Blocked Pathways: Potential Legal Responses to Endocrine Disrupting Chemicals

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I. INTRODUCTION

Recent scientific research indicates that synthetic chemicals may disrupt the human endocrine system, possibly causing decreased fertility, malformed reproductive organs, increased levels of cancer in reproductive organs, impaired fetal development, and neurological, thyroid, and immune disorders.[1] These endocrine disrupting chemicals (EDCs), which mimic, block, or otherwise interfere with normal hormonal signals, are found in pesticides, plastics, detergents, industrial materials, drinking water, and other substances.[2] Over fifty different chemicals may have endocrine disrupting effects,[3] and these chemicals permeate our environment. We may be exposed to them through the products we use, the food we eat, the water we drink, and the air we breath.

Inquiry into endocrine disruption is relatively recent, and scientists still do not have a full understanding of the mechanisms through which EDCs may harm human health. Some of the suspected links between EDCs and health and reproductive disorders may turn out to be false, but if the central hypothesis is confirmed that synthetic chemicals are disrupting hormonal pathways, EDCs could be among the most persistent, widespread, and damaging toxins to which humans have been exposed. Reflecting on the potential danger and current scientific uncertainties, the authors of *Our Stolen Future*,[4] a 1996 book on endocrine

1. *See* 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, 715, 720 (1996).

2. *See* Jorma Toppari et al., *Male Reproductive Health and Environmental Xenoestrogens*, 104 ENVTL. HEALTH PERSPECTIVES, Supplement 4, 741, 758 (1996).

3. *See* THEO COLBORN ET AL., OUR STOLEN FUTURE: ARE WE THREATENING OUR FERTILITY, INTELLIGENCE AND SURVIVAL? – A SCIENTIFIC DETECTIVE STORY 81 (1996). For a list of suspected endocrine disruptors, *see* Known Enodcrine Disruptors (last modified May 6, 1996) <www.vcu.edu/cesweb/ed/disruptors.html>. Some of the suspected endocrine disruptors, such as DDT and PCBs, are no longer produced in the United States, but they persist in the environment and are believed to be significant contributors to EDC risks. See Part III, *infra*.

4. OUR STOLEN FUTURE was aimed at a lay audience and contained a foreword by

disruption, warn that "humans do appear to be gambling with their ability to reproduce over the long term. . . ."[5]

Although there is a clear need for further research into EDCs, the legal and regulatory communities should begin to consider the types of responses that may be appropriate if the scientific evidence grows stronger. Very little legal literature has been produced on EDCs, however, and most academic and governmental discussion of EDCs has focused on understanding their health risks and on identifying new avenues for research. As Mary O'Brian, a toxics and risk assessment specialist, notes, those "trained in law . . . must supply ideas for litigation and legislation that are equal to the problem."[6]

This article describes and assesses potential legal responses to EDCs, focusing on regulation and litigation, and concludes that existing legal tools will probably be inadequate to respond to EDC risks. The main obstacle to legal action is scientific uncertainty, which has frequently posed challenges for our legal system in contexts such as environmental regulation and toxic tort litigation. Scientific uncertainty is likely to plague the risk assessment process for EDCs, delaying potentially effective regulations even though existing statutes provide sufficient authority to regulate EDCs.[7] In the litigation context, scientific uncertainty will make it difficult to connect a given EDC to a given harm.[8] The usual problems with bringing a toxic tort suit, such as long latency periods, proof of causation, and identification of defendants, would be magnified many-fold. Although scientific understanding of EDCs is likely to increase over the next few years, remaining uncertainties can probably be used as a shield to block regulatory efforts and

Vice President Al Gore, who compared the book to Rachel Carson's Silent Spring. *Id.* at vii. For examples of media reports about the book and endocrine disruption generally, *see* Michael Lemonick, *What's Wrong with Our Sperm?* TIME, March 18, 1996, at 78; Sharon Begley, *The Great Impostors*, NEWSWEEK, March 18, 1996, at 48; John Carey, *A Scary Warning – or Scare Story?* BUSINESS WEEK, April 8, 1996, at 18; Rick Weiss & Gary Lee, *Pollution's Effect on Human Hormones: When Fear Exceeds Evidence*, WASH. POST, March 31, 1996, at A14.

5. COLBORN ET AL., *supra* note 3, at 234.

6. Mary O'Brian, *Our Current Toxics Use Framework, Our Stolen Future, and Our Options*, 11 J. ENVTL. L. & LIT. 331, 332 (1996).

7. *See infra* Part IV.

8. *See infra* Part V.

claims by tort litigants. In short, we may be facing a significant new toxic risk with few legal tools available to protect public health.

This article acknowledges the preliminary nature of scientific investigation of EDCs. It undertakes an examination of potential legal responses not to urge immediate legal action, but to explore options so that the scientific and legal communities can begin to share ideas as research progresses. Because EDCs have unusual toxicological characteristics, current risk assessment techniques and regulatory tools may be inadequate. Fundamentally new approaches may need to be developed if the United States is to respond effectively to EDC risks. In the near term, a consideration of the strengths and weaknesses of existing legal and regulatory options can help to identify more effective approaches to EDCs.

Part II of this article provides an overview of EDCs and the current state of scientific knowledge regarding their health effects. The main categories and uses of EDCs are outlined in Part III. Part IV explores the adequacy of current toxic chemical statutes and regulations for addressing EDC risks, and Part V examines the prospects for litigation over EDCs. Finally, Part VI briefly outlines some promising new approaches to EDCs that could be warranted if the scientific case against EDCs continues to strengthen.

II. OVERVIEW OF ENDOCRINE DISRUPTION

A. The Endocrine System

The endocrine system, along with the nervous system and the immune system, is one of the three main integrating and regulatory mechanisms in the human body.[9] The endocrine system is composed of hormone-secreting glands such as the pituitary, hypothalamus, testicles, or ovaries; over 50 different chemical hormones that travel through the bloodstream, such as estrogen or testosterone; and hor-

9. *See* RISK ASSESSMENT FORUM TECHNICAL PANEL, UNITED STATES ENVIRONMENTAL PROTECTION AGENCY, EPA/630/R-96/012, SPECIAL REPORT ON ENDOCRINE DISRUPTION: AN EFFECTS ASSESSMENT AND ANALYSIS 2 (1997) [hereinafter EPA RISK ASSESSMENT FORUM]. <www.epa.gov/ORD/WebPubs/endocrine/endocrine.pdf>.

mone receptors located throughout the body.[10] Hormones regulate many key bodily functions, including blood sugar, ovulation, pregnancy, and development.[11] They act by binding with protein receptors in locations such as the brain, glands, or reproductive organs to change cell activity, including the activation of strands of DNA to express genes.[12] Hormones can act in extremely low doses, as low as parts-per-trillion concentrations in the blood stream.[13]

Endocrine disrupting chemicals can interfere with normal hormonal functioning in several ways. They can disrupt the synthesis, storage, release, transport, and clearance of hormones, and they can disturb receptor recognition, receptor binding, or post-receptor responses within cells.[14] Some EDCs are hormone mimickers that bind to protein receptors in place of the natural hormone. Some enhance (an agonistic effect) or inhibit (an antagonistic effect) the action of hormones.[15] Other EDCs may directly affect the overall production of certain hormones in the body.[16] Much of the scientific research into EDCs has focused on estrogenic compounds: chemicals that mimic estrogen.[17]

B. Animal Studies on Endocrine Disruption

Several animal studies have linked exposure to EDCs to reproductive and developmental problems, as well as to cancer.[18] While inferring human health risks from animal studies has often been controversial, the EPA has noted that the hypothesis that humans are being harmed by EDCs "is supported by observations of similar effects in aquatic and wildlife species. In other words, a common

10. *See* ENDOCRINE DISRUPTOR SCREENING AND TESTING ADVISORY COMM., UNITED STATES ENVIRONMENTAL PROTECTION AGENCY, FINAL REPORT 2-1 (1998) (last modified Jan. 19, 1999) <http://www.epa.gov/opptintr/opptendo> [hereinafter EDSTAC REPORT].

11. *See id.*

12. *See id.* at 2-2 (explaining that as many as 50 to 100 genes in a cell may be controlled by the binding of a single type of hormone to the receptors in the cell).

13. *See* COLBORN ET AL., *supra* note 3, at 74.

14. *See* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 14. *See also* Kavlock, *supra* note 1, at 721.

15. *See* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 13.

16. *See id.* at 14.

17. *See* COLBORN ET AL., *supra* note 3, at 84-85.

18. *See generally id.*, chapter 10 at 167. *See also* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 54-78.

theme runs through both human and wildlife reports."[19]

Alligators in Lake Apopka, Florida, provide one of the most well-known examples of reproductive problems that may be linked to endocrine disruption. The alligators were exposed to a mixture of DDT,[20] DDE,[21] dicofol, chlorobenzilate, and dicholorobenzophenone from a 1980 chemical spill.[22] These suspected EDCs have among them both estrogenic and anti-androgenic (male hormone blocking) properties.[23] Scientists found that the male alligators were demasculinized, with penises one half to one-fourth normal size.[24] Male hatchlings were found to have a ratio of estradiol (a form of estrogen) to testosterone of four times the normal male ratio,[25] and female hatchlings were "superfeminized," with an estradiol/testosterone ratio twice as high as normal.[26] There were severe hatching problems among the alligators: only 5% to 20% of the eggs in each examined nest hatched, compared to a normal hatching rate of 65% to 80%.[27]

Rodent studies have also linked EDCs to developmental and reproductive disorders.[28] The EPA has concluded that "[c]onvincing evidence exists in rodents that exposure to chemicals that have estrogenic activity, reduce androgen levels, or otherwise interfere with the action of androgen during development can cause male reproductive system abnormalities that include reduced sperm production capability and reproductive tract abnormalities."[29] One rodent study involved administering vinclozolin, a fungicide, to pregnant rats.[30] Scientists found that male offspring had impaired penis development, existence of vaginal pouches, prostate gland problems, and reduced or absent sperm pro-

19. *Id.* at 5.
20. Dichlorodiphenyltrichloroethane.
21. Bis(4-chlorophenyl)-1,1dichloroethene. DDE is a DDT metabolite (breakdown product).
22. See EPA RISK ASSESSMENT FORUM, *supra* note 9, at 65-66 for discussion of the Lake Apopka studies.
23. *See* Toppari et al., *supra* note 2, at 756.
24. *See* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 66.
25. *See id.*
26. *Id.*
27. *See id.*
28. *See id.* at 7-8.
29. *Id.* at 3.
30. *See id.* at 35.

duction – all characteristic of interference with the receptors for male hormones.[31]

Scientists have conducted numerous other laboratory and epidemiological studies on EDCs' effects on rodents, birds, fish, seals, and other animals.[32] The research is too abundant to discuss in detail here. The EPA has summarized this animal research, however, by stating that "numerous reports indicate a variety of compounds can modulate the endocrine system and affect reproduction and development in invertebrates, fish, and wildlife . . . ."[33]

C. Health Effects of Endocrine Disruption in Humans

While the data from animal studies are suggestive, the impact of EDCs on human health and reproduction is of course the more important issue from a legal perspective. EDCs have been associated in scientific literature with a wide variety of human health problems, including reduced fertility, birth defects, cancer, endometriosis (a disease of the uterus), malformed reproductive organs, glandular dysfunction, and neurological disorders.[34] The evidence on human health risks from EDCs has been controversial, and the studies are not conclusive, but the research conducted to date offers disturbing warnings that EDCs may pose a hazard to human health.

One of the most widely publicized human health effects hypothesized to be caused by EDCs is a reduced sperm count. Some scientists believe that adult sperm counts may be lowered by exposure of male fetuses to synthetic estrogenic chemicals in the womb.[35] A 1992 article in the *British Medical Journal* reviewed sixty-one studies involving almost fifteen thousand men in over a dozen countries and concluded that sperm counts had dropped forty-five percent

31. *See id.*
32. *See id.* at 56-77.
33. *Id.* at 8-9.
34. *See generally id.* at 21-54.
35. *See id.* at 36. *See also* Elisabeth Carlsen et al., *Declining Semen Quality and Increasing Incidence of Testicular Cancer: Is There a Common Cause?*, 103 ENVTL. HEALTH PERSPECTIVES, Supplement 7, 137-138 (1995). (Discussing possible causal biological mechanisms and concluding that "[t]he physiological basis for a possible role of estrogens in male reproductive dysfunction seems to be feasible," but that "[i]t should be remembered that the estrogen hypothesis remains to be tested." *Id.* at 138.)

from 1938 to 1990.[36] Older men (born in decades when there were fewer synthetic chemicals) appear to have higher sperm counts than younger men, "lend[ing] support to the concept that adverse prenatal factors may influence the sperm production capacity in adult life."[37] Other researchers have criticized the methodology of the sperm count studies or have found no decline in sperm counts in independent tests,[38] but the most recent U.S. study found that sperm counts in the United States have declined by an average rate of about 1.5% per year over the past five decades.[39]

Some scientists believe endocrine disruption has contributed to increasing rates of cancer of the testicles and prostate gland.[40] Both tissues are hormone-sensitive. The reported incidence of testicular cancer in the United States increased 45.4% between 1973 and 1995,[41] and testicular cancer is now the most common cancer among men ages 25 to 34.[42] The reported incidence of prostate cancer increased 119.6% between 1973 and 1995.[43]

Summarizing the data on male reproductive disorders, the Danish scientist Jorma Toppari concludes that "[a]ll of the best evidence available points with some certainty to a rising tide in Europe and many other countries of human male reproductive disorders involving sperm counts (and probably sperm quality), testicular cancer, malformation of

36. *See* COLBORN ET AL., *supra* note 3, at 172-173. *See also* Toppari et al., *supra* note 2, at 742 (also discussing the original study).

37. Toppari et al., *supra* note 2, at 743.

38. *See* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 36-37. *See also*, Stephen H. Safe, *Environmental and Dietary Estrogens and Human Health: Is There a Problem?*, 103 ENVTL. HEALTH PERSPECTIVES 346, 347 (1995). (Noting that this hypothesis was not based on experimental measurements of increased levels of estrogenic compounds in men and discussing reports that reevaluated data to dispute declines in sperm count).

39. *See* Andrew Boswer, *Decline in Sperm Density May be Even Worse Than Reported*, UROLOGY TIMES, February 1998, at 1 (discussing California Department of Health Services Study).

40. *See* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 39, 42.

41. *See* Ries et al., National Cancer Institute, *SEER Cancer Statistics Review, 1973-1995* Table XXIV-1 (1998) (last modified January 21, 1999) <http://www.seer.ims.nci.nih.gov/Publications/CSR7395>.

42. *See* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 39. *See also* Toppari et al., *supra* note 2, at 743.

43. *See* Ries et al., *supra* note 41, at Table XXII-1.

the external genitalia, and possibly testicular maldescent."[44] Toppari asserts that "[a]ll of the described changes in male reproductive health appear interrelated and may have a common origin in fetal life or childhood,"[45] but ultimately determines that "[t]here are insufficient data to prove or disprove that these adverse changes in male reproductive health are the result, wholly or partially, of exposure to environmental estrogens."[46]

The evidence of adverse health effects of EDCs in adult women has been controversial. Some scientists have linked PCBs and dioxin, both suspected EDCs, to endometriosis, but later studies appear to refute this hypothesis.[47] Other studies have linked chlorinated organic compounds such as DDT and PCBs, which exhibit weak estrogenicity, to an increased risk of breast cancer,[48] but these studies have been criticized and other evidence is conflicting.[49] There is scientific consensus, however, on the basic point that a "causal relationship [exists] between female breast cancer and hormonal activity" in general.[50] Reported cases of breast cancer increased 25.2% between 1973 and 1995,[51] and the most notable increase has been in postmenopausal women with estrogen-responsive tumors, that is, tumors that grow when exposed to estrogen or estrogenic chemicals.[52] Numerous studies have found a link between rodent exposure to EDCs and disruption of the timing of menstrual cycles, the development of ovarian follicles, and the speed of embryo transport through fallopian tubes, all of which affect female rat fertility.[53] There is no data on whether human females are experiencing reduced fertility

44. Toppari et al., *supra* note 2, at 764.
45. *Id.* at 768.
46. *Id.* at 764.
47. *See* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 28-29.
48. *See* Wolff, M.S., *Pesticides – How Research has Succeeded and Failed in Informing Policy: DDT and the Link with Breast Cancer*, 103 ENVT'L. HEALTH PERSPECTIVES, Supplement 6, 87-91 (1995) (discussing four recent small studies suggesting link between breast cancer and organochlorine levels in the body and noting that much further research is necessary).
49. *See* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 30. *See also* Safe, *supra* note 38, at 346-347 (discussing studies that dispute the association).
50. Kavlock et al., *supra* note 1, at 718.
51. *See* Ries et al., *supra* note 41, at Table II-1.
52. *See* COLBORN ET AL., *supra* note 3, at 182-183.
53. *See* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 22-25.

due to EDCs, however. A recent study found that girls are entering puberty at an increasingly early age and suggested that EDCs, in mimicking natural hormones, may be a cause.[54]

The greatest health risks from EDCs are probably not to adult males or females, but to human fetuses exposed *in utero* to EDCs taken into the mother's body. Fetuses are particularly vulnerable to EDCs because development and sexual differentiation depend on low-dose hormonal signals received at precise times during gestation. The formation of the testicles in the male fetus around the seventh week of gestation and their first release of testosterone are the key steps in male sexual differentiation,[55] as prior to that time fetuses have unisex gonads.[56] The EPA states that "[t]he development of the male reproductive system pre- and postnatally appears to be particularly susceptible [to hormone disruption] and uniquely sensitive."[57] Problems in sexual differentiation, such as undescended testicles, incomplete penis development, or the presence of rudimentary components of the female reproductive tract, could occur when the hormonal signals in genetic male fetuses are disturbed by exogenous chemicals.[58]

D. DES as a Scientific Parallel for EDCs

The best data on the possible effects of EDCs on male and female fetuses come from the sons and daughters of women who took diethylstilbestrol (DES) during pregnancy. A synthetic estrogen that also has anti-androgenic properties,[59] DES was given to 4.8 million women in the United States from the late 1940's to the early 1970's to prevent

54. *See* Marcia E. Herman-Giddens et al., *Secondary Sexual Characteristics and Menses in Young Girls Seen in Office Practice: A Study from the Pediatric Research in Office Settings Network*, 99 PEDIATRICS 505 (1997) (study of 17,077 girls finding that 48.3% of African-American girls and 14.7% of white girls had begun breast and/or pubic hair development by age 8). This contrasts with earlier studies showing development at later ages. The authors urged investigation into whether the early onset of puberty is related to increasing use of plastics and pesticides that have estrogen-related physiological effects. *Id.* at 511.

55. *See* COLBORN ET AL., *supra* note 3, at 43-44.

56. *See id.* at 42.

57. EPA RISK ASSESSMENT FORUM, *supra* note 9, at 33.

58. *See id.* at 33-34.

59. *See id.* at 38.

miscarriages and other pregnancy complications.[60] The daughters of women who took DES exhibited increased rates of a rare form of vaginal cancer called clear-cell adenocarcinoma,[61] and also showed increased rates of adenosis (abnormal vaginal or cervical growths), T-shaped uteri, damaged cervixes, miscarriages; and ectopic pregnancies.[62] The DES daughters represent the strongest evidence to date that maternal exposures to synthetic estrogen-mimicking chemicals can lead to cancer and reproductive disorders in offspring.

DES has also caused harmful effects in male offspring. Two controlled studies of DES-exposed sons showed increased incidences of genital malformation, testicular disorders, and smaller-than-average penises,[63] all consistent with *in utero* exposure to an estrogenic chemical during development of male sexual characteristics. Overall reproductive tract abnormality in the DES sons was 32%, versus 8% in controls,[64] and sperm count in the DES-exposed sons was 79% that of the non-exposed control group.[65] Cryptorchidism (undescended testicles) has also been observed to occur more frequently in DES-exposed sons.[66]

Starting in the mid-1970's, DES-exposed children began to file lawsuits against DES manufacturers seeking damages for cancer and reproductive abnormalities. Just as the DES experience has provided the scientific community with the best available data on the effects of endocrine disrupting chemicals on fetal development and reproductive abilities of offspring, it also provides the legal community with the principal precedents for intergenerational lawsuits involving endocrine disruption. The DES litigation is discussed in more detail below.[67]

60. *See* Toppari et al., *supra* note 2, at 753.
61. *See* Tracey I. Batt, Note, *DES Third-Generation Liability: A Proximate Cause*, 18 CARDOZO L. REV. 1217, 1221 (1996).
62. *See id.*
63. *See* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 38.
64. *See id.*
65. *See id.*
66. *See* Toppari et al., *supra* note 2, at 754.
67. *See infra* Part V(B).

E. Implications of the Scientific Research

Because scientific inquiry into endocrine disruption is relatively recent, it is not yet possible to demonstrate a causal link between a particular EDC and a given health or reproductive disorder. Numerous associational links have been established, however, and the rise in breast, prostate, and testicular cancer, as well the increasing rate of undescended testicles and declining sperm counts, are cause for concern. The EPA explains that "[w]ith few exceptions (e.g., DES), a causal relationship between exposure to a specific environmental agent and an adverse effect on human health operating via an endocrine disruption mechanism has not been established."[68] Even *Our Stolen Future* acknowledges that "[i]t will never be possible to establish a definitive cause-and-effect connection with [endocrine disrupting] contaminants in the environment."[69]

The absence of conclusive causal links, however, does not mean that the legal and regulatory communities should ignore the dangers of EDCs. Although further research is necessary to understand EDC risks, exposure routes, and mechanisms of harm, the studies linking EDCs to reproductive disorders and other health problems in animals and humans suggest that precautionary legal responses should at least be considered. Though the health effects of EDCs seem to be more subtle than those of DES, the DES experience should serve as a warning of the damage that EDCs may be causing to the human population.

If the scientific case against EDCs grows stronger over the next few years, courts and/or regulatory agencies may respond even if they do not have conclusive evidence of cause-and-effect relationships. Certainly, tort law has never required absolute proof of causation in toxics cases,[70]

68. EPA RISK ASSESSMENT FORUM, *supra* note 9, at 6.

69. COLBORN ET AL., *supra* note 3, at 196. Elaborating on causation problems, Colborn and her colleagues explain that "[a]lthough we know that every mother in the past half century has carried a load of synthetic chemicals and exposed her children in the womb, we do not know what combination of chemicals any individual child was exposed to, or at what levels, or whether he or she was hit during critical periods in their development when relatively low levels might have significant lifelong effects." *Id.*

70. *See infra* Part V(A) for a discussion of causation in toxic tort cases generally and in potential EDC litigation in particular.

and regulatory agencies should require an even lower standard of proof than the courts before considering precautionary regulation of environmental pollutants.[71]

Courts and regulatory agencies have frequently faced the challenge of evaluating new risks about which there is scientific controversy. This challenge arises because technology evolves at a rapid pace, much faster than the law, and continually creates new risks along with its benefits. Some observers advocate caution in crafting legal responses to new or newly-identified risks, advising restraint until the data are more conclusive or until there is greater scientific consensus. Regarding EDCs, for example, John Holtzman of the Chemical Manufacturers Association maintains that "[l]eap[ing] from theory to public policy is pretty risky when there are multiple interpretations of what the data mean."[72] Peter Huber, a leading critic of the U.S. regulatory system, has suggested that the law overreacts to new risks and disregards the hazards of older, established technologies.[73] Judge Posner's dictum that "[l]aw lags science; it does not lead it"[74] rightfully cautions against overzealous legal responses to new risks, especially from the courts.

Protection of public health, however, often requires action in the face of uncertainty. Law should not lead science, but neither should government officials ignore clear danger signals. This article acknowledges the preliminary nature of scientific findings regarding endocrine disruption and examines the *potential* for regulatory action and litigation in the context of scientific understanding becoming stronger

71. Compared to tort judgments, regulation is more appropriate to address uncertain risks and causal mechanisms. For example, regulations might impose minimal restrictions on uses of a chemical when there is some evidence, but not conclusive evidence, that the chemical causes harm. Tort law's remedy of fully shifting injury costs should be imposed only when there is more substantial proof of causation.

72. Cynthia Crossen, *Clamorous Pro and Con Campaigns Herald Book's Launch*, WALL STREET JOURNAL, March 7, 1996 at B1.

73. Peter Huber, *Safety and the Second Best: The Hazards of Public Risk Management in the Courts*, 85 COLUM. L.REV. 277, 307-314 (1985) (arguing that courts focus on new risks from new technology, ignoring potentially greater risks from existing, accepted technology).

74. Rosen v. Ciba-Geigy Corp., 78 F.3d 316, 319 (7th Cir. 1996), *cert. denied*, 117 S.Ct. 73 (1996). (Ruling that expert scientific testimony on link between nicotine patch and heart attack was not valid scientific evidence and inadmissible).

over the next several years. Many observers of the endocrine disruption issue believe that the present state of scientific knowledge is too limited to support a legal response. Because the research conducted to date raises serious questions about the risks of EDCs, however, it is appropriate to examine the legal and regulatory tools that might be used to respond to EDC risks should the scientific case against EDCs continue to strengthen.

III. TYPES OF SUSPECTED EDCS

Since World War II, society has witnessed a "chemical revolution" in which the production and use of synthetic chemicals has increased dramatically. Although we have undeniably benefited from new chemicals in areas such as medicine, agriculture, and packaging, many chemicals are known carcinogens and others may be hazardous but have not been adequately tested.

The overwhelming majority of chemicals in use in the United States have never been required to be tested for health risks. While our primary toxics statute, the Toxic Substances Control Act (TSCA) of 1976,[75] permits the EPA to mandate testing of chemicals, it has been largely ineffective because it creates burdensome factual and legal hurdles for the EPA before the agency can take regulatory action.[76] Since the passage of the Act, EPA has developed testing rules for less than one percent[77] of the 75,000 synthetic chemicals currently listed on EPA's Toxic Substances Inventory.[78] Even when a chemical is tested pursuant to TSCA, it is the manufacturer, and not the EPA, that performs the actual testing. We are essentially "flying blind"[79] in permitting the marketing and use of thousands of chemicals with very little information about their health impacts.

It is partly because so few chemicals are required to be tested that scientists are just beginning to understand the properties and risks of EDCs. Over fifty chemicals are sus-

75. 15 U.S.C. §§ 2601-2692 (1994).

76. *See infra* Part IV for a further discussion of TSCA.

77. DAVID ROE ET AL., ENVIRONMENTAL DEFENSE FUND, TOXIC IGNORANCE 26 (1997). <http://*www.edf.org/pubs/reports/toxicignorance/index.html*>.

78. *See* EDSTAC REPORT, *supra* note 10, at 2-10.

79. COLBORN ET AL., *supra* note 3, at 239; *see generally id.*, chapter 14.

pected of being EDCs,[80] though estimates vary and identification research is still ongoing. Much of the research into endocrine disruption has focused on a few classes of chemicals, such as PCBs or DDT, which scientists have already studied for other reasons.[81] The main classes of EDCs are described below:

Organochlorine Pesticides: This category includes DDT, DDE, and dicofol, as well as the pesticides perthane, aldrin, chlordane, heptachlor, and hexachlorobenzene.[82] The pesticides dieldrin, endosulfan, methoxychlor, and kepone are also suspected EDCs.[83] While the EPA severely restricted use of DDT in 1972,[84] other countries still produce and use it. DDT has a chemical structure similar to DES,[85] and DDT persists in the U.S. environment because it and its break-down products accumulate in wildlife[86] and stay in the foodchain.[87] One DDT break-down product, DDE, has been found to be an androgen receptor antagonist, blocking the binding of natural male hormones to their receptors.[88] DDE can cross the placenta to reach developing human fetuses and has been shown to inhibit male sexual characteristics in rats.[89] Other organochlorine pesticides still used on crops have both estrogenic and anti-androgenic effects,[90] and some studies have suggested a link between body levels of organochlorine pesticides and breast cancer.[91]

80. *See* COLBORN ET AL., *supra* note 3, at 81.
81. *See id.*
82. *See* Toppari et al., *supra* note 2, at 756.
83. *See* Thomas E. Weise & William R. Kelce, *An Introduction to Environmental Oestrogens*, CHEMISTRY AND INDUSTRY, August 18, 1997.
84. 37 Fed. Reg. 13,369 (1972).
85. *See* COLBORN ET AL., *supra* note 3, at 69 (diagramming structures).
86. *See* Richard L. Williamson et al., *Gathering Danger: The Urgent Need to Regulate Toxic Substances That Can Bioaccumulate*, 20 ECOLOGY L.Q. 605, 609-613 (1993) (listing organochlorine pesticides and PCBs as substances with a "high" or "very high" capacity to accumulate in the fatty lipids of wildlife as the chemicals are passed up the food chain).
87. *See id.* at 609, 612.
88. *See* Kelce et al., *Persistent DDT Metabolite p,p'-DDE is a Potent Androgen Receptor Antagonist*, 375 NATURE 581 (June 15, 1995).
89. *See id.*
90. *See* Toppari et al., *supra* note 2, at 756-757.
91. *See* Wolff, *supra* note 48, at 88-89.

Polychlorinated Biphenyls (PCBs): PCBs are industrial chemicals that have been used since 1929 as heat transfer and hydraulic fluids, adhesives, flame retardants, dielectric fluids for electrical equipment, and waxes.[92] Congress banned most production of PCBs in 1976,[93] but like DDT, PCBs persist in the environment and in the food chain, especially in fish that live in polluted waters.[94] PCBs have both estrogenic and anti-androgenic effects.[95] Dioxin, a form of PCB whose cancer-causing properties were brought to public attention in the early 1980's by the contamination of Times Beach, Missouri, is also a suspected EDC.[96] Dioxin is produced as a byproduct of incineration, paper and pulp bleaching, and emissions from steel foundries and motor vehicles.[97]

Alkylphenol Ethoxylates (APEs): APEs, which are estrogenic, are used widely in detergents, paints, herbicides, pesticides, and cosmetics.[98] Over 300 million kilograms of APEs are produced annually worldwide.[99] APEs accumulate in rivers, entering the water directly from fields or through sewage treatment plants, and they also accumulate in the bodies of fish and birds.[100] British studies have indicated that thirty percent of drinking water in the United Kingdom is taken from rivers contaminated with APEs.[101] Denmark has already phased out most uses of APEs because of their endocrine disrupting properties.[102] One APE, nonylphenol ethoxylate, also known as nonoxynol-9, is widely used as a spermicide and condom lubricant.[103]

92. *See* Toppari et al., *supra* note 2, at 757.
93. *See* Toxic Substances Control Act, 15 U.S.C. § 2605(e) (1994).
94. *See* COLBORN ET AL., *supra* note 3, at 26.
95. *See* Toppari et al., *supra* note 2, at 757.
96. *See id.*
97. *See id.*
98. *See id.* at 758.
99. *See id.*
100. *See* Michael Warhurst, *Hormone Disrupting Chemicals Website*, (last modified Nov. 25, 1998) <http://easyweb.easynet. co.uk/~mwarhurst>.
101. *See* Peter Fairley et al., *Endocrine Disruptors: Sensationalism or Science?*, CHEMICAL WEEK, May 8, 1996 at 29.
102. *See id.*
103. *See* Weise & Kelce, *supra* note 83.

Phthalates: This class of suspected EDCs is found in vinyl products, paint, and plastics (including plastic films used for food packaging), as well as in processed foods such as cheese, margarine, and baby formula.[104] One type of phthalate, DEHP (diethylhexylphthalate), is believed to be a testicular toxicant.[105] Another common type, BBP (butylbenzylphthalate), is estrogenic and has been associated with reduced sperm production and testicle size in male rats exposed *in utero* and in early life to levels close to calculated human levels of exposure.[106]

Bisphenol-A: Bisphenol-A has an estrogenic effect and has been found to cause estrogen-sensitive breast cancer cells to proliferate.[107] The chemical has gained particular notoriety because it is used widely in the packaging industry in products such as plastic water bottles and in the inner coating of food cans and bottle caps.[108] Very low levels of Bisphenol-A have been shown to cause endocrine disruption. In one study, doses of two micrograms per kilogram of body weight given to pregnant mice resulted in male offspring with prostate glands 30% larger than male offspring in control groups.[109] Bisphenol-A is used in some dental fillings, where concentrations of 5-30 micrograms per milliliter have been found in saliva one hour after a filling.[110]

Phytoestrogens: Phytoestrogens are naturally-occurring estrogenic and anti-estrogenic chemicals found in plants, including edible plants such as spinach, sprouts, and soybeans.[111] Because soybeans are used so widely in food products, including infant formula, they may be a major

104. *See* Warhurst, *supra* note 100.

105. *See* AMDUR ET AL., CASARETT AND DOULL'S TOXICOLOGY: THE BASIC SCIENCE OF POISONS 499 (1991).

106. *See* R.M. Sharpe et al., *Gestational and Lactational Exposure of Rats to Xenoestrogens Results in Reduced Testicular Size and Sperm Production*, 103 ENVTL. HEALTH PERSPECTIVES 1136-1143 (1995).

107. *See* COLBORN ET AL., *supra* note 3, at 135.

108. *See id.*

109. *See* Weise & Kelce, *supra* note 83.

110. *See id.*

111. *See* COLBORN ET AL., *supra* note 3, at 80. OUR STOLEN FUTURE suggests that plants may have developed phytoestrogenic properties through evolution as a means to disrupt the endocrine systems, and thus the reproduction, of predator animals. *Id.* at 76-77.

source of human EDC intake.[112]

These categories of suspected EDCs are not exhaustive. A number of other chemical types are under investigation, and scientists simply do not know the full range of chemicals that may cause endocrine disruption. Faced with this lack of knowledge about the health effects of most chemicals, the EPA's Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC) recommends investigating 87,000 different chemicals for endocrine disrupting effects.[113]

To be sure, some chemicals that affect the endocrine system have positive applications, including those used for birth control, treatment of osteoporosis and heart disease, and therapies for prostate and breast cancer.[114] Scientists are still trying to understand which types and degrees of impacts on the endocrine system cause harm and which cause no effect or have net benefits.

An important question from both the scientific and legal perspectives is whether the amount of human exposure to EDCs is sufficient to cause harm, especially given that EDCs are generally less potent than natural hormones. The estrogenic activity of DDT in the environment, for example, is one thousand to one million times less potent than natural estradiol in the body.[115] A study funded in part by the Chemical Manufacturers Association found that the daily dose of estrogen-equivalent from a birth control pill was 6.67 billion times greater than the expected daily dose from organochlorine compounds in the environment.[116]

Toppari and his colleagues respond that "[w]hile exposure levels to estrogenic chemicals are not at all well-known for humans, the large number of chemicals in numerous envi-

112. *See* Toppari et al., *supra* note 2, at 758. *See also* Weise & Kelce, *supra* note 83. The EDSTAC has recommended inclusion of naturally occurring estrogens in a screening and testing program because they are ubiquitous and because they may have additive and antagonistic effects with other hormonally active chemicals. *See* EDSTAC REPORT, *supra* note 10, at 7-10.

113. EDSTAC REPORT, *supra* note 10, at ES-3. For further discussion of EDSTAC, see Part IV(C)(1), *infra*.

114. *See id.* at 3-6.

115. *See* Toppari et al, *supra* note 2, at 756.

116. Safe, *supra* note 38, at 349.

ronmental categories suggests adequate availability."[117] That some suspected EDCs can accumulate in fish and wildlife may heighten human dietary exposure, as "biomagnification" in the food chain can concentrate chemicals in fish and wildlife to levels as much as one million times higher than the ambient concentration in water or land environments.[118]

An additional consideration is that fetuses might be damaged by levels of EDCs that would not harm adults because homeostatic mechanisms that maintain hormonal balance are not well-developed in fetuses.[119] Also, fetal hormone receptors might not be as discriminating among hormones as adult receptors.[120] Finally, it might be misleading to put too much weight on the potency of EDCs compared to natural hormones. Because natural hormones can modulate cell activity in concentrations in the parts-per-trillion range, a less potent hormone concentrated in the bloodstream in the parts-per-billion range or less might still have deleterious effects.[121]

IV. POTENTIAL REGULATION OF EDCS

A. Current Endocrine Disruption Programs

Over the past few years, several federal agencies have launched programs to examine the risks of EDCs and to develop testing, screening, and research recommendations. From the public literature, it appears that no government agency is currently considering regulatory action beyond testing and screening. The federal government effort is coordinated by the Committee on Environment and Natural Resources (CENR) of the National Science and Technology Council (NSTC).[122] The CENR's working group on endocrine

117. Toppari et al., *supra* note 2, at 769.
118. *See* Williamson et al., *supra* note 86, at 614.
119. *See* EPA RISK ASSESSMENT FORUM, *supra* note 9, at 19-20.
120. *See* COLBORN ET AL., *supra* note 3, at 74.
121. *See id. See Gina Solomon, Endocrine Disruptors, What Should We Do Now?* (Mar. 19, 1997) <http://*www.nrdc.org/nrdc/nrdcpro/present/gs031997.html*>.
122. *See* UNITED STATES ENVIRONMENTAL PROTECTION AGENCY, ENDOCRINE DISRUPTORS RESEARCH INITIATIVE FACT SHEET (Nov. 25, 1998) <http://www.epa.gov/endocrine/edrifact.html>.

disruption includes representatives from the White House, Environmental Protection Agency, Department of the Interior, National Institute of Environmental Health Sciences, Centers for Disease Control, Department of Agriculture, and a number of other federal agencies.[123]

International efforts to regulate endocrine disruptors have evolved considerably over the past five years. In June and July of 1998, representatives from over one hundred countries met in Montreal to begin negotiations on a global treaty to regulate Persistent Organic Pollutants (POPs): toxic chemicals that persist in the environment and that can be transported globally through water and air pollution and the migration of species.[124] Many of the twelve POPs on which the negotiators focused efforts, such as chlordane, DDT, heptachlor, and PCBs, are also suspected endocrine disruptors.[125] The United States has already banned or restricted most of the twelve chemicals, but it is participating actively in the negotiations because of the government's concern about environmental transport of the chemicals from other countries.[126] Conducted under the auspices of the United Nations Environment Programme, the treaty negotiations are expected to be completed in the year 2000.[127] It is currently unclear whether the global treaty will involve restrictions, phase-outs, or different requirements for the developed and developing world.[128] Assuming that the treaty is completed and that it is ratified by the Senate, domestic legislation might still be needed to implement the

123. *See id.*

124. *See* Joby Warrick, *120 Countries to Try to Reach Pact on Phaseout of Toxic Compounds*, WASH. POST, June 28, 1998, at A3.

125. The twelve chemicals are aldrin, chlordande, DDT, dieldrin, dioxins, endrin, furans, heptachlor, hexachlorobenzene, mirex, PCBs, and toxaphene. *See* United Nations Environment Programme, Press Release, "Treaty talks start on persistent organic pollutants," (June 29, 1998) <http://irptc.unep.ch/pops/POPs_Inc/press_releases/infokite.html#treaty>.

126. *See* Warrick, *supra* note 124 (quoting Rafe Pomerance, the State Department's deputy assistant secretary for the environment, as stating that "[m]any of these problems we cannot solve alone. They exist and are created outside our borders.").

127. UNEP Press Release, *supra* note 125.

128. *See* UNEP document "The international community's response to POPs," (June1998)<irptc.unep.ch/pops/POPs_Inc/press_releases/infokite.html#response> (describing special needs of developing countries, the possibility of technology transfers, and possible treaty provisions including bans on certain chemicals and provisions to promote release reductions).

treaty. Such legislation could become controversial, especially if the treaty regime restricts additional chemicals currently mass-marketed in the United States.

In the United States, the Environmental Protection Agency has taken the lead on endocrine disruption initiatives. In 1996, the EPA's Office of Prevention, Pesticides, and Toxic Substances (OPPTS) established the Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC) to "provid[e] direction to the Agency on the establishment of a comprehensive screening and testing program for pesticides and chemicals for estrogenic and other endocrine effects."[129] The screening program planned by the EDSTAC was mandated by Congress in the Food Quality Protection Act (FQPA),[130] signed into law on August 3, 1996, and by amendments to the Safe Drinking Water Act (SDWA),[131] signed into law on August 6, 1996.

In the FQPA, Congress required the EPA to determine "whether certain substances may have an effect . . . similar . . . to a naturally occurring estrogen, or such other endocrine effect as the Administrator may designate."[132] Congress directed the EPA to screen all registered pesticides and any other substance that may have an effect that is "cumulative" to an effect of a pesticide,[133] and the amendments to the SDWA gave the EPA authority to screen any other substance that may be found in drinking water sources and that may have an endocrine-disrupting effect.[134] Although chemical manufacturers will conduct the actual testing, Congress gave the EPA authority to suspend sale or distribution of a chemical if a firm fails to submit the requested testing data.[135]

In the FQPA, Congress placed the endocrine screening

129. U.S. ENVIRONMENTAL PROTECTION AGENCY, OFFICE OF PREVENTION, PESTICIDES, AND TOXIC SUBSTANCES, FOOD QUALITY PROTECTION ACT IMPLEMENTATION PLAN, (Mar. 1997), at 4.7 <http://*www.pestlaw.com/guide/EPA-70300B.html*>.

130. Food Quality Protection Act of 1996, Pub. L. No. 104-170.

131. Safe Drinking Water Act of 1996, Pub. L. No. 104-182.

132. 21 U.S.C. § 346a(p)(1) (Supp. 1997).

133. *Id.* at § 346a(p)(3).

134. *See* 42 U.S.C. § 300j-17 (Supp. 1996). The EDSTAC decided to expand the scope of the screening program to include "all environmental agents" and is prioritizing which agents to screen first. *See* FOOD QUALITY PROTECTION ACT IMPLEMENTATION PLAN, *supra* note 129, at 4.7.

135. *See* 21 U.S.C. § 346a(p)(5) (Supp. 1997).

program on a very tight schedule, requiring the EPA to develop the screening procedures within two years of enactment (i.e., by mid-1998),[136] and to implement the program within three years of enactment (i.e., by mid-1999).[137] By mid-2000, the EPA is required to report to Congress on its findings, recommendations for further testing of EDCs, and recommendations for possible regulatory action.[138] Because the EPA cannot possibly screen every commercial chemical within that time-period, it must prioritize which chemicals and pesticides will be screened first, and it is requesting voluntary industry compliance with the screening program in order to meet the statutory deadlines. Lynn Goldman, former Assistant EPA Administrator for OPPTS, explained in 1996 that "if we have to do this through rule-making processes it will take an inordinate amount of time and effort."[139]

Most importantly from a regulatory perspective, Congress gave the EPA wide latitude to take protective measures against EDCs. If the EPA finds that a certain chemical does have an "endocrine effect," the FQPA directs the EPA Administrator, "as appropriate," to "take action under such statutory authority as is available to the Administrator . . . to ensure the protection of public health."[140] Congress did not provide new regulatory authority with this provision, but this provision does demonstrate that Congress anticipated that some regulation of EDCs might be necessary under existing statutes. The question naturally arises, then, of whether the current statutes governing toxic chemicals are adequate to address the risks of EDCs.

B. Current Statutory Authority to Regulate EDCs

Because EDCs are found in pesticides, food, air, water, occupational settings, and consumer products, an enormous variety of statutes could potentially be relevant in any regulatory regime for EDCs. These include the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), the Food,

136. *Id.* at § 346a(p)(1). EDSTAC released its final report on schedule in August 1998.
137. *Id.* at § 346a(p)(2).
138. *See id.* at § 346a(p)(7).
139. Fairley et al., *supra* note 101 at 36.
140. 21 U.S.C. § 346a(p)(6) (Supp. 1997).

Drug, and Cosmetic Act (FDCA), the Clean Air Act (CAA), the Clean Water Act (CWA), the Safe Drinking Water Act (SDWA), the Toxic Substances Control Act (TSCA), the Occupational Safety and Health Act (OSHA), and the Consumer Product Safety Act (CPSA). The federal government, rather than the states, has taken primary responsibility for toxic chemical regulation,[141] and the Environmental Protection Agency sets the regulatory standards for most of the federal toxics statutes.[142] This discussion will therefore focus on the EPA.

Regulating EDCs under existing statutes would be problematic for a number of reasons. The existing statutory regime for toxic chemicals has many flaws, including fragmented agency authority,[143] media-specific approaches,[144] inconsistent lists of chemicals regulated under different statutes,[145] command-and-control methodology,[146] and chemical-by-chemical standard setting.[147] Proceeding under existing statutes, then, will merely replicate in the EDC context the same flaws that have plagued regulation of

141. States play a limited role in federal environmental regulation. For example, they grant permits and develop implementation plans for federal standards under statutes such as the Clean Air Act and Clean Water Act. *See* Williamson et al., *supra* note 86, at 646-647 & n.204.

142. EPA shares regulatory authority under the FDCA with the Food and Drug Administration, for example. OSHA, which regulates exposure to toxic chemicals in the workplace, is implemented by the Occupational Safety and Health Administration. The CPSA is implemented by the Consumer Product Safety Commission.

143. *See* Williamson et al., *supra* note 86, at 643-644 (stating that most fields regulated by the U.S. government, from aircraft safety to television and radio broadcasting, fall under a single statute, whereas toxic chemicals are regulated under at least nine statutes).

144. *See* John C. Dernbach, *The Unfocused Regulation of Toxic and Hazardous Pollutants*, 21 HARV. ENVTL. L.REV. 1, 43-45 (1997) (arguing that statutes generally cover a single medium, such as air or water, and ignore pollutant transport between media).

145. *See id.* at 1-6 (noting vast inconsistencies in the lists of pollutants regulated under five different toxics statutes and arguing that these inconsistencies encourage discharges of pollutants regulated under one statute into unregulated media).

146. *See* Williamson et al., *supra* note 86, at 647 (noting lack of market mechanisms in toxics regulation). *See also* Richard Stewart, *The Future of Environmental Regulation: United States Environmental Regulation: A Failing Paradigm*, 15 J.L. & COM. 585, 587 (1996) (noting difficulty of gathering information to implement command-and-control measures and economic inefficiency of such measures).

147. *See* Williamson et al., *supra* note 86, at 647 (noting that chemical-by-chemical testing leads to highly stringent limitations on a few chemicals, while the vast majority of chemicals remain unregulated).

other pollutants. To be sure, U.S. toxics statutes are broadly written to permit the EPA to take regulatory action when new chemical risks are discovered. That is, existing statutes provide sufficient *authority* to regulate EDCs. Because of difficulties that will arise in setting standards, drafting sensible regulations, and defending those regulations in court, however, the overall usefulness of proceeding under existing statutes to protect public health from EDC risks is limited. These obstacles would hinder regulatory action on the hazards of a given EDC even if there was considerable data.

Regulatory obstacles are discussed in more detail in Part IV(C), *infra*, but first it is useful to outline how regulation of EDCs might be incorporated into the existing statutory regime for toxics. This discussion will focus on three major statutes: the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), the Toxic Substances Control Act (TSCA), and the Food, Drug, and Cosmetic Act (FDCA).

FIFRA.[148] The Federal Insecticide, Fungicide, & Rodenticide Act provides that all pesticides must be registered with the EPA before sale or distribution.[149] Before approving a registration, the EPA Administrator must find that the pesticide will perform as intended without "any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide."[150] FIFRA is a classic cost-benefit balancing statute. The burden is on the pesticide manufacturer to produce sufficient safety information to allow the EPA to determine that the benefits of the pesticide outweigh possible hazards. FIFRA also provides the authority to cancel existing pesticide registrations under a similar cost-benefit analysis if new information about pesticide hazards comes to light.[151] In addition, FIFRA grants emergency

148. 7 U.S.C. §§ 136-136y (1994).

149. *Id.* at § 136a(a).

150. *Id.* at § 136(bb). The EPA Administrator shall classify a pesticide for restricted use if the pesticide "may generally cause . . . unreasonable adverse effects on the environment, including injury to the applicator." *Id.* at § 136a(d)(1)(C).

151. In determining whether to suspend a pesticide registration for unreasonable adverse impacts of the environment, the Administrator must consider the impact on "production and prices of agricultural commodities, retail food prices, and otherwise on the agricultural economy." *Id.* at § 136d(b). The EPA is currently re-

authority to the EPA to immediately suspend registrations to address an "imminent hazard."[152] In the context of EDCs, then, FIFRA would provide the EPA with authority to ban or limit the use of new or existing pesticides because of endocrine disrupting effects, as long as such action survived the rough cost-benefit analysis of the statute. Under the statute, the EPA approves the pesticide labels proposed by registrants, and it would have the power to mandate warnings about endocrine disrupting effects on pesticide containers.[153]

TSCA.[154] The 1976 Toxic Substances Control Act is the broadest of the major toxics statutes, as it applies to all new non-pesticide chemicals and to non-pesticide chemicals already on the market. Under TSCA, the EPA Administrator can mandate that a manufacturer test any chemical that "may present an unreasonable risk of injury to health or the environment."[155] Effects for which tests can be mandated include carcinogenesis, mutagenesis, teratogenesis, behavioral disorders, and cumulative or synergistic effects,[156] all of which could be relevant to EDCs.

In practice, the EPA has focused its chemical testing program on "new" chemicals developed after the passage of the Act. The vast majority of so-called "existing" chemicals that were already in commerce when TSCA was passed, including many suspected EDCs, have never been required to be tested for health risks. Even for "new" chemicals, testing is

registering all pesticides registered before November 1, 1984, *Id.* at § 136a-1(a), so older pesticides will be subjected to review over the next decade or so for health risks, including risks of endocrine disruption.

152. *Id.* at § 136d(c). Cancellation procedures can take years, whereas suspension takes effect immediately, but suspension is merely a stopgap measure until full cancellation hearings can be held. Because of the adversarial nature of cancellation proceedings and court delays, they are used infrequently. Suspected endocrine disruptors such as DDT and Kepone were cancelled (not for their endocrine disrupting effects) under such proceedings. *See* MARY DEVINE WOROBEC & GIRARD ORDWAY, TOXIC SUBSTANCES CONTROL GUIDE 60 (1989).

153. 7 U.S.C. § 136a(c)(5)(B) (1994).

154. 15 U.S.C. §§ 2601-2692 (1994).

155. *Id.* at § 2603(a)(1). Testing can also be mandated if the chemical (1) is or will be produced in substantial quantities or (2) if there will be significant or substantial human exposure or if the chemical will enter the environment in substantial quantities. *Id.*

156. *See id.* at § 2603(b)(2)(A).

rarely required. An EPA advisory group, noting that the EPA has ordered testing for only 121 chemicals in twenty years under TSCA, explains that "[t]his is not an indication of how much more information might really be needed but, rather, the administrative challenges of mounting an information request."[157]

Although TSCA mandates that companies submit Pre-Manufacture Notifications (PMNs) to the EPA,[158] EPA regulations require submission only of available toxicity data that is in the company's possession or in scientific literature.[159] Companies are not required to develop their own toxicity information through testing, and over half of PMNs are submitted with no toxicity data whatsoever.[160] Consequently, unlike pesticides under FIFRA, most chemicals in commerce today are being sold without fully developed safety information and without informed EPA review.

The EPA can restrict a chemical pursuant to TSCA only if it can reasonably conclude that the chemical "presents an unreasonable risk of injury to health or the environment."[161] This risk analysis must be balanced with an analysis of the benefits of the chemical, the availability of substitutes, and the "reasonably ascertainable economic consequences" of the regulation.[162] Available restrictions include limiting uses or production volumes, mandating warnings, prohibiting manufacture or distribution, or regulating disposal.[163] These restrictions could in theory ameliorate EDC risks. The EPA rarely restricts chemicals once they are on the market, however, because the required cost-benefit analysis is cumbersome and because regulations must be the "least burdensome" available to address the risk.[164] In TSCA's first twenty years, EPA imposed restrictions on only five types of chemicals.[165]

Although TSCA has many limitations, the statute at the

157. EDSTAC REPORT, *supra* note 10, at 2-11.
158. 15 U.S.C. § 2604 (1994).
159. *See* 40 C.F.R. § 720.50 (1998).
160. *See* ROE ET AL., *supra* note 77, at 27.
161. 15 U.S.C. at § 2605(a).
162. *Id.* at § 2605(c)(1).
163. *See id.* at § 2605(a).
164. *Id.*
165. *See* ROE ET AL., *supra* note 77, at 28. The chemicals are dioxin, hexavalent chromium, PCBs, metal fluids, and lead paint. *Id.* at n.38.

very least would provide the basic authority to ban or limit chemicals or mandate warnings once more information about the endocrine hazards of individual chemicals becomes available.

FDCA.[166] The Food, Drug, and Cosmetic Act gives the EPA the responsibility to set "tolerances," or allowable levels, for pesticide residue on food.[167] Foods that contain pesticide residue above the established tolerance are considered adulterated and violate the Act.[168] The agency must set tolerances that are "safe," defined as "a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information."[169] This language would provide the authority to revoke or modify tolerances for pesticides suspected of causing harm through endocrine disruption. That is, the "harm" that is a precedent for regulatory action is not defined narrowly as a specific disease or cancer, but rather is broad enough to encompass reproductive harm, decreased fertility, or other health problems that have been linked to EDCs.

Significantly, the FQPA directed the EPA to review all current pesticide tolerances within 10 years.[170] In reviewing tolerances, the EPA may now consider "whether the pesticide chemical may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects."[171] Additionally, the FQPA mandated that in setting or reviewing pesticide residue levels, the Administrator shall assess the health risk based on "available information concerning the special susceptibility of infants and children to the pesticide chemical residues, including . . . effects of *in utero* exposure to pesticide

166. 21 U.S.C. §§ 301-395 (1994).
167. *Id.* at § 346a.
168. *See id.* at § 342(a).
169. *Id.* at § 346a(b)(2)(A)(ii). The Administrator is permitted to set a tolerance higher than the "safe" level if the pesticide protects against a health risk greater than the health risk from the residue, or if a higher tolerance "is necessary to avoid a significant disruption in domestic production of an adequate, wholesome, and economical food supply." *Id.* at § 346a(b)(2)(B)(iii).
170. *Id.* at § 346a(q)(1).
171. *Id.* at § 346a(b)(2)(D)(viii).

chemicals."[172] The EPA has stated that it will review 10,000 tolerances by 2006 and that pesticides suspected of endocrine disrupting properties and those used on foods most eaten by children will receive the highest priority.[173] To some extent, then, concerns about endocrine disruption are already being incorporated into the existing regulatory regime.

C. Inadequacies of the Current Regulatory Regime

The problem with regulating EDCs within the existing toxics regulatory framework is not lack of statutory authority. Existing toxics statutes are written broadly enough to authorize the EPA or other agencies to place restrictions on EDCs if scientific data support such measures, and the statutes cover the main exposure routes to EDCs, such as pesticides, occupational exposure, and drinking water. Rather, the problems in potential regulation of EDCs will arise from two other sources: difficulties in risk assessment and bureaucratic and judicial obstacles.

1. Risk Assessment

The existing risk assessment framework for toxic chemicals is likely to be inadequate for determining the health risk from a given EDC and for supporting regulatory action against judicial challenge.[174] As Gina Solomon of the Natural Resources Defense Council writes, "[t]he new problem of endocrine disruptors shows that business as usual [at the EPA] will not protect our health."[175]

The current risk assessment framework for toxic chemicals is premised on (1) testing single chemicals (2) for car-

172. *Id.* at § 346a(b)(2)(C)(i)(II).

173. *See* EPA Press Release, *Riskiest Pesticides Will Be Assessed First Under New Food Safety Act*, August 4, 1997 <*www.epa.gov/epahome/Press.html*>.

174. Risk assessment encompasses four sub-procedures: hazard identification (linking a chemical to a particular health effect); dose-response assessment (determining the relationship between the magnitude of exposure and the probability that the health effect will occur); exposure assessment (determining the level of human exposure to the chemical); and risk characterization (combining the results of the prior steps to determine the overall magnitude of the risk). *See* PERCIVAL ET AL., ENVIRONMENTAL REGULATION – LAW, SCIENCE, AND POLICY 513-514 (1996). The special characteristics of EDCs have the potential to raise problems at each stage of the risk assessment process.

175. Solomon, *supra* note 121.

cinogenic effects (3) by extrapolating backward (4) from high doses (5) given to adult animals.[176] This framework is inadequate for EDC risk assessment for a number of reasons.

First, EDCs can disrupt hormonal pathways and modulate cell activity at extremely low doses, in the parts-per-billion or parts-per-trillion range in the blood stream.[177] The EPA screening program or other risk assessment activities might miss these subtle effects, and the low-dose problem also makes it difficult to set permissible exposures, such as pesticide tolerances for food under FDCA, because it is not known if there is a safe "threshold" below which no harmful effects are expected to occur.[178]

Second, EDCs may have an unusual inverted-U-shaped dose-response curve in which negative health effects increase as the dosage increases, but above a certain dose the effects diminish, possibly because hormone receptors become overloaded.[179] University of Missouri endocrinologist Frederick Vom Saal found this type of dose-response curve for DES.[180] If this inverted-U-shaped dose-response curve exists for other EDCs, it would mean that extrapolating health risks from high doses to low doses, far from exaggerating risks (which is the usual criticism of dose extrapolations), might actually underestimate them.[181] According to Vom Saal, this would be "the end of risk assessment as we know it."[182]

Third, risk assessment for EDCs is complicated by the fact that fertility or reproductive disorders are much more difficult to detect in laboratory animals than cancerous tu-

176. *See* COLBORN ET AL., *supra* note 3, at 198-209.

177. *See* text accompanying note 13, *supra*.

178. *See generally, Short Term Screen for Determining Endocrine Modulation May Not Be Possible*, PESTICIDE & TOXIC CHEMICAL NEWS, June 19, 1996 (on difficulties with developing EDC screening procedures). *See also* Yvonne Sor, *Fertility or Unemployment – Should You Have to Choose?*, 1 J.L. & HEALTH 141, 167 (1986-1987) (discussing lack of evidence regarding threshold levels for chemicals that cause birth-defects).

179. *See* COLBORN ET AL., *supra* note 3, at 169. Most dose-response curves do not turn down above a certain dose. That is, the toxic response continues to increase as the dose increases, leading to the risk assessment axiom that "the dose makes the poison." *Id.* at 205.

180. *See id.*

181. *See id.* at 170.

182. Michael Lerner, *Crossed Signals*, WHOLE EARTH, June 22, 1997, at 78.

mors.[183] Not only are such disorders intergenerational in the case of EDCs, but they are best observed in living animals, so that traditional risk assessment procedures that correlate various doses of a chemical to mortality rates in animals could be inadequate for EDCs.

Finally, because EDCs are thought to have synergistic effects, chemical-by-chemical screening, which is a foundation of our current risk assessment process for toxics, might be inadequate. Yet testing multiple combinations of synthetic chemicals and natural hormones could be extremely time-consuming.

The EPA's Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC) is advising EPA on risk assessment techniques for EDCs. Formed in October 1996, the EDSTAC released its Final Report in August 1998.[184] The report outlines a tiered structure for screening and testing of chemicals.[185] After initial sorting and priority setting stages, Tier 1 screening will identify chemicals capable of interacting with estrogen, androgen, or thyroid hormone systems.[186] Tier 2 testing will then determine whether those interactions are adverse and will identify, characterize, and quantify the adverse effects.[187] After either stage, a chemical may be moved to a "hold box," which indicates that the chemical is not harmful and that no further testing is necessary.[188] The EDSTAC estimates that approximately 87,000 chemicals will need to be screened,[189] an enormous task given the expense of testing and the time needed for each test.

183. *See* Williamson et al., *supra* note 86, at 625 ("Proving human teratogenicity is even more difficult than proving carcinogenicity.").

184. EDSTAC REPORT, *supra* note 10.

185. *See id.* at ES-3 to ES-6.

186. *See id.* at ES-4. Tier 1 screening would be designed for maximum sensitivity to hormonal effects of chemicals, thus minimizing false negatives. The tests would include a range of organisms and assess a range of endocrine disruption endpoints. *See id.* at ES-11.

187. *See id.* at ES-14. Tier 2 testing is designed to be more thorough than Tier 1 screening, and a negative outcome in Tier 2 testing would supercede a positive outcome in Tier 1 screening, thus moving the chemical to the "hold box." EDSTAC recommends that Tier 2 testing include a range of organisms in their most sensitive developmental lifestage and that the tests identify the specific hazard caused by the chemical and establish a dose-response relationship. *See id.* at ES-13.

188. *See id.* at ES-4.

189. *Id.* at ES-3.

The ESDTAC has developed initial sorting recommendations, based on existing data, which could speed the screening process by moving a chemical directly to the "hold box" if it is already known to be benign, or directly to hazard assessment if it is already known to be a harmful EDC.[190] For chemicals where existing data are insufficient, the EDSTAC recommends High Throughput Pre-Screening (HTPS).[191] HTPS is automated, high-volume sampling which would provide preliminary hormonal impact information to assist in placing chemicals in the correct tier.[192] The EPA has indicated that it will begin the screening program in 1999 using HTPS on 15,000 chemicals.[193]

The EDSTAC seems to recognize the difficult risk assessment challenges posed by EDCs. It recommends, for example, that the EPA develop testing protocols for embryonic exposure to chemicals, with evaluation of effects in the fully developed animal.[194] It also recommends that the EPA consider tests that detect multiple hormone interactions and predict long-term or delayed effects.[195] The EDSTAC did not address issues of cost to industry or the length of time needed to develop such sensitive tests, both of which could be substantial.[196] Even currently available tests used to detect the endocrine-modulating activity of a chemical need to be validated and standardized before they could be used on a large scale in Tier 1 screening or Tier 2 testing.[197]

In October 1998, the EPA accepted the recommendations of the EDSTAC report.[198] According to EPA Administrator

190. *Id.* at ES-3 to ES-4.

191. *Id.* at ES-8.

192. *See id.* The EDSTAC recommends that HTPS be performed on all chemicals with current production volumes above 10,000 pounds per year (approximately 15,000 chemicals), all pesticides, and all chemicals proposed to bypass a tier for any reason. *Id.*

193. *See* Glenn Hess, *Endocrine Disruption Screening Will Evaluate 15,000 Chemicals*, CHEMICAL MARKET REPORTER, October 12, 1998.

194. EDSTAC REPORT at ES-12.

195. *See id.* at 3-6.

196. Gary E. Timm, technical advisor to the EPA in the Office of Prevention, Pesticides, and Toxic Substances, estimates that first tier tests could cost industry about $200,000 per chemical, whereas second tier tests could cost about $2 million per chemical. *See* Corinna Wu, *Huge Testing Planned for Hormone Mimics; Endocrine Disruptors*, SCIENCE NEWS, September 5, 1998.

197. *See* EDSTAC REPORT at ES-15.

198. *See* Hess, *supra* note 193.

Carol Browner, the screening program "is a critical first step in our efforts to identify any health threats from these substances and ensure that human health and the environment are protected."[199]

The EDSTAC was not charged with making recommendations for what types of test results should justify regulatory action, but moving from risk assessment to risk management will be controversial. Decisions made during the risk assessment process could potentially affect future regulatory options. There was considerable controversy during the EDSTAC deliberations, for example, over the basic definition of an endocrine disruptor,[200] with opposing camps viewing the definition as potentially important to future regulatory efforts. One camp advocated defining the term as an exogenous agent that "changes endocrine function and causes adverse effects . . . ." Another group of committee members objected to the use of the term "adverse," arguing that the term is subjective and that hormone function is so sensitive that any biochemical alteration may lead to subtle but serious pathologies later in life or in subsequent generations.[201] A definition emphasizing *any* hormone effects, and not just demonstrably adverse ones, could potentially lead to more regulatory activity in the future. To achieve consensus, the EDSTAC finally settled on a definition that retains "adverse" but also cites the precautionary principle.[202] An endocrine disruptor, according to the EDSTAC, is an:

> exogenous chemical substance or mixture that alters the structure or function(s) of the endocrine system and causes adverse effects at the level of the organism, its progeny,

199. *Id.*

200. *See* Peter Fairley, *Low Dose Effects Challenge Risk Assessment Framework*, CHEMICAL WEEK, July 30, 1997, at 10.

201. *See* EDSTAC REPORT, *supra* note 10, at ES-1.

202. The precautionary principle has been formulated in different ways, but in general holds that policy makers should be cautious in the face of uncertain environmental risks. Two authors have described the principle as ensuring "that a substance or activity posing a threat to the environment is prevented from adversely affecting the environment, even if there is not conclusive scientific proof linking that particular substance or activity to environmental damage." James Cameron & Juli Abouchar, *The Precautionary Principle: A Fundamental Principle of Law and Policy for the Protection of the Global Environment*, 14 B.C. INTL & COMP. L. REV. 1, 2 (1991).

> populations, or subpopulations of organisms, based on scientific principles, data, weight-of-evidence, and the precautionary principle.

If the EPA does decide to regulate EDCs under current statutory authority, the agency should recognize the precautionary principle and the limitations of current risk assessment techniques. It should err on the side of safety in deciding whether a suspected EDC should be on the market or in setting permissible exposure levels for EDCs, especially because fetuses and infants appear to be particularly vulnerable to endocrine disruption. Greenpeace has argued that the chemical industry's testing of its own chemicals for endocrine disrupting effects amounts to "cigarette science."[203] Although this charge is perhaps premature, that risk assessment will be based largely on industry data also suggests that the EPA should take a protective, cautious approach to standard-setting for EDCs.

There are limits, of course, to how cautious the EPA can be when regulating EDCs. Because the timing of exposure appears to be as important as the level of exposure, the EPA might theoretically set dosage limits on EDCs so that humans would be "safe" at a specified time in hormonal cycles or development when the body is most vulnerable, even if at other times the chemical would not cause harm to humans. Such stringent limits, however, could be politically unacceptable, as they could possibly mean severe restrictions on certain products and materials.

2. Bureaucratic and Judicial Obstacles

In addition to risk assessment problems, the other major impediment to responding to EDC risks by proceeding under existing statutory authority is that regulations must pass through bureaucratic procedures and court challenges. The labyrinthine process of moving from risk assessment to rule-making to surviving a court challenge and enforcing a regulation will delay implementation of protective measures and could deter agencies from proposing scientifically sound regulations *ex ante*. These delays in proposing, revising, justifying, and implementing rules are one

203. Fairley et al., *supra* note 101.

of the primary flaws in our current toxics regulatory regime. The regulatory process is likely to be particularly vexing in the context of EDCs because there is still much scientific uncertainty and the risks are just beginning to be understood.

Before an agency finalizes any future EDC regulation, the regulation is likely to be subjected to some form of cost-benefit analysis. Statutes such as FIFRA or TSCA require such analysis, as does President Clinton's Executive Order 12,886.[204] This order requires a full Regulatory Impact Assessment, including cost-benefit analysis, for any agency rule that has an annual affect on the economy of $100 million or more or "adversely affect[s] in a material way the economy, a sector of the economy, productivity, competition, jobs, the environment, public health or safety, or State, local, or tribal governments or communities."[205]

Cost-benefit analysis is a useful tool. Agencies should ask the basic question of whether the harm from endocrine disruption, taking all uncertainties into account, appears to outweigh the costs of restricting a given chemical. The problem, however, is that the human health benefits of restricting an EDC may be particularly hard to quantify. The costs of industry compliance with regulations are much easier to determine and may be weighed too heavily in the equation. Further, the burden of performing the cost-benefit analysis can itself delay or deter sound regulation. As Richard Williamson has argued, "given the immense data requirements involved, if the burden of performing the analysis is placed on the government, the rule-making process will slow to a glacial pace and few substances will be regulated."[206]

Court challenges to agency rules probably present an even larger hurdle to the effective regulation of EDCs. Al-

204. 58 Fed. Reg. 51735 (1993)

205. *Id.*

206. Williamson et al., *supra* note 86, at 648. Williamson suggests that the burden of performing cost-benefit analyses might be shifted to industry, where once the government has shown that a substance is toxic, industry would have to show that the benefits of the substance outweigh its costs. But Williamson acknowledges that under such a system, "industry will be forced to endure a staggering burden of cost and delay," *id.*, and he concludes that "allocation of the burden [of performing the cost-benefit analysis] will nearly always be outcome-determinative." *Id.* at n.211.

though an agency might have the statutory authority to regulate EDCs,[207] the substance of agency regulations may still be challenged in court. Courts would apply one of two possible standards of review to agency regulation of EDCs: either the "arbitrary and capricious" standard applicable to informal rule-making under the Administrative Procedure Act,[208] or the "substantial evidence" standard that is mandated by some toxics statutes such as FIFRA and TSCA.[209]

The line between the level of deference granted to agency decision-making under the two standards is difficult to draw. Even under the less exacting arbitrary and capricious standard, courts carefully scrutinize regulations and demand that an agency justify its rule, show that it considered alternatives to the rule (including suggestions from public comments), explain why alternatives were rejected, and compile a record of scientific evidence sufficient to support the rule.[210]

Under either standard of judicial review, a plaintiff such as a chemical manufacturer would have a strong chance of defeating agency regulation of EDCs, at least in the near term, because the scientific understanding of endocrine disruption is still in its infancy, because of the problems with risk assessment discussed above, and because of inherent limitations of the toxic statutes.

To be sure, some courts defer to an agency's scientific judgments on highly technical matters. In *Baltimore Gas &*

207. As discussed above, toxics statutes are written broadly to encompass diverse types of harms. Thus, an agency argument that an existing toxics statute provides authority to regulate a suspected EDC would probably be considered "permissible" under the deferential test enunciated by the Supreme Court in Chevron, Inc. v. NRDC, 467 U.S. 837 (1984), for judicial review of agency interpretations of law.

208. 5 U.S.C. § 706(2)(a) (1998).

209. *See* 7 U.S.C. § 136n(b) (FIFRA substantial evidence standard) and 15 U.S.C. § 2618(c)(B)(i) (TSCA substantial evidence standard). EPA tolerance decisions under the FDCA are reviewed by judges under the arbitrary and capricious standard, except if the EPA Administrator has allowed a public evidentiary hearing on a tolerance revision, in which case the decision will be reviewed under the substantial evidence standard. *See* 21 U.S.C. § 346a(h)(2).

210. Searching judicial scrutiny under the arbitrary and capricious standard is called "hard look" review because courts require agencies to take a "hard look" at possible regulatory responses to the problem that is the subject of the regulation. *See, e.g.*, Motor Vehicle Mfr.'s Ass'n. v. State Farm Mut. Auto. Ins. Co., 463 U.S. 29 (1983) (National Highway Traffic Safety Administration's rescission of passive restraint requirements in automobiles struck down under "hard look" review).

Electric Company v. NRDC,[211] the Supreme Court stated that courts should be at their "most deferential" when reviewing agency determinations that are at the "frontiers of science."[212] Yet even assuming that courts would defer to the EPA's scientific determinations regarding EDCs, the agency would nevertheless be saddled with cumbersome statutes, such as TSCA, that make it nearly impossible to restrict hazardous substances even where the scientific evidence is compelling. In the 1989 case of *Corrosion Proof Fittings v. EPA*,[213] for example, the Fifth Circuit struck down most parts of an EPA ban on asbestos, despite the fact that the EPA had spent ten years developing the rule and had compiled a 45,000 page record to support the ban under TSCA.[214] The Fifth Circuit held that the EPA had not chosen the "least burdensome" course of action, which TSCA requires,[215] and that there were irregularities in the EPA's cost-benefit analysis.[216] The decision was viewed as the "death knell" for EPA attempts to ban toxic chemicals under TSCA, given the time and labor that EPA committed to banning a substance that was widely known to be hazardous.[217] The health risks of asbestos were one of the concerns that had prompted Congress to pass TSCA in the first place.[218]

In general, regulatory agencies have had an abysmal record of incorporating new substances into existing toxics regulatory programs. Cumbersome statutes, notice and comment procedures, and court challenges to agency rules

211. 462 U.S. 87 (1983).

212. *Id.* at 103. The D.C. Circuit has also been generally deferential in reviewing EPA's scientific determinations. *See* Intl. Fabricare Inst., Inc. v. EPA, 972 F.2d 384, 389 (D.C. Cir. 1992) ("The rationale for deference is particularly strong when the EPA is evaluating scientific data within its technical expertise."); Envt'l. Defense Fund, Inc. v. Costle, 578 F.2d 337, 339 (D.C. Cir. 1978) ("In an area characterized by scientific and technological uncertainty . . . this court must proceed with caution, avoiding all temptation to direct the agency in a choice between rational alternatives.").

213. 947 F.2d 1201 (5th Cir. 1991).

214. *See* PERCIVAL ET AL., *supra* note 174, at 568.

215. *See* 15 U.S.C. § 2605(a).

216. 947 F.2d at 1215-1220.

217. *See* DAN FAGIN & MARIANNE LAVELLE, TOXIC DECEPTION: HOW THE CHEMICAL INDUSTRY MANIPULATES SCIENCE, BENDS THE LAW, AND ENDANGERS YOUR HEALTH 138 (1996).

218. *See id.*

have all contributed to this poor record. The EPA, for example, has deleted three pollutants from its list of toxic water pollutants since 1976, and has not been able to add any pollutants to the list.[219] The EPA promulgated standards for only seven hazardous air toxics under the Clean Air Act between the 1970 passage of the Act and the 1990 Clean Air Act Amendments.[220] As Howard Latin of Rutgers University put it, "[u]nrealistic judicial requirements for comprehensive agency assessments of all potentially relevant factors and for a high degree of scientific precision have substantially emasculated environmental control programs in the past decade."[221] This overall record of agency impotence does not augur well for the difficult process of justifying potential restrictions on EDCs.

Assuming courts were ultimately to approve EDC regulations, there is little doubt that the overall process of developing, justifying, and implementing any potential regulation of EDCs would entail enormous cost and delay. For each chemical it tries to regulate, the EPA might be faced with years of litigation. As the columnist Jessica Matthews noted, "[t]he economic stakes involved are so huge and the epidemiology to prove cause-and-effect so difficult that the regulatory quandaries we could soon be mired in will make earlier struggles – over nitrites, saccharin, formaldehyde, Times Beach, Love Canal, cholesterol, alar and even tobacco – look like kids' stuff."[222] Chemical, pesticide, and plastics manufacturers would surely view regulation of EDCs as a major threat and would probably invest heavily in legal services to defeat any proposed rule.

In sum, there is a crucial difference between the statutory *authority* available to regulate EDCs, which appears to be sufficient, and the practical *ability* to promulgate regulations restricting EDCs, which appears to be limited. Although there appear to be numerous problems with regu-

219. *See* Dernbach, *supra* note 144, at 53.

220. *See* Branford C. Mank, *What Comes After Technology: Using an "Exceptions Process" to Improve Residual Risk Regulation of Hazardous Air Pollutants*, 13 STAN. ENVTL. L.J. 263, 268 (1994).

221. Howard Latin, *Good Science, Bad Regulation, and Toxic Risk Assessment*, 5 YALE J. ON REG. 89, 133 (1988).

222. Jessica Matthews, *Overlooking the "POP's" Problem*, WASH. POST, March 11, 1996, at A19.

lating EDCs, the regulatory route should be pursued. The prospects for finalizing and implementing regulations will improve as scientific understanding of EDCs improves, and creative approaches to regulation, possibly involving voluntary testing consent orders or new legislation, could be utilized to address EDC risks even if the science is not completely conclusive. Regulatory approaches to EDCs would be more feasible if reviewing courts were sensitive to the difficult risk assessment problems surrounding EDCs and followed the Supreme Court's admonition to defer to agency findings at the frontiers of scientific research.

V. POTENTIAL LITIGATION OVER ENDOCRINE DISRUPTING CHEMICALS

A. Obstacles to Successful Litigation over EDCs

Private suits have been an important component of environmental protection over the past three decades. Most federal environmental statutes have "citizen-suit" provisions under which any citizen may either directly sue polluting entities to force statutory compliance or sue the EPA to enforce the statute.[223] Common law claims to address toxic risks are more unusual, but a number of analysts have suggested that tort law can act as a "gap-filler" to protect public health and deter polluters where government regulation is incomplete, ineffective, or non-existent.[224] In theory, private litigation over EDCs could supplement potential regulatory efforts and deter manufacturers from producing products with harmful chemicals.

Most common law environmental claims rest on nuisance law or theories of strict liability for abnormally dangerous

223. *See* Shay S. Scott, *Combining Environmental Citizen Suits & Other Private Theories of Recovery*, 8 J. ENVTL. L. & LIT. 369, 372-380 (1994) (overview of statutory citizen suit provisions). This article does not address statutory-based citizen suits because endocrine disruption has not yet been incorporated into any type of toxics regulatory regime. *See infra* Part V.

224. Marshall S. Shapo, *Tort Law and Environmental Risk*, 14 PACE ENVTL. L. REV. 531, 531 (1997) (describing gap-filling role of tort law); *See also* Gwyn Goodson Timms, Note, *Statutorily Awarding Attorneys' Fees in Environmental Nuisance Suits: Jump Starting the Public Watchdog*, 65 S. CAL. L. REV. 1733, 1739 (1992) (same).

activities,[225] but neither of these doctrines is particularly applicable to EDCs. Instead, potential EDC litigation would probably be brought under product liability doctrine, where the claim would likely be that a chemical (or a product containing a chemical) is a defectively designed product that has caused bodily harm through endocrine disruption.[226] Failure-to-warn actions could also be possible.

Although there are some similarities between EDCs and other toxic agents that have been the subject of litigation, such as asbestos or DES, litigation would probably be an ineffective legal response to the health risks of EDCs. Scientific uncertainty and the unusual characteristics of EDCs are likely to foreclose litigation as a means to force manufacturers to internalize their costs and compensate injured parties.

Under traditional tort doctrine, a plaintiff must show that the defendant owed a duty to the plaintiff, that the defendant breached this duty,[227] and that the breach was the cause-in-fact and proximate cause of the plaintiff's injury. Proving cause-in-fact is the single largest barrier to successful litigation over EDCs. Indeed, scientific knowledge about endocrine disruption is unlikely to reach a point in the next decade where it could support legal arguments connecting a harm such as breast cancer, testicular cancer, or reproductive disorders to a given EDC.

This is so despite the fact that the tort system does not require 100% proof of causation, or even statistically significant proof (which most scientists set at 95% or 99% confidence). Rather, courts have established lower (though widely varying) standards for satisfying the burden of proving causation.[228] Some courts require the plaintiff to

225. *See* Shapo, *supra* note 224, at 533-542; Scott, *supra* note 223, at 381-387.

226. A number of product liability suits have already been brought against suspected EDCs, but for health concerns other than endocrine disruption. *See, e.g.*, Conde v. Velsicol Chem. Corp. 24 F.3d 809 (6th Cir. 1993) (chlordane as defective product); Baker v. Monsanto Co., 962 F.Supp 1143 (S.D.Ind. 1997) (failure-to-warn action against former PCB manufacturer).

227. Although product liability law is the most likely paradigm for EDC litigation, the issue of whether an EDC could be found to be a defective product is not discussed in this article. The causation hurdle is so formidable that suits would probably founder on that ground alone.

228. *See generally*, Falcon v. Mem'l. Hosp., 462 N.W.2d 44, 46-48 (Mich.1990) (discussing several causation standards in use in various jurisdictions).

demonstrate that the defendant's action or product "more likely than not" caused the harm.[229] This greater-than-fifty-percent standard conforms with the general "preponderance of the evidence" standard of proof in civil trials. Other courts require that the plaintiff show only that the defendant's action or product was a "substantial factor" in causing the harm, without quantifying "substantial."[230] Finally, some courts require plaintiffs to demonstrate that it is a "reasonable medical probability" that the defendant caused the harm, again without quantifying that phrase.[231]

Regardless of which causation standard is in effect in a given jurisdiction, however, an EDC would be unlikely to be judged the cause of an injury. Although scientists have a general understanding of the natural hormonal processes regulating reproduction and development, too much is yet unknown about how environmental chemicals disrupt the endocrine system and modulate cell activity. Though several studies have associated EDCs with cancer and reproductive disorders in animals and humans, scientists still cannot explain the physical and chemical mechanisms through which EDCs cause harm. In short, there is too much scientific uncertainty to support causation arguments, and this is likely to be the case for the near future.

Of course, epidemiological evidence and associational studies linking exposure to harm are not irrelevant to the causation issue, and in many toxic tort cases such studies are the major evidence offered by the plaintiff. In *Merrell Dow Pharmaceuticals, Inc. v. Havner*,[232] a Bendectin[233] case, the Texas Supreme Court struggled to develop a causation rule that would not completely block plaintiffs in such situations from pursuing their claims. This case contains

229. *See, e.g.*, Parson v. Marathon Oil Co., 960 P.2d 615, 617 (Alaska 1998); Hambrick v. Makuch, 491 S.E.2d 71, 72 (Ga. Ct. App. 1997).

230. *See, e.g.*, Rutherford v. Owens-Illinois, Inc., 941 P.2d 1203 (Cal. 1997).

231. *See, e.g.*, Marks-Brown v. Rogg, 28 S.W.2d 304 (Tex. 1996); Mauro v. Raymark Indus., Inc., 561 A.2d 257 (N.J. 1989).

232. 953 S.W.2d 706 (Tex. 1997).

233. Bendectin is a morning sickness drug that has been linked to birth defects such as malformed limbs. The central issue in most Bendectin litigation is the scientific reliability of expert testimony offered by the plaintiff to establish causation. See *id.* at 708. The U.S. Supreme Court case that developed the standards for admissibility of expert testimony under the Federal Rules of Evidence, Daubert v. Merrell Dow Pharm., Inc., 113 S. Ct. 2786 (1993), was a Bendectin case.

one of the most recent extensive discussions by a state supreme court on causation problems in toxic tort cases and thus provides some indication of how a court might handle causation problems in potential EDC litigation.

The *Havner* court acknowledged "that a disease or condition either is or is not caused by exposure to a suspected agent,"[234] and that "epidemiological studies cannot establish that a given individual contracted a disease or condition due to exposure to a particular drug or agent."[235] The court also recognized, however, that the law should balance the cause-in-fact requirement with "the need to compensate those who have been injured by the wrongful actions of another . . . ."[236] The court concluded that epidemiological studies could be offered to prove cause-in-fact in a probabilistic manner. The studies must show, however, that exposure to the substance at least doubles the risk of contracting the plaintiff's disease to meet Texas' "more likely than not" causation standard.[237] Further, the court held that a plaintiff must show that he or she is similar to the subjects in the studies in terms of the substance involved and the dose levels and that other plausible causes of the injury can be excluded with reasonable certainty.[238] These requirements, according to the court, "strike a balance between the needs of our legal system and the limits of science."[239]

The holding in *Havner* is similar to the conclusions of other courts that have examined difficult causation issues, particularly in requiring a showing of doubling of risk.[240] These cases suggest that plaintiffs might be permitted to rely on epidemiological studies linking EDCs to health problems to support causation, but courts would carefully scrutinize the studies for reliability and ensure that the studies are relevant to the particular circumstances of the

234. 953 S.W.2d. at 718.
235. *Id.* at 715.
236. *Id.* at 718.
237. *Id.* at 717-18.
238. *Id.* at 720.
239. *Id.* at 718.
240. *See* Daubert v. Merrell Dow Pharm. Inc., 43 F.3d 1311, 1320 (9th Cir. 1995) (on remand), *cert. denied*, 166 S.Ct. 189 (1996); DeLuca v. Merrell Dow Pharm., Inc., 911 F.2d 941, 958 (3d Cir. 1990); Hall v. Baxter Healthcare Corp., 947 F.Supp 1387, 1403 (D.Or. 1996).

litigation in terms of the exact substance at issue and the level of exposure. Animal studies alone are probably not sufficient to support causation, leaving plaintiffs with a limited chance of success, as few human epidemiological studies have been conducted and as medical ethics would bar conducting controlled human studies related to EDCs.

A potential defendant, such as a chemical manufacturer, would have several strong approaches to attack a plaintiff's causation arguments, including pointing to conflicting studies on the health effects of EDCs or to the presence of confounding factors that might be the cause of a plaintiff's health problem.[241] These factors would include hormonal agents, such as birth control pills or phytoestrogen in foods, or non-hormonal factors, such as genetics, smoking, or exposure to lead or other heavy metals. Confounding factors would be especially difficult to sort out in EDC litigation because the known health problems associated with EDCs are not "signature diseases" that would strongly implicate EDCs as opposed to other agents. In contrast, asbestos or DES are both associated with a signature disease such as mesothelioma or clear-cell adenocarcinoma. As two critics of the chemical industry have noted regarding chances of success in toxic tort suits, "the presence of fingerprints has become far more important than the severity of the crime."[242]

Even if a plaintiff could show that synthetic chemicals were the cause-in-fact of a health problem, tort law also requires that the plaintiff identify which particular chemical caused the harm.[243] Again, this is a near impossibility because modern society is permeated by synthetic chemicals. United States production of synthetic chemicals was over 435 billion pounds in 1992, or 1,600 pounds per capita,[244] and pesticide use in the United States was over 4.5 billion pounds in 1995,[245] or about 18 pounds per capita. As dis-

241. *See* Kavlock et al., *supra* note 1, at 732 (listing confounding factors in endocrine disruption).

242. FAGIN & LAVELLE, *supra* note 217, at 157.

243. *See* Mary Cabrera, *Legal Remedies for Victims of Pesticide Exposure*, 1 KAN. J. L. & PUB. POL'Y 113, 114 (1991) (discussing difficulties of farmworkers in identifying the specific pesticides to which they have been exposed).

244. *See* COLBORN ET AL., *supra* note 3, at 137.

245. Arnold L. Aspelin, *U.S. EPA Office of Prevention, Pesticides, and Toxic Substances, Pesticide Industry Sales and Usage: 1994 and 1995 Market Estimates*, 3

cussed above, over fifty types of chemicals are suspected of having endocrine-disrupting properties, they have numerous exposure routes, and they act in extremely low doses. Many of the suspected EDCs do not even have chemical structures similar to the natural hormones they disrupt, and the "estrogen receptor system may well be unique in terms of the structural diversity of its effective stimulants."[246] Chemicals such as DDT or PCBs, which have not been manufactured in large quantities in over two decades, may be important contributors to EDC risks, yet they would be very difficult to identify as causal agents because they are dispersed throughout the environment and the food-chain.

In addition, intervening agents would complicate causation arguments in an EDC tort suit. EDCs may not act individually, but rather may act in combination with other chemicals, including natural hormones, through synergism, inhibition, or potentiation.[247] In sum, the legal hurdles to linking a particular health problem to a particular EDC, even under less-than-fifty-percent standards of causation, appear to be insuperable, at least with the current state of scientific knowledge.

Several other problems would hinder both the use of litigation as a response to EDCs and the development of regulations restricting EDCs. Because society is permeated by synthetic chemicals and because we have all absorbed them into body fat to some extent, there is no unexposed "control" group to which scientists, attorneys, or regulators can compare individuals to argue that differential health effects have occurred.[248] As stated in *Our Stolen Future*, "[v]irtually anyone willing to put up the $2,000 for the tests will find at least 250 chemical contaminants in his or her body fat. . . ."[249] The lack of an unexposed control group

(Aug. 1997) <www.epa.gov/oppbead1/95pestsales/95pestsales.pdf>.

246. John A. Katzenellenbogen, *The Structural Pervasiveness of Estrogenic Activity*, 103 ENVTL. HEALTH PERSPECTIVES, Supplement 7, 99, 99 (1995).

247. *See* Kavlock et al., *supra* note 1, at 730.

248. *See id.* at 722.

249. COLBORN ET AL., *supra* note 3, at 106. As an example of the lack of an unexposed control group, OUR STOLEN FUTURE describes villagers living above the Arctic Circle on Broughton Island, Canada, who have some of the highest body-fat PCB concentrations in the world. PCBs from industrialized North America and Europe have bioaccumulated in the Arctic foodweb. *Id.* at 108.

does not mean that all the scientific research on EDCs is flawed, but only that comparisons in humans are necessarily between those suspected of receiving a higher-than-average dose and the general population, which has also been exposed to some extent.

As discussed above, natural hormones and the chemicals that disrupt them act in extremely low concentrations in the bloodstream, often at the parts-per-billion or even parts-per-trillion range.[250] These minute dose effects make it difficult to develop causation arguments in litigation, as well as regulatory programs within agencies. In addition, if many EDCs are found to have the inverted-U-shaped dose-response curve of DES,[251] it might mean that the most common categories of plaintiffs in mass toxics cases, such as workers exposed to a high dose of a toxic agent in an occupational setting, might not be the ones suffering the most severe effects from EDCs.[252]

A final dose-related issue that will cause problems for potential litigation and potential regulation of EDCs is that, at least in the case of *in utero* exposure, the timing of the dose appears to be more important than the amount of the dose.[253] Fetal development depends on precisely timed doses of hormones. A dose of an estrogenic synthetic chemical received by a male fetus during the seventh month of gestation may have no effect, for example, whereas the same dose received during the first trimester, when sexual differentiation occurs, could have a large detrimental impact.[254] Because EDCs can be stored in a mother's body fat, a fetus may be exposed to EDCs taken into the mother's body months or years before her pregnancy.[255] According to Toppari, "[i]t is therefore not the amount . . . to which a mother is exposed during pregnancy that is critical, but rather her lifetime exposure that will determine the level of exposure of the fetus and the breast-

250. *See* note 13, *supra*.
251. *See* Part IV(C), *supra*.
252. *But see* Toppari et al., *supra* note 2, at 758 ("Because of better documentation and higher exposure, [occupational] studies are more likely to reveal adverse effects of chemicals on humans than are the studies on the general population.").
253. *See* COLBORN ET AL., *supra* note 3, at 62.
254. *See id.* at 46.
255. *See* Toppari et al., *supra* note 2, at 756.

fed infant."[256] Without the ability to pinpoint the timing of exposure, proof that a particular exogenous agent caused harm to the fetus would be nearly impossible to obtain. With diseases that have long latency periods, such as certain cancers, it would be difficult to determine whether the cancer stemmed from an adult exposure to an EDC, exposure to the mother while she was pregnant with the fetus, or exposure to the mother before she was pregnant with the fetus.[257]

B. Parallels with DES Litigation

The obstacles to a successful suit over EDCs are formidable under traditional tort doctrine. Over the past two decades, however, courts have shown some willingness to relax traditional tort rules in another context: DES litigation. Because DES, a synthetic estrogen, is an endocrine disrupting chemical with intergenerational effects, it serves as the most relevant precedent for potential litigation over EDCs. Despite the similarities between DES and EDCs, however, the relatively plaintiff-friendly principles enunciated in the DES cases are insufficient to provide a solid legal foundation for EDC litigation. Extensive discussions of DES litigation have appeared elsewhere,[258] but a few examples of the novel theories courts have devised in DES cases are useful for explaining why the DES cases do not improve the prospects for successful EDC litigation.

The courts' willingness to relax traditional tort requirements in DES cases, such as the requirement that a plaintiff identify the specific defendant that caused harm (the identification requirement), was a response to the unique circumstances of the drug and its effects. DES-daughters exposed *in utero* filed suit against DES manufacturers for

256. *Id.*

257. Furthermore, these problems arise only after the cancer could be linked to an EDC as opposed to some other cause.

258. The academic literature on DES is voluminous. *See, e.g.*, Richard Goldberg, *Causation and Drugs: The Legacy of Diethylstilbestrol*, 25 ANGLO-AM L.REV. 286 (1996); Tracy Batt, Note, *DES Third-Generation Liability: A Proximate Cause*, 18 CARDOZO L. REV. 1217 (1996); David M. Schultz, *Market Share Liability in DES Cases: The Unwarranted Erosion of Causation In Fact*, 40 DEPAUL L. REV. 771 (1991); Thomas Currie, *Risk Contribution: An Undesirable New Method for Apportioning Damages in DES Cases*, 10 J. CORP. L. 743 (1985); Glen O Robinson, *Multiple Causation in Tort Law: Reflections on the DES Cases*, 68 VA. L. REV. 713 (1982).

compensation for adenocarcinoma or other disorders, but most DES-daughters could not identify the manufacturer of the specific pills their mothers took during pregnancy.[259] Approximately 300 companies manufactured DES, with companies continuously entering and leaving the market during the three decades in which DES was in widespread use.[260]

In *Sindell v. Abbott Laboratories*,[261] the California Supreme Court resolved the identification problem by employing a "market-share liability" doctrine for DES cases. Under this doctrine, once a substantial share of all DES manufacturers were joined as defendants and the plaintiff made out a successful product liability case, damages would be imposed based on the share of the DES market that each defendant possessed, unless a defendant could prove that it did not manufacture the particular pills taken by the plaintiff's mother.[262] Market-share liability shifted the burden of proof to defendants to show that they did not cause harm. The *Sindell* court reasoned that under this system each DES manufacturer's liability over time would approximate its responsibility for injuries,[263] and the court also added that "as between an innocent plaintiff and negligent defendants, the latter should bear the cost of the injury."[264]

The doctrine that in certain cases a plaintiff will not be required to identify a particular harm-causing defendant could indeed be helpful in potential EDC litigation. A plaintiff alleging harm from an endocrine disrupting chemical is similarly unable to identify particular defendants because the types of EDCs and the mechanisms of exposure and injury are so varied. Significantly, the *Sindell* court prefaced its outline of the market-share theory with a statement advocating judicial activism in response to new types of risks:

259. *See, e.g.*, Hymowitz v. Eli Lilly & Co., 539 N.E.2d 1069, 1072 (N.Y. 1989), *cert. denied*, 493 U.S. 944 (1989).

260. *See id.*

261. 607 P.2d 924 (Cal. 1980).

262. *See id.* at 936-937. Whether the relevant market should be the national market, the California market, or some regional market was not decided in the case. This issue spawned years of litigation, and California finally settled on use of national market share. *See* Hymowitz, 539 N.E.2d at 1076.

263. 607 P.2d. at 935.

264. *Id.* at 936.

> In our contemporary complex 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.[265]

This statement is the strongest in DES case law that plaintiffs might cite to advocate a relaxation of causation or identification requirements in EDC litigation, and it suggests that courts will not be completely inflexible in adapting tort law to meet new circumstances.

Some courts, such as the New York Court of Appeals in *Hymowitz v. Eli Lilly & Co.*,[266] followed the *Sindell* market share theory. Others rejected it and have continued to demand that plaintiffs identify the manufacturer of the particular DES pills the plaintiff's mother ingested.[267] Often these courts are hesitant to make major changes in tort law, such as market-share liability, because they believe such changes should be implemented by legislatures, not courts.[268] Some courts have implemented other types of burden-shifting mechanisms, such as alternative liability, in DES cases.[269] Significantly, federal courts hearing DES cases have been reluctant to devise novel theories of tort liability because federal courts defer to state judges on the evolution of state tort law.[270] Therefore, potential EDC plaintiffs are likely to find state courts to be a more favorable forum.

Although many courts in DES cases have relaxed traditional tort requirements in response to suits by sympathetic plaintiffs who could not identify the defendant that caused

265. *Id.*

266. 539 N.E.2d 1069 (N.Y. 1989), *cert. denied*, 493 U.S. 944 (1989).

267. *See, e.g.*, Morton v. Abbott Lab., 538 F.Supp. 593 (M.D.Fla. 1982); Payton v. Abbott Lab., 512 F.Supp. 1031 (D.Mass. 1981).

268. *See* Mulcahy v. Eli Lilly & Co, 386 N.W.2d 67, 75-76 (Iowa 1986).

269. Under alternative liability, DES manufacturers who could not exculpate themselves were subject to joint and several liability. *See* 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).

270. *See* Thomas J. Currie, *Risk Contribution: An Undesirable New Method for Apportioning Damages in the DES Cases*, 10 J. CORP. L. 743, 747 (1985).

their harm, the importance of the DES cases as precedent for future lawsuits involving EDCs should not be overstated. Most importantly, it should be noted that the traditional tort law requirement that was relaxed in *Sindell* and other DES cases was the requirement of showing *which specific manufacturer* caused the harm. The requirement of proving that *DES* caused the harm, as opposed to some other factor, was not relaxed.[271] This latter requirement was a surmountable hurdle in the DES litigation, despite long latency periods, because most plaintiffs were able to prove that their mothers ingested DES and because the plaintiffs had signature diseases, such as adenocarcinoma, that were strongly linked to *in utero* exposure to DES. DES was a pill, and the exposure route was clear.

In contrast, dozens of compounds are suspected of being EDCs. EDCs permeate the environment, and exposure can occur through many different pathways. Even if a plaintiff could show that an EDC, as opposed to some other environmental agent or genetics, caused his or her health problem (whether through *in utero* or direct exposure), the plaintiff in the vast majority of cases will not know which particular endocrine disrupting agent caused the harm. It is only when a plaintiff gets past that hurdle that the DES cases would be useful precedents.[272] At that point, it would be appropriate for courts to follow the DES precedents and relax the requirement that the plaintiff show which particular company manufactured the EDC that was shown to have caused the injury.[273]

One could imagine a system in which the identification

271. Further, the requirement of showing that DES was a defective product was not relaxed. Plaintiffs still had to proceed under negligence or strict liability theories. In the latter case, plaintiffs had to show that the product was unreasonably dangerous. *See, e.g.*, Collins v. Eli Lilly & Co., 342 N.W.2d 37, 51 (Wis. 1984).

272. Although proving causation would be harder for EDCs than for DES because of the large variety of EDCs and exposure pathways, identifying defendants might be easier in EDC cases once a specific EDC could be shown to have caused the injury. This is because there might be only a handful of manufacturers of a given EDC, such as a pesticide, whereas there were approximately three hundred manufacturers of DES. Indeed, eighty percent of the world pesticide industry is controlled by only twenty companies. *See* JOHN WARGO, OUR CHILDREN'S TOXIC LEGACY – HOW SCIENCE AND LAW FAIL TO PROTECT US FROM PESTICIDES 3 (1996).

273. *See* Cabrera, *supra* note 243, at 118 ("Market share liability may be appropriate in cases of pesticide exposure when the victims can identify the specific product responsible for their injuries.").

requirement could be relaxed (and the burden of proof shifted to defendants) at an even lower threshold of proof by the plaintiff. For example, a rule might be devised so that once a plaintiff has demonstrated that a synthetic EDC caused his or her injury, all manufacturers of all EDCs would be held liable on a market-share or other basis unless they could demonstrate that they did not cause the harm.[274] This is probably the only legal rule that would give plaintiffs in EDC suits any hope of success, but such a rule would stretch tort doctrine to the point of absurdity and would impose unfair burdens on defendants, as the entire chemical industry could be roped into almost every potential EDC case.

It should be noted that courts in cases such as *Hymowitz* and *Sindell*, recognizing the departure they were making from traditional tort doctrine, attempted to limit the scope of the novel theories they enunciated. The New York Court of Appeals in *Hymowitz*, for example, stressed that "the DES situation is a singular case, with manufacturers acting in a parallel manner to produce an identical, generically marketed product, which causes injury many years later. . . ."[275] The California Supreme Court in *Sindell* similarly stressed that a crucial factor in its decision was that DES was a fungible product.[276] If all manufacturers of a

274. *See generally* Wendy E. Wagner, *Choosing Ignorance in the Manufacture of Toxic Products*, 82 CORNELL L. REV. 773 (1997) (advocates shifting the burden of proof on causation in toxics cases because chemical manufacturers have better access to information about the risks of their products).

275. 539 N.E.2d at 1075.

276. 607 P.2d at 936. One commentator suggests that there is no reason to limit the principles of Sindell to fungible products. *See* Glen O. Robinson, *Multiple Causation in Tort Law: Reflections on the DES Cases*, 68 VA. L. REV. 713 (1982). Robinson argues that liability could be imposed based on percentage contribution to the injury in any case in which several factors contributed to a harm. "As long as liability is proportionate to the risks created by a defendant," he asserts, "there is no reason why the Sindell liability rule cannot be applied to cases involving multiple and different risk-creating activities." *Id.* at 750. He adds that "there is no reason even to require that all of the causal agents be identified, so long as it can be proved what contribution a given defendant made to the risk." *Id.* at 753. Robinson's theory could potentially be useful in EDC litigation, as an EDC may be one of many factors that contribute to a health or reproductive disorder. But Robinson is too optimistic about the ability of courts and juries to determine the percentages by which various factors contribute to a harm, especially when all defendants are not before the court. No court has adopted Robinson's theory since it was first outlined in 1982.

certain product can be brought into a case, these courts reasoned, there must be a showing that all defendants contributed equally to the risk through manufacturing an identical, fungible product. EDCs are far from fungible, however. They exist in a huge variety of chemical forms and concentrations in diverse products. Courts have generally declined to extend market-share liability to products other than DES, such as asbestos (where the forms of asbestos and types of exposure varied),[277] and they would probably be similarly hesitant to extend the market-share doctrine to EDCs.

The differences between EDCs and DES highlight a fundamental principle of toxic tort litigation: litigation will be most successful when the toxic substance is potent, associated with a signature disease, and emitted in a concentrated fashion from one or a small number of sources. It will be least successful in cases such as EDCs where the pollution is not highly toxic, is dispersed from a large number of sources, and does not cause a signature disease.[278] Plaintiffs' lawyers are likely to take the former type of case because causal links will be easier to draw, whereas injured individuals will have difficulty finding legal representation for the latter type of case[279] even if the overall health risks from the dispersed pollution are larger. In short, "some environmental injury paradigms lend themselves to institutional responses such as tort litigation that are inappropriate or inapplicable for other paradigms."[280]

C. Other Legal Parallels for EDC Litigation

There have been few non-DES cases in the United States in which plaintiffs have claimed loss of fertility or harm to reproductive organs. Most of these cases involve medical

277. *See* Goldman v. Johns-Manville Sales Corp., 514 N.E.2d 691 (Ohio 1987) (declines to apply market share theory because asbestos products not fungible); Case v. Fibreboard Corp., 743 P.2d 1062 (Okla. 1987) (same); Setliff v. E.I.Dupont de Nemours & Co., 38 Cal.Rptr.2d 763 (Cal. Ct. App. 1995) (declines to apply market share theory because products containing volatile organic compounds are not fungible).

278. *See* Troyen A. Brennan, *Environmental Torts*, 46 VAND. L. REV. 1, 18 (1993).

279. *See id.* at 18-19.

280. *Id.* at 18.

malpractice[281] or defective intra-uterine devices (IUD's),[282] not exposure to environmental toxins.

One body of case law that might be relevant to potential EDC litigation is the so-called "pre-conception torts." In these suits, a plaintiff claims that the defendant caused injury to the plaintiff's mother prior to the conception of the plaintiff[283] and that the injury resulted in a birth defect or other health problem in the plaintiff.[284] For example, the Illinois Supreme Court allowed a child to proceed with a suit where the child's mother had been negligently sensitized to Rh positive blood at the age of thirteen, allegedly resulting in the premature birth eight years later of the child, who was brain damaged and needed frequent transfusions.[285] In contrast, a New York court, concerned that liability be kept within manageable bounds, barred a plaintiff from asserting a claim for birth defects resulting from the negligent perforation of his mother's uterus during an earlier abortion.[286] The New York court distinguished pre-conception torts from torts against existing fetuses, which are cognizable claims in New York, stating that in existing fetus cases "there are two identifiable beings in the zone of danger each of whom is owed a duty independent of the other and each of whom may be directly injured."[287]

The pre-conception tort cases are relevant to any potential EDC litigation because, as discussed above, mothers may be exposed to EDCs years before conception and may store the chemicals in body fat, then damaging a fetus *in utero*.[288] A question thus arises of when the tort occurred. Was it when the mother was exposed to the EDC, or when the EDCs seeped from her body into her child's? Which "be-

281. *See, e.g.*, Battenfeld v. Gregory, 589 A.2d 1059 (N.J. Super. Ct. App. Div. 1991) (plaintiff's allege that delay in removing ruptured appendix during pregnancy damaged uterus).

282. *See, e.g.*, Mackereth v. G.D. Searle & Co., 674 N.E.2d 936 (Ill. App. Ct. 1996); King v. Searle Pharm., Inc., 832 P.2d 858 (Utah 1992).

283. These are thus distinguished from the DES cases, where the claim is that the plaintiff was harmed in utero.

284. *See* Batt, *supra* note 61, at 1235-1240, for a discussion of preconception torts.

285. Renslow v. Mennonite Hosp., 367 N.E.2d 1250 (Ill. 1977).

286. Albala v. City of New York, 429 N.E.2d 786 (N.Y. 1981).

287. *Id.* at 787.

288. See text accompanying note 255, *supra*.

ings" are in the zone of danger, and at what times?

Apart from the DES cases, the case that is most factually analogous to potential EDC litigation and that most closely demonstrates the challenges that a potential EDC plaintiff might confront is *Sanderson v. Intl. Flavors & Fragrances, Inc.*[289] In *Sanderson*, the plaintiff sued several fragrance manufacturers, claiming that their products caused sinus inflammation, toxic encelopathy (brain damage), dysosmia (deranged sense of smell), and "multiple chemical sensitivity."[290] The plaintiff claimed that she was exposed to fragrance products on over 16,000 occasions between April 1994 and October 1995, but for 70 percent of those occasions she was unable to identify the fragrance products to which she was exposed.[291] Her claim was somewhat outlandish, and was treated as such by the court, but her problem of identifying harmful agents after long-term exposure to a profusion of chemicals is similar to that which a potential plaintiff would face in bringing a suit over EDCs.

The court granted the defendant's motion for summary judgment because although a "jury could probably find that defendants' products, as a whole, were a substantial factor in causing her injuries, plaintiff has no evidence whatever from which a jury could find that any particular defendant's products were."[292] As discussed above, this is a likely holding in potential EDC litigation as well, where EDCs in general might be shown to have caused a harm, but not a particular kind or brand of EDC. The court also rejected the plaintiff's suggestion that she could meet her causation burden by showing merely that her injuries were the type caused by the fragrances, that she was exposed to the fragrances, and that there was some temporal connection between the exposure and harm.[293] "At best," the court explained, this "establishes only a 'mere possibility' that defendants' fragrance products were the ones that caused her injuries, and even less of a possibility that any one defendant's products caused them."[294] The *Sanderson* court

289. 950 F.Supp. 981 (C.D.Cal. 1996).
290. *Id.* at 986.
291. *Id.*
292. *Id.* at 985.
293. *Id.* at 988.
294. *Id.*

concluded that the "[p]laintiff's only hope of meeting her causation burden lies in shifting it to defendants," but then proceeded to reject her arguments for imposing market share or alternative liability.[295] The request for burden-shifting and market share liability, and the rejection of those requests, are also likely scenarios in potential EDC litigation.

Finally, the case is telling because the plaintiff's arguments rested on meager scientific evidence and involved a new purported disease, "multiple chemical sensitivity," on which little research had been conducted. Perhaps recognizing her slim chance of establishing causation, the plaintiff implored in a pre-trial motion: "Given the dearth of research on the neurotoxic effects of fragrances and fragrance chemicals, what is a plaintiff to do?"[296] The court responded sardonically in its opinion: "Wait. When a plaintiff can't prove her case with reliable scientific evidence, she can't prove her case."[297]

Fragrances and EDCs both fall into the same category of diffuse exposures, non-signature diseases, and subtle effects. While the scientific evidence linking exposure to harm is stronger for EDCs than for fragrances, *Sanderson* suggests that the prospects for successful EDC litigation, resting on similar facts of wide exposure to chemicals, inability to identify defendants, and controversial scientific linkages, are limited. *Sanderson* also suggests that the courts will not allow plaintiffs to proceed without substantial evidence of causation even where the courts recognize that such evidence will be difficult, if not impossible, to obtain.

D. Drawbacks of Litigation for Addressing EDC Risks

Despite the current practical difficulties with bringing an EDC tort suit, a central normative question is whether the legal system should encourage tort suits over EDCs as a

295. *Id.* at 989-992. The court stated that the plaintiff was not entitled to use the alternative liability doctrine because she had not joined all potential tortfeasors. She was not entitled to use the market-share liability doctrine because the products were not fungible and because she had failed to join a substantial share of the market. *Id.*

296. *Id.* at 1003.

297. *Id.*

means to compensate injured individuals, force EDC manufacturers to internalize their costs, and fill potential gaps in regulation of EDCs. Should courts be as hesitant as they currently appear to be regarding relaxing causation and identification requirements in non-DES cases? Should they be more willing to shift the burden of proof to manufacturers of suspected toxic chemicals to show that their products did not cause harm?

If the effects of EDCs are subtle and causation is hard to prove, proponents of less stringent tort laws might argue, the legal system should adapt to those scientific realities rather than block the courthouse door to injured parties. Indeed, some analysts have made this type of argument in calling for a plaintiff-favorable response from the legal system to the dangers posed by EDCs. Mary O'Brian, a toxics and risk assessment specialist, asserts that "the legal framework must reflect, rather than deny, scientific reality regarding toxic chemicals"[298] and that "litigation must creatively challenge the existing tension between our legal framework for toxics use and science."[299] Other scholars have called for burden-shifting to defendants in toxic tort cases. Wendy Wagner of Case Western Law School argues that when lack of causation evidence results from the manufacturer's failure to test a product, rather than from inherent limits of scientific inquiry, the plaintiff should be entitled to a rebuttable presumption of causation.[300]

Instituting major changes in tort law for EDC cases is unwise, however, on several grounds. If legal rules were relaxed so that a plaintiff did not have to eliminate confounding factors or identify the specific EDC that caused harm, large damage awards might result. Consequently, manufacturers may be overdeterred and beneficial chemicals might be withdrawn from the market without a demonstration that they caused any harm. While burden-shifting

298. O'Brian, *supra* note 6, at 332

299. *Id.*

300. Wagner, *supra* note 274, at 834-836. Wagner draws a useful distinction between inherent scientific uncertainties, which she calls "trans-science," and scientific uncertainties that could have been resolved by manufacturer testing. But her argument would allow a plaintiff to sue almost any manufacturer of any toxic chemical if the plaintiff could not identify the cause of her harm, with the burden of proof shifting to those defendants whom the court determines have not adequately tested their product.

may be appropriate if the plaintiff can identify the specific chemical that caused a health problem (paralleling the DES cases), plaintiffs should not be allowed to sue all or nearly all chemical manufacturers, and shift the burden of proof to them, on the mere assertion that EDCs in general probably caused a harm.

Studies linking EDCs to health and reproductive disorders in animals and humans are cause for concern, but it does not necessarily follow that we should turn to the courts to fashion a remedy. Litigation is not the answer to every toxic risk. There is a fundamental unfairness inherent in imposing liability without a strong showing of causation, and arguments for deterrence, risk-spreading, or novel theories of liability should come into play only once that showing is made. As the California district court held in *Sanderson*:

> Courts are ill-equipped to conduct trials of entire industries, and individual plaintiffs in a private action have no right to put entire industries on trial. Private cases and controversies must sweep more narrowly, catching within the litigation net only those persons whom the plaintiff can link to the harm that has befallen her. That application of these principles may leave an injured person without a remedy in tort is no objection, because the tort system is not designed to provide compensation for every injury.[301]

Compared to litigation, regulatory responses better address diffuse dangers from innumerable chemicals in the environment (though regulations admittedly do not compensate harmed individuals). They can be implemented based on scientific data that a chemical may be harmful to the human population, whereas litigation depends on much more substantial proof that a product actually caused harm to a specific individual.[302] Furthermore, EDCs are similar to

301. 950 F.Supp. at 1003.

302. Regulatory measures could be based on animal tests of EDCs, whereas toxic tort litigation often is not successful until statistically-significant evidence of a danger to the human population has been collected. In the asbestos context, for example, the need for such evidence meant that litigation was not successful until decades after risks from asbestos were first identified, with dangerous exposures occurring in the meantime. *See* Wendy E. Wagner, Note, *Trans-Science in Torts*, 96 YALE L. J. 428, 428 (1986).

other substances, such as air or water pollutants, to which the legal system has responded mainly through regulation rather than litigation: the effects of EDCs are subtle, there is a myriad of manufacturers and exposure pathways, and the entire population is continually exposed to small amounts of EDCs through diet and environmental agents. Because EDCs cross state and even international boundaries, national regulation, rather than a patchwork of state tort laws, is the more appropriate legal response. Although regulatory standard-setting is by no means perfect,[303] regulatory measures could address EDC risks in a more targeted manner than is within the capabilities of the courts.

Litigation over EDCs, on the other hand, could result in scattershot tort damages in which the most serious EDC risks might not be addressed because they may be harder to link to concrete injuries. Litigation would be an *ex post* approach to the EDC problem because it involves claims for compensation for harm that has already occurred. To be sure, damage awards can also deter manufacturers in a prospective fashion, but it might take decades of litigation, proceeding chemical by chemical, before public health is protected to any measurable extent.

While regulation appears to be the preferable route for responding to EDC risks, it would be most effective if Congress acted to reform key aspects of our toxics regulatory system. Existing statutes are blunt instruments, and Congress should provide the EPA with better statutory tools if the agency is to address what could be a significant new type of toxic risk.

VI. PROPOSALS FOR REFORM

It is beyond the scope of this article to outline comprehensively a new statutory/regulatory regime for EDCs. Designing a new regulatory architecture for EDCs is perhaps premature given the recent nature of the science. To some extent, creating a better system for responding to EDC risks would entail overhauling the way all toxic chemicals are regulated, as EDCs would probably be regulated under the

303. See *infra* Part IV for a discussion of problems in regulation of toxic chemicals.

same statutes as other toxics. Other analysts have addressed the issue of reforming toxics regulation in general,[304] and the information that is coming to light about EDCs provides one more argument for revisiting the fundamental assumptions and bases of U.S. toxics policy. Ideas for new legal responses to EDCs are likely to progress as the scientific research progresses, but some general suggestions may be proposed even at this stage.

A. Prospects for Congressional Action

To avoid the limitations discussed previously related to rule-making under existing statutes, congressional action would be necessary. The prospects for further congressional involvement in the EDC issue are uncertain, however. On the one hand, Congress moved swiftly (within four months of the publicity related to *Our Stolen Future*) to mandate the endocrine disruptor screening programs in the FQPA and the SDWA amendments of 1996. On the other hand, these provisions were research-oriented and did not provide the EPA with new regulatory authority. Some of the congressional proponents of further endocrine disruptor research cited concerns about increasing rates of breast cancer in their states,[305] and especially if endocrine disruption is viewed as a women's and children's health issue, it could rise once again on the legislative agenda.

If Congress were to act, one positive approach would be to use the data from the screening and testing efforts that will occur over the next few years to legislate restrictions on those EDCs deemed to be most hazardous. In the few cases where there have been major additions to lists of regulated toxic substances, they have occurred through congressional action, not agency rule-making procedures. In 1984

304. *See, e.g.*, Dernbach, *supra* note 144. *See also* Carl B. Meyer, *The Environmental Fate of Toxic Waters, the Certainty of Harm, Toxic Torts, and Toxic Regulation*, 19 ENVTL. L. 321 (1988).

305. *See* 141 Cong.Rec. S17749-S17752 (daily ed. November 29, 1995) (dialogue between Senator Moynihan (D-NY) and Senator D'Amato (R-NY) on endocrine disruption, with mention of elevated breast cancer rates on Long Island). These two Senators were joined by an influential House Democrat, Henry Waxman (D-CA), in supporting endocrine disruptor research. *See* 142 Cong. Rec. H10960 (daily ed. September 24, 1996) (Waxman explaining support for the Estrogenic Substances Screening Program in FQPA and SDWA Amendments).

amendments[306] to the Resource Recovery and Conservation Act (RCRA),[307] for example, Congress directed the EPA to change its testing procedure for toxicity, leading to the addition of twenty-five toxins to the list of fourteen that were already regulated under RCRA.[308] In the 1990 Clean Air Act Amendments, Congress directed the EPA to develop a broader regulatory program for hazardous air pollutants and listed in the statute the 189 pollutants to be regulated.[309] If Congress were to develop a statutory list of the most hazardous EDCs, with accompanying restrictions, it would speed the process of protecting against EDC risks.

B. Right-to-Know Provisions

The EDSTAC has recommended a broad public outreach strategy as the screening and testing program for EDCs proceeds.[310] This program includes public notification of the testing process and test results, public input into nominating chemicals for screening, and information releases tailored to specific groups, such as farm workers and environmental justice organizations, who may be particularly affected by EPA decisions.[311] Public notification and procedural openness are essential to develop support among various stakeholders for the screening and testing program.

More broadly, Congress and the EPA should consider listing endocrine disruptors under the Toxic Release Inventory (TRI) of the Emergency Planning and Community Right-to-Know Act (EPCRA).[312] The TRI, which is compiled

306. Hazardous and Solid Waste Amendments, Pub. L. No. 98-616, 99 Stat. 3221 (1984).

307. 42 U.S.C. §§ 6901-6992k (1995).

308. *See* David Montgomery Moore, *The Toxicity Characteristic Rule for Hazardous Waste Determination: Has EPA Satisfied Congress' Mandate?*, 7 TUL. ENVTL. L.J. 467, 468 (1994). The change to EPA's toxicity testing procedure is at 42 U.S.C. § 6921(g) (1994).

309. 42 U.S.C. § 7412(b)(1) (1994).

310. EDSTAC REPORT, *supra* note 10, at 7-19.

311. *Id.*

312. 42 U.S.C. §§ 11001-11050 (1995). The TRI is outlined in the Act at §§ 11022-11023. In a single rule-making, EPA was recently able to add 286 chemicals to the list of chemicals required to be reported under the TRI. *See* 40 C.F.R. pt. 372. The relative ease by which so many chemicals were added at one time may be attributed to the fact that the TRI is a reporting statute and does not impose any restrictions on chemical releases. A challenge to this rule-making was

from mandatory submissions of industry data, is the major database of pollutants released by U.S. industry each year. There is an emerging network of environmental, environmental justice, and public health groups focusing on endocrine disruption,[313] so intermediaries might be available to interpret TRI information and to call media attention to large releases of EDCs. Since the TRI covers only point-source discharges, however, its usefulness for detailing EDC risks might be limited.

California's Proposition 65 is a "right-to-know" law that could be even more effective against EDCs. Passed in 1986, it bars the discharge into drinking water of any chemical known to cause cancer or reproductive toxicity and requires warnings before exposing individuals to such chemicals through non-drinking water routes.[314] The California EPA has compiled a list of chemicals subject to the law, many of which are suspected EDCs,[315] and a combination of state investigation of reproductive toxicity through endocrine disruption and private citizen suits to enforce the law could be effective in addressing many EDC risks in California.

Proposition 65 involves burden-shifting. Under the law, dischargers of chemicals must show that the chemical poses "no significant risk" of human disease in order to avoid the warning requirement.[316] In contrast to its usual recalcitrance, industry has supported the California EPA's promulgation of standards for "no significant risk" because such standards provide bright-line rules for whether a warning is required.[317] Furthermore, there is strong evi-

rejected by a district court. *See* Nat'l Oilseed Processors Ass'n. v. Browner, 924 F.Supp. 1193 (D.D.C. 1996).

313. *See* KEYSTONE CENTER, CONVENING REPORT REGARDING THE FORMATION OF THE ENDOCRINE DISRUPTOR SCREENING AND TESTING ADVISORY COMMITTEE (Oct. 1996) <*www.epa.gov/opptintr/opptendo/keystone.htm*>.

314. Cal. Health & Safety Code §§ 25249.5-25249.6. (West 1992 & Supp. 1995).

315. OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT, CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY, CHEMICALS KNOWN TO THE STATE TO CAUSE CANCER OR REPRODUCTIVE TOXICITY, (May 15, 1998) <www.oehha.org/prop65/cas 1598.htm> (listing suspected EDCs such as heptachlor, chlordane, DDT, DDE, and dieldrin).

316. CAL. HEALTH & SAFETY CODE § 25249.10(c) (West 1992 & Supp. 1999).

317. According to David Roe, co-author of the initiative, "California managed to draw bright lines for more chemicals in the first twelve months of the Proposition 65 era than the federal government has managed to accomplish, under the supposedly omnibus Toxic Substances Control Act, in the previous twelve years."

dence that a major effect of Proposition 65 has been to prompt companies to reformulate products containing hazardous chemicals in order to avoid the warnings about cancer or reproductive toxicity.[318] Because such product reformulations have occurred on a nation-wide scale, not just for California,[319] this state "right to know" law has had a positive substantive impact across the country. A national toxics law modeled on Proposition 65 that involves burden-shifting could achieve similar results on a larger scale and may be effective in responding to EDC risks.

C. Burden-Shifting under TSCA

A related step that could be taken at the federal level to respond to EDC and other toxic risks is amending TSCA to shift the burden of proof to chemical manufacturers to demonstrate the safety of chemicals.[320] Currently, the burden is on the EPA to identify synthetic chemicals that are likely to present an "unreasonable risk," to require testing, and, relying on data submitted by the manufacturer, to make the decision on whether to restrict the chemical. Each step in this process is cumbersome and subject to delay, and in the meantime chemicals may be freely sold and distributed. This has led to a situation in which few chemicals are required to be tested and in which no toxicity information is available for 78% of the 12,860 chemicals that are used in quantities exceeding one million pounds per year.[321]

The placement of the burden of proof under TSCA is inconsistent with other statutes. Under FIFRA, for example, the burden is on manufacturers to demonstrate the safety

David Roe, *An Incentive-Conscious Approach to Toxic Chemical Controls*, 3 ECON. DEV. Q. 179, 181 (1989).

318. *See* Clifford Rechtschaffen, *The Warning Game: Evaluating Warnings Under California's Proposition 65*, 23 ECOLOGY L.Q. 303, 341-348 (1996).

319. *See id.* at 341.

320. Manufacturers are required to submit a Pre-Manufacture Notification ("PMN") for new chemicals, but they are not required to develop toxicity data prior to doing so. *See* EDSTAC REPORT, *supra* note 10, at 2-9. The EDSTAC states that in reviewing a notification for a new chemical, the EPA can use risk assessment models to predict likely toxic effects of the chemical, but the EDSTAC also acknowledges that the EPA "often drops review and gives approval for most chemicals." *Id.*

321. *See* Dernbach, *supra* note 144, at 28.

of pesticides prior to receiving a registration from the EPA.[322] Under the FDCA, drug manufacturers must obtain Food and Drug Administration approval prior to marketing new drugs.[323] Similarly, before a manufacturer is permitted to expose thousands or millions of people (many involuntarily) to chemicals whose connection to a health problem might be difficult to prove in an *ex post* tort suit, the manufacturer should be required by law to demonstrate the safety to the chemical to the EPA.

To be sure, every manufacturer of a risk-producing product should not be required to obtain governmental approval prior to sale. Chemicals can be distinguished, however, from products such as power tools, sporting goods, or industrial equipment – products which carry some risks but which normally do not require governmental approval. Individuals do not necessarily know that they have been exposed to chemicals, for example, and chemical manufacturers can generally escape liability for harm because of difficult causation issues.[324] Tort suits alone provide an insufficient incentive for manufacturers to develop toxicity information because it is precisely the lack of such information that may force dismissal of a suit on the grounds of lack of causation evidence.[325] Requiring the development of toxicity information before sale would go a long way toward redressing the imprudence of current regulatory approaches to toxic chemicals.

322. Because the burden of producing safety information is on manufacturers under FIFRA, the EPA has much more data on the hazards of a relatively small number of pesticides than it has on the thousands of industrial chemicals produced in much larger quantities (which are regulated under TSCA). *See* EDSTAC REPORT, *supra* note 10, at 7-16.

323. 21 U.S.C. § 355(a) (1998). As part of the pre-market approval process, applicants must submit reports "to show whether or not such drug is safe for use and whether such drug is effective in use." *Id.* at § 355(b). This is exactly the type of information that should be required of chemical manufacturers under TSCA.

324. As Troyen Brennan put it, in most mass product liability cases "the plaintiffs worked with, or bought, the product. . . . Each plaintiff has encountered the product in a manner that can be documented. The same documentation is not possible in environmental torts unless the pollution leaves a residue." Brennan, *supra* note 278, at 46.

325. *See* Wagner, *supra* note 274, at 774-776. While a "duty-to-test" exists at common law, most courts use it as a means to impose liability once a plaintiff has shown that a product caused his or her harm. If the plaintiff cannot make the initial showing of causation, however, there is usually no "duty-to-test" liability. *Id.* at 803-805.

Although they have not outlined their proposals in detail, several endocrine disruption researchers have proposed shifting the burden of proof about chemical risks to chemical manufacturers. The authors of *Our Stolen Future* argue that "emerging evidence about hormonally active chemicals should be used to identify those posing the greatest risk and to force them off the market and out of our food and water until studies can prove their impact to be trivial."[326] Mary O'Brian states flatly that "[u]se of chemicals should be suspect."[327] She adds that "[s]ociety must end the failed process of estimating how much of each toxic chemical the world can stand, and orient legislation and litigation around the feasible process of determining how little toxic chemical use is necessary."[328]

Under a fair burden-shifting plan, Congress should require that toxicity information for all chemicals be submitted to the EPA by a specified date, with a prohibition upon manufacture or sale as the penalty for non-compliance. Though it would be politically more difficult, Congress could also prohibit manufacture or sale after a certain date unless EPA reviews the toxicity information and gives approval to the chemical as not posing an "unreasonable risk" to human health. Congress would need to provide the EPA with additional resources to sort through the submitted data efficiently and to make scientifically sound determinations on thousands of chemicals. Of course, EPA determinations on whether a chemical could be manufactured or sold based on the submitted data could still be challenged in court, but at least manufacturers would have less incentive to delay.

As discussed in Part V of this article, courts should be reluctant to shift the burden of proof onto defendants in potential EDC lawsuits. Such burden-shifting would permit plaintiffs to sue dozens of chemical manufacturers and force them to prove that they did not cause the plaintiff's health or reproductive disorder. Burden-shifting in the regulatory context, in contrast, would require only that manufacturers demonstrate that a product will not cause

326. COLBORN ET AL., *supra* note 3, at 219.
327. O'Brian, *supra* note 6, at 354.
328. *Id.* at 358.

significant harm to the human population. This is much less onerous than repeatedly requiring companies to disprove their link to injuries of specific individuals.

On October 9, 1998, the Chemical Manufacturers Association (CMA), the Environmental Defense Fund (EDF), and the Clinton Administration announced a six-year program to test the health and environmental effects of 2,800 chemicals produced in excess of 1 million pounds per year.[329] The program, which implements many of the recommendations of EDF's 1997 study, *Toxic Ignorance*, is expected to cost the chemical industry $500-$700 million.[330] The program is additional to the 15,000 chemicals that will be screened for endocrine disrupting effects under the EDSTAC recommendations.[331] That the chemical industry is undertaking voluntary testing for product safety (albeit under the threat of EPA-ordered tests)[332] is a very positive step. It will be a change from past industry practices of claiming that chemicals are safe without conducting safety research. The testing program has a number of safeguards to ensure that the testing will be unbiased and that the results will be transparent.[333] Voluntary testing should not replace reforming TSCA, however, and eventually, testing should be expanded to include lower production volume chemicals, so that the burden of proof on chemical safety becomes fully shifted to the chemical industry.

VII. CONCLUSION

The emerging research into EDCs has raised disturbing warnings about new kinds of health risks from chemicals. The data on falling sperm counts and other fertility problems, the DES experience, and reproductive disorders in animals exposed to suspected EDCs all suggest that there

329. "Industry to Test 2,800 Major Chemicals for Health, Environmental Effects," EDF News Release, October 9, 1998 <www.edf.org/pubs/newsreleases/1998/oct/b%5Fcma.html> [hereinafter EDF News Release].

330. *Id.*

331. *See* Claudia H. Deutsch, *Chemical Industry to Spend $1 Billion to Assess Product Safety*, N.Y. TIMES, January 27, 1999, at A14.

332. *See* EDF News Release, *supra* note 329. Chemical manufacturers will have 13 months to volunteer their products for testing, after which EPA will mandate testing through a TSCA test rule. *See id.*

333. *Id. See also* Deutsch, *supra* note 331.

may be real risks to the human population through endocrine disruption. That these risks are not yet well-defined does not mean that they should be ignored. The susceptibility of fetuses to endocrine disruption, the potential impact on human reproduction, and the fact that EDCs permeate the environment, all warrant caution in the face of uncertain risks. Endocrine disruption must be taken seriously as a public health issue.

While there is considerable scientific controversy surrounding EDCs, it is clear that EDCs do not fit neatly into traditional scientific and legal approaches to toxic chemicals. EDC characteristics such as low-dose effects, numerous exposure routes, and intergenerational impacts make risk assessment difficult. Consequently, crafting any type of legal response to EDCs will be extraordinarily problematic. Indeed, the current state of scientific knowledge regarding EDCs is probably too rudimentary to support legal action.

Because there are so many obstacles to fashioning a response to EDCs, however, the legal and regulatory communities should closely follow the scientific research and begin to consider the legal tools that may be applied as the scientific evidence grows stronger. More research into EDC risks is necessary, but legal analysts should not be excluded from the scientific and policy discourse because of the infancy of the research, nor because of an ill-advised desire to reach scientific clarity before legal solutions are considered. After all, the "endless pursuit of scientific knowledge can be dangerous in a regulatory system in which toxic chemicals are deemed innocent until proven guilty."[334]

At present, both litigation and regulation appear to be blunt instruments to respond to EDC risks. Indeed, litigation over EDCs appears very unlikely to succeed, let alone effectively deter EDC manufacturers. Regulation, with all its flaws, is the preferable route to protect public health, and regulatory capabilities would be greatly enhanced if Congress acts to reform the major toxics statutes.

The application of law in contexts of scientific uncertainty is frequently controversial, inevitably leading to claims of under-regulation from the left and over-regulation from the

334. FAGIN & LAVELLE, *supra* note 217, at 229.

right. When the scientific community begins to research a new type of toxic risk, the legal community should avoid a premature response unsupported by the data, but it should not let scientific uncertainty prevent sensible steps to protect public health.