' compensation and should not have been dismissed.[164]

The *Cunningham* case, however, leaves unclear the result if the conduct pleaded is less deliberately injurious, such as where an employer recklessly ignores safety procedures and warnings but does not undertake the affirmative steps that provided the basis for the Florida court's decision. Maintaining the traditional distinctions—between accidents and non-accidents and between specific intent to injure and reckless endangerment—may no longer be reasonable in light of the new categories and characteristics of injuries confronting the legal system from toxic workplace exposures.

Notwithstanding these few inroads, most states adhere to the stricter, more traditional interpretation of employer misconduct that requires specific intent to injure the worker. Thus, under current law, an employer's failure to adequately warn workers of toxic exposures in the occupational setting, together with concealment of scientific information associated with the risk of harm, usually does not qualify as "intent" for purposes of the employer misconduct exception.[165]

---

164. *Id.* at 96-97. The court refused, however, to allow the plaintiffs' claim for strict liability based upon ultrahazardous activities, ruling that the claim did not constitute an intentional tort. *See id.* at 97.

165. *See, e.g.*, Wilson v. Asten-Hill Mfg., 791 F.2d 30, 32-33 (3d Cir. 1986) (holding that employees exposed to asbestos failed to demonstrate requisite intent necessary under intentional tort exception where employer withheld scientific data regarding risks of inhalation of asbestos and failed to provide warnings of known risks); Miller v. Ensco, Inc., 286 Ark. 458, 461, 692 S.W.2d 615, 617 (1985) (holding that employer's failure to warn of risks or failure to provide safe working conditions deliberately placing employees in hazardous positions in violation of government regulations does not fall within intentional tort exception); Reed Tool Co. v. Copelin, 689 S.W.2d 404, 406 (Tex. 1985) (holding that employer's failure to provide safe place to work is not an intentional tort unless employer believes his conduct will cause injuries). In *Miller v. Ensco, Inc.*, the plaintiff, an employee in the defendant-employer's hazardous waste disposal facility, alleged that his employer specifically and deliberately intended to injure him by exposing him to PCBs without appropriate safety measures. 286 Ark. at 459, 692 S.W.2d at 616. Although Arkansas recognized an intentional tort exception to the exclusivity of the state workers' compensation provisions, the court held that the plaintiff's action fell outside the exception and within the coverage of workers' compensation. *See id.* at 460-61, 692 S.W.2d at 617. The court stated that the acts alleged, rather than the language of the complaint, were determinative of the issue. *See id.* Stating that "intentional torts involve consequences which the actor believes are substantially certain to follow his actions," the court concluded:

> Here, the [employer's] failure to warn of dangers or failure to provide safe conditions, deliberately placing [an employee] in a dangerous position and willfully violating governmental regulations, does not bring the cause of action within the ambit of an intentional tort. That type of activity by an employer, even where flagrant, does not constitute an intentional tort for purposes of the exclusivity provision of the workers' compensation act.

Id. Moreover, the court refused to adopt an aggravation-of-injury exception to exclusivity, as California had adopted in Johns-Manville Prods. Corp. v. Superior Court, 27 Cal. 3d 465, 612 P.2d 948, 165 Cal. Rptr. 858 (1980), despite the fact that the plaintiff had

b. *Aggravation of Injury*

Sometimes called the "dual injury" doctrine, the aggravation of injury exception has been applied primarily in California and New Jersey. In *Johns-Manville Products Corp. v. Contra Costa Superior Court*,[166] the California Supreme Court allowed a tort action brought by a worker against his employer on the basis of asbestos exposure in the workplace. The gravamen of the suit was that the employer had knowingly concealed the hazards of the occupational exposure from the worker, thus causing an aggravation of the physical condition of the employee.[167] Addressing the legal system's concerns that recognition of such an exception would invite a flood of litigation, the court stated:

> We conclude the policy of exclusivity of workers' compensation as a remedy for injuries in the employment [setting] would not be seriously undermined [by this exception], since we cannot believe that many employers will aggravate the effects of an industrial injury by not only deliberately concealing its existence but also its connection with the employment. Nor can we believe that the Legislature in enacting the workers' compensation law intended to insulate such flagrant conduct from tort liability.[168]

This decision led to the amendment of the California workers' compensation statute to include an express exception to exclusivity for aggravation of injury, but limiting recovery in a tort action to damages for aggravation only.[169]

---

alleged that the employer had directed its physician not to test employees for the presence of PCBs in their blood. *See Miller*, 286 Ark. at 262, 692 S.W.2d at 618. For a discussion of the *Johns-Manville* case, see *infra* notes 166-69 and accompanying text. Similarly, in Reed Tool Co. v. Copelin, 689 S.W.2d 404, 405-07 (Tex. 1985), the Texas Supreme Court ruled that knowing failure to provide adequate safety measures, coupled with poor employee training and knowledge of prior injuries, constituted gross negligence, but did not rise to the level of intentional misconduct.

166. Johns-Manville Prods. Corp. v. Superior Court, 27 Cal. 3d 465, 468, 612 P.2d 948, 950, 165 Cal. Rptr. 858, 860 (1980).

167. The relevant portion in the workers' compensation statute provided that willful misconduct of the employer was compensable under the statute by a one-half increase in benefits. *See id.* at 469, 612 P.2d at 951, 165 Cal. Rptr. at 861. Thus, the court further held that the plaintiff's claims of fraud and conspiracy with respect to his primary injury were barred by the statute. *See id.* at 469, 612 P.2d at 950, 165 Cal. Rptr. at 860.

168. *Id.* at 478, 612 P.2d at 956, 165 Cal. Rptr. at 866.

169. The exception allows a tort action "[w]here the employee's injury is aggravated by the employer's fraudulent concealment of the existence of the injury and its connection with the employment, in which case the employers' liability shall be limited to those damages proximately caused by the aggravation." Cal. Lab. Code § 3602(2) (West 1989). In Barth v. Firestone Tire & Rubber Co., 661 F. Supp. 193 (N.D. Cal. 1987), the court sought to define the term "aggravation" within the meaning of the statute. In *Barth*, the plaintiff claimed that his employer had intentionally exposed him to benzene and other workplace toxins, thus creating an increased risk of his developing various diseases and genetic damage. *See id.* at 195. The court found that the plaintiff's initial injury had been to his immune system, with aggravation of that injury in the form of "the presence of diseases in their latency period." *Id.* at 196. The plaintiff was not suffering from any disease at the time of the commencement of the action. Moreover, the court considered

Similarly, in *Millison v. E.I. duPont de Nemours & Co.*,[170] the New Jersey Supreme Court held that the plaintiff could bring an action for aggravation of an existing asbestos-related injury against his employer, but that he must demonstrate "a deliberate corporate strategy" of concealment to prevail in the action.[171] The difficulty of sustaining this burden of proof, however, may deter many would-be plaintiffs from pursuing otherwise meritorious actions.

c. *Dual Capacity*

The dual capacity exception to the exclusivity doctrine permits the injured employee to sue the employer in tort when the employer acts in an additional, distinct role beyond that of employer. The classic example of the dual capacity employer is where the employer is also the manufacturer of a product that injures the employee.[172] In the case of reproductive or genetic injuries, courts could conclude that an employer has acted in the secondary capacity of health care provider if the employer has assumed the obligation to monitor the health of its employees and to provide medical diagnoses and care.[173] In *Duprey v. Shane*,[174] an early case involving this exception, an employee was allowed to bring a malpractice suit against his chiropractor-employer for mistreating a workplace injury on the theory that the chiropractor was acting as a physician rather than as an employer.[175]

The few jurisdictions that recognize this exception have construed it very narrowly, often requiring that the risk created by the employer in the alternate capacity be one owed by the employer to the general public.[176] Thus, if an employer undertakes medical or genetic monitoring of

---

any related emotional distress suffered by the plaintiff to be a further aggravation of the injury for purposes of delineating the damages available to him in his action. *See id.*

170. Millison v. E.I. du Pont de Nemours & Co., 101 N.J. 161, 501 A.2d 505 (1985).

171. *Id.* at 183, 501 A.2d at 517.

172. *See* M. Brown, ed., *Toxic Torts and Product Liability: Changing Tactics for Changing Times* 100 (BNA Special Rep. 1989).

173. Courts are divided as to whether such activities by employers constitute a distinct alternate capacity. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 317.

174. Duprey v. Shane, 39 Cal. 2d 781, 789-94, 249 P.2d 8, 13-16 (1952).

175. *See id.* at 790, 249 P.2d at 13-14.

176. *See, e.g.*, Douglas v. E. & J. Gallo Winery, 69 Cal. App. 3d 103, 107, 137 Cal. Rptr. 797, 799 (1977) (holding that employee injured on scaffolding manufactured by employer and offered for sale to general public was permitted to sue employer in tort); Mercer v. Uniroyal, Inc., 49 Ohio App. 2d 279, 361 N.E.2d 492, 496 (1976), *overruled by* Schump v. Firestone Tire & Rubber Co., 44 Ohio St. 3d 148, 151, 541 N.E.2d 1040, 1044 (1989) (holding that employee could maintain tort action against employer because injury arose out of duty owed by employer to general public). Both *Mercer* and *Schump* involved an employee injured in the course of employment by a blowout of a tire manufactured by the employer. The Ohio turnaround evidences both the general disfavor of the dual capacity exception in product liability cases and the acknowledged difficulty courts have experienced in justifying a separation of the duties owed by the product-manufacturing employer. On the one hand, the employer must provide a safe workplace for its employees; on the other hand, the employer owes the consumer a safe product. Courts

an employee, the dual capacity exception arguably might not apply because the employer's duty does not extend to the monitoring of the general population.

B. *The Adverse Impact of Tort Law*

Although workers' compensation was designed to allow workers to recover for injuries sustained during the course of their employment, in practice the workers' compensation system poses many obstacles to recovery for workers suffering from toxic reproductive and genetic injuries. Unfortunately, the common-law tort scheme often fails to provide a relief mechanism for workers suffering from these injuries as well. Most of these employees will be prevented from commencing a common-law tort action against their employers by the operation of the exclusivity rule of the applicable workers' compensation statute.[177] Nevertheless, some employees may have available a cause of action in tort against their employers or third parties for the injuries sustained in the course of their employment. Similarly, tort law may provide an avenue of relief for some spouses and children of exposed workers.

1. Employee Actions Against Third Parties

In addition to filing for workers' compensation benefits, an injured employee may be able to commence a private action against a third party who manufactured the hazardous substance to which the employee was exposed in the course of employment. Based in products liability, this action makes available to the employee theories of negligence, strict liability, and perhaps breach of warranty, as permitted under the law of the relevant jurisdiction. There are, however, some universal roadblocks to recovery under these theories.

Causation is the primary obstacle to recovery under either negligence or strict liability for victims of reproductive or genetic injuries in the workplace.[178] As in the workers' compensation context, the statistical probabilities offered by epidemiological and toxicological studies may be insufficient to establish legally cognizable individual causation unless they are accompanied by clinical medical evidence that the plaintiff's

---

and commentators have had increasing difficulty justifying a dramatic departure from the traditional operation of the workers' compensation scheme merely because the employee is a user of the product during the course of employment. For a discussion of the abrogation of dual capacity in light of the Ohio cases, see Note, *Schump v. Firestone Tire and Rubber Co.: The Demise of Dual Capacity: Equal Protection Implications of Workers' Compensation Exclusivity as Applied to the Manufacturer-Employer*, 19 Cap. U.L. Rev. 1229 (1991).

177. For a discussion of exclusivity and its exceptions, see *supra* notes 147-76 and accompanying text.

178. For an analysis of causation problems and policies in relation to reproductive and genetic workplace injuries, see *infra* notes 298-317 and accompanying text.

specific injury was connected to a specific occupational exposure.[179]

Availability of the state-of-the-art defense in negligence and strict liability actions is a second concern to workers or others bringing tort actions. This defense forecloses liability if the defendant acted in accordance with state-of-the-art scientific knowledge at the time of the employee's exposure.[180] Although at least one jurisdiction has refused to allow state-of-the-art evidence in some toxic strict product liability suits on the ground that the defendant's conduct is irrelevant,[181] most jurisdic-

179. The report of the Office of Technology Assessment on reproductive hazards in the workplace makes the following observations:

> Clinical physicians are generally concerned with diagnosis and treatment, whereas biomedical researchers and epidemiologists focus more on the etiology of disease. . . . Thus, although animal studies may show a substance to be toxic to an animal fetus, a clinician may be reluctant to draw conclusions based on animal studies alone because of the considerable species variation in effects. Doctors are also likely to stress the role of various environmental and genetic factors outside of the workplace, notwithstanding the fact that such interactions are likely to be legally irrelevant so long as the workplace exposure played a substantial role in the reproductive harm.

Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 323. For a discussion of the problem of causation in the workers' compensation context, see *supra* notes 118-20 and accompanying text.

180. Although industry custom is relevant in determining the state-of-the-art in a given industry with respect to a particular substance, industry custom is not necessarily determinative. *See* Office of Technology Assessment, Reproductive Health Hazards, *supra* note 2, at 307-08. This rule prevents an industry from artificially setting the state of the art at an unsafe level when safer practices could be developed. When raised in strict product liability cases, the state-of-the-art defense is one example of the infusion of negligence principles into the law of strict liability. Because state-of-the-art is essentially fault-based, the defendant's conduct at the relevant point in time will be scrutinized for its propriety in relation to the health of the class of persons to which the plaintiff belongs.

181. *See* Beshada v. Johns-Manville Prods. Corp., 90 N.J. 191, 204-09, 447 A.2d 539, 546-49 (1982). *Beshada* was an asbestos product liability lawsuit brought by an insulation worker. The New Jersey Supreme Court refused to allow the defendant to rely upon the state-of-the-art defense in the failure-to-warn case and emphasized that the safety of the product, rather than the behavior of the defendant, is the relevant consideration. *See id.* at 204, 447 A.2d at 546. The court stated:

> When the defendants argue that it is unreasonable to impose a duty on them to warn of the unknowable, they misconstrue both the purpose and effect of strict liability. By imposing strict liability, we are not requiring defendants to have done something that is impossible. In this sense, the phrase "duty to warn" is misleading. It implies negligence concepts with their attendant focus on the reasonableness of defendant's behavior. However, a major concern of strict liability—ignored by defendants—is the conclusion that if a product was in fact defective, the distributor of the product should compensate its victims for the misfortune that it inflicted on them.

Id. The court then discussed the manner in which disallowing the state-of-the-art defense would advance the policies underlying strict product liability, including compensation, risk spreading, deterrence, and efficiency of the factfinding process. *See id.* at 205-08, 447 A.2d at 547-49.

In 1984, the New Jersey Supreme Court opined, in dicta, that *Beshada* was limited to its specific circumstances, presumably asbestos product litigation. *See* Feldman v. Lederle Laboratories, 97 N.J. 429, 454-55, 479 A.2d 374, 387-88 (1984). *Feldman* was a drug product liability action in which the court allowed the state-of-the-art defense notwithstanding its decision in *Beshada* two years earlier. In 1987, in Fischer v. Johns-Manville

tions have allowed the state-of-the-art defense in this context.[182] This defense is particularly germane to failure-to-warn actions brought on latent-disease claims in which the state of scientific knowledge regarding the toxicity of the relevant substance at the time of the worker's exposure may have been minimal compared to the state of knowledge at the time the action was commenced.

Third-party product liability actions may be available to victims of toxic reproductive and genetic workplace injuries when the toxic substance was conveyed to the victim's employer from a third party for use in the employer's manufacturing business or other enterprise.[183] Such third-party actions may be unavailable to employees of chemical companies, however, if their employers actually manufactured or otherwise created the toxic substance in the course of business, because of the operation of the workers' compensation exclusivity doctrine. This disparity may prove inequitable. While employees of companies that merely use a toxin in their production would be able to bring a tort action against the manufacturer of the toxin, employees of the manufacturer would be limited to workers' compensation due to the mere fortuity of their employer's status as manufacturer.[184]

Corp., 103 N.J. 643, 653-54, 512 A.2d 466, 471-72 (1986), the New Jersey Supreme Court declined to apply the direct holding of *Feldman* to an asbestos case. The court's holding in *Fischer* may have been influenced, however, by the fact that evidence existed that the defendants could have known as early as the 1930s of the dangers of asbestos exposure. *See id.* at 649-50, 512 A.2d at 469. In any event, New Jersey appears to continue to disallow the state-of-the-art defense in asbestos product liability actions, while allowing it in other product liability cases. Indeed, the Third Circuit held, in *In re* Asbestos Litigation, 829 F.2d 1233, 1244 (3d Cir. 1987), *cert. denied*, 485 U.S. 1029 (1988), that New Jersey did not unconstitutionally discriminate between classes of product liability defendants in refusing to allow the state-of-the-art defense in asbestos cases.

182. *See, e.g.*, Fell v. Kewanee Farm Equip. Co., 457 N.W.2d 911, 920 (Iowa 1990) (stating that "the state-of-the-art defense is a complete defense"); Anderson v. Owens-Corning Fiberglas Corp., 53 Cal. 3d 987, 991, 810 P.2d 549, 50, 281 Cal. Rptr. 528, 529 (1991) (ruling that state-of-the-art defense may be relevant to issue of knowability in a product liability action). In *Anderson*, the court, ruling on the availability of the defense in the context of a failure-to-warn action, expressly rejected the plaintiff's argument that the state-of-the-art defense imposes an inappropriate negligence standard on strict liability principles. According to the court, "a manufacturing or design defect *can be* evaluated without reference to the conduct of the manufacturer, [but] the giving of a warning cannot." *Id.* at 1002, 810 P.2d at 558, 281 Cal. Rptr. at 537 (citations omitted) (emphasis in original).

183. Asbestos product liability actions have become the new classic examples of such third-party actions in the area of toxic workplace exposures. Typically, such actions have been brought by asbestos insulation workers against the manufacturers of the asbestos products they installed during the course of their employment for contractors. *See, e.g.*, Karjala v. Johns-Manville Prods. Corp., 523 F.2d 155, 158 (8th Cir. 1975) (suit by asbestos insulation worker against manufacturer of asbestos insulation); Borel v. Fibreboard Paper Prods. Corp., 493 F.2d 1076, 1081-86 (5th Cir. 1973), *cert. denied*, 419 U.S. 869 (1974) (same).

184. It is unlikely that such employees would be successful in arguing that the employer's status as manufacturer would place the employer in a dual capacity outside the scope of workers' compensation because the duty owed by the employer to its employees may be distinct from the employer's duty to the general public. Nevertheless, plaintiffs'

The inequity in the availability of third-party product liability suits is compounded by the fact that virtually all jurisdictions grant the employer a statutory lien against any recovery obtained by the worker from the third party.[185] Thus, negligent employers could conceivably avoid liability altogether: the exclusivity doctrine would bar a direct tort action, and the employer would be entitled to reimbursement for any workers' compensation benefits paid to the injured employee.[186] Although there have been some innovative attempts to rectify these inequitable results by permitting manufacturers to seek contribution from the negligent employer,[187] most jurisdictions still shield employers from any obligation to contribute to the overall damages obtained by the worker in the third-party action.[188]

2. Actions by Related Third Parties Against Employers

Toxic reproductive and genetic injuries do not only harm the employee but, due to their insidious nature, may affect persons beyond the exposed worker. Thus, spouses and offspring may have claims for damages arising out of the worker's exposure to toxins. Like employees, third parties may encounter difficulties in the tort system in recovering for these injuries. Particularly with respect to damaged embryos and fetuses, the legal system is struggling to determine an appropriate remedial framework in which to fit such unconventional claims.

a. *Claims by Spouses*

Claims by spouses arising out of the workers' occupational exposure fall into two categories. The first is the derivative variety, exemplified by suits for loss of consortium. The second involves suits for spouses' direct personal injuries. This distinction is fundamental in establishing the availability of a direct tort action for the spouse against the employer. Even when a cause of action is available, however, the claim may fail.

---

attorneys should investigate the availability and scope of the dual capacity exception in the relevant jurisdiction. For a general discussion of the dual capacity exception to the exclusivity of workers' compensation, see *supra* notes 172-76 and accompanying text.

185. *See* Weiler, *Workers' Compensation and Product Liability: The Interaction of a Tort and a Non-Tort Regime*, 50 Ohio St. L.J. 825, 836 (1989).

186. *See id.* at 837, 840-41.

187. The most dramatic example of judicial manipulation in this manner was effected by the New York Court of Appeals two decades ago. *See* Dole v. Dow Chem. Co., 30 N.Y.2d 143, 282 N.E.2d 288, 331 N.Y.S.2d 382 (1972). In *Dole*, the third-party product manufacturer impleaded the plaintiff's decedent's employer. The court allowed the impleader and established a doctrine that required apportionment of the damages awarded to the plaintiff between the manufacturer and the employer. *See id.* at 148-49, 282 N.E.2d at 292, 331 N.Y.S.2d at 387. Such reform efforts have their inequitable aspects as well. The employer in *Dole* was required to pay its proportionate share of the plaintiff's tort damages only through the fortuitous circumstance that a third-party manufacturer was available for the plaintiff to sue. Had no third party been available, the employer would have been shielded, and payments would have been limited to the extent of workers' compensation benefits.

188. *See* 2B A. Larson, Workmen's Compensation Law § 76.20, at 14-654 (1989).

Loss-of-consortium claims typically are barred by the workers' compensation exclusivity doctrine. States with compensation statutes that define "exclusive liability" in terms of the employer's liability generally,[189] or that list spouses or other parties as within the scope of the covered injury,[190] generally prohibit independent loss-of-consortium suits.[191]

In contrast, when spouses suffer independent injuries arising from the breach of an independent duty owed to the spouse by the employer, the exclusivity doctrine may be circumvented.[192] To bring a personal injury action, however, a spouse must distinguish between the original injury to the worker and the independent injury to the spouse. This task may prove difficult and may result in arbitrary judicial distinctions.

An early example of an independent-injury case is *Price v. Yellow Pine Paper Mill*.[193] In this case, an employer was found liable after bringing a seriously injured employee home to his pregnant wife, causing her such severe shock that she suffered a miscarriage.[194] A more contemporary scenario, however, might involve a male worker who suffers an occupational reproductive or genetic injury that ultimately causes his wife to suffer a spontaneous abortion.[195] While in the former situation the employer clearly breached a separate and independent duty to the spouse after the injury to the worker had occurred, in the latter scenario the line between the worker's injury and the spouse's injury is blurred. An employer could reasonably argue that the spouse's injury was so inextricably merged with the worker's initial injury that in fact the spontaneous abortion was part and parcel of the initial injury, thus falling within the coverage of the relevant workers' compensation statute.[196] Such a result, however, would be inequitable, as it is unlikely that a male worker's claim for his wife's miscarriage would be compensated under most workers' compensation schemes. Apart from the obvious causation problems, by denying the spouse an independent tort action, the current scheme

189. *See* Cal. Lab. Code § 3601 (West 1989); Okla. Stat. Ann., tit. 85, § 12 (West 1970); W. Va. Code § 23-2-6 (1985 & West Supp. 1991).

190. *See* Iowa Code Ann. § 85.20 (West 1984); Minn. Stat. Ann. § 176.061 (West 1966 & Supp. 1992); Mo. Ann. Stat. § 287.120 (Vernon 1965 & Supp. 1992); N.Y. Work. Comp. Law § 11 (McKinney 1965 & Supp. 1992); Tenn. Code Ann. § 50-6-210 (1991); Ga. Code Ann. § 34-9-13 (1988 & Supp. 1991).

191. *See* 2A A. Larson, The Law of Workmen's Compensation § 66.20, at 12-71-92 (1990 & Supp. Nov. 1991) (cataloging cases denying loss-of-consortium actions).

192. *See id.* § 66.30, at 12-92-94.

193. Price v. Yellow Pine Paper Mill Co., 240 S.W. 588 (Tex. Civ. App. 1922).

194. *See id.* at 589.

195. *See* Baker, *Metal Firm to Pay for Lead Tests*, L.A. Times, July 11, 1991, at B1, col. 5 (workers at metal recycling company exposed to lead at work suffered miscarriages).

196. This argument is analogous to the ruling of the California appellate court in Bell v. Macy's Cal., 212 Cal. App. 3d 1442, 1451, 261 Cal. Rptr. 447, 453 (1989). In *Bell*, the court held that prenatal injuries to the offspring of an employee were covered by the workers' compensation system, thus barring the offspring's independent action against the parent's employer. *See infra* notes 205-08 and accompanying text.

forecloses the couple from seeking relief for the spouse's injury under either the compensation or tort systems.

One federal appellate case may shed some light on future judicial analysis of this problem. In *Woerth v. United States*,[197] the spouse of a federal employee contracted hepatitis from the employee. The employee, a nurse at a Veteran's Administration hospital, had contracted the disease in the course of her employment. When the spouse commenced an action against the United States pursuant to the Federal Tort Claims Act,[198] the government argued that the action was barred by the exclusivity provision of the Federal Employee's Compensation Act[199] ("FECA"), a federal scheme analogous to state workers' compensation statutes.[200] The district court concluded that the FECA exclusivity provision barred the spouse's action because the employee was subject to the FECA provisions.[201]

The United States Court of Appeals for the Sixth Circuit reversed, distinguishing the independent injury suffered by Mr. Woerth from the loss-of-consortium claims that typically would be barred by the FECA exclusivity provision.[202] The court stated:

> The proper inquiry . . . is whether the claim is 'with respect to the injury or death of an employee.' While Woerth's hepatitis may derive from his wife as a matter of proximate cause, his cause of action does not. His right to recover for the negligence of the United States is based upon his own personal injury, not a right of 'husband and wife.' The fact that the disease was transmitted through his spouse does not place Woerth in a position different from that of any other unrelated, but similarly injured tort victim.[203]

Accordingly, the court readily recognized that the spouse could maintain this action for personal injuries regardless of the fact that his immediate exposure was through the employee who was covered by FECA. This simple approach opts to draw the exclusivity line along the traditional boundary between derivative claims and individual claims rather than construct a more complicated distinction related to proximate cause. Such an approach clearly would support the independent claims of workers' spouses in toxic reproductive and genetic injury cases. Indeed, without this approach, spouses in these cases might be precluded from any recovery.

b. *Claims by or on Behalf of Offspring*

Offspring also may suffer a variety of injuries from their parents' expo-

197. Woerth v. United States, 714 F.2d 648, 649 (6th Cir. 1983).
198. 28 U.S.C. §§ 2671-2680 (1988).
199. 5 U.S.C. § 8116(c) (1988).
200. *See Woerth*, 714 F.2d at 649.
201. *See id.*
202. *See id.* at 650.
203. *Id.* (quoting 5 U.S.C. § 8116(c) (1988)).

sure to workplace toxins. Like spouses, offspring bringing tort actions for prenatal injuries against a parent's employer must make the threshold showing of an independent injury.

A few jurisdictions have limited the offspring to the parent's workers' compensation benefits.[204] In *Bell v. Macy's California*,[205] for example, the injuries to a pregnant worker's fetus resulting in the death of the offspring at approximately two years of age were deemed to be covered by the California workers' compensation statute. The court, after declaring the underlying precept that "the fetus in utero is inseparable from its mother,"[206] held that the injury to the offspring "was derived from the compensable injury" to the worker and, therefore, was collateral to the covered injury.[207] Although the injuries to the offspring were covered under workers' compensation, the relevant statute failed to provide any actual compensation for the child's medical and other expenses.[208]

While most jurisdictions allow offspring of exposed workers to bring independent personal injury actions against their parents' employers, the conceptual and practical complexities of allowing such actions have pre-

204. *See* Bell v. Macy's Cal., 212 Cal. App. 3d 1442, 1451, 261 Cal. Rptr. 447, 453 (1989); State Indus. Ins. Sys. v. Porter, 103 Nev. 170, 171, 734 P.2d 729, 729-30 (1987). *But see* Adams v. Denny's Inc., 464 So. 2d 876, 877-78 (La. Ct. App. 1985); Dillon v. S.S. Kresge Co., 35 Mich. App. 603, 603, 192 N.W.2d 661, 661 (1971); Witty v. American Gen. Capital Distribs., 697 S.W.2d 636, 638-39 (Tex. Ct. App. 1985), *aff'd in part, rev'd in part on other grounds*, 727 S.W.2d 503, 504-05 (Tex. 1987).

205. Bell v. Macy's Cal., 212 Cal. App. 3d 1442, 1451, 261 Cal. Rptr. 447, 453 (1989).

206. *Id.* at 1453 n.6, 261 Cal. Rptr. at 454 n.6.

207. *Id.* at 1451, 261 Cal. Rptr. at 453. The court explained:

> Our conclusion . . . is founded on the perception that the employer, having given up all common law defenses and undertaken a form of strict liability to employees, should not be held liable in tort for certain collateral consequences of a covered injury . . . . Moreover, were the fetus of a pregnant worker to retain a separate tort cause of action for injury to it, the employer would face a serious risk. . . . The range of common workplace injury that could result in injury or death to a fetus needs little exposition. Trips and falls, car accidents, explosions, fires, and other unfortunate but not unheard-of incidents of employment all may cause serious injury or death to the unborn as well as its parent. Less obvious are cases of subtle poisoning by exposure to toxic substances, genetic damage caused by radiation, and the other numerous and cautionary by-products of the Industrial Revolution.

Id. at 1453-54, 261 Cal. Rptr. at 455. The court then explored the rule permitting an action by the offspring, which could lead to the unacceptable result that employers would exclude women from the workplace to avoid the liability from such accidents and exposures. *See id.* This issue was addressed in United Auto Workers v. Johnson Controls, Inc., 111 S. Ct. 1196, 1209-10 (1991), in which the Supreme Court held that employee protection policies implemented by employers to exclude women from the hazards of particular jobs are in violation of Title VII.

208. *See Bell*, 212 Cal. App. 3d at 1454-55, 261 Cal. Rptr. at 455-56. The court in *Bell* stated that any mitigation of this harsh result would be appropriate for legislative enactment, rather than judicial opinion. *See id.* at 1454-1455, 261 Cal. Rptr. at 455. *Cf.* State Indus. Ins. Sys. v. Porter, 103 Nev. 170, 171, 734 P.2d 729, 729 (Nev. 1987) (holding that prenatal injury was covered under workers' compensation, and mother permitted to recover as compensation benefits medical expenses directly related to neonatal care of premature offspring).

occupied courts for several decades. Generally, all jurisdictions allow children to pursue an action for prenatal injuries if they are born alive. Similarly, parents usually are allowed to bring wrongful death actions to recover for employer conduct that caused a stillbirth.[209] Most jurisdictions at least purport to require that the fetus be viable[210] at the time of the injury in utero.[211]

---

209. *See* W. Keeton, D. Dobbs, R. Keeton & D. Owen, Prosser & Keeton on Torts § 55, at 368-69 (5th ed. 1984) [hereinafter Prosser & Keeton on Torts].

210. "Viability" has been defined as "[c]apability of living; . . . [and] usually connotes a fetus that has reached 500g in weight and 20 gestational weeks." Stedman's Medical Dictionary 1556 (24th ed. 1982). Legal definitions of viability may incorporate other factors as well. In Roe v. Wade, 410 U.S. 113, 160 (1973), for example, the United States Supreme Court stated that medical opinion indicated that viability usually is determined to be approximately 28 gestational weeks, but could be as early as 24 gestational weeks. *Cf.* Webster v. Reproductive Health Servs., 492 U.S. 490, 519 (1989) (rejecting viability as delineating the point at which state could constitutionally regulate abortion) (citing Thornburgh v. American College of Obstetricians & Gynecologists, 476 U.S. 747, 795 (1986) (White, J., dissenting)); *id.* at 528 (O'Connor, J., dissenting). In *Webster*, the Court upheld the Missouri state statute that prohibited nontherapeutic abortions after viability. *See id.* at 519-20. The statute provides that

> [b]efore a physician performs an abortion on a woman he has reason to believe is carrying an unborn child of twenty or more weeks gestational age, the physician shall first determine if the unborn child is viable by using and exercising that degree of care, skill, and proficiency commonly exercised by the ordinarily skillful, careful, and prudent physician engaged in similar practice under the same or similar conditions.

Mo. Rev. Stat. § 188.029 (West Supp. 1992). The statute notes that compliance with this provision could require that the physician perform various medical examinations relating to gestational age, fetal weight, and lung maturity. *See id.* The *Webster* Court rejected an interpretation requiring such medical examinations in all circumstances, finding that in at least some circumstances the "reasonable professional skill and judgment" of the physician in determining viability could be exercised without such testing. *Webster*, 492 U.S. at 515 (plurality opinion). In essence, the Missouri statute creates a "presumption of viability at 20 weeks, which the physician must rebut with tests indicating that the fetus is not viable prior to performing an abortion." *Id.* Thus, the various legal definitions of viability are at best mutable. Efforts to hinge tort liability on fetal viability would merely add another ambiguous dimension to an already slippery concept.

211. This rule may have been perpetuated because most cases in which offspring have recovered for prenatal injuries have in fact involved post-viability injuries. *See* Prosser & Keeton on Torts, *supra* note 209, § 55, at 368. Courts, however, do not always strictly enforce the viability rule. *See, e.g.*, Bennett v. Hymers, 101 N.H. 483, 484-85, 147 A.2d 108, 110 (1958) (infant can maintain an action for prenatal injury occuring prior to viability); Smith v. Brennan, 31 N.J. 353, 367, 157 A.2d 497, 504 (1960) (recovery not necessarily denied if prenatal injury occurred before viability); Sinkler v. Kneale, 401 Pa. 267, 273, 164 A.2d 93, 96 (1960) (notion that child must have been viable when injuries were received has little to do with the basic right to recover); Sylvia v. Gobeille, 101 R.I. 76, 79, 220 A.2d 222, 224 (1966) (employing causation not viability as test); Robertson, *Toward Rational Boundaries of Tort Liability for Injury to the Unborn: Prenatal Injuries, Preconception Injuries, & Wrongful Life*, 1978 Duke L.J. 1401, 1412 (illogic and injustice of viability rule led most courts to abandon the rule and require only a causal connection between injury and damages). Viability as the basis for the existence of a cause of action for prenatal injury apparently had its basis in the concern that the existence of the fetus be verifiable at the time of the injury. Because nearly all jurisdictions allow offspring, and not fetuses, to sue for prenatal injuries, the existence of the fetus at the time of the injury can be verified by objective medical criteria.

Jarvis v. Providence Hospital[212] demonstrates one court's attempt to deal with the strictures of the viability requirement. In this case, a father was permitted to maintain a wrongful death suit for a stillborn child who was exposed to the hepatitis virus prior to viability, but who suffered fatal injury after viability. The child's mother, a medical technician in the defendant hospital's laboratory, was exposed to the virus in the workplace when she was three and one-half months pregnant. The mother was diagnosed with hepatitis when she was eight months pregnant, and approximately one week later her child was stillborn.[213] In allowing the wrongful death action, the court found that although the plaintiff's decedent was not born alive, the fetus had been viable at the time of injury.[214] The court refused to extend the viability requirement to the time of the negligence, however, concluding that the stage of fetal development at which the negligent conduct occurred was irrelevant.[215]

Jarvis presented a situation in which the precise time of both exposure and injury was easily calculable. The timing of most toxic reproductive and genetic injuries, however, is far less precise. Exposures may create acute conditions or may be cumulative. Moreover, injury to the embryo or fetus could be immediate or may require a latency period of days, weeks, or even months before the injury is manifested. Even with current medical developments in prenatal diagnostics,[216] precise information regarding time of exposure and injury may be impossible to ascertain. A worker unaware of a hazardous exposure may have no reason to seek medical attention for the possibility of fetal harm if no other objective medical indicia of injury are presented during gestation. This problem may be particularly characteristic of certain genetic injuries.

The viability requirement is an imprecise and unfair bar to recovery for offspring of exposed workers. Time of viability is imprecise and sub-

212. Jarvis v. Providence Hosp., 178 Mich. App. 586, 444 N.W.2d 236 (1989).

213. *See id.* at 589, 444 N.W.2d at 237.

214. *See id.* at 591, 444 N.W.2d at 238. The common law of Michigan required that for an action based upon prenatal injuries to be allowable, the offspring must have been born alive or the fetus must have been viable at the time of the injury. *See id.* For a historical analysis of the live-birth requirement, see Robertson, *supra* note 211, at 1420-34. *See also* Note, *Recovery for the Wrongful Death of a Fetus*, 25 U. Rich. L. Rev. 391, 394-96 (1991) (discussing live birth and viability as criteria in actions for wrongful death of a fetus).

215. *See Jarvis*, 178 Mich. App. at 593, 444 N.W.2d at 239. The court compared the *Jarvis* case to an earlier case in which the Michigan Supreme Court held that a common-law negligence action for prenatal injury was permissible if brought by an afterborn child for injuries incurred in utero at a gestational age of four months. *See id.* at 591-92, 444 N.W.2d at 238-39; *see also* Womack v. Buchhorn, 384 Mich. 718, 725, 187 N.W.2d 218, 222 (1971) (assuming fetus was nonviable at the time of the injury); O'Neill v. Morse, 385 Mich. 130, 133-34, 188 N.W.2d 785, 785-86 (1971) (extending *Womack* to allow wrongful death claim by viable fetus injured in utero at eight months gestational age). The *Jarvis* court concluded that both *Womack* and *O'Neill* warranted an extension of liability to stillborn fetuses "regardless of the stage of fetal development in which the negligence occurred." *Jarvis*, 178 Mich. App. at 593, 444 N.W.2d at 239.

216. *See generally* G. Kolata, The Baby Doctors—Probing the Limits of Fetal Medicine (1990) (discussing medical and ethical issues in fetal diagnostics and therapy).

ject to conflicting medical and legal interpretations.[217] In addition, because some substances in the workplace can be toxic to the embryo, either before implantation or during the post-implantation embryonic phase, viability as a criterion would preclude recovery for such early pregnancy injuries, as well as for fetal injuries prior to viability.[218] The availability of a cause of action would be dependent purely upon the arbitrary element of gestational age, rather than on notions of legal duty and causation. The injury to resulting offspring could be identical, regardless of gestational age at the time of exposure. Concern for the possibility of nonmeritorious actions is more appropriately addressed within the context of the principles of duty and causation rather than according to arcane concepts of viability and verifiable personhood.[219] Thus, while it may be more difficult for a plaintiff to prove the requisite causation when the injury occurs during preimplantation or prior to viability, the plaintiff, nevertheless, should be afforded the opportunity to present proof for the factfinder to determine.

Moreover, infusing the concept of fetal viability into tort principles makes tort law vulnerable to the political fervor of the abortion debate.[220] Although fetuses traditionally have not been granted legal rights independent from their mothers,[221] current judicial interest in "fetal rights" eventually may give rise to an altered legal status for all fe-

---

217. *See supra* note 210 and accompanying text.

218. *See* Renslow v. Mennonite Hosp., 67 Ill. 2d 348, 350-53, 367 N.E.2d 1250, 1252-53 (1977) (rejecting viability as criterion for common-law action for prenatal injuries). The *Renslow* court observed that "denial of claims for injuries to the previable fetus may indeed cut off some of the most meritorious claims, for there is substantial medical authority that congenital structural defects caused by factors in the prenatal environment can be sustained only early in the previable stages." *Id.* at 352-353, 367 N.E.2d at 1252-53.

219. This focus avoids some of the possible confusion between the treatment of fetuses for the purpose of the tort law and their status within the context of the abortion debate.

220. Indeed, the court in *Jarvis* acknowledged an apparent conflict between the Michigan cases imposing a duty of care upon defendants for conduct toward a nonviable fetus and Roe v. Wade, 410 U.S. 113 (1976), in which the United States Supreme Court permitted a pregnant woman the right to choose to abort her fetus during the first two trimesters of pregnancy. The Michigan court reasoned:

> [A]s *Roe* was balancing only the rights of the mother against the right of the state to protect a "potential human being," *Roe* did not determine whether a state might protect the "potential human being" from conduct of a third party which foreseeably endangers the life of a fetus, whether or not the fetus is viable. We conclude that such protection has been afforded in Michigan under the common law . . . and that such protection is not inconsistent with the mother's right of privacy preserved by *Roe*.

Jarvis, 178 Mich. App. at 596-97, 444 N.W.2d at 240-41. In contrast to *Roe*, the Supreme Court in Webster v. Reproductive Health Services, 492 U.S. 490, 516 (1989), stated in dicta that states may have a legitimate interest in protecting the developing fetus by regulating abortion from the moment of conception. These issues raise a significant question as to what relation such distinctions made within the context of abortion regulation should bear to tort liability.

221. *See* Goldberg, *Of Gametes and Guardians: The Impropriety of Appointing Guardians Ad Litem for Fetuses and Embryos*, 66 Wash. L. Rev. 503, 516-17 (1991).

tuses.[222] The availability of a remedy to children born damaged as a result of a parent's toxic workplace exposure should not depend upon the vicissitudes of viability. Such actions should appropriately be loss-oriented rather than dependent upon the legal status of the developing embryo[223] or fetus. Concurring with the loss-oriented approach, the Rhode Island Supreme Court has rejected the viability standard, focusing instead on proof of causation to determine the existence of legally cognizable prenatal injuries.[224]

Some plaintiffs have advanced other theories of recovery for prenatal injuries, such as "wrongful life." Plaintiffs bringing these claims assert that they never should have been born because the harmful results of the defendants' conduct toward them prior to birth rendered their lives not worth living.[225] Courts are averse to such wrongful life claims on public policy grounds, however, because of the invited comparison between existence and nonexistence.[226]

c. *Preconception and Intergenerational Torts*

Offspring of exposed workers may also seek remedy through preconception torts, alleging that injuries were caused by damage to the sperm or egg prior to fertilization.[227] While only a small minority of courts recognize these kinds of actions,[228] no clear theoretical reason exists to distinguish between such torts and post-conception prenatal torts.[229] Public policy, however, may favor a new approach.

While injury to the egg or sperm arguably is peculiar to the adult male or female because a developing third person does not exist even in the most rudimentary embryonic or fetal form, logic dictates that an in-

222. For a discussion of the potential conflicts between women's rights and the expansion of fetal rights, see Note, *The Creation of Fetal Rights: Conflicts With Women's Constitutional Rights to Liberty, Privacy, and Equal Protection*, 95 Yale L.J. 599, 602-25 (1986).

223. Much has been written by both medical and legal commentators concerning the legal status of the human embryo, particularly as to whether the law should recognize a distinction between preimplantation embryos and postimplantation embryos. For a discussion of the various views of the legal status of the embryo, see Eggen, *The "Orwellian Nightmare" Reconsidered: A Proposed Regulatory Framework for the Advanced Reproductive Technologies*, 25 Ga. L. Rev. 625, 657-64 (1991).

224. *See* Presley v. Newport Hosp., 117 R.I. 177, 187-89, 365 A.2d 748, 753-54 (1976); Sylvia v. Gobeille, 101 R.I. 76, 79, 220 A.2d 222, 224 (1966).

225. *See* Prosser & Keeton on Torts, *supra* note 209, § 55, at 370-71.

226. *See* Gleitman v. Cosgrove, 49 N.J. 22, 25, 227 A.2d 689, 692 (1967), *overruled on other grounds by* Berman v. Allan, 80 N.J. 421, 404 A.2d 8 (1979). More judicial lenience has been shown, however, in analogous actions brought by parents for "wrongful birth," and recovery often is allowed on that theory for the parents' economic losses and emotional distress. *See generally* Robertson, *supra* note 211, at 1439-55 (discussing wrongful conception, failed contraconception, and denied abortion as causes of action).

227. *Cf.* Jorgensen v. Meade Johnson Laboratories, 483 F.2d 237, 238-39 (10th Cir. 1973) (genetic injury resulting from mother's ingestion of drug).

228. *See* Renslow v. Mennonite Hosp., 67 Ill. 2d 348, 10 Ill. Dec. 484, 367 N.E.2d 1250 (1977); Prosser & Keeton on Torts, *supra* note 209, § 55, at 369 & n.26.

229. *See* Prosser & Keeton on Torts, *supra* note 209, § 55, at 369.

dependent duty of the employer to protect foreseeable offspring of its workers should permit a preconception cause of action. This theory formed the basis of the Illinois Supreme Court's decision to allow a claim for a preconception tort in *Renslow v. Mennonite Hospital.*[230] The plaintiff in *Renslow*, suing on behalf of herself and her minor daughter, claimed that a condition resulting from incompatible Rh-positive blood transfusions that she received in 1965 caused the injuries suffered by her daughter, who was born in 1974.[231] The court upheld the existence of a cause of action, ruling that although the negligent conduct of the defendants occurred many years prior to the child's conception, the impact of the conduct on the plaintiff's offspring was foreseeable.[232]

Phrasing its analysis in terms of duty, the court stated:

> In the case at bar, the wrongful conduct took place prior to plaintiff's conception; the plaintiff at the time of the conduct was in no sense a separate entity to whom the traditional duty of care could be owed. Plaintiff herein asks us to reexamine our notions of duty, and to find, in essence, a contingent prospective duty to a child not yet conceived but foreseeably harmed by a breach of duty to the child's mother.[233]

Thus, the court held that the defendant could be held liable because "there is a right to be born free from prenatal injuries foreseeably caused by a breach of duty to the child's mother," regardless of the time of conception.[234]

Earlier, the United States Court of Appeals for the Tenth Circuit had reached a similar conclusion through an analysis of causation rather than duty. In *Jorgensen v. Meade Johnson Laboratories,*[235] the court upheld a cause of action by the father of twins, one deceased and one living, born with genetic defects allegedly caused by the mother's ingestion of the defendant's birth-control pills prior to the twins' conception. The court allowed the plaintiff to present evidence of causation on the theory that the birth control pills had altered the mother's own chromosome structure, thereby inducing the genetic defect in the offspring.[236]

Whether the right of action is couched in terms of duty or causation, actual proof of causation for preconception torts may be especially problematic given the current stage of scientific knowledge regarding reproductive and genetic injuries. Although relief for plaintiffs claiming preconception torts may be illusory in many circumstances, the specter of employer liability may spark further scientific research in this important area.

230. *See* Renslow v. Mennonite Hosp., 67 Ill. 2d 348, 357-59, 367 N.E.2d 1250, 1255-56 (1977).
231. *See id.* at 349, 367 N.E.2d at 1251.
232. *See id.* at 357, 367 N.E.2d at 1255.
233. *Id.* at 355-356, 367 N.E.2d at 1254.
234. *Id.* at 357, 367 N.E.2d at 1255.
235. Jorgenson v. Meade Johnson Laboratories, 483 F.2d 237 (10th Cir. 1973).
236. *See id.* at 239.

In *Renslow*, the Illinois court acknowledged the potential for a Pandora's box of "self-perpetuating" claims by subsequent generations for genetic damage, but it distinguished the case at hand as presenting a finite injury.[237] At least one state high court, however, has rejected a cause of action for a finite preconception tort based upon the potential for limitless actions.

In *Enright v. Eli Lilly and Co.*,[238] the plaintiff's grandmother ingested the drug DES, presumably to prevent a miscarriage while she was pregnant with the plaintiff's mother. The plaintiff claimed that her mother's exposure to the DES in utero resulted in the mother's inability to carry her own baby to term, leading to the plaintiff's premature birth and related injuries.[239] On intermediate appeal, the appellate division[240] held that although prior New York case law[241] foreclosed the plaintiff's negligence claim against the DES manufacturer, the plaintiff could pursue her strict liability claim.[242] The appellate division justified this conclusion by citing New York's strong public policy favoring the fashioning of a remedy for DES injuries.[243]

The New York Court of Appeals reversed the appellate division, concluding that more urgent policies overrode the state's interest in providing a remedy for persons injured by DES.[244] The court stated that the nature of the plaintiff's preconception injuries presented "vexing questions with . . . 'staggering implications.'. . . For all we know, the rippling effects of DES exposure may extend for generations. It is our 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."[245]

Genetic injuries present the exact problems foreseen, but not directly ruled on, by the New York Court of Appeals in *Enright*. The number of genetically impaired plaintiffs arising from a single toxic tort is potentially limitless. Additionally, the severity of the injuries may be completely unknown and unpredictable at the time of the defendant's activities. These factors render the defendant incapable of developing an appropriate risk-management strategy. Defendants would be unable to accurately predict the magnitude of the risks their conduct creates, nor would they be likely to survive the financial onslaught of the generations

237. *See Renslow*, 67 Ill. 2d at 358, 367 N.E.2d at 1255.

238. Enright v. Eli Lilly & Co., 77 N.Y.2d 377, 570 N.E.2d 198, 568 N.Y.S.2d 550 (1991), *cert. denied*, 112 S. Ct. 197 (1991).

239. *See Enright*, 77 N.Y.2d at 380, 570 N.E.2d at 199, 568 N.Y.S.2d at 551.

240. *See* Enright v. Eli Lilly & Co., 155 A.D.2d 64, 553 N.Y.S.2d 494 (1990), *cert. denied*, 112 S. Ct. 197 (1991).

241. The court relied on Albala v. City of New York, 54 N.Y.2d 269, 429 N.E.2d 786, 445 N.Y.S.2d 108 (1981).

242. *See Enright*, 155 A.D.2d at 70, 553 N.Y.S.2d at 497.

243. *See id.* at 68-69, 553 N.Y.S.2d at 496-97.

244. *See Enright*, 77 N.Y.2d at 386-89, 570 N.E.2d at 203-04, 568 N.Y.S.2d at 555-56.

245. *Id.* at 386-87, 570 N.E.2d at 203, 568 N.Y.S.2d at 555 (quoting Albala v. City of New York, 54 N.Y.2d 269, 273, 429 N.E.2d 786, 788, 445 N.Y.S.2d 108, 109 (1981)).

of lawsuits arising out of that conduct.[246] As a result, the deterrent effect of the tort-law system would be diluted. *Enright* responds directly to this threat, and it is probable that other states faced with this issue will rule likewise.

Enright reflected a pure policy decision. The judiciary engaged in arbitrary line-drawing for the purpose of restricting future liability and placing a limit on litigation. While there is nothing new in this approach—statutes of limitations have been enacted for precisely this reason—*Enright* illustrates New York's ambivalence toward toxic-injury litigation. In 1986, the New York state legislature enacted a discovery statute of limitations applicable to all cases arising from exposure to toxic substances.[247] In *Enright*, the state's high court is now saying that only a limited class of persons may take advantage of the tort system to redress those same toxic injuries.

The New York example demonstrates the utter ambivalence and equivocation with which states have approached the legal issues presented by toxic injuries and the mixed messages sent to injured persons and potential defendants alike. Most notably, even where preconception torts are recognized, as in *Renslow*, it is likely that the genetically injured will remain out in the cold. The absence of accountability for such injuries assures not only a lack of deterrence, but also a dearth of incentives for employers to monitor the workplace for potential hazards. If employers know that they will not be held liable for genetic injuries to persons who were not directly exposed—in utero or otherwise—to toxic substances in their workplaces, they will have a powerful incentive to ignore genetic risks. Whether or not the states choose to recognize causes of action for successive generations of genetically injured, the system must at least address these secondary issues in a meaningful fashion.

III. TOWARD A NEW JURISPRUDENCE OF TOXIC LIABILITY

Toxic reproductive and genetic injuries in the workplace challenge the presumptions of the tort and workers' compensation systems in a manner not seen since the Industrial Revolution. Problems arising from addi-

246. Several of the dissenters in *Renslow* were particularly concerned about these issues. The Chief Justice was concerned as to "how to measure the insurance risk and the possible exposure of a defendant to claims by successive generations of plaintiffs who complain of genetic injury." 67 Ill. 2d at 371, 367 N.E.2d at 1261 (Ward, C.J., dissenting). Another dissenting justice opined:

> Considering the likelihood of suits filed decades after the alleged negligence occurred, the difficulty of proving or defending against such claims, the impossibility of actuarial measurement of the risks involved, successive recoveries by unborn abnormal generations affected by genetic changes, . . . I believe this departure from our rule denying recovery for preconception injuries is both unnecessary and undesirable.

Id. at 372, 367 N.E.2d at 1262 (Underwood, J., dissenting).

247. *See* N.Y. Civ. Prac. L. & R. § 214-c (McKinney 1990).

tional related parties, intervening causes, sequential injuries,[248] and a dearth of scientific knowledge relevant to causation may appear singly or in combination in such claims. The law has yet to satisfactorily and cohesively address these concerns. The current operation of the tort and workers' compensation systems has failed in this context. Before these problems can be resolved, the underlying precepts of the tort and workers' compensation systems must be re-evaluated, and any non-functioning myths must be abandoned.

A. *Re-evaluating the Parameters of Liability*

1. The Cause-In-Fact Problem

Causation, as interpreted under both the workers' compensation and tort regimes, traditionally consists of the dual requirements of cause-in-fact and proximate cause. The former requirement has been viewed as factual, while the latter is dependent upon policy determinations as to whether the law should recognize a particular cause for the purpose of assigning liability.[249] The requirement that the plaintiff demonstrate that the defendant's product or conduct was the cause-in-fact of the plaintiff's injury—as defined by the traditional but-for test—is a fundamental threshold principle of the liability system.[250] Placing the burden upon the plaintiff to show cause-in-fact through the introduction of particularized evidence is deemed to serve both the utilitarian and moral goals of the system.[251]

Some commentators maintain that the concept of cause-in-fact has become infused with policy notions, thus blurring the traditional distinction between cause-in-fact and proximate cause.[252] Accordingly, they argue that because the cause-in-fact requirement has shed its original rigid and mechanistic nature, courts should employ greater flexibility in interpreting the requirements of causation. If all aspects of causation were policy choices, considerable elasticity would be available in the judicial process to carve out a doctrine of causation for new and previously uncontemplated types of cases.

Indeed, toxic-exposure cases may be viewed less as an aberration and

248. For example, a worker may learn early on of a diagnosis of infertility that is traceable to an exposure in the workplace. Some time later, the worker may suffer a spontaneous abortion related to the same exposure. Finally, if the worker eventually becomes pregnant and carries a fetus to term, the possibility of birth defects or the expression of genetic damage to the worker could arise.

249. *See* Malone, *Ruminations on Cause-In-Fact*, 9 Stan. L. Rev. 60, 60 (1956).

250. *See* Calabresi, *Concerning Cause and the Law of Torts: An Essay for Harry Kalven, Jr.*, 43 U. Chi. L. Rev. 69, 72 (1975).

251. Rosenberg, *The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System*, 97 Harv. L. Rev. 851, 858 (1984).

252. *See* Delgado, *Beyond Sindell: Relaxation of Cause-In-Fact Rules for Indeterminate Plaintiffs*, 70 Calif. L. Rev. 881, 891-92 (1982); Firak, *The Developing Policy Characteristics of Cause-In-Fact: Alternative Forms of Liability, Epidemiological Proof and Trans-Scientific Issues*, 63 Temp. L. Rev. 311, 312-13 (1990).

more as an extension of the traditional accident case upon which the system was founded. Professor David Rosenberg has argued:

> [T]he entire notion that "particularistic" evidence differs in some significant qualitative way from statistical evidence must be questioned. The concept of "particularistic" evidence suggests that there exists a form of proof that can provide direct and actual knowledge of the causal relationship between the defendant's tortious conduct and the plaintiff's injury. "Particularistic" evidence, however, is in fact no less probabilistic than is the statistical evidence that courts purport to shun. All knowledge of past as well as future events is probabilistic. Inevitably it rests on intuitive or more rigorously acquired impressions of the frequency with which similar events have occurred in like circumstances. "Particularistic" evidence offers nothing more than a basis for conclusions about a perceived balance of probabilities.[253]

This passage suggests that judicial and scholarly antipathy toward novel applications of the causation requirements derives more from rote reverence to inveterate concepts rather than from any logical distinction between forms of proof. What is most apparent, however, is the not-so-novel notion that policy judgments participate in every stage of the process in the most subtle manner.

Causal indeterminacy is a consistent characteristic of reproductive and genetic injury claims, even where the claimants can demonstrate a general, or statistical, probability of causation. Thus, a claimant may have difficulty proving that the particular workplace exposure was causally connected to the injury claimed.[254] Similarly, the claimant may be unable to distinguish workplace exposure as the cause of the injury from the background occurrence level of the injury[255] or from other multiple contributing causes.[256] Strict adherence to traditional notions of proof of causation, therefore, would necessitate the dismissal of virtually all such claims. In contrast, total abrogation of such fundamentals as cause-in-fact might invite overexposure of defendants to liability for injuries that may be associated with background risk levels of the disease rather than with the defendants' enterprises.

While traditionalists might argue that any departure from the established boundaries and burdens of liability would frustrate the policy goals of the tort and workers' compensation systems, courts and scholars have begun to advocate various means by which the causation problems

253. Rosenberg, *supra* note 251, at 870 (footnote omitted). Professor Glen O. Robinson expressed the same sentiment: "One of the illusions fostered by traditional tort doctrine is that events have determinate causes that can be identified by careful investigation. . . . Probably courts recognize these concerns but are reluctant to articulate them; their opinions still largely reflect the dominant mechanistic and deterministic conceptions of causation." Robinson, *Probabilistic Causation and Compensation for Tortious Risk*, 14 J. Legal Stud. 779, 780 (1985).

254. *See supra* notes 126-33 and accompanying text.

255. *See supra* notes 5-6 and accompanying text.

256. *See supra* notes 78-79 and accompanying text.

presented by new toxic injuries can be incorporated into the system.[257] These reform-minded views, to one extent or another, embrace the probabilistic nature of toxic-exposure cases and attempt to refashion the system to achieve an appropriate balance among the goals of compensation, deterrence, and loss-spreading.

For example, several states have adopted judicial rules of alternative liability in actions brought by persons exposed in utero to the drug DES, where the plaintiff is unable to identify the defendant or defendants who manufactured the DES ingested by the plaintiff's mother.[258] Thus, in

257. *See, e.g.*, Delgado, *supra* note 252, at 892-95 (advocating relaxation of causal rules to promote tort goals of compensation, loss-spreading, deterrence, economic efficiency, knowledge, and justice); Firak, *supra* note 252, at 338-40 (discussing proposing alternative liability, industry-wide liability, concerted action, market share liability, and risk contribution liability as alternatives to traditional cause-in-fact liability); Robinson, *supra* note 253, at 785-89 (proposing risk-based liability as an alternative); Rosenberg, *supra* note 251, at 861-86 (considering whether proportional liability for mass exposure torts would serve the goals of tort law more effectively than does the preponderance rule).

258. *See, e.g.*, Sindell v. Abbott Laboratories, 26 Cal. 3d 588, 611-12, 607 P.2d 924, 936-37, 163 Cal. Rptr. 132, 144-45 (market share liability), *cert. denied*, 449 U.S. 912 (1980); Abel v. Eli Lilly & Co., 418 Mich. 311, 325-34, 343 N.W.2d 164, 170-74 (1984) (alternative liability), *cert. denied*, 469 U.S. 833 (1984); Hymowitz v. Eli Lilly & Co., 73 N.Y.2d 487, 511-13, 539 N.E.2d 1069, 1078, 541 N.Y.S.2d 941, 950 (1989) (market share liability); Martin v. Abbott Laboratories, 102 Wash. 2d 581, 603, 689 P.2d 368, 381 (1984) (market share); Collins v. Eli Lilly Co., 116 Wis. 2d 166, 179, 342 N.W.2d 37, 49-50 (1984) (risk contribution liability). The various theories developed within the context of the DES cases arise from more traditionally accepted doctrines. *See, e.g.*, Summers v. Tice, 33 Cal. 2d 80, 86, 199 P.2d 1, 4 (1948) (alternative liability); Restatement (Second) of Torts § 876 (1979) (concerted action). Both the alternative liability and concerted action theories presume that the plaintiff will place before the court all possible defendants who could have performed the acts of which the plaintiff complains. Moreover, these theories assume that the defendants are, in all cases, better able to determine which act was the specific cause of the plaintiff's injury. *See* Collins v. Eli Lilly Co., 116 Wis. 166, 179, 342 N.W.2d 37, 45-46 (1984) (rejecting application of alternative liability or concerted action theories for DES case). In toxic-exposure cases, often neither of these presumptions applies. The DES situation was particularly problematic because the drug was produced generically by a fluid market that included as many as 300 manufacturers during the decades of its production. *See id.* at 175, 342 N.W.2d at 44. Some of the same problems occur in other toxic-exposure cases, such as those involving asbestos insulation, hazardous waste, and AIDS-tainted blood products. In these situations, however, courts generally have demonstrated a reluctance to apply such innovative theories of liability. Even in jurisdictions in which market share liability has been applied to DES cases, controversy ensues over whether market share and similar liability theories should extend to other toxic-exposure cases. Thus, in Hymowitz v. Eli Lilly & Co., 73 N.Y.2d 487, 508, 539 N.E.2d 1069, 1075, 541 N.Y.S.2d 941, 947 (1989), the New York Court of Appeals expressly limited application of the market share theory adopted therein to DES cases. In Hall v. E.I. du Pont de Nemours & Co., 345 F. Supp. 353, 374 (E.D.N.Y. 1972), the court applied enterprise liability—a predecessor to market share liability—in a case involving injuries resulting from blasting caps. The court found significant the fact that the industry engaged in joint safety control by manufacturers. *See id.* at 371-76. Some recent decisions have exhibited judicial interest in applying broader concepts of collective liability outside the context of DES. *See, e.g.*, Nicolet, Inc. v. Nutt, 525 A.2d 146, 147-49 (Del. Sup. Ct. 1987) (ruling that asbestos manufacturer may be liable on theory of fraudulent concealment even if plaintiffs cannot identify manufacturer's products as cause of injuries); Ray v. Cutter Laboratories, 754 F. Supp. 193, 195-96 (M.D. Fla. 1991) (holding that market share theory of liability applied to negligence claims by plaintiffs who alleg-

Sindell v. Abbott Laboratories,[259] the California Supreme Court permitted the plaintiffs to join as defendants manufacturers comprising a substantial share of the DES market, holding that each defendant would be liable for any judgment earned by the plaintiffs in an amount representative of its proportionate share of the market. Market share liability permits a plaintiff to state a cause of action even where specific cause-in-fact cannot be demonstrated. In another variation, the Wisconsin Supreme Court, in *Collins v. Eli Lilly Co.*,[260] applied a risk-contribution theory of collective liability. The court, stating that less than a substantial share of the DES market could be represented among the defendants, held that a defendant would be liable for its proportionate share in contributing to the risk that caused the plaintiff's injury.[261] Both *Sindell* and *Collins* shift the burden of addressing cause-in-fact to individual defendants by requiring each one to come forth with exculpatory evidence.[262]

Both examples dilute the traditional role of cause-in-fact by rendering it unnecessary for the plaintiff to prove cause-in-fact in the prima facie case.[263] Only upon shifting the burden to the defendants does cause-in-

---

edly contracted AIDS from blood clotting product derived from thousands of donors). *But see, e.g.*, Celotex Corp. v. Copeland, 471 So. 2d 533, 538 (Fla. 1985) (rejecting market share theory for action brought by asbestos worker because toxicity of various asbestos products not identical); Shackil v. Lederle Laboratories, 116 N.J. 155, 174-77, 561 A.2d 511, 521-22 (1989) (rejecting market share theory for action alleging defective design of DPT vaccine because such liability would threaten availability of necessary drugs).

259. *See* Sindell v. Abbott Laboratories, 26 Cal.3d 588, 612, 607 P.2d 924, 937, 163 Cal. Rptr. 132, 145, *cert. denied*, 449 U.S. 912 (1980).

260. Collins v. Eli Lilly Co., 116 Wis. 2d 166, 342 N.W.2d 37 (1984).

261. *See id.* at 198-99, 342 N.W.2d at 52-53. The court stated that the Wisconsin rules of comparative negligence would govern the assignment of liability. *See id.* at 198, 342 N.W.2d at 52-53. The factors to be examined included:

> whether the drug company conducted tests on DES for safety and efficacy in use for pregnancies; to what degree the company took a role in gaining FDA approval of DES for use in pregnancies; whether the company had a small or large market share in the relevant area; whether the company took the lead or merely followed the lead of others in producing or marketing DES; whether the company issued warnings about the dangers of DES; whether the company produced or marketed DES after it knew or should have known of the possible hazards DES presented to the public; and whether the company took any affirmative steps to reduce the risk of injury to the public.

Id. at 200, 342 N.W.2d at 53.

262. *See Sindell*, 26 Cal. 3d at 612, 607 P.2d at 937, 163 Cal. Rptr. at 145; *Collins*, 116 Wis. 2d at 197-98, 342 N.W.2d at 52.

263. In *Collins*, the court set forth the following requirements of a prima facie case:

> that the plaintiff's mother took DES; that DES caused the plaintiff's subsequent injuries; that the defendant produced or marketed the type of DES taken by the plaintiff's mother; and that the defendant's conduct in producing or marketing the DES constituted a breach of a legally recognized duty to the plaintiff.

Collins, 116 Wis. 2d at 193, 342 N.W.2d at 50. Thus, the plaintiff need show only that the defendant marketed the *type* of DES taken, not that a specific defendant's product caused the plaintiff's injury. In fact, the court stated that if the plaintiff does not know the type of DES that allegedly caused the injury, the plaintiff is merely required to plead that "the defendant drug company produced or marketed the drug DES for use in preventing miscarriages during pregnancy." *Id.* at 193-94, 342 N.W.2d at 50.

fact become an issue. Both theories base liability on risk: *Collins* turns risk contribution analysis into a multifactorial case-by-case determination, whereas *Sindell* assigns a risk identical to the proportionate economic share of the market held by the defendant. Thus, liability may be imposed even where the named defendant's product or conduct may not actually have caused the plaintiff's injury.

The most extreme rejection of the cause-in-fact requirement in market share cases appeared in *Hymowitz v. Eli Lilly and Co.*,[264] in which the New York Court of Appeals refused to allow defendants to exculpate themselves by proving that they could not have marketed the DES ingested by the plaintiffs' mothers. In *Hymowitz*, therefore, cause-in-fact played virtually no role: liability could be imposed on a party who indisputably could not have caused the specific injury. This result, although desirable to the New York Court of Appeals on policy grounds,[265] raises the traditionalists' concerns that abrogation of the cause-in-fact requirement leads to inaccurate and ultimately incorrect results.

Similarly, the rise of new actions based upon increased risk of future disease evinces an interest in risk over cause-in-fact. In *Ayers v. Jackson Township*,[266] the court recognized that a cause of action for enhanced risk of future disease could lie if a plaintiff could demonstrate a quantified risk of developing the disease. Presumably, this requirement could be satisfied by a showing of physical change in the plaintiff's body consistent with the risk of developing subsequent illness. Moreover, this cause of action would depend upon state rules regarding the accrual of actions for purposes of the statutory limitations on commencement of actions.[267] A primary objection to increased risk claims of this sort is that the defendant could be required to pay, in an all-or-nothing manner, for an injury to which the defendant does not actually contribute and which in fact may never occur.

Increased risk claims have support in a line of cases relating to misdiagnosis of illness. In *Herskovits v. Group Health Cooperative*,[268] the negligent conduct of the defendant in failing to timely diagnose the plaintiff's cancer caused a reduction in his chances of survival from thirty-nine percent to twenty-five percent. Holding the defendant liable for the plaintiff's diminished chance of survival, the court stated:

> Causing reduction of the opportunity to recover (loss of chance) by

264. *See* Hymowitz v. Eli Lilly & Co., 73 N.Y.2d 487, 512, 539 N.E.2d 1069, 1078, 541 N.Y.S.2d 941, 950, *cert. denied*, 493 U.S. 944 (1989).

265. *See id.* at 507-08, 539 N.E.2d at 1075, 541 N.Y.S.2d at 947. The court stated, "[w]e conclude that the present circumstances call for recognition of a realistic avenue of relief for plaintiffs injured by DES. . . . We stress, however, that the DES situation is a singular case." *Id.*

266. *See* Ayers v. Jackson Township, 106 N.J. 557, 598-99, 525 A.2d 287, 308 (1987) (holding no cause of action for an unquantified enhanced risk claim).

267. *See* Anderson v. W.R. Grace & Co., 628 F. Supp. 1219, 1231-32 (D. Mass. 1986).

268. *See* Herskovits v. Group Health Coop., 99 Wash. 2d 609, 612, 664 P.2d 474, 475 (1983).

> one's negligence, however, does not necessitate a total recovery against the negligent party for all damages caused by the victim's death. Damages should be awarded to the injured party or his family based only on damages caused directly by premature death.[269]

The court thus recognized that under some circumstances it may be appropriate to require the defendant to pay the plaintiff according to the defendant's proportionate share of risk.

Such lost-chance cases have proven to be premonitory of the proportionate liability theories that scholars have advanced for toxic-exposure cases. Some commentators have proposed that a defendant should be required to compensate the plaintiff in an amount no greater than that defendant's proportionate share in the risk to which the plaintiff was exposed.[270] Thus, this theory bases liability on probabilities, even where the probability falls far below the fifty-one percent threshold of the preponderance-of-the-evidence standard.[271]

Regardless of the relative merits of the various proposals to assist claimants in proving causation, toxic reproductive and genetic injury cases clearly present problems that can only be remedied by a non-traditional view of the causation requirement. The recommendations set forth in this Article accommodate this need while addressing the policy goals of the tort and compensation systems.[272]

2. The Attenuation of Duty

In *Renslow v. Mennonite Hospital*, the court held that the defendant owed the plaintiff, an unconceived child at the time the defendant negligently transfused the mother with Rh-incompatible blood, a "contingent prospective duty" because the harm to the plaintiff was foreseeable at the

---

269. *Id.* at 619, 664 P.2d at 479.

270. *See, e.g.*, Brennan & Carter, *Legal and Scientific Probability of Causation of Cancer and Other Environmental Disease in Individuals*, 10 J. Health Pol., Pol'y & L. 33, 58-61 (1985) (recommending that legislators adopt a proportionate compensation system under which a plaintiff would receive the proportion of his damages that equals the percent probability that defendant caused plaintiff's disease); Rosenberg, *supra* note 251, at 859, 887-924 (comparing the traditional preponderance rule to a "public law" rule of proportional liability); Comment, *Epidemiological Proof of Probability: Implementing the Proportional Recovery Approach in Toxic Exposure Torts*, 89 Dick. L. Rev. 233, 249-58 (1984) (proposing that an award in a toxic tort case be based on the probability that defendant caused the injury and that this probability should be proved by the plaintiff). In a variation on the proportionate liability approach, Professor Farber has proposed the "most likely victim approach," under which "those plaintiffs whose injuries were least likely to have been caused by the defendant receive nothing, while those with the highest causation probabilities get full compensation." Farber, *Toxic Causation*, 71 Minn. L. Rev. 1219, 1221 (1987) (footnote omitted).

271. *See* Firak, *supra* note 252, at 336-37.

272. For a discussion of the policy goals of the tort and compensation systems in relation to the problems presented by toxic reproductive and genetic hazards in the workplace, see *infra* notes 280-95 and accompanying text.

time of the defendant's acts.[273] Duty, the court declared, "is not a static concept" and is dependent upon foreseeability of injury to a class of persons.[274]

The dissent in *Renslow* criticized the court's couching of its holding in terms of duty, accusing the majority of "abandon[ing] the traditional fault concept of liability premised upon duty and foreseeability and embrac[ing] instead a system which depends wholly upon the element of causation."[275] In *Renslow*, the cause-in-fact of the plaintiff's injuries was clearly traceable to the mother's negligent transfusion. The dissent objected to what it viewed as the majority's imposition of liability based purely upon cause-in-fact. Condemning the majority as mindless of the social policy issues inherent in the concept of duty, the dissent expressed concern that the majority's rule would ultimately lead to the imposition of liability where the consequences are unforeseeable, as in genetic injury cases.[276]

The situation presented in *Renslow* is the converse of the probabilistic causation cases discussed in the preceding section. Many toxic reproductive and genetic injury cases, however, may contain *both* cause-in-fact and duty problems. The most extreme examples of these problems appear in preconception injury cases and in subsequent-generation genetic injury cases. In these cases, courts must decide whether sound reasons exist for extending the defendant's duty beyond certain established relationships to other foreseeable injuries. Thus, although courts generally do extend liability to the injury or death of a fetus—at least where the fetus is viable and often with conditions attached—courts are reluctant to extend liability to the unconceived.[277]

On the one hand, extending duty to the outer limits of foreseeability serves the policy goals of compensation and deterrence. Moreover, extension of liability to remote victims requires no alteration in the substance of the relationships at issue. Thus, the defendant who owes the exposed worker a certain duty arguably would be responsible for all injuries emanating from the worker's exposure.[278]

In contrast, forcing defendants to bear the remote costs of injuries ad infinitum is inimical to the additional goals of finality and risk allocation.

---

273. Renslow v. Mennonite Hosp., 67 Ill. 2d 348, 357, 367 N.E.2d 1250, 1254 (1977); *see also supra* notes 230-34 and accompanying text (discussing *Renslow*).

274. *Renslow*, 67 Ill. 2d at 357, 367 N.E.2d at 1254.

275. *Id.* at 372, 367 N.E.2d at 1262 (Ryan, J., dissenting).

276. *See id.* at 376-377; 367 N.E.2d at 1264 (Ryan, J., dissenting).

277. For a discussion of the current treatment of the claims of injured offspring arising out of workplace exposures of the parent, see *supra* notes 204-26 and accompanying text.

278. The situation presented in *Renslow* differs from the circumstances forming the basis of the New York Court of Appeals' decision in the classic case of Palsgraf v. Long Island R.R., 248 N.Y. 339, 340-41, 162 N.E. 99, 99 (1928). With reproductive and genetic hazards, the possibility of injury extending to later-conceived fetuses or offspring or to future generations related biologically to the worker is far more foreseeable than the possibility that Mrs. Palsgraf would be injured by the series of fortuitous events surrounding the explosion of the unmarked package.

Defendants would be unable to ascertain the risks of their enterprises and would be unable to secure adequate insurance coverage to protect against multi-generational losses. As Dean Prosser stated, "duty . . . could be founded only on the foreseeability of some harm to the plaintiff in fact injured. 'Negligence in the air, so to speak, will not do.' "[279]

B. *Re-examining the Policy Goals of the Tort and Workers' Compensation Systems*

1. Utilitarian Goals

The utilitarian objective of the tort and workers' compensation systems has long been to reduce the net costs of accidents through implementation of appropriate safety measures and efficient allocation of risk.[280] Thus, the workers' compensation system imposed the economic costs of occupational injuries on the enterprise on the theory that the enterprise is most able to bear the loss and can incorporate such losses into its costs by passing those costs on to consumers of its products.[281] Moreover, because workers as a class tend to underestimate their risks in the workplace setting, employers could incorporate this risk factor into their overall costs, including those associated with health and safety measures.[282]

Notwithstanding the overall advantages of the workers' compensation system, the utilitarian goals of the system collapse when confronted with the claims of reproductively or genetically harmed workers. Because proof of causation, embodied in the work-relatedness requirement, has

279. Prosser, *Palsgraf Revisited*, 52 Mich. L. Rev. 1, 5 (1953) (quoting *Palsgraf*, 248 N.Y. at 341).

280. *See generally* G. Calabresi, The Costs of Accidents (1970) (discussing framework under which different systems of accident law may be evaluated); Posner, *A Theory of Negligence*, 1 J. Legal Stud. 29 (1972) (analyzing social function of liability for negligent acts).

281. Peirce & Dworkin, *supra* note 111, at 654-55. The authors note that this well-accepted principle contrasts with nineteenth-century workplace injury theory:

> [I]t was assumed that employees were compensated for the known risks of employment through adjustment in the wage scale. This assumption, in turn, rested on several other [fallacious] assumptions. Among these were that the workers could accurately assess the probability and severity of the risk, that they had sufficient bargaining power to demand higher wages based on those risks, and that the risk-related wage increase would be used to buy insurance or some other buffer against the happening of the risk.

Id. at 655 (footnote omitted).

282. G. Calabresi, *supra* note 280, at 245. Professor Calabresi stated:

> An allocation [of costs] to nonfaulty employers could and did lead to cheaper cost avoidance because it resulted in adequate evaluation of the risk of injury and in its full retention as an economic factor internal to the employment contract. Workmen's compensation thus eased the problem in one area. But it made no change in the fault system, which still largely ignores the question of which category of possible loss bearer is most likely to be adequately informed of the costs involved.

Id. at 245-46.

proved to be a significant barrier to compensability, it is doubtful that substantial numbers of persons claiming reproductive or genetic harm will be compensated under the present interpretation of the system. The absence of an economic burden gives the employer no incentive to conduct safety monitoring or to implement health measures. Moreover, the system frequently forces reproductively and genetically injured workers to bear the costs of their legitimate injuries—a result running counter to the goal of loss-spreading.

The tort system has failed in the same manner. First, the worker must circumvent the exclusivity of workers' compensation to enter the tort system. Second, although theoretically the injured worker can receive greater monetary recovery in the tort system due to the availability of compensation for pain and suffering,[283] the worker must confront the obstacle of cause-in-fact. Third, courts have been reluctant to impose liability on an enterprise when the full extent of the liability may be inestimable at the time the enterprise must purchase insurance to cover the risk of the particular activity.[284] Thus, the very limited availability of the tort system to provide redress for persons with occupational reproductive or genetic injuries demonstrates that a system grounded in the traditional concept of accidents is incapable, absent modification, of handling the new class of environmental injuries that will arise in the 1990s and beyond.

The inherent risk-bearing capacity of industry—in contrast to the individual worker—renders it reasonable that industry be required to bear the full extent of the losses associated with its activities. This makes sense from an economic standpoint because industry is in the superior position to manage those risks through compensation, insurance, and loss-spreading. Assuming this is true, issues arise regarding the respective roles of workers' compensation and tort law in advancing these goals, and regarding the relative merit of the legislature compared to the judiciary in implementing appropriate solutions.

A primary effect of the failure of the tort and compensation systems with respect to toxic reproductive and genetic workplace injuries is the problem of underdeterrence. In the workers' compensation context, accident prevention is subordinate to compensation because, at least theoretically, compensation should be available to an injured worker even in situations in which the employer could not have avoided the injury.[285]

283. Statistics compiled in the 1970s revealed that the average total compensation benefits paid for a worker permanently disabled by a workplace injury were $4,000. Approximately one quarter of those workers brought tort actions that yielded an average of $40,000 per person. *See* Weiler, *supra* note 185, at 829 (citing Bernstein, *Third Party Claims in Workers' Compensation: A Proposal To Do More With Less*, 1977 Wash. U.L.Q. 543, 562-64) (noting that these statistics predate explosion in occupational disease claims, including those related to asbestos).

284. *See* H.R. Moch Co. v. Rensselaer Water Co., 247 N.Y. 160, 168-69, 159 N.E. 896, 898-99 (1928); Prosser & Keeton on Torts, *supra* note 209, § 4, at 25.

285. *See* Haas, *supra* note 104, at 849. "Indeed, workers' compensation requires pay-

When toxic reproductive or genetic injuries fall within the coverage of the workers' compensation system, but are not compensable within that scheme, employers not only are invited to ignore accident prevention, but they also are rewarded for engaging in more egregious behavior such as concealment of occupational hazards.[286]

Furthermore, the problem of overdeterrence arises when the system expands to accommodate novel types of claims. Thus, imposing liability to the outer limits of foreseeability would tend to discourage, rather than encourage, any safety measures by the employer.[287] Employers might conceivably elect to abandon their industrial activities rather than bear the risks associated with those activities. The risk of overdeterrence is especially acute because traditionally liability has been viewed as all-or-nothing once the preponderance-of-the-evidence standard has been met.[288]

The challenge to reformers is to structure the system so as to encourage employers to continue engaging in socially beneficial activities while reducing the hazards associated with them. This result can be achieved exclusively through the market system, by encouraging voluntary accident prevention measures, or by legislative decree.

2. Compensation

Difficulty in distinguishing most reproductive and genetic injuries from background occurrence levels[289] fosters concern that the law will operate to overcompensate some plaintiffs whose injury actually was attributable, in whole or in major part, to a cause other than the workplace environment. This uncertainty arises primarily from scientific uncertainty in identifying the precise cause of a particular reproductive or genetic event.[290] But uncertainty also can arise from an inability to distinguish between multiple factors that combine to cause the injury.[291] If the system operates in the traditional mode, compensation would

---

ment even in cases where the injury is clearly the employee's fault and there is nothing—short of firing or not hiring him—that the employer could have done." *Id.*

286. *See supra* notes 104-76 and accompanying text.

287. This observation also applies to situations in which the state-of-the-art defense has not been allowed in product liability actions. *See generally* Beshada v. Johns-Manville Prods. Corp., 90 N.J. 191, 447 A.2d 539 (1982) (state-of-the-art defense not allowed and therefore medical community's lack of knowledge of dangers of asbestos not a defense).

288. *See* Robinson, *supra* note 253, at 784; Rosenberg, *supra* note 251, at 862-66.

289. Thus, in the "Agent Orange" litigation, the court opined that the plaintiff's alleged injuries, including reproductive injuries, could be attributable to a variety of causes and not just the dioxin to which they were exposed. *See In re* Agent Orange Prod. Liab. Litig., 597 F. Supp. 740, 777 (E.D.N.Y. 1984), *aff'd*, 818 F.2d 145 (2d Cir. 1987), *cert. denied*, 484 U.S. 1004 (1988).

290. *See supra* notes 58-98 and accompanying text.

291. For example, a claimant's infertility may have been the combined result of the occupational exposure and such contributing factors as a pre-existing genetic defect, hormonal imbalances, or previous infection. *See* Office of Technology Assessment, Infertility, *supra* note 24, at 61-76. For a discussion of apportionment of occupational disease

likely be denied for failure to prove the requisite work-relatedness or causation. If causation requirements are eased to recognize such injuries, however, the risk of overcompensation may be substantial.

Because tort law traditionally serves the goal of making the plaintiff whole,[292] the system attempts to avoid payment of money to those whose injuries are speculative. The system has far less of a problem, however, denying compensation to persons whose injuries were probable but whose proof fell below the recognized preponderance-of-the-evidence standard. Thus, while the system has incorporated undercompensation, it has found overcompensation intolerable.[293]

While notions of fairness underlie the compensation goal, whether express or implicit,[294] the requirements of proof of causation tend to act as a check on the system to prevent unbridled overcompensation. Relaxation of the requirements of proof of causation would not necessarily cause compensation to run amok, but would reduce undercompensation and would likely result in aggregate verdicts that more accurately reflect the actual causal effect between exposures and injuries.[295] Moreover, defendant-employers remain in the best position to investigate and mitigate the risks to workers in the occupational setting. Employers are able to spread any incremental increase in loss due to overcompensation by obtaining insurance coverage and passing the costs on to consumers.

IV. THE 1990S AND BEYOND: PROPOSAL FOR RESPONDING TO THE COMING CRISIS IN REPRODUCTIVE AND GENETIC WORKPLACE INJURIES

Industry is in the best position to conduct the necessary research and

benefits in the multiple causation situation, see Peirce & Dworkin, *supra* note 111, at 665-78.

292. Special Committee on the Tort Liability System, Towards a Jurisprudence of Injury: The Continuing Creation of a System of Substantive Justice in American Tort Law 4-29-32 (1984) [hereinafter Special Committee on Tort Liability].

293. The fear of overcompensation accounts, for example, for the reticence of courts to entertain claims for increased risk of illness. *See, e.g.*, Ayers v. Jackson Township, 106 N.J. 557, 597, 525 A.2d 287, 307-08 (1987) (recognizing that enhanced risk claim would be windfall for persons who do not ultimately develop the disease). The *Ayers* court, however, did recognize a cause of action for increased risk of illness. *See supra* note 266 and accompanying text.

294. Special Committee on Tort Liability, *supra* note 292, at 4-41-52. Courts sometimes have used fairness to justify granting compensation to plaintiffs presenting weak evidence. *Id.* at 4-49.

295. Validation of the system in the public mind may be an important reason that supporters of traditional causation rules reject novel approaches. Professor Rosenberg has noted that advocates of adherence to strict requirements of particularistic evidence for the proof of causation may believe that juries are more likely to have strong convictions in the truth of their verdicts under traditional rules. *See* Rosenberg, *supra* note 251, at 872. Thus, a public belief may exist that verdicts should be based on something more than mere probability. Professor Rosenberg states, however, that uncertainty and inaccuracy is inherent in the traditional system as well, and that strict application of traditional causation rules merely "shield[s] the public from having to come to terms with the irreducible uncertainty of court judgments." *Id.* at 873-74.

development to determine the toxicity of the substances it uses and to which its workers and related persons may be exposed. Logic dictates, therefore, that reform measures should revolve around incentives for industry to obtain this information and undertake health and safety measures.

Statistics indicate that reported cases of occupational disease are on the rise in this country,[296] and many of these diseases are reproductive or genetic in nature.[297] The problem requires a two-fold response. First, archaic presumptions inherent in both the workers' compensation and tort systems must be discarded to allow entrance of reproductive and genetic claims into the systems alongside more traditional types of claims. This includes relaxing the standard of proof required to show causation and recognizing a cause of action in tort for employer misconduct in addition to recovery under workers' compensation. Second, some legislative remedy must be enacted in areas where particular urgency or uniformity is warranted.

A. *Modification of the Mechanisms of Liability*

1. Realigning the Burden of Causation

Persons injured by toxic reproductive or genetic exposures in the workplace may encounter a paucity of scientific information regarding the potential effects of the substances to which they were exposed. Typically, these claimants will present evidence of probabilities in the form of epidemiological or toxicological studies[298] but will not be able to make the necessary showing of cause-in-fact. The tribunal's response to the absence of a showing of cause-in-fact, the acknowledged backbone of the American concept of causation, most likely will result in foreclosing the claimant from recovery. Although circumstantial evidence has consistently met with approval as a means of proving causation in tort actions generally,[299] probabilistic causation has encountered a cold reception.[300]

Some adjustment of traditional cause-in-fact requirements is warranted in cases involving reproductive or genetic injuries. Several factors underscore the persuasiveness of this approach. First, in allowing recovery based on market share liability and risk-contribution theories, tort law has moved toward allowing some alteration in causation-related requirements. If any "law of toxic torts" has developed as a discrete cate-

296. Cases of occupational disease reported to the Bureau of Labor Statistics in 1987 demonstrated a 39% increase over such reported cases in 1986. Some explanation for this dramatic increase may be found in the more precise recording of occupational illness as mandated by federal guidelines. *See* Office of Technology Assessment, Genetic Monitoring, *supra* note 9, at 45.

297. *See id.* at 55.

298. *See supra* notes 61-98 and accompanying text.

299. *See* Firak, *supra* note 252, at 332.

300. *See generally supra* notes 249-79 and accompanying text (discussing the courts' antipathy toward such probability-based theories of causation).

gory in response to the special needs of toxic injuries, these special rules—insofar as they have developed—are its salient points.[301]

Second, claimants in toxic exposure cases have severely limited access to the evidence necessary to prove their cases, particularly if the injury is one that is associated with a high background occurrence rate. Statistical studies may be the only available evidence; clinical practitioners may be unable to provide any further insight into the causative factors of the claimants' disorders and may in fact rely on the very same statistical studies in their clinical evaluations.

Third, compelling policy goals justify an alteration in the traditional concepts of causation for workers' compensation and tort. Employers are in a position superior to their workers to determine, assess, and bear the risks associated with the hazards of their enterprises.[302] Indeed, the economic disparity between workers and their employers, and the inherent inequities of the employment relationship, enhance the need for employers to be responsible for the costs of the enterprise. This concept is warranted from an economic, social, and moral standpoint. Furthermore, for deterrence to play an appropriate role in the tort marketplace, compensation must be available to those who are injured as a result of exposures in the workplace.

Accordingly, in cases in which claimants make a showing of general statistical causation, the burden should shift to the employer to disprove causation.[303] The claimant's prima facie case should consist of: (1) epi-

301. *See, e.g.*, Sindell v. Abbott Laboratories, 26 Cal. 3d 588, 612-13, 607 P.2d 924, 937-38, 163 Cal. Rptr. 132, 145-46, (establishing market share liability for DES cases), *cert. denied*, 449 U.S. 912 (1980); Collins v. Eli Lilly Co., 116 Wis. 2d 166, 200, 342 N.W.2d 37, 53 (1984) (establishing risk contribution liability for DES cases); Ayers v. Jackson Township, 106 N.J. 557, 592, 525 A.2d 287, 305 (1987) (recognizing availability of cause of action for enhanced risk of disease upon showing of quantified risk).

302. This disparity between the employer and employee in terms of risk assessment capabilities is particularly noticeable in the occupational disease context. While analysts could argue that a worker can gauge the safety of the workplace vis-à-vis accidents, thus rendering deterrence and economic incentives to employers relatively unnecessary, this same statement could not be true with respect to occupational disease, due to the increased hardship to the worker of obtaining and interpreting information regarding the exposures and injuries. *See* Haas, *supra* note 104, at 875 (citing Smith, *Protecting Workers' Health and Safety*, in Instead of Regulation 311, 327 (R. Poole ed. 1982)).

303. Such a proposal necessarily eschews the cause-in-fact requirement in favor of probabilistic evidence. One commentator argues that while such a violation of the cause-in-fact requirement is acceptable in the unique circumstances of indeterminate defendants in DES cases, it is unacceptable outside that context. *See* Firak, *supra* note 252, at 334-35. DES produces signature diseases that are clinically distinguishable from background levels of related diseases. The DES plaintiffs' sole difficulty was in identifying the specific defendant that manufactured the DES ingested by their mothers, causing injury to them in utero. *See id.* at 334. The author contrasts the "scientific sense" of this situation with the issue of whether epidemiological evidence should be allowed to satisfy the cause-in-fact requirement, concluding that epidemiological evidence should not be so used. *See id.* at 334-35. Many DES plaintiffs are capable of satisfying both the general and specific causation requirements with respect to the relationship between their disease and the substance to which they were exposed. *See id.* Thus, the DES cases are not analogous to most cases involving persons suffering reproductive or genetic impairment through expo-

demiological evidence tending to establish a scientific relationship between the toxic substance and the claimant's reproductive or genetic injury; (2) the presence of the substance in the employer's workplace in a location that would have exposed the claimant to the substance; (3) a temporal relationship between the worker's employment with the employer and the presence of the substance in the workplace; and (4) clinical proof of the presence of reproductive or genetic injury in the claimant.[304] Once the plaintiff makes this prima facie showing, the employer may submit evidence to the factfinder that tends to refute the claimant's epidemiological studies or clinical evidence or that challenges the worker's exposure in time, location, and amount. The mere fact that a claimant's epidemiological study falls short of the highest level of scientific or medical certainty should not prevent its use to satisfy the prima facie showing of causation.[305] Reasonable probabilities suggested by the study will be sufficient.

This proposal only represents a shifting of the burden. Claimants will not necessarily recover merely because they are permitted to reach the trier of fact. If the employer can reasonably refute a claimant's evidence with conflicting studies or clinical information, the claimants may not recover. But if the employer cannot refute the claimant's initial showing—even where the defendant's lack of evidence results from scientific

---

sure to occupational toxins. The persons in the latter cases may not be able to demonstrate the requisite particularized causation through no fault of their own and in the absence of any indication of a signature disease. Perhaps the day will come when signature genetic defects can be associated with specific toxic substances; that day will arrive more quickly if the law emphasizes the goal of deterrence through various means, including incentives to conduct research and development.

304. This framework is similar, although not identical, to the proposal presented in Hall & Silbergeld, *Reappraising Epidemiology: A Response To Mr. Dore*, 7 Harv. Envtl. L. Rev. 441 (1983). Speaking generally in terms of chemical exposure cases, Hall and Silbergeld place the burden upon the plaintiff to demonstrate "exposure significant enough to trigger disease." *Id.* at 445. In most occupational disease cases, workers are underinformed as to the level and extent of their exposure to hazardous substances in the workplace. Although this may be changing from a regulatory standpoint, *see* Emergency Planning and Community Right-To-Know Act, 42 U.S.C. §§ 11001-11050 (1988 & Supp. 1991), numerous potential claims exist at the present time for which pitifully little information exists. Therefore, imposing the burden on the employer to produce information regarding the level and extent of the worker's exposure to the toxic substance is appropriate.

305. *But see* Dore, *supra* note 63, at 432-33 (repudiating the use of epidemiological evidence as sole proof of causation in toxic tort cases because of its lack of scientific certainty). Professor Dore has stated:

> First, epidemiology cannot determine which particular factor caused a particular person's disease, but only what factors are statistically associated with the occurrence of disease in groups of people. Its usefulness to particular plaintiffs therefore relates more directly to issues of risk than of actual occurrence. Second, a host of technical and practical considerations limit epidemiology's usefulness even in assessing general risks.

Id. at 433 (footnotes omitted). The proposal set forth in this Article does not abandon cause-in-fact entirely, however, but shifts the burden to the defendant to produce information tending to refute its existence.

uncertainty—the defendant may be required to bear the loss. Due to broad problems of scientific uncertainty, however, some undercompensation will remain when plaintiffs cannot make out their prima facie cases.[306] The key element to this proposal is that it is incumbent upon the employer to provide exculpatory evidence regarding exposure in the workplace—the area over which the employer exercises the highest control.

Admittedly, the initial effect of implementation of this proposal will be over-recovery. After a period of potential over-payment and adjustment, however, over-payment problems will be minimized as employers become motivated to conduct their own workplace studies and adjust or eliminate the risk of many workplace toxins. This period of adjustment can be shortened by implementing the legislative recommendations set forth in this Article at the same time that the judicial and administrative reforms take effect. Moreover, the employer will have an opportunity to decide whether it is willing to bear the various risks inherent in the enterprise and then may secure adequate insurance against losses related to the risks of overcompensation.

In the workers' compensation context, this burden-shifting would have the effect of a presumption of work-relatedness. For reasons of symmetry, a similar presumption should be allowed in employee tort actions that fall outside of the limitations the workers' compensation exclusivity doctrine.[307] When the employer is the defendant, the identical policy

306. Professor Delgado has proposed a collective liability scheme that reflects some of these concerns regarding causal indeterminacy. He has proposed that a representative plaintiff satisfy the following elements of a prima facie case:

> (i) that plaintiffs have suffered an injury; (ii) that the injury be one that could have resulted from either natural or human causes, acting separately and without synergy; (iii) that the injuries be causally indeterminate—that is, not identifiable as humanly or naturally caused; (iv) that the defendant is the only possible human cause; and (v) that the population injured, mode of risk, and other variables be uniform and stable enough to permit calculation of the increased number of victims.

Delgado, *supra* note 252, at 899-900. Upon the plaintiff's establishment of a prima facie case, the burden would shift to the defendant to establish noncausation in individual cases, to show that the plaintiff has overestimated the number of persons within the class, or to prove the existence of another defendant. *See id.* at 900. The plaintiff would recover from the particular defendant an amount equal to the class's aggregate injuries associated with that defendant's activities. *See id.* at 901. This level of relaxation of the causation requirements results in a kind of judicially enacted insurance program for all persons who were exposed or might have been exposed to toxic substances. As heretofore stated, such a broad-reaching rule is better left to the legislatures for consideration and appropriate action. For an analysis of some of the anticipated criticisms of the Delgado proposal, see *id.* at 902-08. One anticipated problem is the possibility that actions would arise in which a class of indeterminate plaintiffs sues a class of indeterminate defendants, thus invoking both the Delgado suggestion and the theory of market-share liability. *See id.* at 907-08.

307. Shifting the burden of causation in tort actions in which a non-employer third party is the defendant also is consistent with sound remedial goals. Assume the situation in which a third-party chemical manufacturer provides materials to the worker's employer for use in production of the employer's products. Assume further that this manu-

goals are served by allowing this presumption in the litigation context. Similarly, the presumption should also extend to a spouse and offspring who bring tort actions against the worker's employer for injuries arising out of the worker's exposure to occupational toxins. The fact that the employer may not have been able to anticipate the reproductive or genetic risk to persons other than the worker should be irrelevant under this scheme. Cost distribution and market forces eventually will regulate whatever seemingly inequitable results may occur in the initial stages of implementation.[308]

Additionally, states ideally should mandate some form of proportionate recovery reflecting the percentage probability that the employer's activities actually contributed to the employee's injury. Proportionate recovery would increase the efficiency of the employer's cost allocations by holding the employer liable only for the proportion attributable to the employer's workplace environment.[309] Accordingly, the defendant, in

---

facturer has been inattentive to the health and safety risks associated with its chemical, making little or no effort to determine the potential effects of the chemical in the workplace environment. If the employer is required to pay workers' compensation benefits to its injured employees as a result of toxic exposure to the manufacturer's materials, the employer will bear the costs of compensation—albeit at the reduced rate of workers' compensation—and of spreading the loss. Moreover, the employer will assess the risk and may, in fact, decide to eliminate the manufacturer's materials from its production process, if that option is available. If the employee cannot make the requisite showing of causation in a third-party action against the manufacturer, the employee is left undercompensated and the manufacturer is underdeterred. If, instead, the manufacturer is subject to the suggested presumption of causation—with the showing of existence of the substance in the workplace augmented to require the presence of the defendant's substance—the culpable manufacturer will be held accountable for the costs of its conduct. Ideally, this would result in enhanced communication between the manufacturer and the employer regarding use of toxic substances in the workplace.

308. One pair of commentators has found the following advantages in allowing the shifting of the burden of proof to the employer in the workers' compensation context: (1) the superior position of the employer vis-à-vis the information related to the exposure and the litigation process; (2) "the increase in costs due to the employer's added burden is more likely to accurately reflect the true costs of occupational disease"; (3) the priority of compensating victims of occupational disease; and (4) ultimate reduction of costs, as employers will likely not contest many claims. Peirce & Dworkin, *supra* note 111, at 682.

309. *See id.* at 681-83 (proposing shift in burden of proof to defendant to prove non-work-relatedness and apportionment of benefits in workers' compensation context). The authors further propose establishment of a fund to compensate injured workers for the additional amount of total disability above the percentage apportioned to the employer. The taxpayers, compensation insurers, and employees would pay equally into such a fund. *See id.* at 681-82, 685. The authors' rationale for the proposed fund is as follows:

> Requiring the claimant to bear the burden of proof [of percentage of work-relatedness] will not result in claimants going without compensation if special funds are utilized; it would merely more accurately allocate costs and allow businesses to be more competitive. In addition, in the absence of a special fund, an injured employee could go uncompensated for that part of the injury apportioned. That would seem unconscionably harsh and contrary to the rationales behind the workers' compensation system and would encourage the courts to continue avoiding apportionment.

Id. at 685. Employees apparently would receive payments from such a fund even if their injuries were attributable in part to hereditary factors or environmental factors beyond

addressing the plaintiff's case, should submit evidence of the proportional relationship between the workplace exposure and other possible contributing sources of the injury. Although critics of proportionate recovery object to its reliance on proof of probabilistic causation, toxic reproductive and genetic hazards are particularly suited to this mode of liability. Indeed, as one commentator has concluded, "proportional recovery need not be conceptualized as compensation for risk as such, but rather as a means of compensating for actual harm given limited information about causation."[310]

2. Recognizing Tort Actions for Employer Misconduct

The latency periods often associated with reproductive and genetic injuries leave open the door for employer misconduct. Such misconduct may not rise to the level of an intentional tort, but may be manifested in failure to properly disclose either occupational hazards or potentially work-related known adverse health conditions.[311] Long latency periods reduce the probability that the injured worker will be able to make the necessary showing of work-relatedness for workers' compensation. Absent an avenue of liability in the tort system, employers engaging in unscrupulous and deceptive tactics would benefit from the shield of immunity granted by the exclusivity doctrine. The problem is particularly egregious when employers fraudulently conceal workers' health conditions and deliberately fail to conduct health and safety assessments or warn employees of workplace hazards. The latter problem can affect a single employer or, more noxiously, an entire industry.

Piercing the exclusivity doctrine to allow tort actions against employers would be a salutory means of remedying these transgressions. The system should permit an injured worker to collect workers' compensation benefits to the fullest extent available, while at the same time maintaining a tort action against the employer for extreme misconduct. Such an action should be based on intentional misconduct, gross negligence, recklessness, fraudulent concealment, or other willful or wanton misconduct. Any compensatory damages recovered by the claimant in the tort

---

the occupational setting. Allowing employees to recover for such contributing risks would place them in a position superior to other persons whose identical injuries had no workplace connection, and who, even in the most apportionment-minded jurisdictions, would receive no compensation for risks associated with non-defendant activities. Certain kinds of funds may indeed have an appropriate place in handling the problems associated with toxic exposures generally. *See infra* notes 316-17 and accompanying text. The Peirce and Dworkin proposal's inequities lead to the conclusion that a more general insurance scheme for toxic injuries may be necessary. While such a notion may be worth considering, particularly in light of the difficulty the system has had in managing the explosion of asbestos cases, no such scheme would be forthcoming for quite some time.

310. Farber, *supra* note 270, at 1241.

311. Some employers may choose to monitor their employees for either occupational susceptibilities or evidence of occupational illness. For a discussion of the uses and misuses of the results of screening and monitoring in the workplace, see Office of Technology Assessment, Genetic Monitoring, *supra* note 9, at 126-31.

action should be reduced by any amount of workers' compensation benefits paid by the employer with regard to the same injury.

A salient feature of the employer misconduct tort action should be the availability of punitive damages against the employer. Allowing punitive damages would serve deterrence goals and provide relief in tort for the most egregious employer conduct. Moreover, allowing a tort action and subjecting employers to punitive damages would remove the current veil of immunity from employers accused of exposing their employees to toxins and place such employers in positions equal to other tortfeasors engaged in extreme activities injurious to others.

In *Gulden v. Crown Zellerback Corp.*,[312] the United States Court of Appeals for the Ninth Circuit determined that the employees' action against their employer might be maintained outside the exclusivity of the Oregon workers' compensation scheme. The plaintiffs claimed injury as a result of their employer's order that they clean up toxic PCBs on their hands and knees after an accidental spill. The plaintiffs claimed that the levels of PCBs in their bodies exceeded established safety levels and that the employer's conduct rose to the level of an intentional battery. In finding that the action was not barred by the exclusivity doctrine, the Ninth Circuit stated that "a jury could conclude that the intention to injure [the employees] was deliberate where the employer had an opportunity to weigh the consequences and to make a conscious choice among possible courses of action."[313]

The test in *Gulden* should be extended to actions alleging willful, reckless, grossly negligent, and fraudulent conduct. Moreover, courts should allow an action where the employer deliberately chose to remain in ignorance of any hazards. The justifiability of the employer's ignorance would define the distinction between mere negligence, covered only by workers' compensation, and gross negligence giving rise to a tort action.

3. Legislative Solution for Subsequent Generations

Strong arguments exist both in favor of and in opposition to extending tort remedies to reach victims in subsequent generations who develop injuries from an initial workplace harm.[314] On the one hand, such claims in the worker's hereditary line are certainly foreseeable. On the other hand, social policy concerns provide ample reason for the law to disfavor recognition of such actions.

Extending duty to the outer limits of foreseeability raises questions of manageability and risk allocation. In *Enright v. Eli Lilly and Co.*,[315] the New York Court of Appeals refused to extend liability to preconception

312. Gulden v. Crown Zellerback Corp., 890 F.2d 195, 196-97 (9th Cir. 1989).

313. *Id.*

314. *See supra* notes 227-47 and accompanying text.

315. Enright v. Eli Lilly & Co., 77 N.Y.2d 377, 387-88, 570 N.E.2d 198, 204, 568 N.Y.S.2d 550, 553 (1991), *cert. denied*, 112 S. Ct. 197 (1991); *see also supra* notes 239-47 and accompanying text (discussing *Enright*).

injuries on these policy grounds and thus limited liability to those individuals who ingested the DES or were exposed to it in utero. Particularly in genetic-injury cases, it is impossible to accurately predict the probability of expression of abnormalities in subsequent generations. This uncertainty obscures the ability of insurers and employers to accurately measure their risk of exposure to claims. Coupled with a discovery statute of limitations, claim exposure could be virtually infinite, with the difficulty of defending against such claims increasing as each generation passes.

Notwithstanding these arguments, policy reasons exist for allowing some means of recovery for members of subsequent generations. If the employer is not required to pay the costs of these injuries, the full burden falls upon the injured persons and society.[316] Moreover, although the extent of injury to persons born in subsequent generations may be as severe as or even more severe than the initial injury to the worker, identification of the injury's source and proof of causation will be more difficult due to the latency period. Meanwhile, if the initial injury is covered by workers' compensation and the actions for injury to subsequent generations disallowed, the undeterred defendant has little incentive to curtail the hazardous activity.

The need for a balance between the goals of compensation and deterrence on the one hand and manageability on the other warrants legislative establishment of a "contingent injury fund."[317] Financed by industry as an ongoing cost of their enterprises, this fund would compensate subsequent generations as they actually develop illnesses or other injuries related to the initial occupational exposure. Persons presenting claims to the fund for payment would benefit from a relaxed standard of causation similar to the shifting of burdens proposed herein. Similarly, recovery should be on a proportionate basis. For some persons, payment through the fund could result in reduced compensation from that which the claimant might have received if the claim had gone before a jury in a tort action. But on balance, the availability of some compensation for all who can make the necessary showing is a desired result.

Moreover, for employers, ongoing payment to the fund would represent a cost of business that can be absorbed into the cost of their products. The system should allow for adjustment of contribution levels on the basis of relative risk. Thus, if an enterprise becomes less likely to actually cause injuries to subsequent generations, either through implementation of health and safety measures or through abandoning alto-

316. *See* Note, *Preconception Torts: Foreseeing the Unconceived*, 48 U. Colo. L. Rev. 621, 625 (1977).

317. This approach is similar to a proposal made more than three decades ago in the context of radiation injuries. *See* Estep, *Radiation Injuries and Statistics: The Need for a New Approach to Injury Litigation*, 59 Mich. L. Rev. 259, 281-98 (1960). *See generally* Comment, *Radiation and Preconception Injuries: Some Interesting Problems in Tort Law*, 28 Sw. L.J. 414, 425-32 (1974) (analyzing legislative and common-law issues related to genetic injuries from exposure to radiation).

gether certain hazardous enterprises, a reduction of payment would be appropriate. This reward would act as an incentive to undertake injury-prevention measures.

B. *Enhancing the Utilitarian Goals of the System*

1. Medical Monitoring for Employees

Medical monitoring is a direct means of achieving reduction in the costs of workplace accidents by making an ongoing investment in illness detection and early diagnosis. Optimally, the aggregate cost of medical monitoring to the employer would be less than the costs of paying workers' compensation and tort judgments in the absence of monitoring. A principal concern is how employers' insurers will use medical monitoring tests. An appropriate balance of interests is necessary so as not to transgress the privacy rights of employees.

Some form of medical monitoring—either to obtain exposure data or to screen for certain medical conditions—is already required by various OSHA standards related to hazardous occupational exposures.[318] The general OSHA test for monitoring is to avoid any "material impairment of health" by the worker.[319] Moreover, relevant OSHA standards designate specific details regarding medical monitoring rather than leaving them to the employer's discretion.[320] Typically, OSHA-mandated monitoring requires a general medical examination; rarely does it require more invasive testing.[321]

Although medical surveillance is generally predictive and noninvasive, some concern has arisen that monitoring ex ante could result in employers screening out employees who are more sensitive to certain hazards to avoid high costs at a later date.[322] For example, debate has ensued regarding the appropriate OSHA approach to monitoring lead levels in employees' blood. The rule currently requires ambient air lead level

318. *See* OSH Act, *supra* note 3, § 655 (granting authority to OSHA to promulgate occupational safety and health standards). OSHA may determine that a health and safety standard is needed based upon information obtained from individuals (including an individual employee), labor organizations, political groups, or NIOSH. *See id.* § 655(b)(1). OSHA also retains the authority to modify or revoke existing standards. *See id.* § 655(b). In some instances, OSHA may issue emergency temporary standards (ETSs) if, in its determination, employees are exposed to "grave danger" from toxic or otherwise physically harmful substances in the workplace. *See id.* § 655(c)(1). Moreover, OSHA may from time to time issue guidelines for employers, even where no permanent or temporary standard exists. *See, e.g., Safety and Health Program Management Guidelines*, 54 Fed. Reg. 3904, 3904 (1989) (detailing how OSHA constructed new safety and health guidelines to be used by general industry, shipyards, longshoring and marine terminals).

319. OSH Act, *supra* note 3, § 655(b)(5).

320. *See id.* § 655(b)(7).

321. The presence of lead in the workplace requires periodic blood tests of employees and possible removal of an employee from a high lead work area if blood lead levels exceed certain prescribed safety levels. *See* 29 C.F.R. § 1910.1025 (1990).

322. *See* Office of Technology Assessment, Genetic Monitoring, *supra* note 9, at 106.

measurements to determine exposure coupled with periodic blood testing to monitor the workers' health.[323] While this example is the farthest the OSH Act has gone with invasive medical surveillance, it may set a precedent for other standards.[324] Although the OSH Act provides that employees may not be discriminated against for exercising any right granted under the Act,[325] private employer plans for medical surveillance fall outside of the OSHA prohibition unless the program violates an OSHA standard.[326]

Common-law principles of privacy and confidentiality in medical monitoring generally require that the worker have a right to refuse the monitoring, to know the results of the tests and any further information that is relevant to the worker's family, and to have the monitoring tests kept confidential by the examiner.[327] If a valid public interest exists in the worker's medical information, however, a breach of confidentiality may be justified.[328]

These general rules are complicated somewhat when the health personnel conducting the monitoring are employees of the employer. The legal and ethical relationship between the health care personnel and the worker may be compromised by the apparent agency relationship between the health care personnel and the employer.[329] Thus, problems may arise if an employee does not wish the employer to have access to the employee's medical test results.[330] In more extreme cases, a physician or employer might release information regarding the workers' medical screening tests to the public.[331]

---

323. *See* OSH Act, *supra* note 3, § 651.

324. For a summary of the history of the OSHA lead standard, see Office of Technology Assessment, Genetic Monitoring, *supra* note 9, at 106.

325. *See* OSH Act, *supra* note 3, § 660(c).

326. *See* Office of Technology Assessment, Genetic Monitoring, *supra* note 9, at 106.

327. *See id.* at 116.

328. *See id.*

329. *See id.* at 117.

330. *See id.* at 118. *Cf.* 29 C.F.R. § 1904.2, 1904.7 (1972) (providing for employee access to medical records, but silent on employer's access). OSHA requires that employers maintain records of occupational injuries and illnesses. Although some points of difference exist as to the definition of an occupational illness, the most reasonable interpretation was advanced by the Occupational Safety and Health Review Commission in 1980 in holding that the recordkeeping requirement applied not only to illnesses resulting directly from workplace exposures, but also to conditions "in which the occupational environment either was a contributing factor to the illness or aggravated a pre-existing condition." General Motors Corp. (Inland Div.) 1980 O.S.H. Dec. (CCH) ¶ 24,743 (Aug. 29, 1980).

331. A small minority of states has statutory provisions designed to protect patients from unauthorized disclosures of medical information. *See* Cal. Civ. Code § 56.10-16 (West 1985); Mont. Code Ann. § 50-16-525 (1991); R.I. Gen. Laws § 5-37.3-4 (1987); Utah Code Ann. § 63-2-88 (1989); Wis. Stat. Ann. § 146.82 (West 1989). A few states have statutes granting some degree of protection expressly to genetic information. *See* Md. Health-Gen. Code Ann. § 13-109 (1990); R.I. Gen. Laws § 5-37.3-4 (1987); Utah Code Ann. § 63-2-88 (1989). For a general discussion of confidentiality duties, both common-law and statutory, within the context of genetic information, see Office of Technology Assessment, Genetic Monitoring, *supra* note 9, at 116-22.

With respect to workers' compensation benefits, an injured worker is entitled to, among other benefits, unlimited payment of medical and hospital expenses incurred as a result of the occupational injury.[332] Many states require that the medical benefits be reasonably required or necessary[333] and not merely for personal comfort. To extend these workers' compensation benefits to medical and genetic monitoring does not require a significant leap of the imagination. It does require, however, that the worker be able to demonstrate the existence of a covered occupational injury in the first instance. Such a system would be useful, for example, where a male worker suffers from occupationally induced oligospermia,[334] a cause of infertility. Assuming a showing of work-relatedness, the worker trying to conceive should be entitled to full medical payment of reasonable and necessary medical treatments to correct the problem and, ultimately, have children. A worker who is not trying to conceive should be entitled to medical monitoring of his condition to determine whether he continues to suffer damage and, if so, to what extent. Thus, this system provides some measure of equity between the reproducing worker and the non-reproducing worker.

Any further mandated ex post monitoring within the workers' compensation context poses many of the same privacy and confidentiality problems as does mandated ex ante monitoring. Accordingly, this issue would be better left to OSHA rather than to the workers' compensation administrative tribunal. Voluntary ex post monitoring at the insistence of the worker, on the other hand, arguably may be available outside workers' compensation. This concept is supported by *Acevedo v. Consolidated Edison Co.*,[335] in which a New York court held that an employee's action against an employer for medical monitoring for alleged asbestos injuries was not barred, because medical monitoring was not compensable under the state's workers' compensation law.

In *Ayers v. Jackson Township*,[336] the New Jersey Supreme Court set forth a broad standard for allowing medical monitoring claims in the area of toxic exposures. The court allowed the plaintiffs' claims for presymptom medical surveillance where toxic pollutants had leached into a drinking water supply from the township landfill. The court set forth the factors to be weighed in determining the reasonableness of the surveillance:

> The likelihood of disease is but one element in determining the reasonableness of medical intervention. . . . Accordingly, we hold that the cost of medical surveillance is a compensable item of damages where

332. *See* 2 A. Larson, Workmen's Compensation Law § 61.10, at 10-198 (1989).

333. *See, e.g.*, Ky. Rev. Stat. Ann. § 342.035 (Michie Bobbs-Merrill 1983) (reasonable and fair); Neb. Rev. Stat. § 48-120 (1988) (reasonably required and will relieve pain); Wash. Rev. Code § 51.36.010 (1990) (necessary and proper).

334. Oligospermia refers to a low sperm count. *See* Stedman's Medical Dictionary 980 (24th ed. 1982).

335. Acevedo v. Consolidated Edison Co., 572 N.Y.S.2d 1015, 1018 (1991).

336. Ayers v. Jackson Township, 106 N.J. 557, 606, 525 A.2d 287, 312-13 (1987).

> the proofs demonstrate, through reliable expert testimony predicated upon the significance and extent of exposure to chemicals, the toxicity of the chemicals, the seriousness of the diseases for which individuals are at risk, the relative increase in the chance of onset of disease in those exposed, and the value of early diagnosis, that such surveillance to monitor the effect of exposure to toxic chemicals is reasonable and necessary.[337]

The court in *Ayers* imposed a court-supervised fund out of which claims for medical surveillance were to be paid.[338] The court found this method preferable to a lump-sum verdict that would necessarily have to estimate the total future amounts for surveillance.[339]

For medical surveillance claims of this sort to apply to toxic reproductive and genetic workplace injuries, public awareness of the problem must be maximized to disprove the notion that these injuries are not medical problems, but merely social problems. Thus, the "seriousness" factors in *Ayers* should take account of the plaintiff's reasonable well-being and should not be limited to traditional life-threatening conditions. In any event, courts should take advantage of their equitable powers to fashion an appropriate remedial scheme for medical monitoring.

2. Legislation to Enhance the Generation and Use of Information

Focusing on the remedial aspects of toxic reproductive and genetic workplace injuries does not require tunnel vision. Accordingly, certain recommendations are warranted to encourage information gathering, particularly with respect to causation. This is the area in which legislation can be put to particularly good use. To reduce the overall costs of these reproductive and genetic harms, information must be generated. Workers with knowledge can make more informed choices regarding when to conceive and bear children, whether to accept certain workplace risks or seek a different job, and the extent to which medical attention may be necessary. Employers, on the other hand, can make more informed choices regarding the safety and health of their workers vis-à-vis the costs of their products or services.

With the help of a special panel of experts in the field, Congress should establish research priorities with respect to toxic reproductive and genetic occupational hazards. This group of experts should establish the scientific criteria to be used in these studies, with the aims of uniformity and accuracy.[340] Research conducted by public bodies and private in-

337. *Id.* at 605-06, 525 A.2d at 312.

338. *See also In re* Agent Orange Prod. Liab. Litig., 611 F. Supp. 1396, 1399 (E.D.N.Y. 1985), *aff'd in part, rev'd in part*, 818 F.2d 179 (2d Cir. 1987) (establishing distribution of settlement fund in mass litigation to compensate plaintiffs and their families for injuries caused by exposure to "Agent Orange" during Vietnam War).

339. *See Ayers*, 106 N.J. at 609, 525 A.2d at 313.

340. A more detailed proposal, with a similar thrust, has been set forth in Lyndon, *Information Economics and Chemical Toxicity: Designing Laws To Produce and Use Data*, 87 Mich. L. Rev. 1795, 1835-41 (1989). This proposal includes a joint private and

dustry would thus be coordinated for maximum utilization of resources and nonduplicative endeavors. Ultimately, this proposal would enhance the operation of the OSH Act by providing a consistent basis for imposition of OSHA standards with respect to various industries.

Cogent economic justifications exist for focusing increased regulatory attention on the production and distribution of information regarding the reproductive and genetic toxicity of workplace substances. Under the current laissez-faire policy, however, industry has a variety of disincentives to embark upon a course of information gathering. In particular, because an enterprise may not recoup its full investment on research viewed as benefiting the public good, the enterprise may not have sufficient profit motive to warrant the research commitment.[341] From an economic standpoint, some employers may believe that ignorance is financial bliss. The logical solution is to legislatively impose information gathering requirements and to judicially encourage employer accountability for workplace injuries.

CONCLUSION

The legal system has demonstrated ambivalence toward extending traditional rules of liability to cases of toxic exposure in the workplace. Although the incidence of occupational disease has increased dramatically in recent decades, most occupational disease cases encounter considerable institutional resistance from both the workers' compensation and tort systems. As a class, toxic reproductive and genetic workplace injuries pose special problems for both systems. Technological uncertainty, multiple causation, and latent disease processes, often present in a single case, challenge the legal system in an unprecedented fashion.

Heretofore, toxic reproductive and genetic injuries have been undercompensated, resulting in underdeterrence of employer conduct. The workers' compensation system, having developed out of concern for traumatic workplace accidents, has retained the presumptions that nontraumatic illness has its source in non-workplace factors and that injuries not resulting in disability from work are noncompensable. Similarly, the tort system has demonstrated confusion and ambivalence in addressing toxic-exposure cases. Both systems have held on tenaciously, and perhaps well beyond the point of logic, to traditional mechanisms of liability

public research program, based upon the concept of the Superfund and conducted according to a specific plan and protocol. Costs would be borne initially by a general fund, which would then be reimbursed by industries engaged in the use of substances revealed as hazardous. *See id.* at 1837.

341. *See id.* at 1813. Professor Lyndon notes that the "benefits [of research] may not be recovered by individual firms, or their impact may not be easily identifiable as the results of one company's research. Thus, firms are unlikely to undertake costly testing, because the benefits are public and cumulative and not reflected in the corporate balance sheet." *Id.* In a positive sense, however, research can dispel some of the mystery of toxicological harm through identification of toxic substances and processes, as well as enhance the preventive medical care. *See id.*

grounded in the cause-in-fact requirement. These archaic modes and presumptions are inadequate to handle the increasing number of toxic-exposure cases that will enter the system well into the next century. Now is the time to shed the restraints of these presumptions and develop a new jurisprudence to handle the great challenge posed by toxic injuries.

Reformers can undertake several initiatives at the present time to ease these claims into both the tort and workers' compensation systems. First, the cause-in-fact requirement should be relaxed by shifting to the employer the burden of proving noncausation after an initial showing by the claimant. This will reflect more accurately the respective abilities of the parties to determine the role of workplace exposures in the injury. Second, a legislative remedy establishing a fund for compensating descendants of exposed workers must be enacted to eliminate judicial ad-hoc line-drawing. Third, the tort system should be opened up to claims of willful employer misconduct that falls short of an intentional tort. This will assign appropriate liability for egregious fault-based conduct and impose economic incentives on employers to undertake health and safety measures in the workplace.

Furthermore, direct prophylactic measures should be implemented. Medical monitoring claims should be allowed in tort actions brought by workers against their employers and against third-party manufacturers. Federal legislative efforts should include imposition of information-gathering duties on employers and the establishment of research protocols that will, eventually, provide the medical information to abate the problems of causal indeterminacy.

Modification of the doctrines of tort and workers' compensation is desirable to achieve equity within the legal system and to assign appropriate economic burdens. This should be coupled with a comprehensive effort to increase health and safety incentives in the occupational setting. In the most concrete sense, the reproductive and genetic injuries in today's workplace may be with us for generations to come. It is shortsighted to demand that the modes and applications of the tort and compensation doctrines remain intractable. The legal system must enter the twenty-first century armed with the means to handle the harmful consequences of technological progress.