Liability for Prenatal Harm in the Workplace: The Need for Reform*

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I. INTRODUCTION

Fifteen percent of all recognized pregnancies result in spontaneous abortion¹ and seven percent of liveborn infants are afflicted with birth defects.² Although there are innumerable workplace stressors that are known or suspected to cause such injuries, there have been very few prenatal injury lawsuits brought against employers to date.³ In the last fifteen years, however, considerable attention has been focused on the issue,⁴ and the frequency of such lawsuits can be expected to increase.

This Article describes the causes of action available under current Washington law when a workplace hazard contributes to an adverse reproductive outcome such as miscarriage, birth defects, transplacental carcinogenesis, or other prenatal injury.⁵ Part II delineates the wide variety of workplace conditions that may lead to an adverse reproductive outcome, rang-
ing from emotional stress, cigarette smoke, and fall hazards to more traditional teratogen exposures such as lead. Part III describes the types of reproductive harm that can form the basis of a lawsuit in Washington. Part IV notes the theories of liability and the potential defendants, including employers, co-employees, consultants, manufacturers, and others contributing to such an outcome. Part IV also offers a prediction that the number of lawsuits alleging such injury are likely to increase dramatically. Part V discusses defenses available to an employer. Part VI identifies the goals that should be served under any proposed solution. Finally, Part VII argues for a legislative approach towards an equitable balancing of childrens' health interests against womens' employment opportunities and employers' concerns about massive tort liability.

II. Prenatal Harm in the Workplace

Most people's conception of prenatal injury revolves around images of children born with missing or shortened limbs because of their mothers' exposure to teratogenic chemicals. These extreme examples, however, are but a small part of a wide spectrum of prenatal injuries attributable to an even wider assortment of occupational conditions. For an attorney or legislator to comprehend the true magnitude of the potential liability faced by today's employer one must look at the larger picture. This is best accomplished by tracking human reproduction and pointing out the stages where an occupational insult can cause disruption and injury. For the sake of simplicity, this Article will discuss only fertilization and development leading to a single child.  

A. Preconception Congenital Injury

Conception begins when one gamete from each parent join. The male contributes spermatozoa containing half of the genetic material that will direct the development of the child. Spermatozoa originate from a normally inexhaustible pool of progenitor cells in the testes. These progenitor cells number in the many millions and routinely divide and subdivide to produce new

6. See generally Stanley W. Jacob et al., Structure and Function in Man (1978). This reference forms the basis for the following discussion of the biology of reproduction.
7. Id. at 584.
8. Id. at 582, 584.
9. Id. at 580-81.
spermatozoa for fertilization. The average ejaculation contains 200 to 300 million spermatozoa of which only one will fertilize the female's ovum. Throughout his life, the male's sperm cells, at various stages of maturation, are vulnerable to chemical or physical disturbances that may alter their genetic material in such a way that fertilization will not be successful or will lead to a defect in the child.

Many chemicals as well as ionizing radiation have been shown to cause aberrations in spermatozoa. While no "marker" genetic disorders have been isolated for specific exposures, epidemiological studies have correlated several occupations with above average incidences of certain birth defects as well as with higher rates of miscarriage among the spouses of exposed males. Paternally-mediated prenatal harm has also been alleged in civil suits.

10. Id. at 580.
11. Id. at 581.
12. OTA, supra note 1, at 47.
13. See id. at 69-125. A review of the literature showed that abnormal spermatozoa may be caused by several exposures, including arsenic, chlorprene, lead, kepone, and ionizing radiation. Id.
14. A "marker" disorder is one that is definitively attributable to a specific exposure.
15. See Andrew F. Olshan et al., Parental Occupation and Congenital Anomalies in Offspring, 20 Am. J. Indus. Med. 447 (1991). Dr. Olshan's epidemiological study correlates increased incidence of specific congenital anomalies with paternal occupations. Noted associations include the following: (1) janitor with hydrocephaly and heart defects; (2) forestry and logging workers with cataracts, heart valve problems, and fused fingers; (3) printers with clubfoot and urethral defects; and (4) plywood workers with dislocated hips, heart defects, and stomach defects. Id.; see also David A. Savitz & Jianhua Chen, Parental Occupation and Childhood Cancer & Review of Epidemiologic Studies, 88 Envtl. HLTH. PERSP. 325 (1990) (reviewing epidemiological literature and noting that "[s]everal associations have been found with consistency: paternal exposures in hydrocarbon-associated occupations, the petroleum and chemical industries and especially paint exposures have been associated with brain cancer; paint exposures have also been linked to leukemias").
The female contributes one of a finite number of ova, each of which, like the spermatozoon, contains half of the genetic material of the new child.\textsuperscript{18} The female's supply of ova is fixed from conception and declines with age.\textsuperscript{19} By the age of sixteen or so, when a woman can be expected to enter the work force, the number of oogonia (Greek for parents of eggs) number less than 400.\textsuperscript{20} These "parent" cells are frozen in a stage of cell division.\textsuperscript{21} Every ovarian cycle (approximately twenty-eight days) about twenty oogonia proceed through the last stages of division, but only one reaches the mature stage of an ovum.\textsuperscript{22} The same types of chromosomal damage that affect the spermatozoa can similarly impact the ova before they are fertilized and with similar consequences.\textsuperscript{23}

\textbf{B. Miscarriage}

At ovulation, the surviving, dominant ovum is released from its housing, the follicle, and proceeds through the oviduct, where it may be fertilized by the entry of one spermatozoon.\textsuperscript{24} Once the ovum is fertilized, it becomes a "zygote" and proceeds to divide and grow.\textsuperscript{25} Given proper genetic guidance, adequate nutrients, and correct hormonal stimuli from the mother's endocrine system, the zygote will implant in the endometrial lining of the uterus and begin to grow.\textsuperscript{26} While custom varies somewhat, the general rule is that the developing child is termed a zygote through the third week of pregnancy, an embryo during weeks three through eight, and a fetus thereafter until birth.\textsuperscript{27} The point at which the fetus becomes viable is generally presumed to be at twenty weeks and 500 grams (1.1 lbs.).\textsuperscript{28}

\begin{enumerate}
  \item \textsuperscript{18} OTA, \textit{supra} note 1, at 48-49.
  \item \textsuperscript{19} R. Berkow & A. Fletcher, The Merck Manual of Diagnosis and Therapy 1683 (1987).
  \item \textsuperscript{20} OTA, \textit{supra} note 1, at 48-49.
  \item \textsuperscript{21} \textit{Id}.
  \item \textsuperscript{22} \textit{Id}.
  \item \textsuperscript{23} See, e.g., \textit{id}. at 67-126 (comparing the toxic effects on sperm and ova).
  \item \textsuperscript{24} \textit{Id}. at 49-50.
  \item \textsuperscript{25} \textit{Id}.
  \item \textsuperscript{26} \textit{Id}.
  \item \textsuperscript{27} \textit{Id}.
  \item \textsuperscript{28} JACOB et al., \textit{supra} note 6, at 592. The point of transition to viability (when the fetus can survive outside the mother) is significant because it may determine whether the fetus has achieved person status under the law. Different jurisdictions that have ruled on this issue have reached different results. Some have adopted a bright-line demarcation as a matter of law of the 1st trimester (approximately 28 weeks). Roe v. Wade, 410 U.S. 113, 162-65 (1973). Others have considered the issue a fact question for trial. Georgia hinges the personhood question on the date the conceptus is "quick" (first
If the genetic material of the spermatozoon or the ovum has been interfered with by chemicals or radiation in a manner that has significantly diminished its integrity, the conceptus will not successfully implant and flourish in the endometrium of the mother. In human populations, the types and severity of parental exposures (or other factors) that can prevent the successful implantation of fertilized ova is open to speculation. This is because embryos that do not implant and survive for a week or so are neither detected nor counted in epidemiologic studies as a miscarriage.

While genetic harm that prevents successful implantation may be hard to measure, such harm can also lead to later miscarriages that are more easily documented and epidemiologically correlated with many workplace factors. Even exposures as widespread as to video display terminals are suspected of causing increased miscarriage rates. Of note is the

felt to move by the mother, generally around the 16th week), but the determination is submitted as a question of fact to the jury. Porter v. Lassiter, 87 S.E.2d 100, 103 (Ga. Ct. App. 1955). The medical literature is becoming murkier because technology is increasingly able to supplant maternal support mechanisms for preterm babies. To complicate matters further, not all researchers use the same cutoff in epidemiological studies of miscarriage.

29. This phenomenon forms the basis for the "dominant-lethal" test of mutagenicity in which male mice or rats are given an injection of the substance of concern and then mated with a series of unexposed females. The females are then sacrificed and the number of dead or resorbing fetuses are determined. Elevations in these early "miscarriage" rates are taken as evidence of mutagenicity (damage to the chromosomes of the sperm). The predominant effect of sperm mutation is the death of the embryo. LOUIS CASARETT & JOHN DOULL, TOXICOLOGY: THE BASIC SCIENCE OF POISONS 315 (1975).

30. OTA, supra note 1, at 51. It is estimated that approximately 50% of fertilized ova are lost in the first one to two weeks of development. Epidemiologic studies of miscarriage focus on recognizable pregnancies (those lasting more than two to three weeks).

31. Epidemiological studies correlating elevated miscarriage (1st trimester) rates with maternal occupations and exposures abound, even for traditionally clean industries such as electronics. See, e.g., Jane A. Lipscomb et al., Pregnancy Outcomes in Women Potentially Exposed to Occupational Solvents and Women Working in the Electronics Industry, 33 J. OCCUP. MED. 597 (reporting a greater than four-fold risk in spontaneous abortion among women reporting regular solvent exposure over population average); Gayle C. Windham et al., Exposure to Occupational Solvents and Adverse Pregnancy Outcome, 20 AM. J. INDUS. MED. 241 (1991) (identifying miscarriage rates 4.7, 3.1, and 2.3 times the background (control population) among women working with perchloroethylene, trichloroethylene, and paint thinners, respectively); Jun Zhang et al., Occupational Hazards and Pregnancy Outcomes, 21 AM. J. INDUS. MED. 397 (1992) (correlating maternal occupational exposure to noise, radiation, and chemicals with increases in the rates of antepartum death).

32. See Study Links Increased Miscarriage Risk to Workers with High Magnetic Field Exposure, 22 O.S.H. Rep. (BNA) 1314 (1992) (noting a study of three companies in
histologic finding that a large percentage of spontaneous abortions show chromosomal abnormalities that are incompatible with life.33 Accordingly, lawsuits alleging miscarriage and early death because of maternal exposures are beginning to make their way through the courts.34

C. Disruption of Organogenesis

During the first twelve weeks, the embryo undergoes organogenesis, literally the birth of its organs.35 This process is a very precise, genetically programmed sequence of accelerated cell division and differentiation that results in the conversion of a single amorphous cell mass into a rudimentary anthropomorphic organism with all the proper parts.36 The only organ systems that are not completely formed by the twelfth week are the central nervous system and genitalia, which continue to grow and develop until even after birth.37 If this process is interfered with, the result can be undesirable gross congenital malformations that have been labeled terata (literally, monsters).38

During organogenesis, timing is everything. Chemical or physical teratogens (radiation, therapeutic drugs, industrial pollutants) may have completely different effects depending on

Finland that revealed that the use of VDTs having high magnetic fields during early pregnancy correlated with an increased rate of miscarriage). But see Teresa M. Schnorr et al., Video Display Terminals and the Risk of Spontaneous Abortion, 324 New Eng. J. Med. 727 (1991) (finding no excess risk of spontaneous abortion among women who used VDTs during the 1st trimester of pregnancy).

33. See Berkow & Fletcher, supra note 19, at 1759. "Since in 60% of spontaneous abortions the fetus is either absent or grossly malformed, and in 25 to 60% it can be found to have chromosomal abnormalities incompatible with life, spontaneous abortion may be a natural rejection of a maldeveloping fetus." Id. A finding of such chromosomal damage in the aborted fetus of an employee in conjunction with the presence of a mutagenic stressor in the workplace, may serve as the evidence of causation in a lawsuit for wrongful death (assuming the jurisdiction recognizes such actions for miscarriages).


35. Jacob et al., supra note 6, at 588.

36. Id. at 588-90.

37. OTA, supra note 1, at 49.

38. Taber's Cyclopedic Medical Dictionary 1835 (16th ed. 1989) [hereinafter Taber's]
the day incurred. Dramatic manifestations of this phenomenon can be elicited in animal studies. For example, when pregnant rats are given excessive doses of vitamin A on day eight of gestation, skeletal malformations result, while if given on day twelve, the same dose results in cleft palate formation. Technically, teratogenesis is limited to this stage of development.

The most dramatic human experience with teratogens has been associated with pharmaceuticals, of which thalidomide is the most notorious. Thalidomide is a mild sedative that was harmless to pregnant women except between days thirty-five and fifty of pregnancy. Unfortunately, approximately 10,000 European women, unaware of the risks, took thalidomide during that thirty-five to fifty day period and gave birth to children with phocomelia. Pharmaceuticals have also been the most litigated of human teratogens, including Thalidomide, Bendectin, Dilantin, and spermicidal jellies.

Several nonpharmaceuticals have also been implicated as teratogens based on animal studies or epidemiological data. Interestingly, the only litigation involving workplace exposure to a teratogen involved one of the best known, rubella, and was

39. CASARETT & DOULL, supra note 29, at 318.
40. Id. at 319.
41. Id. at 318-19.
42. Id. at 314. In Greek, teratogenesis means literally the "birth of monsters."
43. CASARETT & DOULL, supra note 29, at 313.
44. Id. Phocomelia describes a congenital malformation wherein the long bones in the extremities are absent or poorly developed. TABER'S, supra note 38, at 1388.
49. Known teratogens that may be encountered in the workplace include antineoplastic agents to which health care workers may be exposed. BERKOW & FLETCHER, supra note 19, at 1752. Such drugs have the intended effect of disrupting the rapid growth of tumors; an outcome that is disastrous during the period of fetal organogenesis. Id. at 1911. Rubella (German measles) is also a well-known teratogen that can easily infect the fetus during organogenesis resulting in a wide range of congenital anomalies. See id. at 1752. Animal studies have shown a great number of materials to be potential teratogens: acrylonitrile, arsenic, benzene, benzo-a-pyrene, boron anhydride, cadmium dusts and salts, carbon disulfide, chloroform, chloroprene, chromium, copper dusts and mists, cyanides, di-n-butylphthalate, ethylene glycol mono-ethyl/methyl ethers, formamides, fungicides/fumigants/sterilants (most), inorganic mercury, methyl chloride, nickel, organic mercury, pesticides (most), selenium, vinyl chloride, and xylenes. M. PAUL, OCCUPATIONAL AND ENVIRONMENTAL REPRODUCTIVE HAZARDS: A GUIDE FOR CLINICIANS 394-406 (1992).
the first such case reported in the United States. As more attention is focused on the teratogenic potential of workplace exposures, it can be expected that litigation involving nonpharmaceutical-based teratogenesis will increase.

Chemical or biological agents are not the only way to affect teratogenesis. In fact, one of the most common and potent teratogens is ordinary heat. Additionally, ionizing radiation disrupts rapid cellular growth and can disrupt organogenesis.

D. Fetal Injury

The growing fetus is encased in a placenta upon which it relies for nutrients and elimination of metabolic waste products. This structure also serves to cushion and protect the fetus. Maternal and fetal blood do not merge in the placenta. Rather, fetal capillaries protrude into pools of maternal blood, allowing the diffusion of gases and smaller particulates from the mother's blood into the fetus's blood. This system can screen large molecules or bacteria but not smaller substances such as viruses or most workplace chemicals or metals.

For approximately thirty weeks following organogenesis, the fetus will remain dependent on the placenta. If the placenta is damaged or insufficient nutrients are directed through it, the fetus will be deprived of the necessary life support. During this stage, fetotoxicity may occur whereby the mother's

51. Jacqueline Agnew et al., Reproductive Hazards of Fire Fighting I: Nonchemical Hazards, 19 AM. J. INDUS. MED. 433, 434-37 (1991) (collecting toxicological and epidemiological literature). A rise in core temperature above 38.9°C is thought to pose a teratogenic hazard. Id. at 436-37. It has been reported that one of the greatest fears of toxicologists involved in teratology is that the thermal regulation of some test animals' cages will malfunction. Casarett & Doull, supra note 29, at 328.
52. See Agnew et al., supra note 51, at 440.
53. Jacob et al., supra note 6, at 591.
54. Id.
55. Id.
56. Id.
57. Mark Evans, Reproductive Risks and Prenatal Diagnosis 47-48 (1992). In fact, some chemicals, such as carbon monoxide, may attain higher concentrations in the fetal blood than in that of the mother. Melissa A. McDiamid et al., Reproductive Hazards of Fire Fighting II. Chemical Hazards, 19 AM. J. INDUS. MED. 458, 447 (1991).
58. OTA, supra note 1, at 51.
59. Jacobs et al., supra note 6, at 591.
workplace exposure to toxins is transmitted to the fetus resulting in injury.60

The effect on a fetus of a transplacental toxic exposure will depend on the type of exposure.61 For carcinogens and many chronically toxic substances that increase lifetime risk of illness in some proportion to the long-term dosage, the relatively brief in utero exposure may not be as significant as that of the mother during her working career.62 On the other hand, the mother may have lower susceptibility due to more developed defense and repair mechanisms.63

There is an extensive base of scientific literature respecting transplacental migration and toxicity of various chemical compounds, including animal and human studies.64 There has been litigation alleging direct, post-organogenesis injury to the fetus caused by workplace exposures of the mother to several agents, including carbon monoxide,65 mercury,66 and hepatitis67 as well as "unspecified contaminants."68

More complex scenarios exist in which conduct towards the mother can result in toxic injury to a fetus. An example is the

60. OTA, supra note 1, at 56-57.
61. EVANS, supra note 57, at 47-48; OTA, supra note 1, at 56-57.
62. EVANS, supra note 57, at 91-94.
63. One notable defense mechanism is the blood-brain barrier which, in the adult, limits the infiltration of certain substances. In the child, this barrier is less developed. One effect of this difference is observed with inorganic lead. In adults, because of the blood-brain barrier, lead is distributed from the blood to the peripheral nervous system causing peripheral neuropathy ("wrist drop"). In children, however, the lead accumulates in the brain causing mental defects. CASARETT & DOULL, supra note 29, at 36, 151. Epidemiological studies have suggested increased risks of childhood cancer among the offspring of parents in several occupations (mechanics, aircraft workers, military personnel, painters, chemical workers, welders, pharmacists, physicians, and machinists). Savitz & Chen, supra note 15, at 325.
64. See McDiarmid et al., supra note 57, at 454-56.
67. Jarvis v. Providence Hosp., 444 N.W.2d 236 (Mich. Ct. App. 1989) (alleging in wrongful death action that stillborn birth was due to mother's exposure to hepatitis during 23rd week of pregnancy where the mother, a laboratory technician, was diagnosed as having contracted hepatitis during 8th month and delivered a stillborn baby).
case where a woman whose blood does not contain a protein called the Rh factor is given a transfusion of blood that does contain this component, causing her body to produce Rh antibodies.69 If she subsequently conceives a child who does carry this genetically dictated factor, her placenta will infuse the fetus with an allergy to its own blood.70

Of course, simple physical forces can injure the fetus as well. There have been lawsuits where trauma to the employee/mother has resulted in fetal injury71 or death.72 Less dramatic are the plethora of exposures associated with the birth of a baby that is premature or small for its gestational age.73 In the latter case, while no direct injury may be said to have occurred, the baby is more vulnerable to a variety of problems.74

E. Postpartum Injury

Even after birth, the child can still suffer injury due to the mother's exposure. For example, newborns that are breast-fed

69. BERKOW & FLETCHER, supra note 19, at 1132.
70. Renslow v. Mennonite Hosp., 351 N.E.2d 870 (Ill. App. Ct. 1976). Renslow is a landmark case in which preconception liability to the injured fetus was found where the mother's transfusion was given almost a decade prior to conception. Id. at 871; see Vik Ed Stoll, Note, Preconception Tort-The Need for a Limitation, 44 Mo. L. Rev. 143 (1979). Because many employers provide medical care for their employees, this scenario could also arise in a workplace context leading to employer liability.
71. Cushing v. Time Saver Stores, Inc., 552 So. 2d 730 (La. Ct. App. 1989) (negligence action by employee's child against employer who forced mother to sit on stacked boxes in place of office furniture; the boxes shifted, the mother fell, and an adding machine landed on her abdomen, causing abruptio placentae, which resulted in birth defects including brain damage); Vicknair v. Hibernia Bldg. Corp., 482 So. 2d 95 (La. Ct. App. 1986) (personal injury action by parents and child of woman who was born with hyaline membrane disease attributable to mother's running down 21 flights of stairs in response to a false fire alarm).
73. While many workplace factors may be associated with low birth weight including exposure to anesthetic agents, and several solvents, the most consistent associations are with cigarette and alcohol consumption. See, e.g., OTA, supra note 1, at 1.
74. Premature infants and those small for gestational age are prone to difficulty in regulating body temperature, respiratory distress, necrotizing enterocolitis, inadequate sucking and swallowing reflexes, apnea, cerebral hemorrhage, meningitis, and other problems. BERKOW & FLETCHER, supra note 19, at 7.
may share the toxic exposures of the mother through the contamination of her breast milk. Moreover, these shared exposures may be followed by long latency periods and result in disease and lawsuits years after birth.

Thus, parentally-mediated prenatal harm can occur in many ways, most of which have been tested in the laboratory and the courts of various jurisdictions. While a surprising variety of workplace factors are known or suspected to adversely affect fetal development, what is most alarming is what is not yet known. Regarding chemicals, for example, a recent General Accounting Office (GAO) report estimated that information on reproductive toxicity exists for only five percent of the 104,000 chemicals registered with the Environmental Protection Agency (EPA). Thus, there are 98,800 chemicals in use in the United States for which we have no data upon which to determine acceptable risk levels. The paucity of scientific data can, of course, result in unknowing exposures to harmful circumstances. Also possible, however, is the situation where legal action is brought because scientific evidence at the time of the lawsuit implicates an agent with a prenatal injury but, upon later investigation, the causal association is refuted. One such case may be the anticonvulsant Dilantin (phenytoin) upon which Harbeson v. Parke-Davis, Inc., discussed below, is based. Studies have revealed that the children of untreated epileptic mothers experience the same types of anomalies attributed to Dilantin, a finding that tends to refute the causal association between the drug and these anomalies.

III. COMPENSABLE REPRODUCTIVE OUTCOMES

While the scientific evidence may implicate an agent with an adverse reproductive outcome and the courts of another jurisdiction may have recognized it, that association must also

75. Washington Dep't of Labor and Indus., Pub. No. P-413-035-000, Workplace Hazards to Reproductive Health 27 (1991) [hereinafter Workplace Hazards]. Nonpolar, low molecular weight contaminants such as polyhalogynated biphenyls and DDT are likely to be excreted via the breast milk.


78. Id.

79. 98 Wash. 2d 460, 656 P.2d 483 (1983).

80. Berkow & Fletcher, supra note 19, at 1753.
be cognizable under Washington law to sustain a lawsuit here. In Washington, both statutory and case law exists to support causes of action for most adverse reproductive outcomes.

Washington has recognized prenatal injury to a child born alive as actionable for over thirty years. Thus if an employer’s culpable actions injured a viable fetus via the parent(s), the child could sue once born. Further, in Harbeson v. Parke-Davis, Inc., a medical malpractice case, the Washington Supreme Court also recognized the child's right to sue for birth defects caused by conduct prior to conception. Damages available to the child born with birth defects under such a “wrongful life action” include extraordinary medical and educational expenses, and other expenses attributable to the disability.

In Harbeson, the court also recognized actions for wrongful birth on behalf of the parents for prenatal negligence resulting in the birth of malformed children. The court allowed the parents to recover damages including increased medical and other expenses associated with the birth and rearing of the child as well as compensation for “emotional injury caused by the birth of the defective child.”

Where the injury to the fetus is so severe that it results in postpartum death, Washington statutory law steps in to provide remedies. Where such death occurs before the age of majority, the parents may maintain a wrongful death action for dam-

81. Seattle-First Nat'l Bank v. Rankin, 59 Wash. 2d 288, 367 P.2d 835 (1962) (recognizing a cause of action where a child was born with brain damage caused by the physician's failure to treat the mother's anemia during pregnancy).
82. 98 Wash. 2d 460, 656 P.2d 483 (1983).
83. Id. at 477-78, 656 P.2d at 494 (holding that the physician negligently failed to warn the female patient of teratogenic risk of “dilantin,” an anticonvulsant prescribed for epileptics).
84. Id. at 480, 656 P.2d at 495. The court provided that the costs of extraordinary care may only be recovered once. Id. If the parents recover the costs incurred during the child's minority, the child's recovery would be limited to lifetime costs thereafter. Id.
85. Id. at 476-78, 656 P.2d at 493-94. Although the court relied on sections of various statutes when determining the standard of care and damages, it stated that this action was not based on statute but was a common law negligence action. Id. The parents were deemed to have a right to avoid the birth of children with congenital defects, a right the physicians had a duty to protect. Id. at 476, 656 P.2d at 493. The physicians negligently breached that duty by failing to apprise the parents of the risk of birth defects due to the mother's anticonvulsant medication. Id. at 477-78, 656 P.2d at 493-94.
86. Id. at 475, 656 P.2d at 493.
Where death occurs later in life, the child's estate or dependents may file a wrongful death or survival action. Examples of occupational parental exposures that could result in the later death of offspring are transplacental carcinogens such as vinyl chloride and benzene.

In cases of miscarriage due to prenatal injury, Washington has joined most states in recognizing a viable fetus as a person for purposes of applying the Washington wrongful death statute, Revised Code of Washington (RCW) 4.24.010. However, in *Moen v. Hanson*, the court expressly reserved the issue of whether such actions could be brought for the death of a nonviable fetus. The court also refused to establish a bright line demarcation of fetal viability, leaving that to the finder of fact at trial.

Wrongful death actions respecting the preborn loom as the most onerous source of liability facing employers. As illustrated in Part II, miscarriage and stillbirth are by far the most com-

87. Wash. Rev. Code § 4.24.010 (1992). Section 4.24.010 grants an action by the natural parents for injury or death of a child. *Id.* Damages include all expenses associated with the care of the child as well as loss of love and companionship, destruction of the parent-child relationship, and other damages as "may be just." *Id.*

88. Ordinarily, an employment-related prenatal injury action would be tolled during the injured child's minority. *Id.* § 4.16.190. Thus, a negligence cause of action, assuming it were known, would not accrue until age 21. In the case of transplacental carcinogens, death may not ensue until later in life because of long latency periods.

89. *Id.* § 4.20.010. Section 4.20.020 specifies the beneficiaries to include the direct family including spouse, children, grandchildren, and, if none exist, then dependent parents or siblings. *Id.* § 4.20.020. No limitation on damages is set.

90. *Id.* § 4.20.046. Section 4.20.046 grants a survival cause of action to the deceased's estate for all damages except pain, suffering, anxiety, emotional distress, or humiliation personal to and suffered by the deceased. *Id.*

91. See Workplace Hazards, supra note 75, at 8.

92. See McDiarmid et al., supra note 57, at 457; see also Savitz & Chen, supra note 15, at 325 (correlating various parental occupations with increased incidence of various childhood cancers).

93. Sheldon R. Shapiro, Annotation, Right to Maintain Action or to Recover Damages for Death of Unborn Child, 84 A.L.R.3d 411, 422 (1978 & Supp. 1993). Of the states that have ruled on the issue, 34 have recognized a wrongful death cause of action based on the death of a viable fetus and 10 have refused to allow such actions, regardless of viability. *Id.* Michigan, Louisiana, and Rhode Island have recognized the action regardless of viability, while Georgia requires that the fetus be quick. *Id.* Many states, such as Washington, have not expressly ruled on the issue of viability. *Id.*


95. 85 Wash. 2d 597, 537 P.2d 266 (1975) (alleging wrongful death under RCW 4.24.010 based on the death of a fetus when the mother was killed in an automobile collision during the 8th month of pregnancy).

96. *Id.* at 601, 537 P.2d at 268.

97. *Id.* at 601-02, 537 P.2d at 268.
mon and obvious endpoints of developmental harm. In fact, virtually no employment exists that does not involve at least one factor correlated by at least one researcher with an increased risk of miscarriage.

IV. THEORIES OF LIABILITY

Assuming the law recognizes the compensability of an adverse reproductive outcome, it must also provide a basis for employer culpability. In Washington, strict and negligence theories of liability are possible.

A. Strict Liability

Liability without fault may lie where (1) the employer's business is deemed an ultrahazardous activity, (2) it poses a substantial risk of harm no matter how much care is exercised, and (3) it is not a common activity in the community. Possible workplace scenarios that might result in strict employer liability for fetal injury include higher risk operations such as hazardous waste responders, asbestos abatement, fire fighting, and other jobs where significant risks of exposure are not under perfect control and where the use of stressful personal protective equipment is necessary.

Finally, where the injury is caused by a consumer product, the Washington Product Liability Act may apply where the employee was exposed to the employer's products in the same manner as a member of the public.

98. Because most germ cell mutations result in early fetal death in test animals, it would seem reasonable to assume that an employee's exposure to a mutagenic stressor could result in miscarriage. See CASARETT & DOUILL, supra note 29, at 314. There are thousands of known mutagens in the work environment. U. S. DEPT OF HEALTH & HUMAN SERVS., REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTCECS) (1990) (listing over 3000 substances that have elicited mutagenesis as well as over 900 that have caused teratogenesis).

99. In fact, the act of working alone may increase the rate of miscarriage in many jobs. See, e.g., Agnew et al., supra note 51, at 48 (reviewing literature that shows an increased risk of miscarriage associated with standing and performing tiring jobs).


101. The use of personal protective equipment such as self-contained breathing apparatus and limited permeability or heat resistive garments can quickly elevate core body temperatures resulting in well-documented fetal risk. Agnew et al., supra note 51, at 436-37.


103. Id. § 7.72.030(1). Although the statute expressly states that liability for design defects or inadequate warnings is predicated on a finding of negligence, the courts have interpreted this requirement rather loosely. See, e.g., Ayers v. Johnson & Johnson, 59 Wash. App. 287, 797 P.2d 527 (1990). In Ayers, the court noted that
B. Negligence

Since relatively few occupational exposures are likely to meet the requirements of strict liability, most actions are likely to proceed under a negligence theory. A negligence action would aver that the employer had a duty to the unborn child or parents, the breach of which proximately caused the compensable injury.104

1. Duty

In Seattle-First National Bank v. Rankin,105 the Washington Supreme Court held that when a physician negligently failed to diagnose and treat a mother’s anemia and such anemia was a proximate cause of injury to the fetus, a personal injury cause of action arose in the live-born fetus.106 While the court did not analyze the issue expressly, by implication it found the duty to the mother to be a source of a duty owed to the child.107 The court later spelled out the duty owed to the unborn child by physicians or others in Harbeson:

[A] duty may extend to persons not yet conceived at the time of the negligent act or omission. Such a duty is limited, like any other duty, by the element of foreseeability. A provider of health care, or anyone else, will be liable only to those persons foreseeably endangered by his conduct.108

While the presence of a duty is normally a question of law,109 the determination of the exact bounds of foreseeability is a question of fact for the jury unless “reasonable minds cannot differ.”110

The scope of the employer’s duty to protect against prenatal harm will turn on the foreseeability of such harm as a result of workplace hazards. Clearly, if the employer knows of the harm-

"foreseeability is not an element of a strict liability claim. Confusion caused by legislative tinkering should have been erased by now: liability under RCW 7.72.030(1)(a) and (b) rests on traditional strict liability principles in which the concept of negligence plays no part." Id. at 294, 797 P.2d at 531 (citations omitted).

106. Id. at 291, 367 P.2d at 838.
107. Id.
108. Harbeson, 98 Wash. 2d at 480, 656 P.2d at 489 (citation omitted) (emphasis added).
ful effects of a workplace hazard and an employee's susceptibility to that hazard, the duty will be established. The question remains, however, whether the average employer will be charged, as were the physicians in Harbeson, with the responsibility of continually researching the literature to ascertain obscure or newly-recognized prenatal hazards.111 While no cases on the issue exist, the answer is probably yes. Under the Washington Industrial Safety and Health Act of 1973 (WISHA),112 the employer is charged with maintaining the workplace "free from recognized hazards that are causing or likely to cause serious injury or death to his employees."113 By implication, the employer is charged with doing the research necessary to know of those hazards.114 Additionally, regulations promulgated under WISHA expressly recognize "reproductive toxins" as occupational health hazards.115 Because most of the literature respecting reproductive hazards to the employees also includes prenatal hazard information, an employer would likely be charged with constructive knowledge of both.116

The duty to protect against prenatal harm does not end with the employer. Individual co-employees, consultants, contractors, and others who bear responsibility for managing workplace risks, as well as manufacturers and suppliers of chemicals, equipment, and other instrumentalities of prenatal

111. Harbeson, 98 Wash. 2d at 476, 656 P.2d at 493.
113. Id. at § 49.17.060 (emphasis added).
114. RCW 49.17.180(6) requires employers to exercise "reasonable diligence" to discover violations of WISHA standards. Wash. Rev. Code § 49.17.180(6) (1992). The Division of Industrial Safety and Health states that "as a general rule, if the (safety and health) inspector was able to discover a violation, he/she can presume that an employer could have discovered the same condition through the exercise of reasonable diligence." Division of Indus. Safety & Health, Dep't of Labor & Indus., WISHA Operations Manual ch. VIII (1985).
116. Implicit in the employers duty to become familiar with prenatal hazards to the employees may be the duty to know of the reproductive status of those employees. This may be impeded by regulatory prohibitions such as Washington Administrative Code (WAC) 162-12-140. Wash. Admin. Code § 162-12-140 (1992). Section 162-12-140 defines "[a]ll questions as to pregnancy, and medical history concerning pregnancy and related matters" as unfair preemployment inquiries. Id. WISHA deals with the issue in a gingerly manner in its lead standard by mandating pregnancy testing if the employee requests it. Id. § 296-62-07521(11)(c)(ii)(F).
injury, may also face liability.\textsuperscript{117} For co-employees, this liability can be particularly onerous because they may no longer be employed or indemnified by the employer at the time of the lawsuit.\textsuperscript{118} In addition, because statutory time limitations may be tolled for many years or, in preconception cases, generations, co-employees (or their estates) may be the only parties remaining to be sued if the employer's business has been dissolved.

2. Breach

The regulations promulgated under WISHA also serve as a baseline for determining the standard of care in discharging an employer's duty.\textsuperscript{119} Under the WISHA hazard communication provision, the employer would have a duty to warn of such hazards.\textsuperscript{120} Likewise, the employer would be bound to adhere to specific WISHA safety standards for exposures of employees. The duty to protect beyond mere compliance with WISHA safety regulations would be determined on a case-by-case basis in light of traditional negligence factors such as the probability and gravity of the harm balanced against the feasibility and cost of risk-reducing measures.\textsuperscript{121}

\textsuperscript{117} See, e.g., Namislo v. AKZO Chem., Inc., 620 So. 2d 573 (Ala. 1993) (complaint included claims for fraud, outrage, and willful conduct against co-employees); Cushing v. Time Saver Stores, Inc., 552 So. 2d 730 (La. Ct. App. 1989) (complaint included a claim against a supervisor for negligent supervision and failure to provide a safe workplace).

\textsuperscript{118} The employer who has been sued may name a current or former co-employee as a codefendant or may seek contribution or indemnification. See Wash. Rev. Code § 4.22.040 (1992) (Washington's general contributory fault and indemnification statute).


\textsuperscript{120} Wash. Admin. Code § 296-62 (1992). WAC 296-62 enunciates a lengthy list of employer responsibilities respecting the gathering and dissemination of workplace hazard information. Id. The focus of the standard is on "chemicals" and does not include "intangible" exposures or "safety" hazards. Id. The requirement of WAC 296-24-073(2) that the employer "do every other thing necessary to protect the life and safety of employees" could fill this gap. Id. § 296-24-073(2).

\textsuperscript{121} Because of inadequate data and the delay in the promulgation of safety and health regulations, most workplace standards do not contemplate prenatal harm. See OTA, supra note 1, at 1, 9-10, 60. Thus, employers who merely comply with applicable WISHA regulations do so at their own peril. Lead exposure is a good example. The current WISHA standard allows blood lead levels as high as 50 micrograms per deciliter of blood (ug/dl). Wash. Admin. Code § 296-62-07521(1)(a)(i)(D) (1992). Meanwhile, the American Conference of Governmental Industrial Hygienists (ACGIH) has adopted a 20 ug/dl standard to prevent prenatal cognitive developmental harm. Note, ACGIH Considering Adopting Limit for Blood Levels of Exposed Workers, 23 O.S.H. Rep. (BNA) 6 (1993). The scientific community, however, is far from certain that even the ACGIH levels are safe. See, e.g., Ellen K. Silberg, Implications of New Data on Lead Toxicity for
Conflicting policy considerations make the task of defining due care problematic in prenatal injury cases. Such problems start with the very definition of safety. In the parlance of safety and health regulation, "safe" means that level of exposure for which the risk of death or disability is not deemed significant.122 In tort litigation, however, that same risk may result in culpability if it is deemed unreasonable in the particular case. Thus, it is unlikely that mere compliance with WISHA regulations will allow an employer to escape tort negligence liability for injury to the unborn child of an employee.123

There are, of course, other consensus safety standards that often are more risk averse than those of regulators such as WISHA.124 Even those standards, however, can serve as no

Managing and Preventing Exposure, 89 ENVTL. HEALTH PERSP. 110 (1990) ("Current biomedical consensus accepts that blood lead levels as low as 5 to 15 μg/dl are risky to fetuses, young children, and adults.").

122. See Industrial Union Dep't v. American Petroleum Inst., 448 U.S. 607, 642 (1980) (stating that "safe is not the equivalent of 'risk-free'" and that the statutory mandate of the Occupational Safety and Health Administration (OSHA) was to be limited to the control of significant occupational risks only). This decision has been interpreted by OSHA to allow an asbestos-related cancer mortality risk of 6.7 deaths per 1000 workers over a lifetime. 51 Fed. Reg. 22,647 (1986). In the case of agents that are carcinogenic or mutagenic, policy decisions respecting risk are generally based on the assumption that there is no level of exposure that is risk free. See, e.g., 29 C.F.R. § 1990.111 (1992). Thus "safety" is defined as that statistical probability of cancer that is deemed acceptable under the circumstances. With noncarcinogens (including most fetotoxic or teratogenic stressors), safety limits tend to represent the level of exposure below which the body is able to resist or repair damage or for which the effect of exposure is not deemed to result in a "material impairment of health or functional capacity." 54 Fed. Reg. 2361 (1989). Because of interpersonal variations in susceptibility, however, there will always be some proportion of the population who will experience disability at levels below the safety standards. A good example is the exposure limit for noise. Exposure at the legal limit is known to protect only 85% to 90% of the population from compensable hearing loss. It is, as a practical matter, impossible to set exposure levels for most occupational health hazards that are risk free. The best that can be hoped for is low risk. See William W. Lowrance, Of Acceptable Risk 11 (1976).

123. While the balance of various broad policy interests may restrict regulators in the management of occupational risks to employees, the courts are not similarly restricted in civil suits. It has been held that risks far below one in a thousand may form the basis for liability. See, e.g., Davis v. Wyeth Labs., Inc., 399 F.2d 121, 129-30 (9th Cir. 1968) (holding that the failure to warn of a one in a million risk of polio from a vaccine rendered defendant liable); see also Ayers v. Johnson & Johnson, 59 Wash. App. 287, 297, 797 P.2d 527, 553 (1990) (finding liability for the failure to warn of the risk of aspirating baby oil in spite of the company having sold over 500 million bottles of the product without a single reported case of aspiration).

124. The major standard-setting body respecting occupational health hazards is the American Conference of Governmental Industrial Hygienists (Lansing, Michigan). Their consensus standards formed the basis of the original standards promulgated under the federal Occupational Safety and Health Act of 1970. Occupational Safety and
more than evidence of the requisite standard of care, leaving compliant employers with a lingering doubt as to future liability.

Irrespective of the care taken by an employer, the plaintiff is likely to present expert testimony at trial to establish that more protective efforts were possible.\footnote{125} The employer will then have to justify its reasons for not making those efforts in cases where the jury is presented with a child who may not have been injured if the employer had spent a few more dollars.

3. Causation

Some commentators have argued that prenatal injury suits are difficult to prove because of the lack of extensive data on prenatal hazards and the inability to definitively point to a particular chemical as the cause of an injury.\footnote{126} It is true that very few suits have been filed to date and that most of those have alleged traumatic injury or exposure to acute toxins such as carbon monoxide.\footnote{127} A more appropriate analysis, however, should focus on how such cases will be treated when they are pursued.\footnote{128}

With respect to obscure, tenuous associations, Washington courts have been fairly generous to plaintiffs in prenatal injury litigation to date. In \textit{Rankin}, the court noted that “[w]e are not

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Health Act of 1970, Pub. L. No. 91-596, 84 Stat. 1590 (codified at 29 U.S.C. §§ 651-678 (1988)). These standards are updated yearly to reflect changes in the science. Because they are not hampered by administrative procedures requiring notice or comment, the standards tend to be more responsive to contemporary scientific research.

125. Even if an exposure has not been definitively determined to pose a prenatal hazard, entrepreneurs are capitalizing on concerns about safety and developing safe alternatives. See, \textit{e.g.}, \textit{Radiation-Free Computer Monitor Introduced; Said To Avoid Potential Miscarriage Problems}, 18 O.S.H. Rep. (BNA) 1888 (1989) (advertising “Radiation-free” video display monitor expected to “prevent miscarriages and other reproductive problems”).

126. See, \textit{e.g.}, \textit{Williams}, \textit{supra} note 4, at 646 n.25 (“Indeed, the problems of proving causation may be so great that professed employer concern about liability may be a pretext for sex discrimination.”).

127. Acute causes of occupational injury also receive disproportionate representation in the workers’ compensation arena. This may be because many occupational diseases are not recognized as such by employees. See, \textit{e.g.}, Elinar P. Schroeder \& Sidney A. Shapiro, \textit{Responses to Occupational Disease: The Role of Markets, Regulation, and Information}, 72 GEO. L.J. 1231, 1245 (1984) (noting that only 2\% to 3\% of workers’ compensation payments are for occupational diseases while 97\% of those afflicted with serious occupational diseases are not compensated at all).

128. In light of the increasing awareness of new and varied cause-and-effect relationships, an increasing number of attorneys joining the field, and the recent expansion of the rights of the unborn, it should be assumed that more and more plaintiffs will be seeking redress in the future.
unmindful of the fact that a claim for prenatal injuries is prone to present difficult causation issues. This, however, is no reason to deny the sufficiency of the pleading. Difficulty of proof does not prevent the assertion of a legal right.”

The court went on to hold that liability could be found on the basis of testimony that the incremental loss in the mother’s oxygen-carrying ability due to an undiagnosed anemia could compound the effect of her blood loss during delivery and ultimately cause cerebral palsy in her child. In Harbeson, the court upheld a finding of causation of a child’s “fetal hydantoin syndrome” on the basis of epidemiological studies in the literature that showed an increased incidence of the syndrome among users of the prescription drug used by the mother.

Other jurisdictions have also been very reluctant to take prenatal injury cases away from a jury on the basis of tenuous causation evidence, preferring to leave the issue to a battle of the experts. As in Harbeson, much of that battle has turned on epidemiological studies. The court in Ferebee v. Chevron Chemical, Co. made the following observation:

Judges, both trial and appellate, have no special competence to resolve the complex and refractory causal issues raised by the attempt to link low level exposure to toxic chemicals with human disease. On questions such as these, which stand at the frontier of current medical and epidemiological inquiry, if experts are willing to testify that such a link exists, it is for the jury to decide whether to credit such testimony.

In Wells v. Ortho Pharmaceutical Corp., the court held that liability for failure to warn could arise “as soon as there was a

130. Id. at 293, 367 P.2d at 838.
133. Harbeson, 98 Wash. 2d at 477, 656 P.2d at 493-94; see also Oxendine v. Merrell Dow Pharmaceuticals, Inc., 506 A.2d 1100 (D.C. 1986). Of note in Oxendine is the fact that none of the epidemiological studies relied on by the plaintiffs showed a statistically-significant association of birth defects with the defendant’s product. Id. at 1107-09.
134. 736 F.2d 1529 (D.C. Cir. 1984).
135. Id. at 1534.
hint of a possibility that the [spermicide] causes birth defects." Application of the Wells hint-of-a-possibility test to the array of epidemiological and toxicological studies already in existence would probably yield lawsuits for prenatal injury by employees in thousands of occupations.

A coup de grace to the notion that a scientific consensus must support the casual association between an alleged exposure and a fetal injury was delivered by the U.S. Supreme Court in Daubert v. Merrel Dow Pharmaceutical, Inc. In that case, the Court ruled that plaintiffs' experts could testify as to a drug's teratogenicity on the basis of their own reanalyses of the data in peer-reviewed epidemiologic studies that refuted such association. This rejection of the test adopted in Frye v. U.S. opens the door to anyone who can qualify as an expert and opine in plaintiff's favor, so long as the scientific method underlies the expert's reasoning.

Another causation issue concerns indirect injury. Many exposures in the workplace, as well as personal habits such as smoking and nutrition, result in the birth of babies that are preterm or small for their gestational age. These conditions, while not injuries in the strictest sense of the term, do predispose babies to illness and death. While Washington courts have not ruled on this scenario, the Court of Appeals for the Fifth Circuit has. Applying Louisana law, the court said that if an infant's right "to the start in life that results from nature's scheme of granting a nine months' period of gestation" is interfered with by the negligence of another, and if it is shown that such interference substantially increased the chances of death from an outcome that actually did cause death, then the jury could attribute the death to the negligent act that caused the premature birth. In light of the holdings in Rankin and Harbeson, it is likely that Washington courts would adopt a similar position.

Most workplace fetal injury lawsuits to date have involved well known prenatal hazards such as trauma, rubella, or hyp-

137. Id. at 294.
139. Id. at 2792, 2799.
140. 293 F.2d 1013 (D.C. Cir. 1923) (holding that scientific evidence must attain general acceptance in the professional community before it may be admitted at trial).
141. Daubert, 113 S. Ct. at 2795.
142. See BERKOW & FLETCHER, supra note 19, at 1853-54, 1856.
143. Pan-American Casualty v. Reed, 240 F.2d 336, 340 (5th Cir. 1957).
oxia. In such cases, the question of causation is relatively straightforward. However, actions involving more insidious workplace fetal hazards are also beginning to be reported.

4. Damages

Although the types of damages allowable in Washington have been discussed earlier in this Article, the potential judgment amounts also deserve discussion. Tort damage awards for prenatal injuries in the workplace are not governed by the limits that apply to workers' compensation claims nor are they paid out over years as are disability pensions. They are left to the jury and are payable at once.

The Washington cases involving childhood injuries have been telling. In Rankin, the jury awarded $89,000 in 1962 to a child who suffered cerebral palsy due to anoxia caused by her mother's untreated anemia. In Ayers v. Johnson & Johnson, a product liability action, the jury awarded $500,000 to the parents and $2,000,000 to the child who suffered cardiac arrest leading to paralysis, mental retardation, and seizures due to aspiration of baby oil. In contrast, under industrial insurance, an employee's permanently and totally disabling

144. See Berkow & Fletcher, supra note 19, at 1871, 1875, 1911.


146. See discussion supra part III.

147. Wash. Rev. Code § 51.32.010 (1992). RCW 51.32.010 limits eligibility to workers injured within the course of employment. Id.


150. Id. at 289, 797 P.2d at 529.
occupational impairment is compensated by monthly payments at or below the average monthly wage in the state.\textsuperscript{151}

Other jurisdictions have yielded large verdicts in prenatal injury cases. In \textit{Wells v. Ortho Pharmaceutical Corp.}, the plaintiff was awarded $5.1 million for birth defects caused by spermicidal jelly, including the lack of a left arm, an under-developed left shoulder, a cleft lip, a nostril deformity, and an optic nerve defect in one eye.\textsuperscript{152}

Such large isolated damage awards are likely to be intimidating to future defendants. Although some cases will be decided in favor of defendants, it will be the stalwart employer who will refuse to settle when faced with a prenatal injury case that cannot be dismissed on motion before trial.\textsuperscript{153} The unpredictability of damage awards is also likely to lead liability insurance carriers to attempt to exclude such risks in the future.

The aggregate cost to industry is potentially overwhelming if a cause of action for previability fetal death (miscarriage) gains acceptance. Because the background rate of recognized miscarriage is approximately fifteen percent,\textsuperscript{154} a two or three-fold increase in that rate in a particular occupation could mean that fifteen to thirty percent of all conceptions in that population result in a prima facie wrongful death action.\textsuperscript{155} To date, only Louisiana,\textsuperscript{156} Michigan,\textsuperscript{157} and Rhode Island\textsuperscript{158} would permit a wrongful death action based on the death of a nonviable

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\textsuperscript{151} Wash. Rev. Code § 51.32.060 (1992). Different compensation is afforded to employees based on marital status and number of children. Provision is also made to pay for a full time attendant if the worker is rendered "physically helpless." \textit{Id.}

\textsuperscript{152} 788 F.2d 741, 743 (11th Cir. 1986). In the district court's breakdown of the award, $3,000,000 was apportioned to compensate for the child's past pain and suffering and $500,000 for the mother's mental distress. \textit{Id.} The balance covered past and present pecuniary losses of mother and child. \textit{Id.}


\textsuperscript{154} While the true miscarriage rate is 43%, a large percentage of these occur prior to detection and confirmation of pregnancy. The rates reported for purposes of epidemiological studies vary with different populations but range from about 10% to 15%. OTA, supra note 1.

\textsuperscript{155} For an individual employer, miscarriage clusters could be catastrophic. See, \textit{e.g.}, Note, 18 O.S.H. Rep. (BNA) 1700 (noting a cluster of 14 miscarriages among 11 women in one year in a company whose drinking fountains were later found to contain 3.5 times the acceptable level of lead).

\textsuperscript{156} Adams v. Denny's Inc., 464 So. 2d 876 (La. Ct. App. 1985) (recognizing that a "human being exists from the moment of fertilization and implantation").


\end{flushleft}
fetus, while Georgia would permit such an action after the fetus became quick.\textsuperscript{159}

If such actions were to extend to the moment of conception (as in Louisiana) and science advances to the point where it can reliably detect the estimated forty percent of abortions that occur in the first couple of weeks following conception, the numbers of such lawsuits could skyrocket.\textsuperscript{160}

V. DEFENSES TO EMPLOYER LIABILITY

In most workplace injury scenarios, employers may invoke certain defenses to liability. These defenses may be based on statutory immunity, intervening forces, or the conduct of the plaintiff. In the case of prenatal hazards, however, these defenses are compromised.

A. Workers' Compensation Exclusivity

The quid pro quo of workers' compensation is that the employer will pay or insure for scheduled compensation to employees injured on the job in exchange for immunity from tort liability.\textsuperscript{161} This immunity applies to co-employees and to contractors providing personal services.\textsuperscript{162}

Washington courts have not directly ruled on the issue of whether RCW 51.24\textsuperscript{163} bars prenatal injury actions attributable to the parent's workplace. The issue turns on the determination of whether prenatal injury to the offspring constitutes injury to the employee/parent.

Most of the jurisdictions that have considered the issue have not barred actions based on injury or death of the fetus. In Witty v. American General Capital Distributors, Inc.,\textsuperscript{164} the

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\textsuperscript{159} Porter v. Lassiter, 87 S.E.2d 100, 103 (Ga. Ct. App. 1955). When the fetus becomes quick is a question for the jury. \textit{Id.} In this case, the plaintiff miscarried in the 5th month. \textit{Id.} at 102.

\textsuperscript{160} OTA, \textit{supra} note 1, at 51.


\textsuperscript{162} Kerr v. Olson, 59 Wash. App. 470, 477, 798 P.2d 819, 822 (1990) (holding that contractor physicians who worked in employer's clinic are also entitled to immunity). The immunity of federal co-employees is based on the determination of whether they are performing discretionary functions and the exclusive remedy provision of the state law that governs the lawsuits. Andrews v. Benson, 809 F.2d 1537 (11th Cir. 1987). Safety engineers have been held to be performing nondiscretionary duties and to not be protected by sovereign immunity. \textit{Id.}


\textsuperscript{164} 697 S.W.2d 636 (Tex. App. 1985), rev'd on other grounds, 727 S.W.2d 503 (Tex. 1987).
Texas Court of Appeals ruled that the workers' compensation exclusive remedy provision barred a mother's claim for emotional distress but not a wrongful death claim. In *Thompson v. Pizza Hut of America*, the Illinois' workers' compensation statute was interpreted to allow an action by an infant born with neurological damage due to its mother's workplace exposure. In *Cushing v. Time Saver Stores*, a Louisiana court noted that the "Louisiana Workers' Compensation Act was neither intended nor purports to affect the rights of an employee's child who is injured on the employee's job site." The court went on to allow the child's claim for brain damage caused by the mother's workplace trauma during pregnancy. Michigan has also refused to interpose exclusivity in prenatal injury actions. For example, in *Jarvis v. Providence Hospital*, the court noted the potential burden on employers of pregnant women but determined that "[a]ny decision to extend the exclusive remedy provision of the Workers' Disability Compensation Act to limit the protection given to fetuses must be made by the Legislature." Alabama and Tennessee have also refused to bar actions based on the injury or death of an employee's offspring.

Only California has reached a different result. In barring wrongful death and survival actions based on the death of an employee's child, the court in *Bell v. Macy's California* said that to hold otherwise would result in "adverse consequences to female employees, who could easily find themselves the victims of financially driven gender discrimination by liability conscious employers." The court reasoned that the fetal injury was

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167. *Id.* at 919.
169. *Id.* at 732; see also *Adams v. Denny's Inc.*, 464 So. 2d 876 (La. Ct. App. 1985) (holding that parents' wrongful death claim for miscarriage due to fall at work is not barred). In *Cushing*, the court analogized to the case where the employee brought her child to work and the child was injured and killed because of the employer's negligence. *Cushing*, 552 So. 2d at 732.
170. *Cushing*, 552 So. 2d at 732.
172. *Id.* at 291.
175. 261 Cal. Rptr. 455 (Ct. App. 1989).
176. *Id.* at 447. The court likened in utero injuries via the mother to loss of consortium cases where the family of the employee is injured by the loss of a family
derivative of injury to the mother and thus barred by the state's workers' compensation statute.\textsuperscript{177} In response, the California Legislature passed a bill permitting tort actions based on workplace prenatal injuries but the bill was vetoed by the Governor.\textsuperscript{178}

**B. Compliance with Title VII**

An employer might try to avoid liability by claiming that federal antidiscrimination laws left it no choice but to expose the female employees to prenatal risks. The basis for this assertion is found in *International Union, UAW v. Johnson Controls*,\textsuperscript{179} where the U.S. Supreme Court held that an employer's policy of excluding fertile or pregnant women from jobs that entailed exposure to fetal hazards was impermissible sexual discrimination under Title VII of the Civil Rights Act of 1964.\textsuperscript{180} Yet for apprehensive employers, the Court held out the following hope:

Without negligence, it would be difficult for a court to find liability on the part of the employer. If, under general tort principles, Title VII bans sex-specific fetal-protection policies, the employer fully informs the woman of the risk, and the employer has not acted negligently, the basis for holding an employer liable seems remote at best.\textsuperscript{181}

The Court went on to say that such liability may be preempted if state tort law "impede[s] the accomplishment of Congress' goals in enacting Title VII."\textsuperscript{182}

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\item \textsuperscript{177} *Id.* at 454. It also made a comparison with a case barring a family's claim against an employer for injuries caused by the father who was driven insane by work. *Id.* As the dissent pointed out, however, although they were both injured at the same time, the injury to the fetus did not accrue from an injury to the mother. *Id.* at 457.
\item \textsuperscript{178} *Id.* at 454. Applying this tendentious reasoning, the court would, presumably, bar a personal injury action by children who are injured, along with their mothers, at work. Thus, an employee who is negligently caused to fall while carrying a child at work would be compensated but the child would not.
\item \textsuperscript{179} *California Civil Rights Act Amendment Sought To Prohibit Pregnant Workers From Some Jobs*, 21 O.S.H. Rep. (BNA) 549 (1991) (noting that as he vetoed Assembly Bill 489, the Governor called for legislation that would permit employers to institute fetal protection programs to prevent such harm).
\item \textsuperscript{180} *Id.* at 197.
\item \textsuperscript{181} *Id.* at 208.
\item \textsuperscript{182} *Id.* at 209. In response, the concurring opinion pointed out that (1) preemption of state tort law by Title VII is not supported by precedent, (2) warnings to employees would not preclude their children's cause of action, and (3) employers cannot determine in advance what conduct will constitute negligence because compliance with
\end{itemize}
By invoking Johnson Controls, an employer who has complied with WISHA standards and has apprised its employees of the risks of prenatal injury in the workplace may try to avoid liability in the event of prenatal injury. The employer would contend that, had it not been for Title VII, the employer would have protected the fetus from injury by removing the mother from the injurious exposure. However, as was discussed earlier in this Article, most prenatal injuries derive from very mundane occupational hazards that are ubiquitous in the workplace. Thus, assuming, arguendo, that a preemption defense were valid, an employer seeking to claim the defense would have to contend that, freed from the restrictions of Title VII, it would have excluded most women from most jobs. It is doubtful that such a claim could prevail in most prenatal injury cases.

C. Compliance With Safety Regulations

It is black letter tort law that compliance with regulatory standards is not a defense to an allegation of negligence. In Washington, while evidence of compliance with a standard will be admissible as evidence of due care, the only circumstance where compliance with a government mandate will preclude liability occurs under the Products Liability Act in the case of products made to government specifications respecting design or warnings. As previously discussed, WISHA standards entail risks that may exceed those deemed appropriate for fetal exposures and thus expose the employer to liability.

D. Parental Negligence

In many cases of prenatal injury, the employer and the employee may share responsibility. For example, a father who is exposed to a toxin at work may not adhere to all safety rules and thereby increase his exposure. A mother may be exposed to solvents at work while also consuming alcohol at home. Such occupational safety and health regulations has been held not to be a defense to state tort liability. Id. at 211 (White, J., concurring).

183. See supra notes 53-80 and accompanying text.
186. See supra notes 112-125 and accompanying text.
187. Fetal alcohol syndrome and fetal solvent syndrome manifest themselves in similar reproductive outcomes. Other factors, notably cigarette consumption, can magnify the risks from workplace exposures. See OTA, supra note 1, at 60, 82.
behavior may result in different legal outcomes depending on the claims asserted.

In most cases where the child is born with an injury, parental misconduct would probably not be attributed to the child to reduce or defeat recovery. Washington courts have long held that a parent’s negligence will not be imputed to the child in personal injury actions.\textsuperscript{188} Similarly, in a wrongful death action based on the death of a child, the negligence of one parent would not be imputed to the other.\textsuperscript{189} The employer may, however, have a right of contribution against the negligent parent proportionate to that parent’s share of fault.\textsuperscript{190}

However, in cases where the parental behavior was reckless and extreme, a court may determine that such recklessness serves to cut off the causal connection between the employer’s conduct and the child’s injury. In \textit{Walker v. Rinck},\textsuperscript{191} for example, the Indiana Court of Appeals held that a parent’s negligent conduct could constitute an intervening, superseding cause of their child’s birth defects.\textsuperscript{192} The parents knew of the mother’s Rh sensitization and of the consequent risk of fetal harm.\textsuperscript{193} Yet they proceeded to conceive anyway, giving birth to three children, all of whom suffered injury.\textsuperscript{194} The court held that their conduct cut off any liability for prior negligence by health care providers.\textsuperscript{195}

\section*{E. Parental Assumption of Risk}

In Washington, “[a] parent does not have legal authority to waive a child’s own future cause of action for personal injuries resulting from a third party’s negligence.”\textsuperscript{196} Thus, in most cases, an employer’s receipt of parental consent would prove

\begin{itemize}
\item \textsuperscript{188} Vioen v. Cluff, 69 Wash. 2d 306, 316, 418 P.2d 430, 437 (1966).
\item \textsuperscript{189} \textit{WASH. REV. CODE} \textsection 4.22.020 (1992).
\item \textsuperscript{190} \textit{Id.} at \textsection 4.22.040.
\item \textsuperscript{191} 566 N.E.2d 1088 (Ind. Ct. App. 1991).
\item \textsuperscript{192} \textit{Id.} at 1090.
\item \textsuperscript{193} \textit{Id.}
\item \textsuperscript{194} \textit{Id.}
\item \textsuperscript{195} \textit{Id.} In fact, the parents conceived twice. The first time, the baby was premature, anemic, and suffered respiratory problems. The second time, they conceived twins, one of whom suffered birth defects including hearing impairment, motor skills deficiencies, and possible mental retardation, the other one being asthmatic. \textit{Id.} at 1089.
\item \textsuperscript{196} Scott v. Pacific West Mountain Resort, 119 Wash. 2d 484, 495, 834 P.2d 6, 10 (1992).
\end{itemize}
ineffective. Similarly, an agreement to waive a future claim based on strict liability would also fail.197

The best that an employer might hope to accomplish is a waiver of the parents' rights in a wrongful death or survival action. Additionally, an employer could seek indemnification from the parents concerning injury to the child. However, such indemnification would only be as valuable as the parents' resources. Furthermore, such agreements would have to survive court scrutiny respecting public policy.198

VI. THE POLICY GOALS OF A SOLUTION

A problem clearly exists. Many prenatal injuries are probably accruing daily from occupational exposures. Yet very few victims are receiving compensation through the tort system because the system is ill-equipped to handle such cases. Nevertheless, the fear of massive, unpredictable tort liability results in an incentive for employers to exclude fertile and pregnant women from certain occupations. In addition, the trend is such that the employers' liability fears may soon come to pass. All but one of the employment-related prenatal injury cases cited herein arose within the last ten years.

As several commentators and courts have pointed out, only legislation can ameliorate this problem.199 In crafting such legislation, all affected interests should be accommodated and balanced. First and foremost, for obvious reasons, legislation should work toward preventing prenatal injury. However, contrary to the suggestions of some, the occurrence of prenatal injuries cannot be eliminated, but merely reduced.200 Thus, we must also be prepared to fairly compensate persons whose injuries fall through the cracks in those legislative safeguards, just as we do for persons who suffer other work-related injuries and illnesses.

197. Id. at 494, 834 P.2d at 11 ("Under Washington law parents may not settle or release a child's claim without prior court approval.").

198. The agreement would be ineffective with respect to gross negligence or where the terms were unduly oppressive or unfair. In the employment context, it is likely that most agreements would be presumed unconscionable based on the respective bargaining power of the parties.


200. See, e.g., Buss, supra note 4, at 592-96. Buss argues that most prenatal hazards are attributable to exotic chemicals that should be banned. While there are a few stressors that can be replaced with functionally-equivalent substitutes, most prenatal injury is attributable to everyday exposures, which are an inexorable part of modern civilization.
The incentive to exclude women should also be eliminated. The ability to bear a child should be considered a gift, not a liability. Fetal protection policies in isolated industries have generated some of the most acrimonious debates concerning the workplace to date.\textsuperscript{201} As these debates raged, however, scientists and lawyers around the country have worked to obviate the discussions by demonstrating, in the laboratory and the courtroom, that the only effective fetal protection policy would entail shutting down virtually all business. The law should eliminate the need for such measures or for contrivances that seek to judicially accomplish what should be done by the legislature.

Finally, employers, who are the backbone of the economy, have a right to be able to predict the costs of doing business. An employer who meets contemporary standards of care in its occupational safety and health program, pays its workers' compensation premiums, and otherwise follows the law should not have to worry about "end runs" such as a tort suit for prenatal injury.

VII. A Proposed Approach

In order to meet the policy goals stated above, legislation incorporating the following elements is proposed.

1. Require that all WISHA occupational safety and health standards expressly address prenatal risk.

Exposure standards should comprise limits that are based on prenatal hazard data, where available. Permissible exposure levels might need to be lowered in some cases.\textsuperscript{202} Based on exposure routes and susceptibility, standards may also require ongoing exposure or biological monitoring.\textsuperscript{203} Specific procedures to accommodate pregnancy may be appropriate for early-acting teratogens. For example, temporary removal with full pay and seniority may be indicated for some pregnant employees to the extent compatible with Title VII strictures.\textsuperscript{204}

\textsuperscript{201} See articles cited supra note 4.

\textsuperscript{202} Although not enough data exists to set many intelligent limits, an exception is lead, for which consensus standards are already being lowered. See AGGIH Considering Adopting Limit for Blood Levels of Exposed Workers, 23 O.S.H. REP. (BNA) 6 (1993).

\textsuperscript{203} Monitoring of radiation dosage and blood lead levels is already routine.

\textsuperscript{204} If needed, state antidiscrimination law should be amended in a manner that accommodates the least intrusive measures reasonably necessary. Testing for early pregnancy indicators such as human chorionic gonadotropin might also have a place in
tal hazard training that is specific for the stressor in question should also be required. All of these requirements could be grafted easily onto existing WISHA standards.

2. Create a WISHA prenatal hazard warning standard.

Employers should be required to provide specific warnings to each employee. The contents of a minimal warning should be promulgated by WISHA and should be aimed at making employees fully aware of all incurred risks. Accordingly, those warnings should address workplace prenatal hazards, hazards of individual lifestyle, and the interrelation of the two. The warnings should be based on tort law principles of informed consent rather than scientific dissertations. Those warnings should be documented, archived, updated, and refreshed through mandatory posters and other means.

3. Compensate, through the industrial insurance program, children suffering from prenatal injuries and birth defects resulting from the workplace exposures of their parents.

Such compensation should include medical, rehabilitative, and, after majority or emancipation, disability pensions. The standards of causation that apply to occupational disease claims should be used to determine eligibility, although a rebuttable presumption in favor of causation should be raised in the case of certain well-documented parental exposures to known teratogens/fetotoxins such as lead. Transplacental carcinogens should also be covered. Statutory time limitations should accommodate latent periods.

4. Compensate, through the industrial insurance program, employees for the medical expenses of miscarriages associated with workplace hazards.

This compensation should also be administered under industrial insurance and should include costs of grief and pregnancy counseling. The standards of causation should be lenient and a presumption of causation should be drawn for any substance that has been linked to miscarriage by statistically significant epidemiological studies.205

5. Provide employers who pay into the industrial insurance system and who have, at a minimum, complied with the prenatal hazard warnings promulgated by WISHA, immunity from tort liability for prenatal injury and miscarriage.

limited circumstances. At a minimum, employees should be afforded the risk-reducing options on a voluntary basis.

205. Some formula for reconciling conflicting studies may be necessary.
This immunity should include all co-employees and personal services contractors and should extend in perpetuity. Exceptions to this immunity should be made for intentional conduct, defined as an intention that the parent be exposed above a WISHA standard.\footnote{206} Employers who fail to comply with WISHA prenatal hazard warnings should lose the immunity but such noncompliance should not affect the immunity of co-employees unless a showing is made that no solvent business or successor remains against which a judgment could be enforced.\footnote{207}

VIII. CONCLUSION

The science respecting prenatal hazards in the workplace is rapidly advancing, showing heretofore unknown associations. It is incumbent on the law to keep up. As it stands today, the law is not able to provide for children injured by their parents' exposures, and the law is causing unproductive tension between employers and women employees. It is time for a more rational approach.

\footnote{206}{The higher, inappropriate standard being the intention that the prenatal harm occurs. Where no WISHA standard exists, tort standards of recklessness should govern.}
\footnote{207}{Analogous to Wash. Rev. Code § 7.72.040 (1992).}