

June 2000

The Science of Endocrine Disruption - Will It Change the Scope of Products Liability Claims?

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The Science of Endocrine Disruption— Will it Change the Scope of Products Liability Claims?

KAREN FASSULIOTIS*

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The author wishes to express her heartfelt gratitude and love for the sacrifices and support given by her family, especially her mother, Despina, father, William, and sister, Connie. Special thanks are also extended to her many friends, particularly Claire and Vincent Campisi, as well as her fellow students at Pace and on the law review, for their assistance, editing skills and encouragement in preparing this article.

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WE ARE SUBJECTING WHOLE POPULATIONS TO EXPOSURE TO CHEMICALS WHICH ANIMAL EXPERIMENTS HAVE PROVED TO BE EXTREMELY POISONOUS AND ARE, IN MANY CASES, CUMULATIVE IN THEIR EFFECTS. THESE EXPOSURES NOW BEGIN AT OR BEFORE BIRTH AND—UNLESS WE CHANGE OUR METHODS—WILL CONTINUE THROUGH THE LIFETIME OF THOSE NOW LIVING. NO ONE KNOWS WHAT THE RESULTS WILL BE BECAUSE WE HAVE NO PREVIOUS EXPERIENCE TO GUIDE US.¹

I. Introduction

In 1996, Congress passed the Food Quality Protection Act of 1996 (FQPA)² and amendments to the Safe Drinking Water Act (SDWA).³ Both Acts require the Administrator of the Environmental Protection Agency (EPA), in consultation with the Secretary of Health and Human Services, to develop a screening program for endocrine disrupting effects.⁴ The requirements of this screening program and its potential impact on products liability claims will be examined in this Comment.

In 1996, Theo Colborn, Dianne Dumanoski and John Peterson Myers published the book, *Our Stolen Future: Are We Threatening Our Fertility, Intelligence, and Survival?—A Scientific Detective Story*.⁵ Their book began a debate over endocrine disruptors and the regulation of synthetic chemicals and pesticides. These authors argue that synthetic chemicals that have the characteristics of being persistent, toxic and bioaccumulative, are adversely affecting humans and wildlife by disrupting normal endocrine path-

1. Vice-President Al Gore, *Foreword to* THEO COLBORN ET AL., *OUR STOLEN FUTURE, ARE WE THREATENING OUR FERTILITY, INTELLIGENCE, AND SURVIVAL?—A SCIENTIFIC DETECTIVE STORY*, v-vi (Penguin Group 1996) (quoting Rachel Carson).

2. See The Food Quality Protection Act of 1996, Pub. L. No. 104-170 (1996) (codified at 21 U.S.C. § 346 (1996)).

3. See Safe Drinking Water Act Amendments of 1996, Pub. L. No. 104-182 110 Stat. 1613 (1996) (codified at 42 U.S.C § 300f:j (1996)).

4. See 21 U.S.C. § 346a(4)(f)(1)(C)(v) (1996); 42 U.S.C. § 300j-17 (1996).

5. THEO COLBORN ET AL., *OUR STOLEN FUTURE: ARE WE THREATENING OUR FERTILITY, INTELLIGENCE, AND SURVIVAL?—A SCIENTIFIC DETECTIVE STORY* (Penguin Group 1996).

ways.⁶ They also argue that synthetic chemicals should be assumed guilty until proven innocent.⁷ The authors call for basic revisions in the U.S. laws that govern environmental health standards to ensure protection from chemicals that interfere with hormones.⁸ They also call for international agreement for the worldwide phase out of the production and use of persistent hormone-disrupting chemicals, and urge that new financial support be provided for the containment, retrieval, and clean-up of such chemicals.⁹ While acknowledging that “many unknowns and uncertainties” remain in the scientific understanding of the threat these chemicals pose to humans, they assert the evidence exists and warrants immediate action.¹⁰

Not surprisingly, the chemical industry, through trade associations such as the Chlorine Chemistry Council (CCC), Chemical Manufacturers Association (CMA) and the Specialty Organic Chemical Manufacturers Association (SOCMA), has refuted the theory, citing flaws in the scientific studies cited by these authors.¹¹ Nonetheless, EPA, in 1996, as a result of the passage of the endocrine testing requirements in the SDWA and FQPA, established the Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC).¹² EDSTAC was formed to recommend a testing program that will allow EPA to meet its congressional mandate of having testing in place by the year 2000.¹³

Many of the chemicals implicated by scientists supporting the endocrine disrupting theory are contained in products that consumers are in contact with every day—either directly or through food and water sources.¹⁴ The health effects these chemicals are

6. *See id.* at 122-41.

7. *See id.* at 219.

8. *See id.* at 220-22.

9. *See id.* at 225-26.

10. Colborn et al., *supra* note 5, at 212.

11. *See, e.g.*, Chlorine Chemistry Council, *Our Stolen Future – Question and Answers* (visited Jan. 20, 1999) <<http://c3.org/aol/library/stolenqu396.html>>.

12. *See* UNITED STATES ENVIRONMENTAL PROTECTION AGENCY ADVISORY COMMITTEE CHARTER, ENDOCRINE DISRUPTOR SCREENING AND TESTING COMMITTEE (Office of Prevention, Pesticides, and Toxic Substances, Nov. 20, 1996) [hereinafter ESTAC CHARTER] (visited Jan. 20, 1999) <http://www.epa.gov/oscpmont/ospendo/history/endo4_1.htm>.

13. *See id.*

14. *See* A. Krishnan et al., *Bisphenol-A: An Estrogenic Substance Is Released from Polycarbonate Flasks During Autoclaving*, 132(8) *ENDOCRINOLOGY* 2279-86 (1993); *see also* S. Jobling et al., *A Variety of Environmentally Persistent Chemicals, Including some Phthalate Plasticizers, Are Weakly Estrogenic*, 103(6) *ENVTL. HEALTH PERSPECTIVES* 582-87 (1995); C. Purdom et al., *Estrogenic Effects of Effluents from Sewage*

alleged to have range from attention deficit disorder in children,¹⁵ to breast cancer in women.¹⁶ The legal ramifications, in terms of potential products liability and other claims, can be significant as these chemicals are found in almost all consumer goods.

Our Stolen Future has raised some important potential legal and policy issues, including whether current health standards are adequate and whether the current science will support potential claims by consumers for adverse health effects suffered due to alleged exposure to chemicals implicated as causing effects on the endocrine system. The goal of this Comment is to examine the current state of the science and comment as to whether U.S. policy is directed toward adequately addressing the health issues in a timely manner. The paper also examines whether the current science supports a successful products liability claim and comments as to why this may or may not be possible.

II. Background

A. *Our Stolen Future*

In order to understand the current EPA and FDA (Food and Drug Administration) philosophy concerning the safety testing of chemicals intentionally added to pesticides, foods, products contacting food, and consumer products, it is necessary to examine the claims made in *Our Stolen Future*. This book has been compared by some to Rachel Carson's *Silent Spring*¹⁷ and has resulted in a major research initiative by EPA, in cooperation with the FDA.¹⁸ *Our Stolen Future* is the foundation for much of this research.¹⁹ Therefore, it is important to understand the scientific hypotheses contained in the book in order to grasp the potential legal issues arising from these authors' allegations.

Our Stolen Future focuses on a growing body of scientific research implicating synthetic industrial chemicals and pesticides in interfering with the normal function of the endocrine system in

Treatment Works, 8 CHEMISTRY AND ECOLOGY 275-85 (1994); S. Jobling and J. Sumpster, *Detergent Components in Sewage Effluent Are Weakly Oestrogenic to Fish: An In Vitro Study Using Rainbow Trout (Oncorhynchus mykiss) Hepatocytes*, 27 AQUATIC TOXICOLOGY 361-72 (1993).

15. See Colborn et al., *supra* note 5, at 122-41.

16. See Colborn et al., *supra* note 5, at 122-41.

17. See Colborn et al., *supra* note 5, at 167.

18. See EPA Advisory Committee, EDSTAC FINAL REPORT, ES-1 (Aug. 1998) [hereinafter EDSTAC FINAL REPORT] (visited Jan.20, 1999) <<http://www.epa.gov/scu-poly/ospendo/history/finalrpt.htm>>.

19. See *id.*

humans and wildlife.²⁰ Such disruption causes a variety of problems with development, behavior and reproduction.²¹ The fundamental question raised by the endocrine issue is whether exposure to small amounts of synthetic chemicals in food, water or air can interfere with the hormonal systems of humans and wildlife to cause adverse health effects.²² The legal question is whether the intentional addition of these synthetic chemicals to pesticides, foods, food containers and consumer products can give rise to products liability actions by those individuals exposed to these chemicals who are suffering from the adverse health effects claimed.

Endocrine modulation is not new to science.²³ *Our Stolen Future* raises important issues with regard to the use of science in the regulation and policy issues concerning the effects of man-made chemicals on human health and the environment.²⁴ The authors call for basic revisions in domestic laws governing environmental health standards to ensure protection from chemicals that interfere with hormones.²⁵ Specifically, they urge that these new health standards include: 1) shifting the burden of proof to chemical manufacturers to show that a chemical is safe before a new chemical is used; 2) an emphasis on preventing exposure; 3) setting standards that protect the most vulnerable, namely children and the unborn; 4) considering the interactions among chemicals, not just the effects of each chemical individually; 5) considering the cumulative exposure from air, food, water and other sources; 6) having a "right to know" provision for products; 7) requiring companies that sell products to monitor their products for endocrine disrupting chemicals; 8) broadening the Toxic Release Inventory (TRI) to include endocrine disrupting chemicals; and 9) reforming health data systems to enable them to provide information needed to make sound and protective policies.²⁶ With these provisions in place, consumers could make informed decisions as to what types and amounts of exposure they are willing to accept.

20. See COLBORN ET AL., *supra* note 5; see also Robert J. Kavlock et al., *Research Needs for the Risk Assessment of Health and Environmental Effects of Endocrine Disruptors: A Report of the U.S. EPA-Sponsored Workshop*, 104 ENVTL. HEALTH PERSPECTIVES (Supplement 4) (Aug. 1996).

21. See Colborn et al., *supra* note 5, at 121.

22. See Colborn et al., *supra* note 5, at 121.

23. See Colborn et al., *supra* note 5, at 121.

24. See Colborn et al., *supra* note 5, at 218-22.

25. See Colborn et al., *supra* note 5, at 218-22.

26. See Colborn et al., *supra* note 5, at 218-22.

B. The Endocrine System

The endocrine system is a complex biological system defined as any glandular tissues or cells that release hormones or chemical messengers to cause an effect on a target tissue or cell to produce a response.²⁷ If the well-being of the endocrine system is compromised it can lead to adverse effects on the metabolic system, produce developmental abnormalities or reproductive dysfunction.²⁸ It should be noted that a chemical action on the endocrine system may not be an undesirable effect; chemical or drug suppression of target organ hormones has been used and can be therapeutically useful.²⁹

The fundamental concept of the endocrine system is that endocrine cells release a hormone that is then transported to a receptor site at a target tissue.³⁰ Almost all tissues in the human body are "target organs" for one or more hormones.³¹ The hormone then binds to the receptor and exerts a biological effect.³² Hormones can also act locally.³³ This effect, known as a "paracrine effect," occurs when a cell, known as an effector cell, releases a hormone on an adjacent target cell to produce a local effect.³⁴ An example of this is through the action of growth hormones.³⁵ Another pathway for hormonal action is known as the "autocrine system."³⁶ The autocrine system activates when a particular cell releases a hormone that then acts on the same cell to augment a particular response.³⁷ Examples of these types of cells are found in the nervous, gastrointestinal and immune systems.³⁸

Hormones have been categorized into four different chemical categories.³⁹ These include proteins (e.g. insulin, growth hormones), polypeptides (e.g., thyrotropin-releasing hormones),

27. See JOEL G. HARDMAN ET AL. EDS., GOODMAN & GILMAN'S THE PHARMACOLOGICAL BASIS OF THERAPEUTICS 1363 (McGraw-Hill, 9th ed. 1996).

28. See *id.*

29. See *id.* at 1291-1308.

30. See *id.* at 30-37.

31. See *id.*

32. See JOEL G. HARDMAN ET AL. EDS., GOODMAN & GILMAN'S THE PHARMACOLOGICAL BASIS OF THERAPEUTICS 579 (McGraw-Hill, 9th ed. 1996).

33. See *id.*

34. See *id.*

35. See *id.*

36. See *id.*

37. See JOEL G. HARDMAN ET AL. EDS., GOODMAN & GILMAN'S THE PHARMACOLOGICAL BASIS OF THERAPEUTICS 579 (McGraw-Hill, 9th ed. 1996).

38. See *id.* at 1363.

39. See *id.* at 29-37.

amines (e.g., epinephrine) and steroids (e.g., estrogens, androgens).⁴⁰ In addition, chemicals that do not fall into these categories have been shown to act on hormone receptors,⁴¹ making it difficult to predict the potential action of chemicals based on structure alone.⁴²

C. Biological Effects Due to Endocrine Disrupting Chemicals

1. Cancer-Causing Effects

Examination of cases in which pregnant women were exposed to diethylstilbestrol (DES) supplies support for one hypothesis implicating a causal association between endocrine disruptors and cancer in humans.⁴³ DES exposure of pregnant women has been shown to cause clear-cell adenocarcinoma of the vagina and cervix in their female children.⁴⁴ This finding led to a number of important conclusions.⁴⁵ First, maternal exposures during pregnancy can lead to cancer in offspring.⁴⁶ Also, it demonstrates that a synthetic estrogen can cause cancer.⁴⁷ Additionally, some of the male children of women who took DES during pregnancy were found to display pseudohermaphroditism⁴⁸ and malformations of the genitalia, including testicular abnormalities such as small testes, reduced semen quality and epidymal cysts.⁴⁹ These findings are offset by follow-up surveys of DES-exposed male children who did not show impaired fertility or sexual function,⁵⁰ or evidence of an increased risk of testicular cancer.⁵¹

40. See *id.* at 1363.

41. See *id.* at 29-37.

42. See HARDMAN ET AL., *supra* note 27, at 29-37.

43. See A. Herbst et al., *Adenocarcinoma of the Vagina. Association of Maternal Diethylstilbestrol Therapy with Tumor Appearance in Young Women*, 284 NEW ENG. J. MED., 878-81 (1971).

44. See *id.*

45. See *id.*

46. See *id.*

47. See *id.*

48. See N.M. Kaplan, *Male Pseudohermaphroditism; Report of a Case with Observations on Pathogenesis*, 261 NEW ENG. J. MED. 641-44 (1959).

49. See W.H. Gil et al., *Association of Diethylstilbestrol Exposure in utero with Cryptorchidism, Testicular Hypoplasia and Semen Abnormalities*, 122 J. UROLOGY 36-39 (1979); see also S.G. Driscoll, S.H. Taylor, *Effects of Prenatal Estrogen on the Male Urogenital System*, 56 OBSTETRICS & GYNECOLOGY 537-42 (1980); R. Penny, *The Effects of DES on Male Offspring*, 136 WESTERN J. MED. 329-30 (1982).

50. See F.J. Leary et al., *Males Exposed in utero to Diethylstilbestrol*, 252 JAMA 2984-89 (1984); A.J. Wilcox et al., *Fertility in Men Exposed Prenatally to Diethylstilbestrol*, 332 NEW ENGL. J. MED. 1411-15 (1995).

51. See *id.* at 2984.

In the United States, the most common type of cancer is breast cancer.⁵² Several risk factors associated with an increased risk of breast cancer related to hormonal activity include age, race, decreased parity, age at first delivery and age at the onset of menstruation.⁵³ Breast tumors can also be characterized by their degree of positive estrogen-receptor activity.⁵⁴ Consequently, "[t]he evidence supports a causal relationship between female breast cancer and hormonal activity."⁵⁵

Studies have shown that a number of organochlorine pesticides or metabolites of pesticides are found in human breast milk and fat tissue.⁵⁶ Several recent studies suggest a possible relationship between an increased breast cancer risk and the level of organohalides in human tissues, but a clear relationship across the studies has not been established.⁵⁷ In general, these studies suggest that levels of a metabolite of dichlorodiphenyl trichloroethane (DDT), known as dichlorodiphenyl dichloroethane (DDE), and total polychlorinated biphenyls (PCBs), were higher in the serum or fat of women who had breast cancer.⁵⁸ However, the meaning of these findings is unclear as both DDE and PCB metabolites have little estrogenic activity.⁵⁹ The data are further confounded in that studies of women occupationally exposed to high levels of PCBs have not demonstrated an excess risk of breast cancer mortality.⁶⁰ Thus, based on the results of these studies, fur-

52. See ROCKFORD REGISTER STAR, Apr. 7, 1998, available in 1998 WL 5624998. As reported in this 1998 article, one in nine women in North America will develop breast cancer in her lifetime.

53. See R.J. Kavlock et al., *supra* note 20, at 4.

54. See R.J. Kavlock et al., *supra* note 20, at 4.

55. R.J. Kavlock et al., *supra* note 20, at 4.

56. See A.A. JENSEN, S.A. SLORACH, CHEMICAL CONTAMINANTS IN HUMAN MILK (CRC Press, 1991); B.R. Sonawane, *Chemical Contaminants in Human Milk: An Overview*, ENVTL. HEALTH PERSPECTIVES, 103 (Supplement 6), 197-205 (1995).

57. See M. Unger et al., *Organochlorine Compounds in the Adipose Tissue of Deceased Persons with and without Cancer: a Statistical Survey of some Potential Confounders*, 29 ENVTL. RES. 371-76 (1984); see also H. Mussalo-Rauhamaa et al., *Occurrence of Beta-hexachlorocyclohexane in Breast Cancer Patients*, 66 CANCER 2124-28 (1990); H. Austin et al., *A Prospective Follow-up of Cancer Mortality in Relation to Serum DDT*, 79 AM. J. PUBLIC HEALTH 43-46 (1989); F. Falck et al., *Pesticides and Polychlorinated Biphenyl Residues in Human Breast Lipids and Their Relation to Breast Cancer*, 47 ARCH. ENVTL. HEALTH 143-46 (1992); M.S. Wolff et al., *Blood Levels of Organochlorine Residues and Risk of Breast Cancer*, 85 J. NAT'L CANCER INST. 648-52 (1993); A.K. Henderson et al., *Breast Cancer among Women Exposed to Polybrominated Biphenyls*, 6(5) EPIDEMIOLOGY 544-46 (1995).

58. See *id.*

59. See Kavlock et al. *supra* note 20, at 4.

60. See D.P. BROWN, *Mortality of Workers Exposed to Polychlorinated Biphenyl - An Update*, 42 ARCH. ENVTL. HEALTH, 333-39 (1987); see also T. Sinks et al., *Risk*

ther research is needed to definitively establish a causal relationship between endocrine-disrupting chemicals and breast cancer in women.⁶¹

2. Reproductive Effects

The endocrine system is not the only mechanism by which adverse effects on reproduction can occur.⁶² There are, however, examples of chemical agents that have been shown to alter reproductive development by an endocrine mechanism.⁶³ These chemicals include DES, pesticides including DDT and kepone, and other chemicals, including dioxins and some PCBs.⁶⁴ In humans, evidence linking endocrine disrupting chemicals to adverse reproductive effects has been shown in the children of DES mothers.⁶⁵ Males exposed to kepone have been reported to have reproductive dysfunction,⁶⁶ while in females, lactation has been found to decrease with increasing DDE levels in breast milk.⁶⁷ There has also been a report of declining sperm quality in males over the last several decades but the cause is still not known.⁶⁸ Several other chemical or chemical classes, such as alkylphenols, some phthalates and bisphenol A, have been shown to interfere with some

Factors Associated with Excess Mortality among Polychlorinated Biphenyl Exposed Workers, 136 AM. J. EPIDEMIOLOGY 389-98 (1992).

61. See D.P. Brown, *Mortality of Workers Exposed to Polychlorinated Biphenyl – An Update*, 42 ARCH. ENVTL. HEALTH, 333-39 (1987); see also T. Sinks et al., *Risk Factors Associated with Excess Mortality among Polychlorinated Biphenyl Exposed Workers*, 136 AM. J. EPIDEMIOLOGY 389-98 (1992).

62. See PRINCIPLES AND METHODS OF TOXICOLOGY, 998 (A. Wallace Hayes ed., 3d 1994).

63. See P.S. Guzelian, *Comparative Toxicology of Chlordecone (kepone) in Humans and Experimental Animals*, 22 ANN. REV. PHARMACOLOGY & TOXICOLOGY 89-113 (1982); see also W.J. Rogan et al., *Polychlorinated Biphenyls (PCBs) and Dichlorodiphenyl Dichlorethane (DDE) in Human Milk: Effects on Growth, Morbidity and Duration of Lactation*, 77 AM. J. PUBLIC HEALTH 1294-97 (1987); B.C. Gladen, W.J. Rogan, *DDE and Shortened Duration of Lactation in a Northern Mexican Town*, 85 AM. J. PUBLIC HEALTH 504-08 (1995).

64. See generally *supra* note 63.

65. See *supra* notes 49 and 50.

66. See Guzelian, *supra* note 63.

67. See W.J. Rogan et al., *Polychlorinated Biphenyls (PCBs) and Dichlorodiphenyl Dichlorethane (DDE) in Human Milk: Effects on Growth, Morbidity and Duration of Lactation*, 77 AM. J. PUBLIC HEALTH 1294-97 (1987); B.C. Gladen, W.J. Rogan, *DDE and Shortened Duration of Lactation in a Northern Mexican Town*, 85 AM. J. PUBLIC HEALTH 504-08 (1995).

68. See E. Carlsen et al., *Evidence for Decreasing Sperm Quality of Semen during the past 50 Years*, 304 BRITISH MED. J. 609-13 (1992); J. Auger et al., *Decline in Semen Quality among Fertile Men in Paris during the past 20 Years*, 332 NEW ENG. J. MED. 281-85 (1995).

endocrine-mediated pathways, but there is currently no direct evidence of an effect in humans or other animals.⁶⁹

3. Effects on the Nervous System

Chemicals can exert an effect on the nervous system, thereby affecting the endocrine system through multiple mechanisms.⁷⁰ There can be a direct effect on an endocrine gland, such as the thyroid, to change the hormonal balance to affect the nervous system, causing neurotoxicity.⁷¹ Alternatively, endocrine disruptors can act on the central nervous system to affect the endocrine system.⁷² The end result is an adverse effect on behavior, learning and memory, attention, sensory function and psychomotor development.⁷³

4. Effects on the Immune System

Studies have suggested that exposure of humans to DES, TCDD, PCBs, carbamates, organochlorides, organometals and certain heavy metals change the immune system to cause immunosuppression.⁷⁴ Evidence of an increased rate of autoimmunity associated with prenatal DES exposure suggests that other endocrine disruptors may cause a similar pathological state.⁷⁵ Evidence indicates that the incidences of allergy and asthma (which are forms of hypersensitivity) are increasing in humans.⁷⁶ It is not known whether exposures to endocrine disrupting chemicals are responsible.⁷⁷

69. See R.J. Kavlock et al., *supra* note 20, at 7.

70. See Theo Colborn et al., *Developmental Effects of Endocrine-disrupting Chemicals in Wildlife and Humans*, 101 ENVTL. HEALTH PERSPECTIVES 378-84 (1993).

71. See *id.*

72. See *id.*

73. See *id.*

74. See K.L. Noller et al., *Increased Occurrence of Autoimmune Disease among Women Exposed in utero to Diethylstilbestrol*, 49(6) FERTILITY & STERILITY J. 1080-82 (1988); M.I. Luster et al., *Immunotoxicology: Review of Current Status*, 46 ANNALS OF ALLERGY 427-32 (1990).

75. See *id.*

76. See A.S. Buist, W.M. Vollmer, *Reflections of the Rise in Asthma Morbidity and Mortality*, 264 JAMA 1719-20 (1990); see also K.B. Weiss, D.K. Wagener, *Changing Patterns of Asthma Mortality. Identifying Target Populations at High Risk*, 264 JAMA 1688-92 (1990); P.J. Gergen, K.B. Weiss, *Changing Pattern of Asthma Hospitalization Among Children: 1979-1987*, 264 JAMA 1688-92 (1990).

77. See *id.*

III. United States Laws and Research Initiatives

A. Federal Initiatives

In response to the growing concern over environmental endocrine disruptors, in August 1996, Congress passed both the FQPA⁷⁸ and amendments to the SDWA.⁷⁹ Both of these laws contain provisions requiring the screening and testing of pesticides and chemicals for potential endocrine disrupting effects.⁸⁰ Specifically, these laws require EPA, in consultation with the Secretary of Health and Human Services, to “develop a screening program, using appropriate validated test systems and other scientifically relevant information, to determine whether certain substances may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen and other such endocrine effect as the Administrator may designate.”⁸¹ These laws also require EPA to develop a screening program by August 1998 and implement the program by August 1999.⁸² A report on the progress of the program must be presented to Congress by August 2000.⁸³

The two laws target different sets of chemical substances. Section 304 of the FQPA states that in carrying out the program, the Administrator shall “(A) provide for the testing of all pesticide chemicals; and (B) may provide for the testing of any other substance that may have an effect that is cumulative to an effect of a pesticide chemical if the Administrator determines that a substantial population may be exposed to such a substance.”⁸⁴

Section 136 of the SDWA amendments states that “in addition to the substances referred to in the FQPA, the Administrator may provide for testing under the screening program authorized by the FQPA for any other substance that may be found in sources of drinking water if the Administrator determines that a substantial population may be exposed to such substance.”⁸⁵ It should be noted that the FQPA and amendments to the SDWA supplement

78. See 21 U.S.C. § 345 (1996).

79. See 42 U.S.C. § 136 (1996).

80. See 42 U.S.C. § 300j-17 (1996).

81. *Id.*

82. See 21 U.S.C. § 345 (1996).

83. See 42 U.S.C. § 136 (1996).

84. 21 U.S.C. § 304 (1996).

85. *Id.* § 136.

testing requirements already in place for new and existing pesticides and industrial chemicals.⁸⁶

As a result of the passage of the FQPA and the SDWA, the EPA formed the Endocrine Disruptor Screening and Testing Advisory Committee, or EDSTAC.⁸⁷ The EDSTAC is composed of individuals representing various stakeholder groups and scientific experts, including representatives from the EPA, FDA, state agencies, industry, worker protection and labor organizations, national environmental groups, public health groups, and research scientists.⁸⁸ The EDSTAC goals were defined to:

- 1) develop a flexible process to select and prioritize chemicals and pesticides for screening, recognizing the need to obtain and utilize appropriate exposure information in setting priorities; 2) develop a process for identifying new and existing screening tests and mechanisms for their validation; 3) agree on a set of available, validated screening tests for early application; and 4) develop a process for deciding when additional tests, beyond screening tests, are needed and how many of these additional tests will be validated.⁸⁹

86. See the Federal Food, Drug and Cosmetic Act of 1938, as amended. In 1958, the FFDCA regulated the use of pesticides as food-additives and established pesticide tolerances for food. The Act defines a tolerance as the maximum amount of residue allowed to remain on an agricultural commodity at the time of harvest; the Clean Water Act (Federal Water Pollution Control Act, 1972, as amended) regulates toxic water pollutants; the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA) (1947) as amended, provides a regulatory framework for the registration and use of pesticides; the Safe Drinking Water Act (1974) sets enforceable standards for substances in drinking water; the Toxic Substance Control Act (TSCA) (1976) requires chemical producers to notify EPA prior to introducing new chemicals into commerce and gives EPA authority to require testing and information reporting, and control of new and existing industrial chemicals.

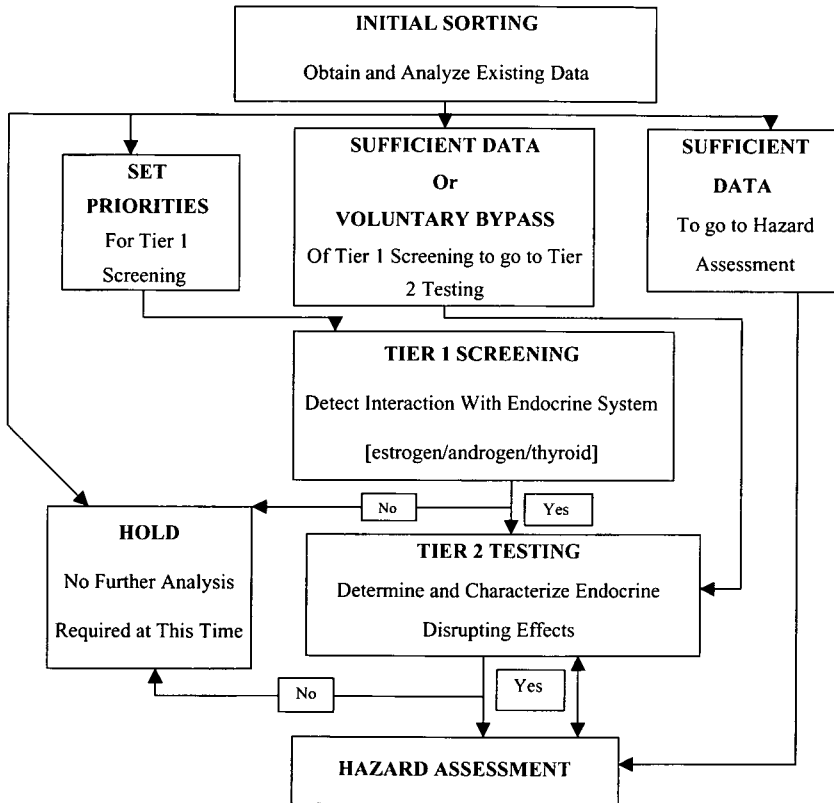
87. See EDSTAC CHARTER, *supra* note 12, at ES-1-15. The Charter established the EDSTAC in accordance with the requirements of the Federal Advisory Committee Act, 5 U.S.C., App. 2 Section 9. The EDSTAC provides advice and counsel to the EPA on a strategy to screen and test potential endocrine disruptors in order to reduce or mitigate risk to human health and the environment. See *id.*

88. See Environmental Protection Agency, ENDOCRINE DISRUPTORS, KEYSTONE CONVENING REPORT REGARDING THE FORMATION OF THE ENDOCRINE DISRUPTOR SCREENING AND TESTING ADVISORY COMMITTEE, THE KEYSTONE CENTER (Office of Prevention, Pesticides and Toxic Substances, Oct. 1996) (visited Jan. 20, 1999) <<http://www.riskworld.com/nreports/1996/endocrin/nr6acool.htm>>.

89. *Id.*

The EDSTAC final recommendations were published in August 1998.⁹⁰ A summary of the testing approach recommended by this committee is presented in Figure One.⁹¹

FIGURE 1: EDSTAC CONCEPTUAL FRAMEWORK PROVIDING THE STRUCTURE FOR SCREENING AND TESTING FOR ENDOCRINE DISRUPTORS⁹²



90. See EDSTAC FINAL REPORT, *supra* note 18, at ES-1-2. The EDSTAC recommended that EPA's endocrine disruptor screening and testing program (EDSTP) should "address both human and ecological (wildlife) effects." *Id.* at ES-2. It also recommends evaluating "endocrine disrupting properties of both chemical substances and common mixtures." *Id.* Recognizing that there are over 87,000 chemicals that must be prioritized for endocrine disrupting screening, the EDSTAC recommended that "high throughput pre-screening" (HTPS) be established. *Id.* at ES-3, 8. Priority setting would be based on different combinations of information and criteria, including exposure and effects information. See *id.* at ES-9. Testing would involve *in vitro* and *in vivo* screening tests for the various endocrine endpoints. See *id.* at ES-11-15.

91. See EDSTAC FINAL REPORT, *supra* note 18, at ES-11-15.

92. See EDSTAC FINAL REPORT, *supra* note 18, at ES-5.

B. State Initiatives

In addition to the federal initiatives, various states sought to implement their own programs.⁹³ The California Assembly Health Committee, in 1996, rejected legislation that called for the state to convene a special task force to determine if further studies were needed on the relationship between chemicals, breast cancer and the environmental presence of endocrine disruptors.⁹⁴ The committee granted the bill an opportunity for reconsideration at a future date.⁹⁵

The Minnesota Pollution Control Agency forwarded a proposal in June 1996 to the Legislative Commission on Minnesota Resources.⁹⁶ The proposal listed nonylphenols and phthalates from plastics, along with organochloride pesticides, surfactants from detergents, petrochemicals and residuals from pharmaceuticals as possible endocrine disruptors.⁹⁷ The proposal called for a two-tiered statewide screening process for endocrine disruptors.⁹⁸ The process would involve physical studies and bioassays of fish and frogs, followed by an analysis of water chemistry at sites where endocrine anomalies were detected.⁹⁹ It also required the analysis of drinking water intakes and the evaluation of the suitability of tests for various classes of chemicals for future monitoring.¹⁰⁰ The proposal also called for a follow up to a study conducted by the Minnesota Department of Natural Resources and the EPA, which found elevated yoke protein (a female attribute) in male carp on the Mississippi River near Minnesota.¹⁰¹ Minnesota's governor approved the project in 1997.¹⁰²

C. U.S. Industry Group Initiatives

In addition to the EPA and state initiatives, various U.S. industry groups have established programs to defend the products their companies manufacture. The CCC, CMA and SOCMA have

93. See 1996 Cal. Legis. Serv. 1023 (S.B. 1497) (West). See also 1997 Minn. Sess. Law Serv. 216 (West).

94. See 1996 Cal. Legis. Serv. 1023 (S.B. 1497) (West).

95. *Id.*

96. See 1997 Minn. Sess. Law Serv. 216 (West).

97. See *id.*

98. See *id.*

99. See *id.*

100. See *id.*

101. See 1997 Minn. Sess. Law Serv. 216 (West).

102. See *id.*

all established research programs to examine the effect of their chemicals on the endocrine system.¹⁰³

In 1997, the CMA committed four million dollars towards a generic endocrine research program.¹⁰⁴ Based on the concept of “Responsible Care,” the CMA outlined the following priorities of that research:

1. Development of tools that will allow industry to evaluate its products (this is particularly important to U.S. chemical companies because of recent mandates laid out by the Food Quality Protection Act and Safe Drinking Water Act);
2. Set priorities among chemicals for screening and testing which will be based on a combination of factors including production volume, potential exposure, physical and chemical properties and modeling and structural activity relationships;
3. Ensure that standardized and validated screening tests are available;
4. Examine risk assessment methods to see if new approaches are needed for summarizing hazard and exposure information into a form that is useful for decision-makers;
5. Increase industry’s understanding of underlying biological mechanisms (i.e. what are the thresholds for adverse effects? What is the shape of the dose-response curve?);
6. Seek further opportunities for collaborations with other interested parties including government agencies, environmental interest groups and universities to foster scientific consensus and help resolve some of the risk management and inventory aspects of the endocrine disruptor issue.¹⁰⁵

At that time, CMA also called for the exchange of information among international groups to avoid duplication of research efforts and to minimize public confusion.¹⁰⁶ In 1999, CMA announced that its Board of Directors had approved the first three years of a research initiative that will be designed to “investigate

103. See Chemical Manufacturers Association, *The Chemical Industry’s Health and Environmental Effects Research Initiative* (visited Feb. 28, 1999) <<http://www.cmahq.com/healthresearch.nsf/pages/about>>.

104. See International Workshop on Endocrine Disruptors: Workshop Report, Smithsonian Institution, Washington, D.C., 4 (Jan. 23-24, 1997). The CMA is a non-profit trade association of more than 190 member companies. It represents approximately 90% of the product capacity for basic industrial chemicals in the U.S.

105. *Id.* at 4-5.

106. See *id.* at 5.

the basic mechanisms by which chemicals interact and react with human health and the environment."¹⁰⁷

CCC has published a Question and Answer document specifically directed at questions raised by *Our Stolen Future*.¹⁰⁸ In 1996, CCC discounted concerns about declining sperm counts and stated that better cancer detection methods were responsible for the apparent rise in prostate and breast cancer rates.¹⁰⁹ Recently, CCC has also discounted any health risks due to the presence of phthalate esters in blood contained in polyvinyl chloride (PVC) plastic bags.¹¹⁰

In March 1996, the Competitive Enterprise Institute (CEI), a non-profit, non-partisan public policy group, downplayed the human health risks of synthetic chemicals, and pointed to naturally occurring toxins instead.¹¹¹ CEI noted that "naturally-occurring compounds, known as phytoestrogens, are many times more potent than the synthetic compounds most identified with threats to endocrine systems, DDT and PCBs, both of which were banned in the 1970s."¹¹²

D. The Response from Environmental Groups

Taking up the call of Theo Colborn, the environmental groups have focused on persistent organic pollutants, saying that "people should be considered innocent until proven guilty; chemicals should not."¹¹³ Greenpeace has focused on chemicals such as dioxin and PVCs that are used widely in modern consumer products or are produced as byproducts.¹¹⁴ Greenpeace has stated "there is now more than enough scientific evidence to begin a phase out of toxic hormones such as dioxin and dioxin producing vinyl plas-

107. Chemicals Manufacturers Association, *supra* note 103. The CMA estimates that this initial program, totaling \$67 million, will grow \$25 million per year within five years. *See id.*

108. *See* Chlorine Chemistry Council, *supra* note 11.

109. *See* Chlorine Chemistry Council, *supra* note 11, at 5.

110. *See* Chlorine Chemistry Council, *Key Concerns with Anti-Vinyl Arguments Made by Health Care Without Harm* (visited Feb. 1999) <http://www.c3.org/library/ivbag_concerns.html/>.

111. *See* Competitive Enterprise Institute Press Release, *Risks to Endocrine Systems Overstated: New Studies Challenge Latest Environmental Scare Campaign* (Mar. 13, 1996) (visited Jan. 20, 1999) <<http://www.cei.org/press.asp>>.

112. *Id.*

113. M. M. Malkin and M. Fumento, *Rachel's Folly: The End of Chlorine*, 3 (Chlorine Chemistry Council, pub.1996) (visited Jan. 20, 1999) <<http://c3.org/library/rachelchlor.html>>.

114. *See* Greenpeace Press Release, Mar. 14, 1996 (visited on Feb. 28, 1999) <<http://www.greenpeace.ch/press/1996/133.html/>>.

tics.”¹¹⁵ The Sierra Club has called for “immediate action to stop exposing men, women and children to [chlorine] based chemicals. Regulation of [dioxin and other chlorinated] chemicals as a class is the only way that we can adequately address this issue.”¹¹⁶

IV. Products Liability

A. A Discussion of Products Liability

Before an analysis is presented as to whether the current state of science would support a successful products liability claim due to harm from endocrine disrupting chemicals, it is useful to review the various theories under which such a claim may be brought. The phrase “products liability” has its roots in case and statutory law and allows recovery of money damages from the manufacturers and sellers of defective products that injure people or property.¹¹⁷ There are four principal theories that form the foundation for products liability suits.¹¹⁸ These include: 1) negligence; 2) breach of one or more warranties; 3) strict liability, or liability without fault or negligence; and 4) misrepresentation.¹¹⁹

Practically all products liability actions require the plaintiff to show the product was either: 1) manufactured incorrectly; 2) was defective in design or formulation; 3) failed to give satisfactory warnings or instructions for safe use; or 4) failed to truthfully represent the quality of a product.¹²⁰ In all cases the plaintiff must show there was liability on the part of a manufacturer of the product, causation and damages.¹²¹

1. Negligence

In negligence actions, the law looks to compensate an individual for personal injury or property loss that is foreseeable and that was caused by another person’s failure to act with due care under the circumstances.¹²² From this rule, a seller is liable for negli-

115. *Id.*

116. J. Robert Cox, Sierra Club President, quoted in THE PLANET (Washington, D.C.: Sierra Club), Nov. 1994 (visited Jan. 20, 1999) <<http://www.sierraclub.org/planet/199411/alert-dioxin.html>>.

117. See JOHN L. DIAMOND ET AL., UNDERSTANDING TORTS, § 17.01, at 291 (1996) [hereinafter DIAMOND ET AL.].

118. See *id.*

119. See *id.*

120. See *id.*

121. See David Owen, *Products Liability Law Restated*, 49 S.C. L. REV. 273 (1988); see also DIAMOND ET AL., *supra* note 117, at 292.

122. See DIAMOND ET AL., § 3.01, *supra* note 117, at 45-46.

gence in a products liability action if he or she acts or fails to act in a way that creates an unreasonable risk of harm or loss to the user of a product.¹²³ Liability also attaches to a seller if a person might be foreseeably injured through the use of the product.¹²⁴ To prevail, a plaintiff must prove that there is harm to his or her person or property, and that there was a proximate cause between the seller's conduct and the harm suffered.¹²⁵

To determine whether a risk is reasonable or unreasonable, most courts use Judge Learned Hand's risk-benefit model.¹²⁶ This model weighs the risk and benefits of the product to determine liability.¹²⁷ Thus, the manufacturer has a duty of care that must be weighed against the potential risk when considering the design, formulation, fabrication, testing and warnings concerning a product.¹²⁸ The manufacturer of the product has the burden to assure that the benefits of the product outweigh the harm that it may cause.¹²⁹

To be successful in a products liability negligence action it is not necessary to show that the product is "inherently dangerous."¹³⁰ Rather, the duty of due care requires the manufacturer to select the appropriate materials and method of manufacture that will produce a safe product.¹³¹ Where there is more than one manufacturer of the product, the duty of due care in the final product may rest on the final expertise of each manufacturer.¹³² Additionally, a manufacturer's liability may be limited in cases where the purchaser of a product has superior knowledge in the operational or safety requirements of the particular use of the

123. See DIAMOND ET AL., § 3.01, *supra* note 117, at 292.

124. See DIAMOND ET AL., § 3.01, *supra* note 117, at 292.

125. See DIAMOND ET AL., § 3.01, *supra* note 117, at 292.

126. See *United States v. Carroll Towing Co.*, 159 F.2d 169 (2d Cir. 1947). In *Carroll Towing Co.*, Judge Hand stated that an actor's conduct is considered in breach of his duty when B, the burden of taking measures to avoid the harm, is less than P, the likelihood or probability that the harm will occur, multiplied by L, the magnitude of the harm or liability should it occur. See *id.*

127. See *id.*

128. See DIAMOND ET AL., *supra* note 117, at 309.

129. See DIAMOND ET AL., *supra* note 117, at 309.

130. DIAMOND ET AL., *supra* note 117, at 311.

131. See DIAMOND ET AL., *supra* note 117, at 311.

132. See *Elliott v. Century Chevrolet Co.*, 597 S.W.2d 563, 565 (Tex. App. 1980). This case involved a suit against the manufacturer of truck chassis by a worker who was injured when a beer truck backed up and pinned him between the truck and loading dock. Since the chassis manufacturer sold trucks to secondary manufacturers who would then install other units on the chassis the court held that the secondary manufacturer, not the chassis manufacturer, had the necessary expertise to assess the safety of its design. See *id.*

product.¹³³ Finally, a manufacturer's liability can be limited when a product leaves its possession and control, is substantially altered, and is the proximate cause of the plaintiff's injuries.¹³⁴

In addition to the element of due care, reasonably foreseeable use or misuse also limits a manufacturer's liability.¹³⁵ The plaintiff must also be a person who might reasonably be foreseen to use, consume, or be affected by the product.¹³⁶ It should be noted that a manufacturer cannot be held liable for failing to foresee beyond what is scientifically or technologically discoverable at the time of manufacture.¹³⁷ Thus, a products liability action may fail if the plaintiff's harm could not have been anticipated based on the scientific knowledge that existed when the product was introduced into commerce. Similarly, the majority rule is that the safe use of a product over time is admissible evidence to show that a manufacturer met his duty of safe manufacture, but it is not the only factor that determines when the duty has been met.¹³⁸

2. Warranties

The concept of warranty in products liability law merges contract with tort.¹³⁹ A warranty provides remedies for persons who have bought or been exposed to products that either do not satisfy ordinary expectations, or are dangerous, or both.¹⁴⁰ Under this

133. See *Biss v. Tenneco, Inc.*, 409 N.Y.S.2d 874 (N.Y. App. Div. 1978), *appeal denied*, 389 N.E.2d 841 (N.Y. 1979). In this case the plaintiff's employer had bought a loader for its logging operations. The loader rolled over causing the plaintiff injury. The plaintiff's employer, not the manufacturer of the loader, was held liable for negligence for failing to provide the safety option of a roll-over protection structure. See *id.*

134. See *Robinson v. Reed-Prentiss Div. of Package Machinery Co.*, 403 N.E.2d 440 (N.Y. 1980).

135. See *Wallace v. Owens-Illinois, Inc.*, 389 S.E.2d 155 (S.C. Ct. App. 1989).

136. See *id.* This case involved a defective soda bottle that exploded. The explosion did not injure the plaintiff. Rather, the plaintiff was injured in the clean up. The court held that plaintiff had the requisite foreseeability of his injury as evidenced by the finding that the manufacturer intended to hold liquid under pressure and, as a consequence, should foresee that a bottle can fracture and spill and cause a situation that will invite the user or others to clean-up the broken glass.

137. See *DIAMOND ET AL.*, *supra* note 117, at 312.

138. See *Fredericks v. American Export Lines*, 227 F.2d 450, 452 (2d Cir. 1955), *cert. denied*, 350 U.S. 989 (1956). The court's comments are appropriate to the majority view. In affirming a jury verdict that the manufacturer was negligent for a broken skid that had been used safely for two and a half years the court stated, the "mere passage of time confers no immunity upon a negligent wrongdoer; but it has relevance to the likelihood, depending upon the circumstances of a particular case, that deterioration due to use, perhaps accelerated by misuses, will be mistaken by a jury for a defect due to negligent manufacture or fabrication." *Id.*

139. See *DIAMOND ET AL.*, *supra* note 117, at 317.

140. See *DIAMOND ET AL.*, *supra* note 117, at 317.

theory, a product can have either an express or an implied warranty.¹⁴¹ An express warranty occurs when the seller makes representations to the buyer of quality, performance, construction or durability of a product.¹⁴² Under the Uniform Commercial Code (UCC) section 2-313(1), a seller's assertion of fact can be considered an express warranty if it "becomes a benefit of the bargain."¹⁴³ As a general rule, to be considered part of the basis of the bargain of the sale, the seller's statement must precede or accompany the sale of the product.¹⁴⁴

In addition to an express warranty, the UCC establishes an implied warranty of merchantability.¹⁴⁵ Many courts have defined merchantability to mean reasonable fitness for the general purposes for which the article is sold and used.¹⁴⁶ Thus, to be merchantable a product need not be perfect or even of high quality.¹⁴⁷ It must merely conform to ordinary standards, be of average grade or quality and have the value of similar goods that are sold in commerce.¹⁴⁸

UCC section 2-314(2)(c) also requires that a product be "fit for [its] ordinary purpose."¹⁴⁹ To be found liable for a breach under a theory of implied warranty of merchantability it is necessary for a plaintiff to show that the product failure or accident happened in an ordinary use of the product.¹⁵⁰ UCC section 2-315 also creates

141. See *DIAMOND ET AL.*, *supra* note 117, at 317.

142. See *DIAMOND ET AL.*, *supra* note 117, at 317.

143. U.C.C. § 2-313(1), cmt. 7 (1977).

144. See *id.*

145. See U.C.C. § 2-314. This section lists six standards to which a product must conform to be merchantable. To be merchantable, goods must "(a) pass without objection in the trade under the contract description; and (b) in the case of fungible goods, are of fair average quality within the description; and (c) are fit for the ordinary purposes for which such goods are used; and (d) run, within the variations permitted by the agreement, of even kind, quality and quantity within each unit and among all units involved; and (e) are adequately contained, packaged, and labeled as the agreement may require; and (f) conform to the promises or affirmations of fact made on the container or label if any." Additionally, absent exclusion or modification, "other implied warranties may arise from course of dealing or usage of trade." *Id.*

146. See *DIAMOND ET AL.*, *supra* note 117, at 317.

147. See *DIAMOND ET AL.*, *supra* note 117, at 317.

148. See *DIAMOND ET AL.*, *supra* note 117, at 317.

149. U.C.C. § 2-314(2)(c).

150. See *Hardman v. Helene Curtis Indus., Inc.*, 198 N.E.2d 681, 691 (Ill. App. Ct. 1964). This case involved a child who was injured after she sprayed flammable hair spray on her hair and dress because of the pleasant fragrance. In holding that it was for a jury to decide whether this was ordinary use, the court stated "the essential question presented by a claim of breach of implied warranty of merchantability is whether the product failed to safely and adequately satisfy the uses to which products are ordinarily put." *Id.*

two conditions for showing that a product is fit for a particular purpose.¹⁵¹ First, the buyer must rely “on the seller’s skill or judgment to select or furnish suitable goods.”¹⁵² Secondly, at the time of sale or at the point when the parties enter into a contract to sell, the seller must have reason to know of the buyer’s purpose in buying the goods and must also have reason to know that the buyer is relying on the seller’s skill or judgment.¹⁵³ Unlike UCC section 2-314, under section 2-315 the person need only be a seller and not “a merchant with respect to goods of that kind.”¹⁵⁴

The UCC also allows sellers to disclaim warranties and limit the remedies available to the buyer.¹⁵⁵ As a general rule, an express warranty cannot be disclaimed once it is made, particularly when the express warranty is made in writing.¹⁵⁶ This is in contrast to an implied warranty of merchantability or fitness for a particular purpose that can be disclaimed.¹⁵⁷ However, in the latter instance, the seller must follow disclosure and conspicuousness requirements.¹⁵⁸ Conspicuous is defined by UCC section 2-201(10).¹⁵⁹ The issue of conspicuousness is for resolution by the court.¹⁶⁰ Generally, the main purpose of a warranty under UCC section 2-316 is to avoid surprise to the buyer and the knowledge of the disclaimer should be sufficient to give it effect.¹⁶¹

151. See U.C.C. § 2-315.

152. *Id.*

153. See *id.*, “Where the seller at the time of contracting has reason to know any particular purpose for which the goods are required and that the buyer is relying on the seller’s skill or judgment to select or furnish suitable goods, there is unless excluded or modified under the next section an implied warranty that the goods shall be fit for such purpose.” *Id.*

154. U.C.C. § 2-314. See also § 2-315.

155. See U.C.C. § 2-316.

156. See U.C.C. § 2-316(1).

157. See U.C.C. § 2-316(2) and (3).

158. See *id.* Section 2-316(2) states that any language that is intended to modify the implied warranty of merchantability “must mention merchantability and in case of writing must be conspicuous.” *Id.* But to be able to exclude or modify any implied warranty of fitness, the exclusion must be in writing and conspicuous. There are no particular words that must be used to disclaim the implied warranty of fitness for a particular purpose. Section 2-316(3) gives an example of the type of language that might be used to exclude all implied warranties of fitness (e.g. “there are no warranties, which extend beyond the description on the face hereof.”) *Id.*

159. See U.C.C. § 2-201(10). This section states that the language must be “so written that a reasonable person against whom it is to operate ought to have noticed it.” *Id.*

160. See *id.*

161. See *id.*

"As is" disclaimers are also allowed by the UCC in section 2-316(2).¹⁶² Using words such as "as is," "with all faults," or "as they stand," for example, the seller may effectively disclaim all warranties.¹⁶³

A seller can also limit the buyer's remedies through use of warranty limitations, but there are ways for a buyer to overcome this limitation.¹⁶⁴ A warranty limitation on consequential damages will not be given effect when they are found to be unconscionable.¹⁶⁵ A warranty limitation on consequential damages is considered prima facie unconscionable in circumstances where a buyer is seeking consequential damages for injury to a person.¹⁶⁶

3. Strict and Fault-Based Liability

In May 1997, the American Law Institute (ALI) adopted the Restatement (Third) of Torts.¹⁶⁷ Like section 402A of the Restatement (Second) of Torts,¹⁶⁸ the Third Restatement imposes strict liability on manufacturers for manufacturing defects.¹⁶⁹ However, design and warning cases now utilize a fault-based liability.¹⁷⁰

The Restatement (Third) provides a new black letter rule that imposes a continuing duty to warn on the manufacturer.¹⁷¹ Further, once a product is marketed with adequate warnings, a manufacturer can still incur liability if he does not act as a reasonable

162. See U.C.C. § 2-316(3)(a). "As is" disclaimers can be used as an alternative to U.C.C. § 2-316(2). This section does not require the seller to follow the guidelines of section 2-316(2). Instead it requires that the disclaimer be in a "language which in common understanding calls the buyer's attention to the exclusion of warranties and makes plain that there is no implied warranty." *Id.*

163. *Id.* See also U.C.C. § 2-316(3), cmt. 7.

164. See U.C.C. § 2-719.

165. See U.C.C. § 2-719(3).

166. See *id.* See also *Collins v. Uniroyal, Inc.*, 315 A.2d 16 (N.J. 1974). This case is a leading decision involving the interpretation of this Code provision. In this case, five months after buying new tires for his car the plaintiff's decedent was killed in a car accident as a result of a tire blowout. Uniroyal disclaimed consequential damages and the warranty limited the seller's liability to repair or replacement of the tire. The New Jersey Supreme Court noted that where a manufacturer makes express representations as to the safety of the product an ordinary buyer would likely buy the product relying on the safety assurances and not the repair or replacement remedy noted in the warranty limitation. Thus, the warranty limitation on damages and liability were unconscionable.

167. See RESTATEMENT (THIRD) OF TORTS: PRODUCTS LIABILITY (1998).

168. See RESTATEMENT (SECOND) OF TORTS (1965).

169. See RESTATEMENT (THIRD) OF TORTS: PRODUCTS LIABILITY § 1-2 (1998).

170. See *id.* § 2(b), (c).

171. See *id.* § 10.

person with respect to a duty to warn about risks discovered after the product is on the marketplace.¹⁷²

Under the Restatement (Third), a court, in extraordinary circumstances, may consider imposing liability on a manufacturer for harms caused by a product that is so dangerous it should never have been made, even if there is no other way to make the product.¹⁷³ This type of claim is also allowed for pharmaceuticals that no reasonable practitioner would prescribe.¹⁷⁴

The Restatement (Third) also allows plaintiffs to win liability claims due to defective design, warnings and mismanufacture under the doctrine of *res ipsa loquitur*.¹⁷⁵ This doctrine states that direct evidence is not needed to prove a defect and the plaintiff need not be an expert.¹⁷⁶ There is almost no case law to support this view with respect to defects based on either design or warnings.¹⁷⁷

With regard to warnings, the Restatement (Third) also revised the Restatement (Second) approach. Under Section 402A of the Restatement (Second), a manufacturer could be insulated from liability by providing a warning.¹⁷⁸ The Restatement (Third) expressly rejects this approach. Rather, it states that a warning is one factor that a court should weigh in deciding whether a product is defective.¹⁷⁹

Two recent decisions have followed the Restatement (Third). In *Uniroyal Goodrich Tire Co. v. Martinez*, the Texas Supreme Court affirmed a \$10.3 million judgment for the plaintiff.¹⁸⁰ In agreeing with the trial court judge, the Texas Supreme Court utilized comment f in section two of the Restatement (Third), which states that a warning is one factor in determining whether a prod-

172. See *id.* § 10(b).

173. See *id.* § 2, cmt. e.

174. See RESTATEMENT (THIRD) OF TORTS: PRODUCTS LIABILITY § 6(c).

175. See *id.* § 3.

176. See *id.* § 3.

177. See, e.g., *Uniroyal Goodrich Tire Co. v. Martinez*, 977 S.W.2d 328 (Tex. 1998); *Rogers v. Ingersoll-Rand. Co.*, 144 F.3d 841 (D.C. Cir. 1998).

178. See RESTATEMENT (SECOND) § 402A, cmt. j. Comment j states, "where warning is given, the seller may reasonably assume that it will be read and heeded; and a product bearing such a warning which is safe for use if it is followed, is not in defective condition nor is it unreasonably dangerous." *Id.*

179. See RESTATEMENT (THIRD) § 2 cmt. l. Comment l states, "when an alternative design to avoid risks cannot reasonably be implemented, adequate instructions and warnings will normally be sufficient to render the product reasonably safe." However, warnings are not "a substitute for the provision of a reasonably safe design." *Id.*

180. See *Uniroyal*, 977 S.W.2d at 331.

uct is defective.¹⁸¹ In *Uniroyal*, the plaintiff admitted seeing a prominent warning highlighted in yellow and red that told him not to place a sixteen-inch diameter tire on a 16.5-inch rim.¹⁸² The warning was supplemented with a picture of a worker being thrown into the air with an exploding tire.¹⁸³ Despite the warning, the plaintiff proceeded to mount a smaller tire on the larger rim.¹⁸⁴ The tire ultimately exploded causing him severe injury.¹⁸⁵ The plaintiff argued that the tire was defective because it did not incorporate a safer, reasonable, alternative design used by other tire manufacturers.¹⁸⁶ The court agreed with him.¹⁸⁷

In another case, the United States Court of Appeals for the District of Columbia Circuit also followed section 2, comment l of the Restatement (Third) in holding that a manufacturer was liable for injuries a worker incurred in a milling machine accident.¹⁸⁸ As the driver backed up, the milling machine alarm did not sound to alert the workers in the area, and the driver failed to see the plaintiff due to a blind spot in the rear view mirror.¹⁸⁹ The plaintiff's pelvis was crushed in the incident, and internal injuries were sustained.¹⁹⁰ The manufacturer did provide warnings in both its operations and maintenance manual, stating that personnel should stay ten feet from the rear of the machine when it was in operation; that the operator should confirm that the back-up alarm was working; and that personnel should examine the area to assure that it was free of other personnel.¹⁹¹ In holding for the plaintiff, the court found that an adequate warning by itself does not "immunize a manufacturer from any liability caused by its defectively designed product"¹⁹² and that warnings cannot "trump all other factors."¹⁹³

181. *See id.* at 335. *See also* RESTATEMENT (THIRD) § 2 cmt. f.

182. *See Uniroyal*, 977 S.W.2d at 332.

183. *See id.*

184. *See id.*

185. *See id.*

186. *See id.*

187. *See Uniroyal*, 977 S.W.2d at 331.

188. *See Rogers v. Ingersoll-Rand Co.*, 144 F.3d 841 (D.C. Cir. 1998).

189. *See id.* at 842.

190. *See id.* at 842.

191. *See id.* at 843.

192. *Id.*

193. *Rogers*, 144 F.3d at 844.

4. Toxic Tort Litigation

In looking toward whether exposures to potential endocrine disrupting chemicals can lead to successful litigation, it is also useful to examine the litigation involving DES for guidance.¹⁹⁴ DES was a prescription drug prescribed to pregnant women from the late 1940s through the early 1970s for a variety of reasons.¹⁹⁵ The drug was marketed in the United States by hundreds of pharmaceutical companies.¹⁹⁶

In 1971, the FDA determined that DES was both ineffective and dangerous for use by pregnant women and withdrew approval for its use.¹⁹⁷ Studies revealed a link between a form of gynecological cancer in daughters born from women who took DES during pregnancy.¹⁹⁸ By the time FDA approval was withdrawn, the drug had been used by millions of pregnant women, and many of their daughters faced the possibility of developing cervical cancer.¹⁹⁹

As a result of these exposures, thousands of lawsuits were filed against DES manufacturers²⁰⁰ based on products liability claims.²⁰¹ In most of these claims the plaintiffs were able to prove: 1) that DES caused their injuries; 2) the defendant drug companies manufactured the DES for the prevention of miscarriages; 3) the defendants knew or should have known that DES was carcinogenic; and 4) the defendants failed to warn the plaintiffs' mothers of the hazards of the drug.²⁰²

The plaintiffs also had the burden of identifying which drug company manufactured the DES their mothers took.²⁰³ However, many brands of DES were not patented and fungible DES pills

194. See HARDMAN ET AL., *supra* note 27, at 1420. DES is the common abbreviation for diethylstilbestrol, a synthetic female hormone with estrogen-like effects.

195. See Note: *Market Share Liability: An Answer to the DES Causation Problem*, 94 HARV. L. REV. 668, 677 (1981) [hereinafter "Note"]. The FDA approved the use of DES for the prevention of certain complications during pregnancy. See also Comment: *DES and a Proposed Theory of Enterprise Liability*, 46 FORDHAM L. REV. 963 (1978) [hereinafter "Comment"].

196. See Comment, *supra* note 195, at 964.

197. See *Certain Estrogens for oral and parental use*, 36 Fed. Reg. 21,537-38 (1971).

198. See Comment, *supra* note 195, at 964.

199. See Note, *supra* note 195, at 668.

200. See Note, *supra* note 195, at 669.

201. See Note, *supra* note 195, at 669.

202. See Note, *supra* note 195, at 669.

203. See Note, *supra* note 195, at 669-70.

were essentially interchangeable.²⁰⁴ As a result, a number of plaintiffs were not able to recover damages for their injuries because they could not identify the specific drug company that manufactured the DES.²⁰⁵ Other courts, however, aware of the difficulties in applying traditional causation theories to DES cases, allowed plaintiffs to recover notwithstanding their inability to prove a causal connection between their injuries and a particular manufacturer.²⁰⁶

In *Sindell v. Abbot Laboratories*, the daughters of women who were prescribed DES while pregnant brought a class action suit.²⁰⁷ The plaintiffs' claim was that their cancerous or pre-cancerous conditions resulted from *in utero* exposure to DES.²⁰⁸ The plaintiffs were able to show that the drug caused their injuries.²⁰⁹ Their products liability claim was that the drug manufacturers were negligent for failing to adequately test the drug and warn consumers of its potential dangers.²¹⁰ The trial court dismissed the complaint because the plaintiffs were unable to identify the precise manufacturers of the actual pill that caused their harm.²¹¹

On appeal, the appellate court found that the existing exceptions to the traditional causation theories could not apply, which meant the plaintiffs would not be allowed a recovery.²¹² However, the court did recognize that harm had occurred due to exposure to DES, and devised a theory that would allow the plaintiffs a remedy.²¹³ The theory is known as market share liability.²¹⁴ The

204. See *Diethylstilbestrol: Extension of Class Action Procedures to Generic Drug Litigation*, 14 U.S.F.L. REV. 461, 466 (1980).

205. See *Gray v. United States*, 445 F. Supp. 337 (S.D. Tex. 1978); see also *Namm v. Charles E. Frosst & Co.*, 427 A.2d 1121 (N.J. Super. Ct. App. Div. 1981).

206. See *Sindell v. Abbott Laboratories*, 607 P.2d 924 (Cal. 1980), *cert. denied*, 449 U.S. 912 (1980); see also *Abel v. Eli Lilly & Co.*, 289 N.W.2d 20 (Mich. Ct. App. 1979); *Ferrigno v. Eli Lilly & Co.*, 420 A.2d 1305 (N.J. Super. Ct. Law Div. 1980); *Bichler v. Eli Lilly & Co.*, 436 N.Y.S.2d 625 (N.Y. App. Div. 1981), *aff'd*, 436 N.E.2d 182 (1982).

207. See *Sindell*, 607 P.2d at 924.

208. See *id.* at 926.

209. See *id.*

210. See *id.*

211. See *id.*

212. See *Sindell*, 607 P.2d at 928.

213. See *id.* at 937. See also *Summers v. Tice*, 199 P.2d 1 (Cal. 1948).

214. See *Sindell*, 607 P.2d at 937. Market share liability modifies the alternative liability rule that was established in *Summers*. See *Summers v. Tice*, 199 P.2d 1 (Cal. 1948). In *Summers*, the plaintiff was injured by the bullet of one of two hunters who had fired their guns in his direction. The plaintiff was awarded damages despite being unable to identify which of the two negligent defendants fired the shot that caused his injury. See *id.* at 2. In market share liability each defendant is liable for that

court reasoned that "as between an innocent plaintiff and negligent defendants, the latter should bear the cost of the injury."²¹⁵ The *Sindell* court also noted:

In our contemporary complex industrialized society, advances in science and technology create fungible goods which may harm consumers and which cannot be traced to any specific producer. The response of the courts can be either to adhere rigidly to prior doctrine, denying recovery to those injured by such products, or to fashion remedies to meet these changing needs.²¹⁶

The court also stated that the imposition of liability would encourage defendant companies to manufacture safer products.²¹⁷

About the same time the *Sindell* case was decided, a New Jersey court, in another DES products liability case, held for the plaintiffs on a theory known as alternative liability.²¹⁸ The court, relying on *Anderson v. Somberg*,²¹⁹ determined that the burden of proof shifts to the defendants to show their innocence when plaintiffs, through no fault of their own, cannot establish which of a group of negligent defendants caused their harm.²²⁰ Those defendants who cannot prove they are not liable remain jointly and severally liable.²²¹

In *Abel v. Eli Lilly & Co.*, the Michigan Court of Appeals announced another theory under which plaintiffs might prevail when they stated a cause of action without proof of the precise causative agent.²²² Like the *Ferrigno* court, the Michigan Court of Appeals recognized the market share theory and stated that it could be applied in the case.²²³ The court also discussed another theory, known as the concerted action theory, under which:

portion of the judgment proportionally representative of his market share. See *Sindell*, 607 P.2d at 937. In alternative liability, each defendant is jointly and severally liable for the entire judgment. See *id.* at 928.

215. *Sindell*, 607 P.2d at 936 (citing *Summers v. Tice*, 199 P.2d 1 (Cal. 1948)).

216. *Id.* at 936.

217. See *id.*

218. See *Ferrigno v. Eli Lilly & Co.*, 420 A.2d 1305 (N.J. Super. Ct. Law Div. 1980).

219. See *Anderson v. Somberg*, 338 A.2d 1 (N.J. 1975), *cert. denied*, 423 U.S. 929 (1975).

220. See *Ferrigno*, 420 A.2d 1305 at 1313.

221. See *id.* at 1316.

222. See *Abel v. Eli Lilly & Co.*, 280 N.W.2d 20 (Mich. Ct. App. 1979). The plaintiffs developed cancer after their mothers took DES while pregnant, but they could not identify the manufacturer of the DES. See *id.*

223. See *id.*

those who, in pursuance of a common plan or design to commit a tortious act, actively take part in it, or further it by cooperation or request, or who lend aid or encouragement to the wrongdoer, or ratify and adopt his acts done for their benefit, are equally liable with him. . . . Express agreement is not necessary, and all that is required is that there be a tacit understanding²²⁴

Under this theory, if it can be shown that the defendants engaged in concerted activity, all the defendants are liable even though only one directly caused the harm.²²⁵ The court concluded that the plaintiffs' allegations that the manufacturers of DES acted in concert by wrongfully producing and marketing a dangerous drug without adequate testing or warnings, were sufficient to state a cause of action without identifying the precise cause of their harm.²²⁶

In *Bichler v. Eli Lilly & Co.*, the concerted action theory was modified to allow the plaintiff to recover even though she was not able to identify with certainty the causative agent of her harm.²²⁷ The *Bichler* court agreed with the *Abel* decision that "the law, especially in the products liability area, was not so rigid as to preclude an injured party, with an otherwise valid claim, from a remedy"²²⁸ solely because she could not discern the cause of her harm.²²⁹ For this reason, the court upheld the trial court's modified definition of concerted action.²³⁰ This definition states that defendants are deemed to have acted in concert even though they "act[ed] independently of each other in committing the same wrongful act, [if] their acts ha[d] the effect of substantially encouraging or assisting the wrongful conduct of the other, which in this case, was the alleged failure to adequately test."²³¹ As a result, the plaintiff was able to recover without proving causation.²³²

224. W. PROSSER, *THE LAW OF TORTS* at 292 (4th ed. 1971).

225. *See Abel*, 280 N.W.2d at 20.

226. *See id.* at 25.

227. *See Bichler v. Eli Lilly & Co.*, 436 N.Y.S.2d 625 (N.Y. App. Div. 1981), *aff'd*, 436 N.E.2d 182 (1982).

228. *Id.* at 632.

229. *See id.*

230. *See id.* at 631.

231. *Id.* at 632.

232. *See Bichler*, 436 N.Y.S.2d at 632.

V. Analysis

A. Does the Current Science Involving Endocrine Disruptors Support a Successful Products Liability Claim?

A plaintiff alleging harm due to exposure to endocrine disruptors has a number of options with regard to the theory of products liability under which he or she might bring the claim.²³³ In all cases, whether the claim is brought under negligence, failure to warn, or strict liability under the Restatement (Third), the plaintiff must prove liability, causation and damages.²³⁴ Only liability and causation will be discussed here.

1. Liability

A plaintiff in a suit to recover damages for exposure and harm from endocrine disruptors would first have to show that the manufacturer of the product was liable.²³⁵ Since these chemicals are in a wide variety of products to which a person can be exposed, this task is not unlike that facing plaintiffs in the DES cases.²³⁶ Like DES victims, endocrine disruptor plaintiffs cannot be certain of who caused their exposure to the toxic substance at the root of their injuries.²³⁷ Many DES plaintiffs were not able to recover damages for their injuries because they could not identify the specific company that manufactured the DES they had taken.²³⁸ Other courts, however, aware of the difficulties in applying traditional causation theories to DES cases, allowed plaintiffs to recover notwithstanding the fact that they were unable to prove a causal connection between their injury and a particular manufacturer.²³⁹ Thus, the courts will need to apply a similar reasoning if a plaintiff alleging harm due to exposure to endocrine disruptors is to prevail.

To determine which theory should be applied to determine who is liable, the circumstances surrounding the injury must be

233. See notes 114-87. The various theories that could be explored in filing a claim include those typically filed in products liability actions, including negligence, failure to warn and a breach of warranty.

234. See David Owen, *Products Liability Law Restated*, 49 S.C. L. REV. 273 (1998); see also DIAMOND ET AL., *supra* note 117, at 289.

235. See Owen, *supra* note 234, at 273.

236. See *Abel*, 280 N.W.2d at 22.

237. See *id.*

238. See *Gray v. United States*, 445 F. Supp. 337 (S.D. Tex. 1978); *Namm v. Charles E. Frost & Co.*, 427 A.2d 1121 (N.J. Super. Ct. App. Div. 1981).

239. See *Sindell*, 607 P.2d at 924. See also *Abel*, 289 N.W.2d at 20; *Ferrigno*, 420 A.2d at 1305; *Bichler*, 436 N.Y.S.2d at 625.

examined. Where a person alleges an injury due to exposure to an identifiable substance emanating from a consumer product, food or food package, he or she may be able to trace it to a number of defendants.²⁴⁰ If this can be done, an action could be brought against all those manufacturers for allowing the toxic substance to escape and cause harm.²⁴¹

It is likely, however, that the plaintiff will not be able to determine the actual manufacturer of the chemical that caused his or her harm.²⁴² If the market share theory were applied, proving defendant liability would be difficult. This is because it is unlikely the plaintiff would be able to determine the manufacturers' shares of the market since the sale of these chemicals are usually to multiple sources and it is difficult to obtain accurate records.²⁴³

Applying the concerted action theory of liability may be a better option for the plaintiff who cannot identify which defendant caused his injury.²⁴⁴ Using this theory the plaintiff will allege that the defendants acted in concert by wrongfully adding chemicals into a product without adequately providing for their safety or showing the products are safe.²⁴⁵ If the court followed the ruling in *Abel v. Eli Lilly & Co.*,²⁴⁶ they would be able to find that a manufacturer could be liable despite the plaintiff's inability to show an exact cause. Alternatively, a court could use the modified version of the concerted action theory announced in *Bichler v. Eli Lilly & Co.*²⁴⁷ to allow a plaintiff to recover. The plaintiff could argue that, although the defendants acted independently, their acts of intentionally adding the endocrine modulating substances encouraged or assisted the wrongful behaviors of the other defendants.²⁴⁸ The named defendants would then be jointly and

240. See COLBORN ET AL., *supra* note 5, at 213-22.

241. See *Sindell*, 607 P.2d at 924.

242. See COLBORN ET AL., *supra* note 5, at 211-22.

243. See COLBORN ET AL., *supra* note 5, at 211-22. See also Aaron M. Levin, *Identification of Manufacturer of Diethylstilbestrol (DES)*, 370 P.L.I. LIT. 191, 209-11 (1989); Myra P. Mulcahy, Note: *Proving Causation in Toxic Torts Litigation*, 11 HOFSTRA L. REV. 1299, 1324 (1983). Mulcahy considered this for litigation concerning hazardous waste and her theory is appropriate here.

244. See Mulcahy, *supra* note 243. Various other commentators have also proposed this theory for toxic tort cases in which a specific cause could not be found and for which testing has not been adequately performed. See, e.g., Allan Kanner, *Environmental and Toxic Tort Issues*, SC24 A.L.I.-A.B.A. 713 (1998).

245. See Kanner, *supra* note 244, at 733.

246. See *Abel v. Eli Lilly & Co.*, 289 N.W.2d 20 (1979).

247. See *Bichler v. Eli Lilly & Co.*, 436 N.Y.S.2d 625 (N.Y. App. Div. 1981), *aff'd*, 436 N.E.2d 182 (1982).

248. See *id.*

severally liable unless they could absolve themselves.²⁴⁹ Defendants could force unnamed parties who may be culpable and not named by the plaintiff to be joined as third party defendants. A separate action could also be filed by the defendants against such parties for their contribution.²⁵⁰

In applying the concerted action theory, it could be argued that it is not an equitable approach for the defendants. It should be remembered, however, that it is the defendants who produced the endocrine disrupting chemicals, and they were the ones who intentionally added endocrine disrupting chemicals to products, resulting in exposure and serious risk of harm to the plaintiff. Since the equities weigh in favor of the plaintiff, the courts should adopt this theory for endocrine disruptor cases.

The alternative liability approach is also an attractive alternative for showing liability, but the result may vary with the jurisdiction, depending upon which view is followed.²⁵¹ The court, for example, in *Ferrigno*, disagreed with *Sindell*.²⁵² In *Ferrigno*, a recovery under alternative liability was allowed even though the court could not determine that the defendants actually caused the harm.²⁵³ Thus, if the courts were to follow the decision in *Ferrigno* the plaintiff first needs to establish that the defendants breached their duty of care by adding the endocrine disruptor to the product, that harm resulted from exposure to the substance, and that the defendant produced the chemical.²⁵⁴ Once the plaintiff has established these elements, the burden of proving causation then shifts to the defendants.²⁵⁵ Those defendants who cannot meet this burden will be held jointly and severally liable.²⁵⁶ Thus, a plaintiff harmed by endocrine disruptors bringing a products liability suit under an alternative liability theory would have a chance for a remedy even where it is not possible to name all potential defendants.

While it may be observed that shifting the burden of proving causation to defendants appears to be unfair, it should be remembered that the defendants intentionally added the chemical

249. See *id.* See also Mulcahy, *supra* note 243, at 1324.

250. See Mulcahy, *supra* note 243, at 1324.

251. See *Sindell*, 607 P.2d at 931; see also *Ferrigno*, 420 A.2d at 1305; Mulcahy, *supra* note 243, at 1324.

252. See *Ferrigno*, 420 A.2d at 1305.

253. See *id.*

254. See *id.*

255. See *id.*

256. See *id.*

to the product and the chemical caused the harm. While it may be true that adopting an alternative liability theory for endocrine disruptor torts may cause traditional concepts and basic principles of tort law to be distorted or abandoned,²⁵⁷ the endocrine disruptor tort is not a “traditional” injury. Rather, as observed by the *Sindell* court, it is “the result of our industrialized society.”²⁵⁸ Therefore, as one commentator has noted, “it is important that our traditional legal judgments evolve to keep up with our progressing society.”²⁵⁹

2. Causation

In determining whether a plaintiff would be able to establish that they were harmed from exposure to products containing endocrine disruptor chemicals, it is first necessary to look at toxic tort litigation for guidance.²⁶⁰ Toxic tort litigation has forced the courts to articulate the basis for causation in chemical-related injuries.²⁶¹ The courts have looked at how scientific methods and scientific theories that may not conform to the scientific norm should be evaluated in terms of applicability and adequacy.²⁶² To survive a motion for summary judgment the plaintiff must diminish the uncertainty in the scientific methods or theories that surround proof of causation.²⁶³ The courts have addressed this issue in a number of ways.²⁶⁴

In order to recover damages in a suit alleging harm due to exposure to endocrine disruptors, a plaintiff must prove that a chemical or chemicals are the cause of his or her disease or harm.²⁶⁵ Specifically, the plaintiff must prove: 1) the toxic substance has the ability to cause the alleged harm; 2) the plaintiff's exposure to the toxic substance was of sufficient quantity to cause the disease; and 3) the injury or harm was caused by exposure to the toxic substance.²⁶⁶ Thus, a plaintiff hoping to recover in a

257. See *Namm*, 427 A.2d at 1127.

258. *Sindell*, 607 P.2d at 936.

259. Mulcahy, *supra* note 243, at 1324.

260. See Shawn A. Copeland et al., *Current Issues in Toxic Tort Litigation*, SC 64 A.L.I.-A.B.A. 33, 76-86 (1998).

261. See *id.* at 77.

262. See *id.* See also *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993).

263. See Copeland et al., *supra* note 260, at 77.

264. See Susan R. Poulter, *Science and Toxic Torts: Is There a Rational Solution to the Problem of Causation?*, 7 HIGH TECH L.J. 189, 198 (1992).

265. See *id.*

266. See *id.*

products liability suit for harm suffered from exposure to endocrine disruptors would first have to identify a specific chemical or chemicals in the product to which he or she was exposed.²⁶⁷ In this respect, individuals alleging harm due to endocrine disruptors face problems in proving this part of causation similar to those encountered by DES plaintiffs.²⁶⁸ Since any potential injury due to endocrine disruptors would be caused by fungible goods, they confront the problem of identifying the cause of their injuries.²⁶⁹ Since endocrine disruptors may be found in a number of consumer products²⁷⁰ and their packaging,²⁷¹ the plaintiff does not have an easy burden to overcome. However, analytical methods do exist to measure these chemicals,²⁷² so it is not something that is impossible to accomplish. Of course, the plaintiff would need to determine the amount present, which can be accomplished using analytical methods.²⁷³ Thus, it would be possible for a plaintiff to show he or she was exposed to a specific endocrine disruptor or multiple endocrine disruptors.

The plaintiff must also prove that exposure to the endocrine disruptor was in an amount sufficient to cause a disease.²⁷⁴ It is in this element of causation that the plaintiff faces his or her most formidable obstacle. Traditionally, in the absence of a specific cause-effect relationship following exposure to a chemical, courts have relied on epidemiological studies to support a direct cause of injury.²⁷⁵ Epidemiological risk analysis has been required as a minimal requirement by some courts to infer causation, especially when the cause of the disease or injury cannot be definitively proven through a plaintiff's medical record.²⁷⁶ The courts have

267. *See id.*

268. *See Sindell*, 607 P.2d at 937.

269. *See id.*

270. *See COLBORN ET AL.*, *supra* note 5, at 122-41.

271. *See generally supra* note 14.

272. *See generally supra* note 14.

273. *See Poulter*, *supra* note 264, at 231.

274. *See Poulter*, *supra* note 264, at 198.

275. *See Michael D. Green, Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of Agent Orange and Bendectin Litigation*, 86 Nw. U. L. REV. 643, 646-53 (1993). Epidemiological studies compare two groups of populations – one exposed to a particular chemical and one not exposed – and attempt to determine whether that exposure has resulted in an increased incidence of disease or injury in that particular population. *See id.* A chemical is said to cause a disease or injury if it increases the relative frequency of that disease or injury when it is present and decreases that frequency when it is absent. *See id.*

276. *See, e.g., In re Agent Orange Product Liab. Litig.*, 11 F. Supp. 1223, 1231, 1261-62 (E.D.N.Y. 1985), *aff'd*, 818 F.2d 187 (2d Cir. 1987).

not been consistent in determining what constitutes an acceptable epidemiological study in terms of statistical significance and relative risk.²⁷⁷ Epidemiological studies are able to uncover weaker toxic effects,²⁷⁸ but some toxic effects are too small to be detected in an epidemiological study, even though the effect was caused by the substance.²⁷⁹ These findings cannot satisfy the burden of proof under a preponderance of the evidence standard.²⁸⁰

In a toxic tort case, plaintiffs generally do not use epidemiological studies alone.²⁸¹ When epidemiological studies are available, a plaintiff will normally supplement these studies with medical testimony.²⁸² Even when epidemiological studies are not available, the medical testimony offered for a plaintiff will attempt to establish: 1) that the exposure to the chemical was the cause of the plaintiff's disease; 2) why the diagnostic tests used to establish the cause of the condition were appropriate; and 3) why other factors the plaintiff has are not relevant to the existence of the disease.²⁸³ The courts have not been consistent with regard to the use of medical testimony with or without the presence of epidemiological studies.²⁸⁴ At least one court has determined that medical testimony need not be dependent on an epidemiological study which first establishes a cause-effect relationship between the chemical and the disease.²⁸⁵ Other courts have stated that medical testimony should be considered additional supporting evidence to an epidemiological study.²⁸⁶

277. See Green, *supra* note 275, at 653.

278. See Green, *supra* note 275, at 653.

279. See Green, *supra* note 275, at 653.

280. See Green, *supra* note 275, at 653. See also Harold Ginsburg, *Use and Misuses of Epidemiological Data in the Courtroom: Defining the Limits of Inferential and Particularistic Evidence in Mass Tort Litigation*, 12 AM. J.L. & MED. 423, 431 (1986); Diana L. Mitts, *Epidemiological Evidence as a Basis for Causation: Implications for Suspected Pesticide-Induced Cancer*, 8 SANJALR 187, 188 (1998).

281. See Poulter, *supra* note 264, at 231.

282. See Poulter, *supra* note 264, at 232.

283. See Green, *supra* note 275, at 653.

284. See *Ferebee v. Chevron Chem. Co.*, 736 F.2d 1529, 1535-36 (D.C. Cir. 1984) (jury verdict upheld for plaintiff where plaintiff's case was based on expert testimony). *But see In re Joint Eastern and Southern Dist. Asbestos Litig.*, 827 F. Supp. 1014, 1027, 1030 (S.D.N.Y. 1993), *rev'd on other grounds*, 52 F.3d (2d Cir. 1994) (district court set aside jury verdict on grounds that epidemiological evidence combined with clinical evidence was insufficient as a matter of law to meet preponderance of evidence standard).

285. See *In re Joint Eastern and Southern Dist. Asbestos Litig.*, 827 F. Supp. at 1027, 1030.

286. See Poulter, *supra* note 264, at 231.

Epidemiological studies have recurrent biases and flaws, and because of their design of requiring a statistical evaluation by aggregation, they can obscure effects on individuals.²⁸⁷ Thus, when courts insist on epidemiological studies and reject other types of evidence on causation, such as animal studies or *in vitro* research, individuals who may have been harmed by a chemical are denied recovery in tort. In those cases, courts have told plaintiffs, in effect, there may be good evidence of a toxic effect due to chemical exposure, but because the effect is only seen in animals or *in vitro*, or have little to no probabilistic or statistical significance, it does not meet the causation element and no recovery is possible.²⁸⁸

Until 1993, the courts were divided as to the standard required for admitting expert scientific evidence.²⁸⁹ In 1993, the Supreme Court, in *Daubert v. Merrill Dow Pharmaceutical Co.*, articulated a new standard to govern the admissibility of scientific evidence.²⁹⁰ Rejecting the rigid "general acceptance" requirement established by the United States Court of Appeals for the District of Columbia Circuit in *Frye v. U.S.*,²⁹¹ the Supreme Court relied on Federal Rule of Evidence 702 (FRE 702).²⁹² Under *Daubert*, a trial judge, in considering the admissibility of scientific evidence, must first decide whether: 1) the evidence constitutes scientific knowledge; and 2) the evidence will have a valid connection to the issues in the case (will assist the trier of fact).²⁹³

287. See Troyen A. Brennan, *Causal Claims and Statistical Links: The Role of Scientific Uncertainty in Hazardous Substance Litigation*, 73 CORNELL L. REV. 469, 498 n. 155 (1988). See also Heidi L. Feldman, *Science and Uncertainty in Mass Exposure Litigation*, 74 TEX. L. REV. 1, 25 (1995).

288. See *Turpin v. Merrell Dow Pharms. Inc.*, 959 F.2d 1349, 1360 (6th Cir. 1992) (court rejects animal studies because they raise only a possibility rather than a probability of harm in humans); *Lynch v. Merrell-Nat'l Lab.*, 830 F.2d 1190, 1194 (1st Cir. 1987) (requiring confirmatory epidemiological evidence in addition to animal-studies evidence); *Hupp v. United States*, 563 F. Supp. 25, 30 (S.D. Ohio 1982) (only epidemiological studies could prove that a swine flu caused multiple sclerosis). *But see Ferebee v. Chevron Chem. Co.*, 736 F.2d 1529, 1535 (D.C. Cir. 1984) (holding a "cause effect relationship need not be clearly established by animal or epidemiological studies"); *Wells v. Ortho Pharm. Corp.*, 615 F. Supp. 262,266 (N.D. Ga. 1985) (the plaintiff's burden was not "to produce an unassailable scientific study on causation), *aff'd and modified in part*, 788 F.2d 741 (11th Cir. 1986).

289. See *United States v. Shorter*, 809 F.2d 54, 59-60 (D.C. Cir. 1987) (applying the "general acceptance standard"), *cert. denied*, 484 U.S. 817 (1987).

290. See *generally* *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993).

291. See *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923).

292. See *Daubert*, 509 U.S. at 588.

293. See *id.* at 590-91.

To be considered scientific knowledge, the Court said that the testimony must be derived from a scientific method.²⁹⁴ In analyzing whether a scientific method or technique is reliable the Court offered four non-exclusive, general observations that are designed to assist the trial judge.²⁹⁵ The general observations noted by the Court include: 1) whether a theory or technique “can (and has been) tested;”²⁹⁶ 2) whether it had been subjected to peer review and publication (although the publication or lack of publication in a peer review journal is not dispositive in considering the scientific validity);²⁹⁷ 3) its potential rate of error;²⁹⁸ and 4) whether the theory or technique is “generally accepted.”²⁹⁹ In outlining these criteria, the Court stressed that the methodology used was what should be considered in the evaluation of the reliability of the scientific knowledge, not the conclusion that the method generated.³⁰⁰ Further, the subject of scientific testimony does not have to be “known to a certainty.”³⁰¹ The only requirement is that the process used to derive the inference must be based on a scientific method.³⁰²

The Court used FRE 702 to state that to help determine the relevance of the testimony it must “assist the trier of fact to understand or to determine a fact or issue.”³⁰³ Thus, to determine the relevancy of the testimony the judge must ask whether the methodology or reasoning used by the scientific evidence is useful for resolving the issue in dispute.³⁰⁴ This is a more flexible approach than the “general acceptance” test under *Frye*.³⁰⁵ An epidemiological study under *Daubert* “is only probative if the correlation between the exposure and the disease supports an inference that exposure was more likely than not the cause of injury.”³⁰⁶ In addition, the Court recognized that evidence could be excluded by the

294. *See id.* at 595.

295. *See id.* at 592-94.

296. *Id.* at 592.

297. *See Daubert*, 509 U.S. at 592.

298. *See id.* at 594.

299. *Id.*

300. *See id.* at 595.

301. *Id.* at 590.

302. *See Daubert*, 509 U.S. at 595.

303. *Id.* at 589.

304. *See id.*

305. *Id.* at 594. In fact, the Supreme Court specifically stated that “the inquiry envisioned by Rule 702 is, we emphasize, a flexible one.” *Id.*

306. Jeffrey D. Cutler, *Comment: Implications of Strict Scrutiny of Scientific Evidence: Does Daubert Deal a Death Blow to Toxic Tort Plaintiffs?*, 10 ENVTL. L. & LITIG., 189, 200 (1995).

trial judge.³⁰⁷ Using summary judgment and judgment as a matter of law were “appropriate safeguards” when the plaintiff did not have sufficient evidence to present to a jury.³⁰⁸

Thus, in considering whether or not an epidemiological study is admissible, *Daubert* states that a plaintiff must only demonstrate scientific validity, as well as statistical significance.³⁰⁹ More importantly, however, *Daubert* does not preclude the admissibility of other evidence that may be relevant to the plaintiff's case, such as an expert medical opinion and toxicological studies.³¹⁰ This observation is an important consideration if a plaintiff is to prevail in a suit alleging harm from exposure to an endocrine disruptor. To date, there are no definitive human epidemiological studies to support a causal relationship between exposure to an endocrine disruptor and a disease, such as cancer, an immune deficiency, or other harm.³¹¹ However, there is increasing toxicological evidence in animal models that a link exists between exposure to these chemicals and a toxic response.³¹² Thus, the Supreme Court's decision in *Daubert* supports the call by various commentators urging the courts to adopt a preponderance of the available evidence standard to enable plaintiffs to prove causation in cases alleging toxic exposure.³¹³ By adopting a preponderance of the evidence standard, a plaintiff alleging harm due to exposure to endocrine disruptors would then be able to introduce animal toxicological studies and expert medical opinion in lieu of having substantial, reliable and consistent epidemiological studies.³¹⁴ Of course, the toxicological studies and medical opinions would have

307. See *Daubert*, 509 U.S. at 596. In so stating, the Supreme Court addressed the concern voiced by the lower courts that a lesser standard would open the floodgates in toxic substance litigation. The Court thus recognized that products liability is grounded in the premise that an individual should be compensated for damages to himself or his property. See *id.* at 597. Evidence may be excluded by a court if it does not have probative value or the expert testimony did not rely on facts and data that are reasonably relied on by experts in the particular field. See *id.* See also Carl F. Cranor et al., *Judicial Boundary Drawing and the Need for Context-Sensitive Science in Toxic Torts After Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 16 VA. ENVTL. L.J. 1, 4 (1996) (suggesting that those judges who follow *Daubert* are excluding too much evidence).

308. *Daubert*, 509 U.S. at 596.

309. See *id.* at 594.

310. See *id.* at 597. The Court stated “the scientific project is advanced by broad and wide-ranging consideration of a multitude of hypotheses, for those that are incorrect will eventually be shown to be so, and that in itself is an advance.” *Id.* at 597.

311. See COLBORN ET AL., *supra* note 5, at 122-41.

312. See COLBORN ET AL., *supra* note 5, at 122-41.

313. See Green, *supra* note 275, at 680. See also Mitts, *supra* note 280, at 208.

314. See Mitts, *supra* note 280, at 208.

to meet the standards for reliability and relevance set forth in *Daubert*.³¹⁵ If they did not meet these standards then the court would be free to use the judicial controls available to them.³¹⁶ As more than one commentator has noted, "imposing a heightened evidentiary threshold when the required scientific evidence may never be forthcoming, is contrary to judicial notions of fairness and social responsibility."³¹⁷ Furthermore, "[w]here epidemiological studies are lacking or inconclusive it is unjustifiable to exclude other toxicological evidence."³¹⁸ Thus, by adopting a preponderance of the available evidence standard the question would properly be placed before a jury. The jury would then decide whether, based upon the evidence before it, the chemical was a substantial factor in causing the harm claimed.³¹⁹ In other words, it is up to the jury to decide "whether that cause had such an effect in producing the harm as to lead reasonable men to regard it as the cause."³²⁰

A plaintiff alleging harm due to exposure to endocrine disruptors may still have a formidable task in proving causation. The EPA is currently working to develop a screening program to identify potential endocrine disruptors.³²¹ Many manufacturers do not test chemicals for toxic potential unless required by regulation.³²² If studies are conducted, they examine one substance rather than combinations,³²³ and do not investigate low dose and slow exposure.³²⁴ Additionally, the current regulatory environment does not support the development of data other than those

315. See *Daubert*, 509 U.S. at 590-91. But see *Daubert v. Merrell Dow Pharm. Inc.*, 43 F.3d 1311 (9th Cir. 1995). It should be noted that on remand the Texas Appellate Court, using the Supreme Court's opinion in *Daubert*, refined the test announced by the Supreme Court to enable them to maintain a restrictive approach in allowing novel scientific evidence. Thus, the approach advocated here would not be applicable for the Ninth Circuit.

316. See *Daubert*, 509 U.S. at 596-97.

317. Mitts, *supra* note 280, at 208. See also Green, *supra* note 275, at 681.

318. Green, *supra* note 275, at 681.

319. See Green, *supra* note 275, at 681. As noted by one commentator, the substantial factor test "acknowledges the fact that in the usual course, '[an] event without millions of causes is simply inconceivable; and the mere fact of causation, as distinguished from the nature and degree of the causal connection, can provide no clue of any kind to singling out those which are to be held legally responsible.'" *Id.*

320. Green, *supra* note 275, at 681.

321. See EDSTAC FINAL REPORT, *supra* note 18, at ES 1-15.

322. See Wendy E. Wagner, *Choosing Ignorance in the Manufacture of Toxic Products*, 82 CORNELL L. REV. 773, 784-90 (1997).

323. See COLBORN ET AL., *supra* note 5, at 220.

324. See Maurice Zeeman, *Our Fate is Connected with the Animals*, 46 BIOSCIENCE 542, 544 (1996).

examining the risk of cancer.³²⁵ However, the body of evidence implicating these substances in causing harm is growing.³²⁶ For example, products containing PVCs are made with chemicals called phthalates.³²⁷ Most recently, phthalates have been found in blood stored in intravenous (IV) bags made from PVCs.³²⁸ Animal studies suggest that phthalates can damage the heart, kidney, liver, and testicles, and may cause cancer.³²⁹ While PVC manufacturers maintain that their products are safe,³³⁰ at least one PVC maker, Abbott Laboratories, has admitted there are “too little data to draw hard conclusions.”³³¹ Abbott Laboratories has included a warning to that effect with some of its IV bags.³³²

VI. Conclusions

Our Stolen Future has raised concerns about the exposure of individuals to chemicals that are capable of disrupting the endocrine system and causing harm.³³³

This paper has examined the issue of whether potential plaintiffs could successfully pursue a products liability claim. Courts have been willing to dispense with traditional causation tests to allow victims of DES-related injuries to recover without having to prove who manufactured the product that caused their injury.³³⁴ The approaches may have differed with respect to what the plaintiffs must prove for recovery, but in all of these cases the burden of proving causation shifted to the defendants.³³⁵ For a successful

325. See COLBORN ET AL., *supra* note 5, at 221. As noted by these authors the FDA, through the Delaney Clause, bans food additives that cause cancer in any other animal species. See 21 U.S.C. § 348(c)(A) (1998). Other adverse effects, such as immunotoxic or effects on reproduction, are regulated on a risk-benefit approach or not at all.

326. See COLBORN ET AL., *supra* note 5, at 122-41.

327. See Jeffrey Kluger, *Poisonous Plastics?*, TIME, Mar. 1, 1999, at 28.

328. See *id.*

329. See *id.*

330. See *id.* As noted in this article, PVC manufacturers and other manufacturers of chemicals added to food, drugs and cosmetics, as well as to products that contact food, drugs and cosmetics (such as plastics and can coatings) argue that animal toxicity studies show toxic effects at levels much higher than a human would ever absorb. However, these tests do not take into account the potential synergistic effects of these chemicals. Also, in at least one experiment, rats were given low levels of PVC doses and still exhibited adverse effects.

331. *Id.*

332. See *id.*

333. See COLBORN ET AL., *supra* note 5, at 122-41. See also EDSTAC FINAL REPORT, *supra* note 18, at ES-1-15.

334. See *Sindell*, 607 P.2d at 937.

335. See *id.*; *Anderson*, 338 A.2d at 1; *Abel*, 280 N.W.2d at 22.

products liability claim for exposure to an endocrine disruptor, the plaintiffs would be required to prove that they have sustained an injury, and that the injury was caused by exposure to a chemical causing endocrine disruption.³³⁶ It would also be necessary to show that the defendants produced or intentionally added these substances to the product.³³⁷ The burden of proof would then shift to the defendants.³³⁸ The defendants could either disprove the claims or establish that the particular substance could not have caused the injuries.³³⁹

Plaintiffs hoping to prevail in a lawsuit alleging products liability face numerous hurdles in bringing a successful suit. The most formidable challenge is to prove the injury was due to exposure to the endocrine disrupting chemical. Currently, EPA and the FDA are establishing a screening program for elucidating whether a chemical is an endocrine disruptor.³⁴⁰ Until this testing is validated and performed, the issue surrounding potential products liability actions with regard to endocrine disruptors leaves plaintiffs to rely on existing animal toxicological data and limited epidemiological studies.³⁴¹ Until *Daubert*, the courts were reluctant to use animal toxicity data in the absence of more definitive epidemiology studies.³⁴² However, *Daubert*³⁴³ and the Restatement (Third) of Torts³⁴⁴ offer potential mechanisms by which plaintiffs may be able to prevail by a preponderance of the evidence standard. Those studies will come down to a battle of the experts and a sympathetic jury. The issue surrounding potential products liability action with regard to endocrine disruptors is also confounded by a skepticism about toxic risks: "Innocent until proven guilty may sound fine in theory, but it lets the bodies pile up before the truth gets written."³⁴⁵ Many manufacturers do not test chemicals for toxic potential unless required by regulation.³⁴⁶ Even if studies are conducted, manufacturers often examine one substance rather than combinations,³⁴⁷ or do not investigate low

336. See *Sindell*, 607 P.2d at 937; *Anderson*, 338 A.2d at 1; *Abel*, 280 N.W.2d at 22.

337. See *Sindell*, 607 P.2d at 937; *Anderson*, 338 A.2d at 1; *Abel*, 280 N.W.2d at 22.

338. See *Sindell*, 607 P.2d at 937; *Anderson*, 338 A.2d at 1; *Abel*, 280 N.W.2d at 22.

339. See *Sindell*, 607 P.2d at 937; *Anderson*, 338 A.2d at 1; *Abel*, 280 N.W.2d at 22.

340. See EDSTAC FINAL REPORT, *supra* note 18, at ES-1-15.

341. See COLBORN ET AL., *supra* note 5, at 110-21.

342. See *Daubert*, 509 U.S. at 589.

343. See *id.*

344. See RESTATEMENT (THIRD) OF TORTS: PRODUCTS LIABILITY (1998).

345. Mark Hertsgaard, *Benefit of the Doubts*, 10 NATION (July 8, 1996).

346. See Wagner, *supra* note 322, at 784-90.

347. See COLBORN ET AL., *supra* note 5, at 220.

dose and exposure over time.³⁴⁸ Thus, the plaintiff may need to bear the burden of having tests and studies conducted. Subsequent plaintiffs, however, will have the data available to them. As more is learned about the characteristics of endocrine disruptors, plaintiffs' expenditures to collect data for proof will decrease.

At the same time, the proposal is not unfair to the defendants who, by producing and inadequately testing a fungible item, have created a situation where innocent plaintiffs are harmed and are unable to trace the injury-causing substance back to its source.³⁴⁹ The defendants are given the chance to show they did not cause the harm.³⁵⁰ Lessening the plaintiff's burden of proof is justified.

This paper has examined the current issue of endocrine disrupting chemicals and has explored various legal theories that might be pursued in order for a plaintiff to sustain a successful suit in products liability. The Supreme Court's holding in *Daubert*,³⁵¹ as well as the Restatement (Third) of Torts,³⁵² have afforded plaintiffs the possibility that such an action will be sustained. The courts must now recognize the theories of causation that, traditionally, they were reluctant to recognize.

348. See Zeeman, *supra* note 324, at 544.

349. See Kanner, *supra* note 244, at 733.

350. See Kanner, *supra* note 244, at 733.

351. See *Daubert*, 509 U.S. at 590.

352. See RESTATEMENT (THIRD) OF TORTS: PRODUCTS LIABILITY (1998).