

DO YOU WANT TO BET YOUR CHILDREN'S HEALTH ON
POST-MARKET HARM PRINCIPLES? AN ARGUMENT
FOR A TRESPASS OR PERMISSION MODEL
FOR REGULATING TOXICANTS¹

BY CARL F. CRANOR²

I. INTRODUCTION

Scientific discoveries can lead to paradigm shifts, not only within science, but also in society at large.³ In turn, this can modify how citizens respond to the world around them. Recent developments in monitoring toxicants in United States citizens and developmental toxicology provide the background for rethinking regulatory strategies for toxic substances.⁴

There is now considerable evidence that substantial numbers of human-made chemicals, many of them known toxicants, are present in the bodies of United States citizens. In addition, these toxicants are also highly likely to be present in the womb, and thus, likely to be found in the bodies of developing fetuses and neonates. The chemical contamination of a mother's body will in all likelihood be shared with her child *in utero* or while nursing. Such exposure to toxicants is often greater on a per-weight basis than a mother's exposure to the same substances. Where there is comparable exposure, adverse effects on children tend to be greater than

1. Previous versions of this Article were presented at the Southern California Law and Philosophy Group, Villanova University Law School, Oxford University, and the University of California Environmental Toxicology Program. They were commented on by a number of colleagues to whom the Author is grateful: Tracey Woodruff, Coleen McNamara, Stephen Munzer, Sharon Lloyd, Chris Naticchia, Marshall Cohen, Craig Ihara, Matthew Liao, Julian Savulescu, David Eastmond, and Mary Lyndon. The author is deeply grateful to Philippe Grandjean for an invitation to the Faroe Islands Conference on Prenatal Programming and Toxicity and for introducing him to these issues.

2. Carl Cranor is Professor of Philosophy at the University of California, Riverside. He specializes in philosophic issues arising at the interface of science and the law. His books are two major publications on the subject. *See* CARL CRANOR, *REGULATING TOXIC SUBSTANCES: A PHILOSOPHY OF SCIENCE AND THE LAW* (Oxford Univ. Press 1997) (1993); CARL CRANOR, *TOXIC TORTS: SCIENCE, LAW AND THE POSSIBILITY OF JUSTICE* (Cambridge Univ. Press 2007) (2006).

3. *See* THOMAS KUHN, *THE ESSENTIAL TENSION: SELECTED STUDIES IN SCIENTIFIC TRADITION AND CHANGE* 233-37 (Univ. of Chi. Press 1977) (noting methodological results of scientific developments).

4. As used in this Article, a "toxicant" is the toxicologists' preferred term for a substance that is toxic. A "toxin" usually refers to poisons such as snake venom.

in adults. Developing organ systems can be disrupted and permanently damaged as a result of fetal and neonatal exposure to exogenous insults.⁵ In some cases, lifelong adverse effects will result. Moreover, some diseases such as cancer are exacerbated or appear sooner as a result of early exposures.

The main legal strategy for regulating toxicants in the United States is by means of post-market laws, which allow substances to enter the stream of commerce without required testing or agency approval. If substances pose health problems, a government agency must show harm or risks of harm to require regulation. These laws, however, tend to function poorly in protecting adults from harmful toxic effects. They will function much worse in the protection of developing children. Under the existing United States laws which apply to most substances, firms in effect have a right to expose members of the public, particularly children, to toxicants until the government satisfies a high scientific and legal burden of proof to show that exposures cause harm or pose risks of harm. Such laws can create serious health risks before toxicants are reduced or eliminated. The difficulties in identifying and controlling toxic substances result from the legal requirements of post-market laws. In part, they rest on requirements to show harm or risks of harm to developing children.

The above discussion highlights the need for a new regulation model. This Article proposes that the United States move toward either a "trespass" or a "permission" model for regulation. A trespass model would hold chemical invasions in children or adults as *one kind of wrong* with any resulting risks or harms viewed as *additional wrongs*. Firms seeking to market products should test substances to determine whether they can invade human (mammalian) bodies and cross the placenta or be present in breast milk. If they can, companies should further test them on non-human systems to ensure, to the satisfaction of a government agency, that products will not pose significant harms or risks of harm to developing children. With a permission model, like the European Union's REACH legislation, citizens have assurances, based on non-human tests and certified by government agencies, that products in the market will not pose harm or significant risks

5. As used in this Article, an "exogenous insult" refers to an insult from outside the entity's body.

of harm to adults or developing children.⁶ As the aphorism describing REACH puts it: No (safety) data, no market.

Some might not initially be persuaded of the need for a new model for regulation. They should ask themselves whether they want to bet the health of their children or their grandchildren on post-market risk or harm-based legal structures. This is a dicey bet to make. This Article argues that these laws function poorly in preventing harm to developing children. Thus, this Article suggests a new paradigm for the legal regulation of chemical substances that can potentially invade human bodies, often exposing adults, developing fetuses, and newborns to health risks.

II. THE CENTERS FOR DISEASE CONTROL BIOMONITORING PROJECT: THE PRESENCE OF CHEMICALS IN CITIZENS' BODIES

Recent studies conducted by the biomonitoring project at the Center for Disease Control (CDC) have revealed that human bodies contain varying levels of manmade environmental chemicals.⁷ The CDC's aim is to measure the amount of a substance that can be scientifically identified, and for which good exposure protocols can be written to test for it in the blood and urine of a randomly selected group of United States citizens in order to obtain some more systematic information about human exposure.⁸

The overall purpose of the research is to provide unique exposure information to scientists, physicians, and health officials to help prevent disease that results from exposure to environmental chemicals. Specific public health uses of the exposure information in the CDC's Report are:

- To determine which chemicals get into Americans and at what concentrations.
- For chemicals with a known toxicity level, to determine the prevalence of people with levels above those toxicity levels.

6. See Registration, Evaluation, Authorization and Restriction of Chemicals (REACH), No. 1907 (2006) (U.K.) (providing text of legislation establishing a European Chemicals Agency).

7. Biomonitoring is "the direct measurement of people's exposure to toxic substances in the environment by measuring the substances or their metabolites in human specimens, such as blood or urine." See Dep't of Health and Human Servs., Ctrs. for Disease Control and Prevention, National Biomonitoring Program, <http://www.cdc.gov/biomonitoring/> (defining biomonitoring).

8. Dep't of Health and Human Servs., Ctrs. for Disease Control and Prevention, Nat'l. Ctr. for Env'tl. Health, Third National Report on Human Exposure to Environmental Chemicals (Sept. 11, 2007), <http://www.cdc.gov/exposurereport/report.htm> (stating project's goal).

- To establish reference ranges that can be used by physicians and scientists to determine whether a person or group has an unusually high exposure.
- To assess the effectiveness of public health efforts to reduce exposure of Americans to specific chemicals.
- To determine whether exposure levels are higher among minorities, children, women of childbearing age, or other potentially vulnerable groups.
- To track, over time, trends in levels of exposure of the population.
- To set priorities for research on human health effects.⁹

The most recent CDC report on biomonitoring shows that United States citizens' bodies contain at least 148 manmade substances.¹⁰ This raises some concern about their effects. A CDC report due out in summer 2008 will show that approximately 270 substances can be measured in the human body.¹¹ This increase in numbers does not mean that there are more substances in citizens' bodies, but that more are now identifiable by reliable exposure markers. As measurements are created, standardized, and completed, it seems highly likely that more substances will be found in citizens' bodies. A Canadian report describing the results of testing on eleven people found many chemicals in measurable levels in their bodies.¹² Another recent report by the Body Burden Work Group & Commonwealth Biomonitoring Resource Center, which conducted the biomonitoring of thirty-five ordinary citizens from seven different states within the United States, found that all tested positive for at least seven of twenty chemicals.¹³ Of these subjects, thirty-three contained phthalates, all thirty-five had polybrominated diphenyl ethers (a fire retardant in furniture and electronic equip-

9. DEP'T OF HEALTH AND HUMAN SERVS., CTRS. FOR DISEASE CONTROL AND PREVENTION, THIRD NATIONAL REPORT ON HUMAN EXPOSURE TO ENVIRONMENTAL CHEMICALS 3 (2005) [hereinafter REPORT ON HUMAN EXPOSURE], <http://www.cdc.gov/exposurereport/pdf/thirdreport.pdf> (listing research's agenda).

10. *Id.* at 1-2 (providing list of chemicals).

11. Email from Larry Needham, Director of the Ctrs. for Disease Control Biomonitoring Project (Sept. 10, 2007) (on file with author) (indicating that anticipated release date of next CDC biomonitoring report is summer of 2008).

12. ENVTL. DEF., TOXIC NATION: A REPORT ON POLLUTION IN CANADIANS 1 (Nov. 2005), http://www.toxicnation.ca/files/toxicnation/report/PATN_English.pdf (detailing mix of toxic chemicals found in every person tested).

13. BODY BURDEN WORK GROUP & COMMONWEAL BIOMONITORING RES. CTR., IS IT IN US? CHEMICAL CONTAMINATION IN OUR BODIES: TOXIC TRESPASS, REGULATORY FAILURE AND OPPORTUNITIES FOR ACTION 1-52 (2007) [hereinafter CHEMICAL CONTAMINATION], <http://www.isitinus.org/documents/Is%20It%20In%20Us%20Report.pdf> (detailing study methods and findings).

ment), and the thirty-three who provided urine samples had phthalates and bisphenol A in their bodies.¹⁴ The last three substances are of substantial concern in the scientific community, but none has received much regulatory attention.

The presence of manmade industrial chemicals in citizens' bodies does not necessarily mean that they pose risks or are harmful. Their mere presence is a wrong on a trespass model, and should be viewed with concern. Moreover, there has not been appropriate research on the effects of low levels of exposure and on risks of harm to children or developing fetuses. As if to confirm this point, an expert panel of the Food and Drug Administration (FDA) recommended that the FDA recall cold and flu remedies for young children on grounds that they did not seem to be effective and that they had not been properly tested for use by children.¹⁵

The CDC is careful about what it infers from the biomonitoring. For example, with respect to one class of pesticides, the CDC Report notes that "[t]he health effects of exposure to organochlorine pesticides on the general population at current levels of exposure are unknown."¹⁶ The report contains similar comments about many of the substances it considers; for some chemicals it simply does not address this issue.¹⁷ Yet, there are substances of concern. Some are known or probable human carcinogens, some are known or probable reproductive or developmental toxicants, some are only possible toxicants, and some are perhaps unknown for their toxicity, but are on the list simply because they are suspected of posing risks to human health at certain concentration levels.

14. *Id.* at 18 (providing results).

15. Gardiner Harris, *Ban Sought on Cold Medicine for Very Young*, N.Y. TIMES, Sept. 29, 2007, available at <http://www.nytimes.com/2007/09/29/health/29fda.html> (describing ineffectiveness of cold medicines used in children as well as dangers posed); Rob Stein, *Cold Remedy Makers Pull Infant Medicines*, WASH. POST, Oct. 11, 2007, available at 2007 WL 19957065 (describing movement to ban cold medicines for children under six); Gardiner Harris, *Makers Pull Infant Cold Medicines*, N.Y. TIMES, Oct. 11, 2007, available at <http://www.nytimes.com/2007/10/11/health/11cnd-cold.html?hp> (describing voluntary withdrawal of cold medicines from market); Editorial, *Children and Cold Medicines*, N.Y. TIMES, Nov. 3, 2007, available at <http://www.nytimes.com/2007/11/03/opinion/03sat3.html> (describing efforts on part of drug companies to soften impact of negative studies).

16. See REPORT ON HUMAN EXPOSURE, *supra* note 9, at 309 (describing organochlorine pesticides).

17. See *id.* (discussing other chemicals).

III. RISKS TO FETUSES AND INFANTS

A. Women's Chemical Contamination Will Be Shared with Developing Fetuses and Nursing Newborns

As already noted, many substances that are in a woman's body will be transmitted to the unborn fetuses or to newborns that are being nursed.¹⁸ Some might have thought that the womb is a sheltered, capsule-like environment safe from the intrusions and dangers of the outside world. Apparently, this was a widely accepted view until the mid-1960s. As Needleman and Bellinger wrote in 1995:

Only three decades ago, the prevailing image of the womb was that of a time capsule with a short lease, relatively impermeable to circulating drugs or toxicants. The mother's body was considered an altruistic reservoir, prepared to sacrifice itself to the fetus's sustenance.¹⁹

Moreover, the layer of biological material known as the placenta was once was called "the *placental barrier* because at one time it was believed to afford great protection to the embryo and fetus. [Scientists] now know that the degree of protection is often modest at best, and that instead of being a barrier, the placental membrane acts more as an ultrafilter."²⁰ In the 1960s, children born to women exposed to methyl mercury and thalidomide raised the first alarms.²¹ Female fetuses' exposure to diethylstilbestrol in the womb was soon found to cause various reproductive tract cancers when the women reached their early twenties.²² Subsequently, to-

18. Philippe Grandjean et al., *The Faroes Statement: Human Health Effects of Developmental Exposure to Chemicals in Our Environment*, 102 BASIC & CLINICAL PHARMACOLOGY & TOXICOLOGY 73, 74 (2007), available at <http://www.blackwell-synergy.com/doi/pdf/10.1111/j.1742-7843.2007.00114.x> (describing susceptibility of infants to toxic exposure).

19. HERBERT L. NEEDLEMAN & DAVID BELLINGER, PRENATAL EXPOSURE TO TOXICANTS ix (Herbert L. Needleman & David Bellinger eds., Johns Hopkins Univ. Press 1994) (describing outdated views on susceptibility of infants in womb to toxic substances). A similar view was reported to this Author by Prue Talbot, a University of California, Riverside, developmental biologist.

20. RONALD D. HOOD, PRINCIPLES OF DEVELOPMENTAL TOXICOLOGY REVISITED, DEVELOPMENTAL AND REPRODUCTIVE TOXICOLOGY: A PRACTICAL APPROACH 8 (Ronald D. Hood ed., CRC Press 2d ed. 2006) (introducing fundamental toxicology concepts). See generally E.M. FAUSTMAN & P. RIBEIRO, PHARMACOKINETIC CONSIDERATIONS IN DEVELOPMENTAL TOXICITY, DEVELOPMENTAL TOXICITY: RISK ASSESSMENT AND THE FUTURE (Ronald D. Hood ed., 1990).

21. See NEEDLEMAN & BELLINGER, *supra* note 19, at ix (positing the event that brought attention to effects of toxicity to infants in womb).

22. JAMES L. SCHARDEIN & OREST T. MACINA, HUMAN DEVELOPMENTAL TOXICANTS: ASPECTS OF TOXICOLOGY AND CHEMISTRY 286-87 (CRC Press 2007) (detailing

bacco smoke, alcohol, polychlorinated biphenyls, vitamin A, and numerous other drugs and chemicals have been identified as posing risks of harm. A recent textbook author states that “[i]t is clearly evident that there really is no placental barrier per se: The vast majority of chemicals given the pregnant animal (or woman) reach the fetus in significant concentrations soon after administration.”²³

Whether a given substance will pass through the placenta depends on a variety of factors, including its molecular size (the smaller, the more likely), its charge (uncharged molecules more readily cross), its fat solubility (the more soluble, the more likely to cross), its degree of ionization (the less ionization, the more likely to cross), and its molecular complexity (the less complex, the more likely to cross).²⁴ There are a number of developmental toxicants, so classified by different authors or agencies for different purposes.²⁵

Developmental toxicologists classify adverse developmental effects into four categories: “death [of the fetus], malformation, growth retardation, and functional deficit.”²⁶ Fetal death or malformations tend to be obvious, although the cause may not be clear, but growth retardation and the various kinds of functional deficits are more subtle, consequently being more difficult to detect and to ascribe causation.

At a recent scientific conference in the Faroe Islands, scientists presented results of the current understanding of the effects of

a wide range of reproductive problems in males and females with DES intake during pregnancy); *see also* *Sindell v. Abbott Labs.*, 607 P.2d 924, 926 (Cal. 1980) (describing danger of DES).

23. JAMES L. SCHARDEIN, *CHEMICALLY INDUCED BIRTH DEFECTS* 5 (Marcel Dekker, Inc. 3d ed. 2002) (rejecting notion of placental barrier with respect to most chemicals).

24. HOOD, *supra* note 20, at 8-9 (discussing factors influencing whether placental barrier allows chemical passage).

25. *See* Philippe Grandjean & Phillip J. Landrigan, *Developmental Neurotoxicity of Industrial Chemicals*, 368 *LANCET* 2167, 2168-72 (2006) (estimating 200 neurotoxins alone based only upon human studies); JAMES L. SCHARDEIN & OREST T. MACINA, *Preface to HUMAN DEVELOPMENTAL TOXICANTS: ASPECTS OF TOXICOLOGY AND CHEMISTRY* (Taylor & Francis 2007) (estimating that there are about 70 developmental toxicants and reviewing 50 present developmental toxicants arbitrarily selected based upon their commercial impact on public health considerations, availability of quality data in humans and animals, and representations of various classes of development toxicity).

26. *See* KIM N. DIETRICH & DAVID BELLINGER, *Assessment of Neurobehavioral Development in Studies of the Effects of Prenatal Exposure to Toxicants*, in *PRENATAL EXPOSURE TO TOXICANTS: DEVELOPMENTAL CONSEQUENCES* 57 (H. L. Needleman & D. C. Bellinger eds., Johns Hopkins Univ. Press 1994) (identifying four classifications for adverse developmental effects).

chemical pollutants on human fetal and mammalian developmental processes.²⁷ The conference results were summarized as follows:

Three aspects of children's health are important in conjunction with developmental toxicity risks. First, the mother's chemical body burden will be *shared with her ff]etus or neonate*, and the child may, in some instances, be exposed to *larger doses relative to the body weight*. Second, *susceptibility to a wide range of adverse effects is increased* during development, from preconception through adolescence, depending on the organ system. Third, developmental exposures to [toxicants] *can lead to life-long functional deficits and manifestations of increased disease [risks]*.²⁸

In utero exposure to pollutants can yield adverse consequences over a lifetime.²⁹ Animal studies show that changes produced during development can be permanent and in some cases transmitted to later generations.³⁰ There is some evidence for similar long-term consequences in humans.³¹ One dramatic example involves prenatal exposure to diethylstilbestrol (DES), which caused cancer in female children twenty years after birth, and now appears to affect male offspring by causing genital abnormalities.³² Other long-term effects may be far more subtle and more difficult to detect. Another very recent study found that:

High levels of serum *p,p'*-DDT predicted a statistically significant 5-fold increased risk of breast cancer among women who were born after 1931. These women were under 14 years of age in 1945, when DDT came into widespread use, and mostly under 20 years as DDT use peaked. Women who were not exposed to *p,p'*-DDT before 14 years of

27. See Prenatal Programming and Toxicity, Conference Programme (May 20-24, 2007), <http://www.pptox.dk/Programme/tabid/68/Default.aspx> (discussing susceptibility of fetal life and early infancy to environmental hazards).

28. See Grandjean, *supra* note 18, at 74 (emphasis added) (discussing effects of toxicants on developing human being).

29. See *id.* (summarizing aspects of toxicity to children).

30. See Grandjean & Landrigan, *supra* note 25, at 2168 (discussing animal studies).

31. See *id.* (discussing identification of neurological developmental problems in humans).

32. See SCHARDEIN & MACINA, *supra* note 22, at 287 (noting wide range of reproduction and developmental problems associated with pregnant intake of DES).

age showed no association between *p,p'*-DDT and breast cancer³³

Consequently, since women heavily exposed to DDT have not yet reached the age of fifty, the public health significance of their exposure may be quite large.³⁴ In addition, recent studies suggest that lead exposure early in life can contribute to neurological degeneration late in life, “a proportion of what has been termed ‘normal’ age-related cognitive decline may, in fact, be due to exposure to neurotoxins such as lead.”³⁵ The lead content in the bodies of persons may exacerbate such neurodegenerative conditions as Alzheimer’s disease, Parkinson’s disease, and Lou Gehrig’s disease.³⁶

Moreover, as the Faroe’s Statement emphasizes, children exposed *in utero* or during the immediate postnatal period may be much more susceptible to toxic insults than mature adults.³⁷ There are a variety of reasons for this, which will be discussed below.

Finally, once children are born and begin their own lives outside the womb, they will be subjected to most of the same toxic insults and invasions that adults are. Consequently, children have at least three major routes of exposures: (1) in the womb, (2) through nursing, and (3) from general environmental exposures following birth.

B. Developing Children Often Experience Greater Exposures

As the Faroe’s Statement indicates, toxicants can concentrate in placental cord blood, increasing toxic concentrations in a developing fetus to higher levels than in the mother’s body tissues.³⁸ If newborns are breast-fed, any toxicants that are lipophilic or soluble in body fats can have greater concentrations in breast milk than in

33. Barbara A. Cohn et al., *DDT and Breast Cancer in Young Women: New Data on the Significance of Age at Exposure*, 115 ENVTL. HEALTH PERSPECTIVES 1406, 1406 (2007) (noting results of DDT exposure investigation forecasting predictably of cancer, specifically focusing on young women exposed during peak of DDT usage).

34. *See id.* (noting danger of DDT exposure and future consequences).

35. *See* Brian S. Schwartz & Walter F. Stewart, *Lead and Cognitive Function in Adults: A Questions and Answers Approach to a Review of the Evidence for Cause, Treatment, and Prevention*, 19 INT’L REV. OF PSYCHIATRY 671, 671 (2007) (noting potential link between lead and neurodegenerative disease).

36. *See id.* at 685 (noting potential for exacerbating well-recognized neurological diseases of old age).

37. *See* Grandjean, *supra* note 18, at 73 (noting increased risk of harm from exposure in children).

38. *See id.* (finding developing fetus’ concentration of toxicants can reach higher levels than their mother’s).

the nursing mother's body. Toxicants in breast milk can be as much as one hundred times more concentrated than in the mother's body.³⁹ A toxic substance, such as lead, that is in the bones of a mother's body can be mobilized by the same processes that mobilize calcium for nursing (the "calcium stream"), resulting in substantial concentrations to nursing infants.⁴⁰

Newborns and small children have a number of features that can increase their exposures to toxicants. They have higher metabolisms and breathing rates,⁴¹ as well as "augmented absorption rates, and diminished ability to detoxify many exogenous compounds, relative to that of adults."⁴² Behaviorally, they tend to play close to the floor, increasing exposure to toxicants present in their environment. The pesticide Chlorpyrifos, for example, can be found in carpeting exposing children who play there. In addition, children tend to "mouth" most objects in their environment, to be more active, and to have higher fluid and food intake rates relative to their body weight than adults.⁴³

This is merely a sketch of some of the differences, but it indicates that developing fetuses and newborns have a number of physiological and behavioral characteristics that can increase their exposures to any toxicants.

C. Developing Children Often Have Greater Susceptibility

Developing fetuses, newborns, and young children exposed to low levels of toxicants tend to be more susceptible to adverse effects than older children or adults subject to the same exposure. For example, the blood-brain barrier that normally protects adults by keeping many exogenous toxicants out of the brain does not de-

39. See Grandjean & Landrigan, *supra* note 25, at 2168 (finding maternal breast milk passes lipophilic substances to infant at over 100 times mother's own concentrations).

40. David Bellinger & Herbert L. Needleman, *The Neurotoxicity of Prenatal Exposure to Lead: Kinetics, Mechanism and Expressions*, in *PRENATAL EXPOSURE TO TOXICANTS* 91-92 (Herbert L. Needleman & David Bellinger eds., Johns Hopkins Univ. Press 1994) (stating risk of breastfeeding children by mothers whose bodies are contaminated by toxicants, including lead).

41. See Mark D. Miller et al., *Differences Between Children and Adults: Implications for Risk Assessment at California EPA*, 21 INT'L J. TOXICOLOGY 403, 406 (2002) (reporting that higher metabolism and breathing rates in children make them more susceptible to toxicants).

42. See Grandjean & Landrigan, *supra* note 25, at 2168 (finding children are less able than adults to detoxify foreign substances).

43. See Miller et. al., *supra* note 41, at 405 (identifying greater susceptibility of children to toxicants than adults).

velop in children until about six months of age.⁴⁴ Moreover, the “brain continues to change throughout life . . . with possible age-specific periods of susceptibility to neurotoxicants.”⁴⁵ Consequently, any toxicants in the mother’s body that cross the placenta and invade the womb will expose the developing fetus and may not be prevented from entering the brain because the blood-brain barrier is not yet effectively functional.

In general, a developing child’s susceptibility to a wide range of adverse effects is increased during development, from pre-conception through adolescence, depending on the organ system. A textbook on developmental toxicity describes this point as follows:

Organisms tend to be significantly more sensitive to many adverse environmental influences during early developmental stages, although this differential may not be quite as universally applicable in mammals as was once thought Many tissues are undergoing rapid cell division, and the embryo, and to a considerable extent, the fetus, has much less capacity to metabolize xenobiotics than does the adult.⁴⁶

Moreover, “[t]he toxicology and pharmacology literature documents that children often react quantitatively and/or qualitatively differently to many toxins and drugs as compared to adults. In most organ systems, these differences amount to an increased susceptibility to many hazardous environmental chemicals.”⁴⁷ Adverse developmental effects in several organ systems illustrate this vulnerability.

[For example, t]he developing human brain is inherently much more susceptible to injury caused by toxic agents than is the brain of an adult. This susceptibility stems from the fact that during the 9 months of prenatal life, the human brain must develop from a strip of cells along the dorsal ectoderm of the fetus into a complex organ consisting of billions of precisely located, highly interconnected,

44. See Grandjean & Landrigan, *supra* note 25, at 2168 (stating blood-brain barrier does not develop in children until six months after birth).

45. See Schwartz & Stewart, *supra* note 35, at 672 (explaining that changes in brain continue for life).

46. HOOD, *supra* note 20, at 7 (stating that there are greater risks to mammals during developmental stages of life).

47. Luz Claudio et al., *Testing Methods for Developmental Neurotoxicity of Environmental Chemicals*, 164 TOXICOLOGY & APPLIED PHARMACOLOGY 1, 2 (2000) (finding children often react to toxicants differently than adults).

and speciali[z]ed cells. Optimum brain development requires that neurons move along precise pathways from their points of origin to their assigned locations, that they establish connections with other cells, both nearby and distant, and that they learn to communicate with other cells via such connections. All these processes have to take place within a tightly controlled time frame, in which each developmental stage has to be reached on schedule and in the correct sequence. Because of the extraordinary complexity of human brain development, windows of unique susceptibility to toxic interference arise that have no counterpart in the mature brain, or in any other organ. If a developmental process in the brain is halted or inhibited, there is little potential for later repair, and the consequences can therefore be permanent.⁴⁸

Moreover, the brain has reduced capacity to repair damage.⁴⁹

The immune system is similar to the brain in the precise, sequential nature of development and potential sensitivity to exogenous insults:

The development of the immune system results from a series of carefully timed and coordinated events during embryonic, fetal, and early postnatal life. There is evidence for a number of immunotoxic chemicals that exposure of pregnant animals at doses causing only transient effects in adults produces long-lasting or permanent immune deficits in their offspring. During critical developmental stages, the future immune system cells are increasing in number and becoming specialized in function.⁵⁰

The development of the immune system goes through several "discrete functional changes representing critical windows of differential vulnerability to toxicants."⁵¹ First, "early-life stages have in-

48. See Grandjean & Landrigan, *supra* note 25, at 2167-68 (observing complexity of brain's sensitive development state). See generally J. Dobbing, *Vulnerable Periods in Developing Brain*, in APPLIED NEUROCHEMISTRY 287-316 (A. N. Davidson & J. Dobbing eds., 1968); P. M. Rodier, *Developing Brain as a Target of Toxicity*, 103 ENVTL. HEALTH PERSP. 73, 73-76 (1995); D. Rice & S. Barone, Jr., *Critical Periods of Vulnerability for the Developing Nervous System: Evidence from Humans and Animal Models*, 108 ENVTL. HEALTH PERSP. 511, 511-33 (2000).

49. See Miller et. al., *supra* note 41, at 413 (explaining brain's reduced capacity to repair damage during developmental stage).

50. See *id.* at 411 (describing developmental growth of immune system).

51. See Rodney R. Dietert & Michael S. Piepenbrink, *Perinatal Immunotoxicity: Why Adult Exposure Assessment Fails to Predict Risk*, 114 ENVTL. HEALTH PERSP. 477,

creased dose sensitivity to most toxicants.”⁵² This has been well documented for lead, dioxin, and mercury.⁵³ Second, toxicants that adversely affect the developing immune system produce “a different and unpredictable array of alterations when the exposure occurs *in utero* or in the early neonate versus the adult.”⁵⁴ Lead, methoxychlor, ethanol, and genistein, among others, illustrate this point. Third, alterations in the immune system following early exposure can cause persistent changes that show up later in life, as occurred, for example, with DES. Finally, “sublethal exposure to a toxicant may produce an unrecognizable immunotoxic alteration until the postnatal immune system is placed under subsequent stress.”⁵⁵ DES and lead have caused such stress resulting in adverse effects. Thus, the assessment of risks in adults does not predict perinatal sensitivity to toxicants. In addition, as long as the immune system is immature or partially disabled because of any adverse toxicity effects, it will be unable to fully protect other organ systems from toxic insults.

Lung development goes through a long process that can be subject to external insults and perturbations along the way. One researcher has called attention to these issues as follows:

The developing lung is highly susceptible to damage from exposure to environmental toxicants particularly due to the protracted maturation of the respiratory system, extending from the embryonic phase of development in utero through to adolescence. The functional organization of the lungs requires a coordinated ontogeny of critical developmental processes that include branching morphogenesis, cellular differentiation and proliferation, alveolarization, and maturation of the pulmonary immune, vasculature, and neural systems. Therefore, exposure to environmental pollutants during crucial periods of prenatal and/or postnatal development may determine the course of lung morphogenesis and maturation. De-

477 (2006) (describing various stages of immune system development). See generally R. R. Dietert et al., *Workshop to Identify Critical Windows of Exposure for Children's Health: Immune and Respiratory Systems Work Groups Summary*, 108 ENVTL. HEALTH PERSP. 483, 483–90 (2000).

52. See Dietert & Piepenbrink, *supra* note 51, at 480 (discussing how dose levels impact perinatal immunotoxic sensitivity).

53. See *id.* (noting dose sensitivity to several different toxicants).

54. See *id.* (discussing how toxicants can adversely affect developing immune systems and produce unpredictable alterations).

55. See *id.* (describing latency).

pending on the timing of exposure and pathobiological response of the affected tissue, exposure to environmental pollutants can potentially result in long-term alterations that affect the structure and function of the respiratory system.

Besides an immature respiratory system at birth, children possess unique differences in their physiology and behavioral characteristics compared to adults that are believed to augment the vulnerability of their developing lungs to perturbations by environmental toxins. Furthermore, an interaction between genetic predisposition and increased opportunity for exposure to chemical and infectious disease increase the hazards and risks for infants and children.⁵⁶

Ozone, environmental tobacco smoke and particulate matter are substances that pose special risks to the developing lung.

Finally, the reproductive system develops, not only *in utero*, but throughout childhood and puberty. During this long period there are a variety of opportunities for toxicant exposure to interfere with development.

Because the reproductive process is critical for perpetuation of any species of organisms, factors or agents that alter or disrupt this process can have devastating consequences.

We now realize that such agents can arise from varying sources—which can be pharmacological, environmental, and natural—having extensive chemical structural diversity. In addition, the effects can be through a single action, or in combination, and can influence either individual or multiple cellular signaling pathways in a tissue or organ system. As such, it is difficult to ascribe a single action or effect to certain agents

Most important in recent toxicology studies is the number of examples showing that exposure during specific periods of development results in long-term effects that occur following sexual maturity and adulthood. Certain organ systems are more susceptible to toxicants during these

56. Radhika Kajekar, *Environmental Factors and Developmental Outcomes in the Lung*, 114 PHARMACOLOGY & THERAPEUTICS 129, 129 (2007) (discussing interaction between genetic predisposition and increased opportunity for exposure to chemicals and infectious diseases).

developmental periods. It is extremely important to realize that not only adults, but also children, infants, and the developing fetus, are potential targets for toxicological insult. Exposure during these sensitive periods either alters normal development, resulting in immediate or acute effects, or may subsequently compromise normal physiology and function later in life.⁵⁷

D. Specific Substances Can Cause Adverse Effects During Development

Apart from general biological reasons and background evidence about the vulnerability of developing organ systems, some particular substances have been identified as causing specific adverse effects.⁵⁸ For instance, exposures to comparatively high doses of dioxins have altered pigmentation, been associated with developmental delays and lower IQs, have caused cognitive delays, and have probably affected sex-related behaviors, with none of the effects being reversible.⁵⁹ PCB exposures can also result in male deficits in spatial reasoning, lack of endurance, clumsy movement, and IQs approximately six points lower at age eleven.⁶⁰ Children have greater susceptibility prenatally than during the immediate postnatal period.⁶¹ Background levels of dioxins "can influence the human immune system."⁶²

Taken together, the human studies involving complex mixtures of dioxin-like compounds, including both dioxin and non-dioxin-like PCBs, suggest that levels present in the general population may be associated with subtle signs of neurological dysfunction, delays in psychomotor devel-

57. Kenneth S. Korach, *Preface*, in *REPRODUCTIVE AND DEVELOPMENTAL TOXICOLOGY V* (Kenneth S. Korach ed., Marcel Dekker Inc. 1998) (noting how exposure to toxicants during sensitive periods either alters normal development or subsequently compromises normal physiology).

58. *See id.* (identifying various sources causing adverse effects).

59. Linda Birnbaum, *Developmental Effects of Dioxins*, in *REPRODUCTIVE AND DEVELOPMENTAL TOXICOLOGY 103* (Kenneth S. Korach ed., Marcel Dekker Inc. 1998) (discussing impact of exposures to comparatively high doses of dioxins).

60. Grandjean & Landrigan, *supra* note 25, at 2172 (noting role that PCB exposures can have in certain male deficits).

61. Joseph L. Jacobson & Sandra W. Jacobson, *The Effects of Perinatal Exposure to Polychlorinated Biphenyls and Related Contaminants*, in *PRENATAL EXPOSURE TO TOXICANTS 144* (Herbert L. Needleman & David Bellinger eds., Johns Hopkins Univ. Press 1994) (discussing how prenatal children have greater susceptibility than postnatal children).

62. *See* Birnbaum, *supra* note 59, at 105 (discussing how background levels of dioxins influence human immune system).

opment, alterations in thyroid hormone status, and changes in immunological functions.⁶³

Recent research suggests that PCB exposures also skew sex ratios in newborns among the Inuits and other groups inhabiting the Northern Hemisphere.⁶⁴ Traditionally, out of one hundred births, 51 will be boys and 49 girls; in the high northern latitudes researchers are finding that females are born at twice the rate of males.⁶⁵

A great deal of research has been done on lead, and the results are both revealing and disturbing. Lead, now a ubiquitous metal widely detected in the environment, is toxic to most living things.⁶⁶ Its toxic effects "were known in Roman times In the 1940s . . . acute [exposures caused] severe learning and behavioral problems."⁶⁷ Lead exposure can cause both clinically identifiable and more subtle adverse effects. Most sensitive is the nervous system, but other target organs include the gastrointestinal tract, reproductive system, and skeletal system.⁶⁸ The first toxic effects of lead were noticed following substantial exposure levels, but over time researchers found much lower concentrations of lead may cause diseases or functional deficiencies.⁶⁹ Consequently, the United States Environmental Protection Agency (EPA) has set a cap at 15µg of lead per liter in drinking water with an action level of 10 µg/dL (micrograms /deciliter) adopted by the CDC for children.⁷⁰

There is considerable evidence that umbilical cord blood levels (thus, prenatal exposures) in excess of 10 µg /dL cause delays in early cognitive development and remain persistent when blood lead

63. *See id.* at 106 (discussing results from human studies involving complex mixtures of dioxin-like compounds).

64. Francesca Lyman, Are Boys An Endangered Species?, <http://www.b-e-a-c-h.org/news%20images/Are%20Boys%20An%20Endangered%20Sp.pdf> 1-2 (discussing suggestion that PCB exposures skew sex ratios in newborns).

65. *Id.* (providing information on population in Arctic Circle).

66. Robert A. Goyer & Thomas W. Clarkson, *Toxic Effects of Metals*, in CASARETT AND DOULL'S TOXICOLOGY 623, 639 (4th ed. 1991) (noting that lead is toxic to most living organisms).

67. Grandjean & Landrigan, *supra* note 25, at 2169 (discussing when toxic effects of lead became known to humans).

68. *See* Goyer, *supra* note 66, at 641 (noting target organs of human body).

69. Grandjean & Landrigan, *supra* note 25, at 2169-70 (discussing effects of lead on humans).

70. Dep't of Health and Human Servs., Agency for Toxic Substances & Disease Registry, Lead Toxicity What Are U.S. Standards for Lead Levels?, http://www.atsdr.cdc.gov/csem/lead/pb_standards2.html (setting drinking water standards).

levels remained above 10 µg /dL.⁷¹ In addition, fetal exposures are more harmful than postnatal exposures.⁷² More recent research suggests that there is likely no threshold for adverse effects from childhood lead poisoning.⁷³ Moreover, researchers found that children are much more sensitive to blood lead levels than adults, although some recent research suggests that even adults can be harmed at fairly low exposures, causing concern for occupationally exposed individuals.⁷⁴ Finally, exposures to lead early in life appear to contribute to “‘normal’ age-related cognitive decline[s] . . .” later in life.⁷⁵ Beyond the specific substances just noted, Grandjean and Landrigan have summarized the scientific literature finding about 200 neurotoxic development toxicants based solely on human studies.⁷⁶

E. Particular Diseases Can Be Exacerbated by Exposures During Development

Particular diseases can be exacerbated as a result of *in utero*, immediate postnatal, or childhood exposure to toxicants. Consider cancers as one such example. There has been a general increase in childhood cancers since the 1970s that researchers indicate may not be due merely to improved diagnosis of the disease.⁷⁷ Moreover, there is a documented increased risk of cancer as a result of DES exposure, as well as “higher rates of radiation-induced breast cancer among women exposed during puberty, compared with those exposed after puberty”⁷⁸ For women, exposure to to-

71. David Bellinger & Herbert L. Needleman, *supra* note 40, at 89 (finding children with high umbilical cord blood levels achieved lower Mental Development Index scores).

72. *Id.* at 104 (stating difference between post-natal and fetal lead exposure).

73. See Richard L. Canfield et al., *Low-Level Lead Exposure and Executive Functioning in Young Children*, 9 CHILD NEUROPSYCHOLOGY 35, 35-53 (2003) (concluding lead exposure places children at disadvantage in associative learning and academic success); see also David Bellinger & Herbert L. Needleman, *Intellectual Impairment and Blood Lead Levels*, 349 NEW ENG. J. MED. 500, 500 (Jul. 2003) (concluding there are extensive adverse effects of lead poisoning).

74. Ellen K. Silbergeld & Virginia M. Weaver, *Exposures to Metals: Are We Protecting the Workers?*, 64 J. OCCUPATIONAL & ENVTL. MED. 141, 141-42 (Mar. 2007) (showing harmful effects of lead in adults).

75. Schwartz & Stewart, *supra* note 35, at 671 (indicating cognitive decline may be due to exposure to neurotoxicants).

76. Grandjean & Landrigan, *supra* note 25, at 2175 (finding over two hundred chemicals known to cause neurotoxic effects in humans).

77. Miller, *supra* note 41, at 411-12 (concluding increased cancer levels may not be function of better testing).

78. *Id.* at 412 (citing NRC Report describing effects of radiation during puberty). See generally NATIONAL RESEARCH COUNCIL, HEALTH EFFECTS OF EXPOSURE

bacco smoke during puberty may increase the risk of breast cancer among those with certain gene-based physiological characteristics.⁷⁹ The effect of women's exposure to DDT during adolescence appears to hasten the onset of breast cancer.⁸⁰ Finally, there is substantial evidence among experimental animals (our mammalian relatives) of increased cancer risks following early life exposures to a number of compounds such as urethane, vinyl chloride, DES, tamoxifen, nitrosourea compounds and alkenylbenzene compounds,⁸¹ as well as aspartame.⁸² EPA funded research supports the finding that animal exposures to mutagenic carcinogens "indicate a 5- to 60-fold increased carcinogenic sensitivity in the . . ." immediate postnatal period and a somewhat similar but smaller increase for exposures to radiation, but there is no such effect for carcinogens that depends upon metabolic activation (in part because the metabolic processes for reducing compounds is less developed in immature mammals).⁸³

Additionally, in some cases children either develop diseases or adverse conditions not seen in adults at the same exposure levels, or adverse effects in children are worse than the same disease in adults, or the disease may emerge earlier.⁸⁴ Early exposures can lead to life-long adverse effects, especially neurological effects.⁸⁵ In some cases exposure may even result in late-life adverse functioning, on the hypothesis that subclinical injuries "silently kill a fraction of the cells needed to sustain brain function in later life (e.g., in the substantia nigra)."⁸⁶ Prenatal exposures to lead, tobacco smoke, and pesticides are associated with poor cognitive function-

TO LOW LEVELS OF IONIZING RADIATION: BEIR V (1990) available at http://www.nap.edu/openbook.php?record_id=1224&page=R1

79. Miller, *supra* note 41, at 411-12 (finding tobacco smoke increases risk of breast cancer).

80. Cohn, *supra* note 33, at 1413 (discovering early life exposure to DDT may increase breast cancer risk).

81. See Miller, *supra* note 41, at 411-12 (finding increased cancer risk in numerous compounds).

82. Morando Soffritti et al., *Life-Span Exposure to Low Doses of Aspartame Beginning during Prenatal Life Increases Cancer Effects in Rats*, 115 ENVTL. HEALTH PERSP. 1293, 1293-97 (2007) (finding carcinogenic effects of aspartame in animals after prenatal exposure).

83. Dale Hattis et al., *Age-Related Differences in Susceptibility to Carcinogenesis: A Quantitative Analysis of Empirical Animal Bioassay Data*, 112 ENVTL. HEALTH PERSP. 1152, 1152 (2004) (showing increased carcinogenic sensitivity in fetal period).

84. See Grandjean & Landrigan, *supra* note 25, at 2169-73 (finding effects of lead, methyl mercury, ethanol, toluene, and perchlorate on children).

85. *Id.* at 2174 (discussing effects of early exposure to toxicants).

86. *Id.* (discussing effects of exposure in later life).

ing, attention deficit hyperactivity disorder, antisocial activity,⁸⁷ and exacerbated neurogenerative diseases late in life.⁸⁸ This evidence leads scientists to propose that low level perinatal exposures may represent “a silent pandemic in modern society.”⁸⁹

The above findings led the scientists at the Faroe Island Conference to adopt the view that, for children exposed prenatally and during the immediate postnatal period, the *timing* of a dose may be as important as the particular amount of a dose.⁹⁰ An important assumption of toxicology has been that “the dose makes the poison.”⁹¹ For developmentally linked effects, however, it appears that “the timing makes the poison.”⁹² Ill-timed and even very low exposures can be harmful.

F. Scientific Summary

The scientific picture that emerges from the above review is incomplete in some respects, but compelling nonetheless. As a matter of general knowledge scientists know that developing organ systems in mammals, including humans, are more susceptible to adverse effects from toxicants. Developing fetuses and children often have greater exposures on a per body weight basis than adults, higher metabolisms, breathing rates, and absorption rates along with behavioral tendencies increasing exposures. At the same time, developing children have fewer defenses against potential toxicants than mature adults. Moreover, there is compelling evidence that particular compounds, such as lead, mercury, DES, pesticides, and radiation have substantial adverse effects on developing humans and animals. Some diseases are exacerbated or triggered sooner as a result of early exposures. Early prenatal or neonatal exposures may cause adverse effects much later in life.⁹³

87. BRUCE LANPHEAR ET AL., CINCINNATI CHILDREN'S HOSP. MED. CTR. & UNIV. OF CINCINNATI, *ROLE OF ENVIRONMENTAL TOXICANTS IN THE DEVELOPMENTAL ORIGIN OF LEARNING AND BEHAVIOURAL PROBLEMS 1* (May 21, 2007), <http://www.pptox.dk/portals/0/o6.pdf> (reporting neurological risks associated with prenatal exposure to lead, tobacco smoke, and other toxicants).

88. Schwartz & Stewart, *supra* note 35, at 682-83 (discussing exacerbation of neurogenerative diseases from exposure).

89. See Grandjean & Landrigan, *supra* note 25, at 2174 (arguing that evidence suggests that low level prenatal and perinatal exposures to industrial chemicals have created a silent pandemic of neurological disorders).

90. See Grandjean et al., *supra* note 18, at 74 (discussing conclusions).

91. *Id.* (discussing paradigm developed by Paracelsus).

92. *Id.* (suggesting when exposure occurs is quite important in causing toxic effects perinatally).

93. *Id.* (suggesting that early prenatal exposure can likely lead to later effects).

Nonetheless, there are gaps in the science. The evidentiary picture may be something like a pointillist painting with parts of the picture filled in with some data points, other parts blank, but the general background reasonably solid. Scientists almost certainly do not yet know all the compounds that might have such adverse effects and may never have an exhaustive list, because new developmental toxicants are likely to be discovered. They may not yet know all the disease processes that can be accelerated or exacerbated by early exposures to toxicants. They may not know the lowest exposures during development that can pose adverse effects, either immediately or later in life. The resulting picture is troubling, however. There is substantial scientific concern that substances remarkably similar to those with known toxicity are only now being appropriately appreciated, such as polybrominated diphenyl ethers, Bisphenol A and phthalates.⁹⁴

Just because there are gaps in the scientific picture, should we be willing to bet our children will be safe in the future from adverse effects? This is a poor bet. Do we want to bet that the currently incomplete picture means the country need not take steps to better protect our children's health? This too would be a poor bet.

Polybrominated diphenyl ethers (PBDEs), illustrate the manner in which the science of toxicants emerges (although in some respects the science is much better for this class of products than for many because of similarities to known toxicants). The PBDE example also shows the difficulties of using post-market laws to regulate substances to which children will be exposed in the womb and early in life.

94. Kim Hooper & Thomas A. McDonald, *The PBDEs: An Emerging Environmental Challenge and Another Reason for Breast-Milk Monitoring Programs*, 108 ENVTL. HEALTH PERSP. 387, 387-88 (2000) (discussing scientific concern that persistent organic pollutants with known toxicity are only now being appropriately appreciated); see also Lucio G. Costa & Gennaro Giordano, *Developmental Neurotoxicity of Polybrominated Diphenyl Ether (PBDE) Flame Retardants*, 28 NEUROTOXICOLOGY 1047, 1047-67 (2007) (discussing scientific concern that toxicity of PBDEs are only now being appropriately appreciated); Prasada Rao S. Kodavanti, *Neurotoxicity of Persistent Organic Pollutants: Possible Mode(s) of Action and Further Considerations*, 3 DOSE-RESPONSE 273, 273-305 (2005) (emphasizing scientific concern regarding appreciation of persistent organic pollutants); Arnold Schecter et al., *Polybrominated Diphenyl Ether Flame Retardants in the U.S. Population: Current Levels, Temporal Trends, and Comparison with Dioxins, Dibenzofurans, and Polychlorinated Biphenyls*, 47 J. OCCUPATIONAL & ENVTL. MED. 199, 200 (Mar. 2005) (discussing scientific concern that PBDEs are only now being appropriately appreciated and comparing them with other persistent organic pollutants).

PBDEs are a class of flame retardants that since the 1960s⁹⁵ have been “extensively used in a variety of consumer products such as textiles, carpets, polyurethane foams, electronic cables, television sets and computers.”⁹⁶ Some chemical compounds can be chemically bound in polymers so that it is difficult for them to escape from the products and enter the environment; this is not the case with PBDEs. They “are not fixed in the polymer product through chemical binding, and can thus leak into the environment.”⁹⁷ Moreover, they have a number of chemical and biologically active properties that are quite similar to other compounds known to cause human developmental effects, and there is good reason to believe that they will pose similar problems in humans once they are well studied. Any such problems have not shown up to date, either because they have not been studied in humans sufficiently long enough, have not had a sufficiently long period to manifest their adverse effects, or the adverse effects are sufficiently subtle that they have not been detected with the crude tools of epidemiology.

PBDEs are part of a general class of long-lived organic compounds, which includes polychlorinated biphenyls (PCBs), organochlorine pesticides, and dioxins that tend to “remain in the environment for a long period of time due to their high persistence, and bioaccumulate in the food web.”⁹⁸ Many of these substances accumulate in animal and human tissues, especially body fat. More specifically, they belong to a family “of pollutants called polyhalogenated aromatic hydrocarbon [compounds, that include] dioxins, polychlorinated dibenzofurans (PCDF), polychlorinated diphenyl ethers, DDT, and polybrominated diphenyl ethers (PBDEs)”⁹⁹ Many members of this class are known to cause developmental problems. PBDEs are specifically quite similar chemically to PCBs, which have been extensively studied in both animals and humans. There is substantial research showing that PCBs cause developmental effects in both animals and humans.¹⁰⁰

95. Hooper & McDonald, *supra* note 94, at 388 (stating use of PBDEs began in 1960s).

96. Lucio G. Costa & Gennaro Giordano, *Developmental Neurotoxicity of Polybrominated Diphenyl Ether (PBDE) Flame Retardants*, 28 NEUROTOXICOLOGY 1047, 1048 (2007) (noting various products where PBDEs can be found).

97. *Id.* (suggesting reason why PBDEs are exceedingly dangerous).

98. Kodavanti, *supra* note 94, at 274 (explaining why PBDEs remain in environment for extended period of time).

99. *See id.* (discussing chemical properties of PBDEs).

100. *Id.* at 276 (illustrating exposure and effect of PCBs on humans and various other mammals).

PBDEs are of "concern as a result of their association with endocrine disruption, reproductive and developmental toxicity, including neurotoxicity, and cancer" ¹⁰¹

Animal studies of PBDEs alone provide reasons to believe that there will be adverse neurotoxic effects in humans, since animal models are routinely utilized to predict adverse effects in humans. ¹⁰²

(1) Animal studies, carried out with different PBDEs, have indicated that pre-and postnatal exposures to PBDEs may cause long-lasting behavioral alterations, particularly in the domains of motor activity and cognitive behavior.

101. Arnold Schecter et al., *Polybrominated Diphenyl Ether Flame Retardants in the U.S. Population: Current Levels, Temporal Trends, and Comparison with Dioxins, Dibenzofurans, and Polychlorinated Biphenyls*, 47 J. OCCUPATIONAL & ENVTL. MED. 199, 200 (2005) (explaining why PBDEs are such great concern). See generally L. Birnbaum & D. Staskal, *Brominated Flame Retardants: Cause for Concern?*, 112 ENVTL. HEALTH PERSP. 9, 9-17 (2003); S. Hallgren & P. Darnerud, *Effects of Polybrominated Diphenyl Ethers (PBDEs), Polychlorinated Biphenyls (PCBs) and Chlorinated Paraffins (CPs) on Thyroid Hormone Levels and Enzyme Activities in Rats*, 35 ORGANOHALOGEN COMPD. 391, 391-94 (1998); S. Hallgren & P. Darnerud, *Polybrominated Diphenyl Ethers (PBDEs), Polychlorinated Biphenyls (PCBs) and Chlorinated Paraffins (CPs) in Rats - Testing Interactions and Mechanisms for Thyroid Hormone Effect*, 177 TOXICOLOGY, 227, 227-43 (2002); M. Gillner & E. Jakobsson, *Structure-affinity Relationships for Thyroid and Dioxin Receptor Binding of Halogenated Naphthalenes and Diphenylethers*, 29 ORGANOHALOGEN COMPD. 220, 220-21 (1996); I. Meerts et al., *In Vitro Estrogenicity of Polybrominated Diphenyl Ethers, Hydroxylated PDBEs, and Polybrominated Bisphenol A Compounds*, 109 ENVTL. HEALTH PERSP. 399, 399-407 (2001); I. Meerts et al., *Placental Transfer of a Hydroxylated Polychlorinated Biphenyl and Effects on Fetal and Maternal Thyroid Hormone Homeostasis in the Rat*, 68 TOXICOL SCI. 361, 361-71 (2002); D. Morse et al., *Interference of Polybrominated Biphenyls in Hepatic and Brain Thyroid Hormone Metabolism in Fetal and Neonatal Rats*, 22 TOXICOL APPL PHARMACOL 27, 27-33 (1993); I. Branchi et al., *Effects of Perinatal Exposure to a Polybrominated Diphenyl Ether (PBDE 99) on Mouse Neurobehavioural Development*, 23 NEUROTOXICOLOGY 375, 375-84 (2002); P. Eriksson et al., *A Brominated Flame Retardant, 2,2',4,4',5-pentabromodiphenyl Ether: Uptake, Retention, and Induction of Neurobehavioral Alterations in Mice During a Critical Phase of Neonatal Brain Development*, 67 TOXICOL SCI. 98, 98-103 (2002); H. Viberg et al., *Neonatal Exposure to the Brominated Flame Retardant, 2,2',4,4',5-pentabromodiphenyl Ether, Decrease Cholinergic Nicotinic Receptors in Hippocampus and Affects Spontaneous Behavior in the Adult Mouse*, 17 ENVIRON TOXICOL PHARMACOL. 61, 61-65 (2004); Env'tl. Prot. Agency, 2,2',3,3',4,4',5,5',6,6' - Decabromodiphenyl ether (BDE-209) (CASRN 1163-19-5), <http://www.epa.gov/iris/subst/0035.htm#reforal>.

102. See JOHN M. ROGERS & ROBERT J. KAVLOCK, *DEVELOPMENTAL TOXICOLOGY*, CASARETT AND DOULL'S TOXICOLOGY (Curtis Klaassen ed., Pergamon Press, 6th ed. 2001) (discussing use of animal studies as close correlation for effects on humans). "There have been several extensive reviews of the similarity of responses of laboratory animals and humans for developmental toxicants. In general, these studies support the assumption that results from laboratory tests are predictive of potential human effects." *Id.* at 374. See generally Lucio G. Costa & Gennaro Giordano, *Developmental Neurotoxicity of Polybrominated Diphenyl Ether (PBDE) Flame Retardants*, 28 NEUROTOXICOLOGY 1047, 1061 (2007) (discussing further use of animal studies as close correlation for effects on humans).

Neurochemical changes have also been found following developmental exposure to PBDEs. (2) PBDEs affect thyroid hormone homeostasis, which may result in developmental neurotoxicity. (3) There is indication that young animals may have reduced ability to excrete PBDEs, and pups have higher tissue (including brain) concentrations than the dams. (4) PBDEs are excreted in milk, and relatively high concentrations are found in North America. (5) Dust has been found to be a major source of exposure. (6) Infants and toddlers have the highest body burden of PBDEs, due to exposure via maternal milk and house dust.¹⁰³

The known structural similarities of PBDEs to PCBs and the known similarity of PCBs' adverse effects in animals and humans, together with known similarities between adverse effects of PCBs and PBDEs in animals also predict adverse effects of PBDEs in humans.¹⁰⁴ "There is sufficient evidence that PCBs can cause neurotoxicity in humans . . . and it is believed that in utero exposure is more important than lactational exposure in causing the neurotoxic effects."¹⁰⁵ This evidence includes both accidental high dosage exposures in Japan and other events revealed in several epidemiological studies.¹⁰⁶ PCBs and related compounds can cause neurotoxicity in rats, mice, and monkeys.¹⁰⁷ These include behavioral changes and learning deficits resulting from both adult and perinatal exposure.¹⁰⁸ Some of these effects resemble Attention Deficit Hyperactivity Disorder. Exposure can also decrease cognitive function in rats, primates and mice, impaired visual discrimination, altered spatial perception function, as well as deficits in learning and memory.¹⁰⁹ Animal studies of PBDEs reveal similar

103. Costa & Giordano, *supra* note 96, at 1051 (quoting various findings from animal studies with regards to PBDEs).

104. Hooper & McDonald, *supra* note 94, at 1051 (discussing known similarities between PBDEs and PCBs).

105. See Kodavanti, *supra* note 94, at 275 (noting there is sufficient evidence that PCBs have been shown to cause neurotoxicity in humans).

106. *Id.* at 275-77 (describing effects of PCB exposure).

107. *Id.* at 276 (illustrating exposure and effect of PCBs on humans and various other mammals).

108. *Id.* at 277 (showing children of exposed women could be hypoactive or hyperactive with varying degrees of behavioral problems).

109. *Id.* at 278 (describing both motor and cognitive effects of exposure).

neurotoxic effects in animals. Exposure of animals to PCBs and PBDEs produce similar neurotoxic outcomes.¹¹⁰

There appears to be a similar mechanism at work with both PCBs and PBDEs. In general, the physical and chemical properties of molecules affects their toxicity.¹¹¹ In PCBs the *shape* of the molecule affects its toxicity: so-called coplanar congeners (lying in the same geometric plane) tend not to be active in disrupting intracellular signaling, whereas non-coplanar congeners (not lying in the same plane) tend to disrupt this signaling. Congeners are related chemical substances with different numbers of key molecules attached. Intracellular signals are "essential not only for the function of the nervous system, but also play a key role in nervous system development. Any interference with these processes would have the potential for profound effects on the function of neuron[s] as well as their development."¹¹² There are also reasons to believe that PCBs produce their toxic effects by disrupting intracellular signaling because of effects on learning and memory seen in human epidemiological studies and effects at the molecular level.¹¹³ The important point is that PBDEs' non-coplanar structure is so similar to PCBs non-coplanar structure that they will be active in disrupting intracellular signaling as PCBs are.

PCBs and PBDEs have been shown [to] exert neurobehavioral effects and cause changes in intracellular signaling pathways in neuronal cells at equimolar doses/concentrations suggesting a common mode of action for these chemicals. Considering the structural similarities of PBDEs and PCBs and the known health effects of PCBs in

110. See Kodavanti, *supra* note 94, at 297-98 (describing potential application of PCB data to PBDEs).

111. INST. OF MED. AND NAT'L RESEARCH COUNCIL, COMM. ON THE FRAMEWORK FOR EVALUATING THE SAFETY OF DIETARY SUPPLEMENTS, DIETARY SUPPLEMENTS: A FRAMEWORK FOR EVALUATING SAFETY 205-06 (Nat'l Acads. Press 2005) (discussing structure of toxic compounds). "The physical-chemical properties and biological effects of a substance are derived from its chemical structure. If the chemical structure of dietary supplement is known, but addition insight into the biological activity is needed, then it is scientifically appropriate to consider the information about the biological activity of structurally related substances. It is assumed that the biological effects of chemicals, including toxic effect, are implicit in their molecular structures" *Id.* at 205-06.

112. See Kodavanti, *supra* note 94, at 281 (detailing significance of intracellular signaling). See generally S. Murphy et al., *Phorbol Ester Stimulates Proliferation of Astrocytes in Primary Cultures*, 428 BRAIN RES. 133, 133-35 (1987); P. R. Girard & J. F. Kuo, *Protein Kinase C and its 80-kilodalton Substrate Protein in Neuroblastoma Cell Neurite Outgrowth*, 54 J. NEUROCHEM 300, 300-06 (1990).

113. See Kodavanti, *supra* note 94, at 281-83 (noting PCB's effects on intracellular signaling).

humans, these two groups of chemicals (may be other structurally related chemicals) could conceivably work through the same mechanism(s), to cause developmental neurotoxicity.¹¹⁴

The dose at which the adverse effects of PBDEs in animals occurs is nearly equal on a per body weight basis to concentrations of PBDEs in human tissues and bodies. In animals, the adverse effects of PBDEs are seen “at exposure levels relevant to humans, at least in North America.”¹¹⁵ Concentrations of PBDEs in individuals’ bodies are one to two orders of magnitude higher in the United States than in Europe and the highest in the world.¹¹⁶ In addition, whereas the half-life of PBDEs in rodents “is in the order of several days or months, the terminal total body half-lives in humans have been estimated to be much longer, in the order of years . . .” for some congeners and months for others.¹¹⁷ The longer a substance remains in a mammalian body, the more time it has to inflict molecular damage that can lead to more serious organism damage. In humans, the concentrations of PBDEs on a per body weight basis seem to be highest in infants, next highest in children one to five years of age, next highest in children six to eleven years of age, next in children twelve to nineteen years old, and lowest (but still of concern) in adults.¹¹⁸

Finally, it is important to recall that individuals are not merely exposed to one substance at a time, nor are their bodies contaminated by one substance at a time. Researchers have found that some low doses of PCBs alone or PBDEs alone do not show adverse effects in rodents, but “co-exposure [of] the same low doses . . . produced significant behavioral alterations.”¹¹⁹ Individuals’ bodies are also contaminated with “other developmental neurotoxicants (e.g. lead, methylmercury, perchlorate, dioxins, etc.),” which could

114. *Id.* at 298 (recognizing similarity in PCBs and PBDEs). See generally P. Eriksson et al., *Brominated Flame Retardants: A Novel Class of Developmental Neurotoxicants in Our Environment?*, 109 ENVTL. HEALTH PERSP. 903, 903-08 (2001).

115. Costa & Giordano, *supra* note 96, at 1061 (describing need for reliance on animal data for PBDEs).

116. *Id.* at 1049 (detailing sources for alarmingly high PBDE levels in humans in North America).

117. *Id.* at 1061 (recognizing longer half-life of PBDEs in humans). The half-life of substance in a mammalian body is the amount of time it takes for one-half of the substance to be eliminated from the body.

118. *Id.* at 1050 (listing estimated exposure to PBDEs across different age groups).

119. *Id.* at 1062 (illustrating potential synergistic effect of PCBs and PBDEs and other neurotoxicants).

have additive or synergistic effects, enhancing the adverse effects.¹²⁰ Since many of these compounds have long half-lives, they remain in human bodies for substantial periods of time interacting with one another.

There are some gaps in the PBDE-neurotoxicity picture, but these are largely because of the absence of human studies. The problem with gaps in the evidence is that they introduce some uncertainty about conclusions and they provide an opportunity for adversaries to argue that substances may not cause health problems in humans as they do in many other biological systems. They open the possibility of some scientific doubt that can be exploited to argue that the substances are less worrisome than the bulk of scientific data supports. The question for the remainder of this Article is whether the post-market legal structure governing most substances (including PBDEs) is sufficiently protective for fetuses and developing children. Existing evidence concerning PBDEs and other substances reviewed in this Section suggests that they are not. It would be a poor bet to rely on post-market laws to protect our children. The chemical invasion revealed by the CDC biomonitoring and the results of scientific research showing that chemical invaders can enter, disrupt, and potentially cause adverse effects during the developmental process with possible further lifetime consequences suggests that we should reconsider laws and legal structures for controlling toxicants. The examples of PBDEs only reinforce this concern.

IV. EXISTING HARM-BASED OR RISK OF HARM-BASED REGULATION OF TOXICANTS

A. Regulatory Law

Current regulation of toxicants under administrative law is *in effect* harm-based, or *risk-of-harm* based. In addition, to the extent that tort or personal injury law serves to *regulate* exposures to toxicants, except for some marginal causes of action, it is explicitly harm-based. Moreover, to the extent that our laws are harm-based or risk-of-harm based, it is arguable that existing laws do not work or work well enough to protect our children.

Regulatory or *administrative* laws governing environmental chemicals seek to prevent harms from exposures occurring in the first place. They try to accomplish this aim through pre-market no-

120. Costa & Giordano, *supra* note 96, at 1062 (recognizing potential exposure to other neurotoxicants).

tification laws, pre-market testing and approval laws, and several varieties of post-market laws.

1. *Pre-Market Screening Laws*

Some laws require manufacturers to notify the EPA that they propose to manufacture new chemicals or use existing chemicals for substantial new uses. Manufacturers must provide the EPA with what they know about the products proposed for manufacture. The Toxic Substances Control Act (TSCA) authorizes the agency to review these products as they enter the market.¹²¹ The EPA is then given ninety days to review each substance for any toxicity properties, with the possibility of time extensions for further review or to permit the EPA to request more data.¹²² There is, however, no legally required amount of information a manufacturer must submit to the agency and no minimal testing data; companies need only report what they know about the product. Typically this includes some chemical and physical properties as well as the chemical structure and some biological activity data about it. If the EPA identifies some feature of concern about a substance, typically a chemical structure that resembles other toxicants, under the TSCA new substances provisions it can require other data about or testing of the product to try to determine whether it poses risks or harm to humans or the environment.¹²³

In 1983, the Office of Technology Assessment found that about one-half of the substances submitted for review contained *no toxicity information* and “only 17 percent of [the chemicals proposed for manufacture had] any test information about the likelihood of the substance’s causing cancer, birth defects or mutations—three biological effects that were singled out for special concern in TSCA.”¹²⁴ In 1987, when revisiting the pre-market screening provisions of TSCA, the Office of Technology Assessment reported that

121. Toxic Substances Control Act (TSCA), 15 U.S.C. §§ 2601-2692 (2006) (providing text of Act).

122. U.S. Congress, Office of Technology Assessment, *Identifying and Regulating Carcinogens*, OTA-BP-FI-42, 216-17 (Washington, D.C.: U.S. Government Printing Office, Nov. 1987) (stating EPA’s obligations under Toxic Substances Control Act).

123. *Id.* at 127 (discussing TSCA regulations with regard to environmentally hazardous chemicals).

124. *Id.* (quoting U.S. Congress, Office of Technology Assessment, *The Information Content of Premanufacture Notices*, OTA (Washington, D.C.: U.S. Government Printing Office, 1983) (noting short-term tests on PMNs)).

"most PMNs [Premanufacture Notifications] do not contain any toxicity test information" ¹²⁵

2. *Pre-Market Testing and Approval Laws*

Some laws, including laws under the authority of the FDA or the EPA, are authorized to review substances, such as drugs and new food additives, under the FDA, or pesticides, under the EPA, *before* they enter commerce and substantial human exposure occurs. Such laws impose testing requirements for the substances, agency review of the test data, some level of demonstrated safety (or minimization of risks), and explicit agency approval before the products are permitted to enter commerce. The language of many of these statutes is explicitly that of avoiding *risk of harm*. According to a 1984 National Research Council (NRC) report, however, the substances governed by pre-market testing and approval laws cover only a relatively small portion of the chemical universe that was in existence at that time, at most about twenty percent and it could be less than ten percent.¹²⁶ Even though the goal is to detect toxicants before they enter commerce and there is substantial pre-market testing, unless the laws authorize tests specifically for developmental toxicants, they will not be detected. Moreover, the pre-market testing provisions do not work perfectly, so some toxic effects will be missed despite the best and most conscientious efforts. When this occurs, if manufacturers and agencies have been vigilant, this may be the best situation for which citizens can reasonably ask. The regulatory law of the United States has not achieved this goal for the vast majority of substances.

3. *Post-market Laws*

Finally, a large majority of substances (approximately eighty percent, and possibly as high as ninety percent) are regulated under *post-market laws*. Post-market laws permit products to enter commerce without any legally required testing. Since 1980, substances that would otherwise only be subject to post-market laws have been subject to the TSCA pre-market screening provisions described above. Under this provision the EPA is provided whatever data manufacturers know about the substance, but there is no legally prescribed testing or minimally required data. Agencies, such

125. *Id.* (stating PMNs do not contain toxicity test information).

126. NAT'L RESEARCH COUNCIL, TOXICITY TESTING: STRATEGIES TO DETERMINE NEEDS AND PRIORITIES 11 (Nat'l Acad. Press 1984) (reporting statistics on health hazard information).

as the Occupational Safety and Health Administration (OSHA), the Consumer Product Safety Commission (CPSC), the FDA, and the EPA must then review substances under laws that require them to identify risks of harm from the products *after they are in commerce* and take steps to reduce or eliminate any risks or harms from them.

Under virtually all post-market laws regulatory agencies bear the burden of proof to show risks of harm, actual harm or at least the toxicity of substances before any legal regulation can occur.¹²⁷ In addition, substantive legal provisions central to some post-market laws exacerbate the agencies' difficulties. Scientific documentation of risks or harms creates further and high hurdles.

Some laws require agencies to identify toxicants and then authorize the use of technology to reduce exposures to the lowest achievable level or perhaps to force the development of improved technology to ensure lower exposures.¹²⁸ Other "warning laws" require the identification of toxicants and might require those causing the exposure to warn the public about the risks.¹²⁹ Technology-forcing and warning laws simplify some government tasks depending upon the statute. In general, they only require agencies to *identify* toxicants and then utilize the best available technology to reduce exposures or to post warnings, respectively.¹³⁰ While these laws appear to be comparatively simple and straightforward, there can be a number of disputes concerning to which industries the law applies, the technologies manufacturers are required to adopt and the extent to which technologies should reduce exposures to toxicants, all taking considerable time.¹³¹

127. California's Proposition 65 does not follow this pattern, since once the state has listed a substance as a reproductive or carcinogenic toxicant, the burden of proof falls on firms that expose the public to the toxicant. See Office of Env'tl. Health Hazard Assessment, Proposition 65, <http://www.oehha.org/prop65.html> (providing additional information about Proposition 65).

128. U.S. Congress, Office of Technology Assessment, *Identifying and Regulating Carcinogens*, OTA-BP-FI-42, 199-200 (Washington, D.C.: U.S. Government Printing Office, Nov. 1987) (detailing regulatory carcinogen laws).

129. California's Proposition 65 is one example of such a law. See Safe Drinking Water and Enforcement Act of 1986, CAL. HEALTH & SAFETY CODE § 25249.5 (1989) (requiring public warning of risks associated with hazardous chemicals).

130. See U.S. Congress, Office of Technology Assessment, *Identifying and Regulating Carcinogens*, OTA-BP-FI-42, 127 (Washington, D.C.: U.S. Government Printing Office, Nov. 1987) (discussing how few toxicants submitted as part of PMNs have any testing); see also Carl F. Cranor, *Information Generation and Use Under Proposition 65: A Model for Other Post-market Laws?*, 83 IND. L. REV. (forthcoming 2008) (stating California's Proposition 65 requires firms to post clear and reasonable warnings).

131. See Bruce A. Ackerman & Richard B. Stewart, *Comment: Reforming Environmental Law*, 37 STAN. L. REV. 1333, 1333-365 (1985) (discussing criticisms of technology forcing laws).

Other laws require the setting of "ambient-exposure" levels to protect citizens. Government agencies bear the burden to prove that substances pose risks and the burden to determine exposure levels in the relevant environment so that statutorily mandated protection will be met. Such laws are especially onerous from the point of view of the regulator. When agencies are authorized to act under ambient-exposure laws, they must not only identify a substance as toxic to humans or the environment, or as posing risks thereto, but specify the approximate amount of exposure that poses a risk or harm, and then estimate whether there are exposure levels in the environment that necessitate regulation. Substantive legal provisions might require regulations to prevent "unreasonable risks of harm to health," to prevent human health risks "with an adequate (or ample) margin of safety," to prevent exposure to substances which cause "cancer in humans or animals," or to "prevent material impairment of health or functional capacity."¹³² Ambient exposure laws typically require all four stages of risk assessment: hazard identification; potency assessment; exposure assessment; and an overall risk characterization as part of the regulatory process.¹³³ Carrying out these tasks is quite science-intensive and time-consuming. Such laws impose a burden on the agency to provide evidence of risk or harm sufficient to justify regulation and to avoid being overturned on appellate review. These legal structures consequently invite manufacturers who are subject to regulation to raise skeptical concerns about the evidence, to delay, to be slow in producing any needed data about their products, to argue that there is insufficient information to justify the regulation, or to argue that there is too much uncertainty to permit an agency to issue a regulation at the current time. All this tends to delay regulatory action, to increase the implicit scientific and legal standards of proof that must be satisfied, to keep products in commerce longer, and to leave the public exposed to risks of harm longer while an agency deliberates about the issues.¹³⁴ Appellate review has been increas-

132. U.S. Congress, Office of Technology Assessment, *Identifying and Regulating Carcinogens*, OTA-BP-FI-42, 122, 127-28, 200-01, 215 (Washington, D.C.: U.S. Government Printing Office, Nov. 1987) (discussing potential legal requirements).

133. NAT'L RESEARCH COUNCIL, *RISK ASSESSMENT IN THE FEDERAL GOVERNMENT* 3 (Nat'l Acads. Press 1983) (delineating four typical stages of risk assessment covered by ambient exposure laws).

134. In addition, there appears to be poor monitoring of products once they are in the market to alert the company or the relevant agency of risks or harms that might be caused by their products.

ingly intrusive since the Supreme Court's benzene decision¹³⁵ in 1980 that affected not only the Occupational Safety and Health Administration (whose regulation was at issue) but other regulatory agencies as well by producing a "chilling effect" on their activities.¹³⁶

Consumer products, subject to national regulation, including plastic consumer products as well as lead in toys or jewelry, appear to be poorly regulated by the Consumer Product Safety Act and the Federal Hazardous Substances Act.¹³⁷ In 1985, the Consumer Product Safety Commission was quite small and had only a few toxicologists responsible for carcinogens. It is now even smaller and has fewer scientists to test products. Other agencies may not be so eviscerated, but Congressional funding for inspections and enforcement has tended to be substantially curtailed.

In theory, post-market laws authorize or encourage the use of surrogate means to identify risks before they materialize into actual human health and environmental *harm*. For example, the laws encourage the use of animal studies and other non-human evidence. In the early periods of regulatory activity, beginning in the 1970s, typically the risk information was based upon animal studies seeking to identify risks in mammalian models before substances caused harm to humans.¹³⁸ Despite the early emphasis on surrogates to identify risks to humans, there is constant pressure from affected industries to challenge the scientific value of animal and other kinds of evidence, as well as pressure to which agencies may acquiesce to support their regulations by epidemiological evidence of *human harm*. For example, a National Academy of Sciences (NAS) committee recommends "the most stringent criteria and requires epidemiologic evidence for drawing any positive conclusions about potential carcinogenicity; animal evidence and other test informa-

135. See *Indus. Union Dept., AFL-CIO v. Am. Petroleum Inst.*, 448 U.S. 607 (1980) (providing opinion regarding issue of exposure to benzene).

136. Carl F. Cranor, *The Regulatory Context for Environmental and Workplace Health Protections: Recent Developments*, in THE BLACKWELL GUIDE TO BUSINESS ETHICS 77-101 (Norman Bowie ed., Blackwell Publishers 2002) (identifying chilling effect stemming from benzene decision); THOMAS O. MCGARITY & SIGNED A. SHAPIRO, WORKERS AT RISK: THE FAILED PROMISE OF THE OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION 257-58 (Greenwood Publ'g Group 1993) (describing adverse consequences to agencies of overzealous judicial review of agency actions).

137. See Consumer Product Safety Act, 15 U.S.C. §§ 2051-2084 (2006) (providing text of Act); Federal Hazardous Substances Act, 15 U.S.C. §§ 1261-1278 (2006) (providing text of Act).

138. U.S. Congress, Office of Technology Assessment, *Identifying and Regulating Carcinogens*, OTA-BP-FI-42, 122, 26, 32-33, 45-52 (Washington, D.C.: U.S. Government Printing Office, Nov. 1987) (describing cancer risk assessment policies).

tion are used only to confirm cancer causation once epidemiological associations have been demonstrated.”¹³⁹

Fortunately, for public health protections, more recent NAS committees have disagreed with this view, recognizing that animal and other kinds of evidence can legitimately support regulatory actions.¹⁴⁰ To the extent that agencies are under pressure and regulate on the basis of human harm, risk-based statutes or statutes that explicitly anticipated the use of surrogates as a basis for regulation become human harm-based laws and lose any protective effects resulting from early detection of toxicants. In other words, humans must suffer harm before toxicants can be justifiably regulated to prevent harm to others.

This concern is heightened if one understands the commonly used tool for discovering evidence of human harm - human epidemiological studies. Epidemiological studies are statistics-based studies comparing either persons exposed to a toxicant with persons not so exposed (a cohort study), or comparing persons with disease to persons without (a case-control study). In each instance, the aim is to isolate and identify what diseases the exposures cause.¹⁴¹ Epidemiological studies are notoriously insensitive, however. As the joke goes, a catastrophe is something that even an epidemiological study can detect. Epidemiological studies can detect more common diseases and identify gross adverse effects with comparative ease where exposures are reasonably precise. They will, however, have much greater difficulty detecting diseases that have long latency periods, that are comparatively rare, that have new causes for common conditions (difficulty distinguishing the noise of a common disease from a new causal effect), that lack signature effects, or

139. COMM. ON TOXICITY TESTING AND ASSESSMENT OF ENVTL. AGENTS ET AL., TOXICITY TESTING FOR ASSESSMENT OF ENVIRONMENTAL AGENTS: INTERIM REPORT 134-38 (Nat'l Acads. Press 2006) (requiring epidemiologic evidence of human harm for positive conclusions on carcinogenicity and assigning animal evidence to merely supporting role).

140. See INST. OF MED., COMM. ON EVALUATION OF THE PRESUMPTIVE DISABILITY DECISION-MAKING PROCESS FOR VETERANS, IMPROVING THE PRESUMPTIVE DISABILITY DECISION-MAKING PROCESS FOR VETERANS 139-60 (J. M. Samet & C. C. Bodurow eds., Nat'l Acads. Press 2000) (discussing the need to combine diverse types of evidence in making judgment about whether exposure causes health concerns); see also NAT'L RESEARCH COUNCIL, TOXICITY TESTING IN THE TWENTY-FIRST CENTURY: A VISION AND A STRATEGY 21-38 (Nat'l Acads. Press 2007) (discussing risk assessment).

141. See CARL F. CRANOR, TOXIC TORTS: SCIENCE, LAW AND THE POSSIBILITY OF JUSTICE 96-97 (Cambridge Univ. Press, 2006) (providing description of different types of epidemiological studies); KENNETH J. ROTHMAN & SANDER GREENLAND, MODERN EPIDEMIOLOGY 73-74 (Lippincott Williams & Wilkins 2d ed. 1998) (discussing different types of epidemiological studies).

that have subtle adverse effects (which are likely to be typical of many developmental effects).¹⁴² Where exposures are difficult to determine, scientific conclusions become much more tentative. Adverse effects triggered during development could take years, if not decades, to be identified by epidemiological studies. Scientists could identify obvious adverse outcomes, such as shortened limbs, but would have much greater difficulty tracing the causes of shortened attention spans or reduced IQ from lead exposures.¹⁴³

Consequently, under post-market statutes, substances enter commerce without any legally mandated testing and no independent certification of some degree of safety. They remain in commerce providing financial benefits to manufacturers. If they cause harm, this harm continues until a regulatory agency presents a sufficiently strong scientific and legal case proving harm or risk of harm based upon animal and human evidence. If human evidence is insisted upon before there is regulation, these difficulties will be substantially exacerbated. For developmental toxicants that are subtle and that take years to manifest adverse effects, many cohorts of children will be subject to adverse outcomes before the risks are identified and eliminated. Under post-market laws the American citizenry are, in effect, human guinea pigs for the commercial creations of American industry. Our children are even more likely to be experimented upon and the harms may be more difficult to detect. This is not a health-protective approach toward our children.

Even risk-based laws utilizing non-human evidence, however, may not function well. Long-term animal studies commissioned by a government agency can easily take seven years to authorize, conduct, and analyze in order to produce usable results for regulation.¹⁴⁴ If manufacturers do not test products before

142. See CRANOR, *supra* note 141, at 225-27 (noting strengths and weaknesses of epidemiological studies); see also Rall et al., *Alternatives to Using Human Experience*, 355-85; Huff & Rall, *Relevance to Humans*, 433; ELAINE FAUSTMAN & GILBERT S. OMENN, *RISK ASSESSMENT, CASARETT AND DOULL'S TOXICOLOGY: THE BASIC SCIENCE OF POISONS* 86 (Curtis D. Klaassen, ed., McGraw-Hill 6th ed. 2001); S. R. Silver et al., *Effect of Follow-Up Time on Risk Estimates: Longitudinal Examination of the Relative Risks of Leukemia and Multiple Myeloma in Rubber Hydrochloride Cohort*, 42 AM. J. INDUS. MED. 481, 481-89 (2002).

143. Grandjean & Landrigan, *supra* note 25, at 2167-178 (discussing difficulties in determining cause of some adverse effects).

144. CARL F. CRANOR, *REGULATING TOXIC SUBSTANCES: A PHILOSOPHY OF SCIENCE AND THE LAW* 27 (Oxford Univ. Press 1993) (stating difficulty of animal studies commissioned by government agency); see also U.S. Congress, Office of Technology Assessment, *Identifying and Regulating Carcinogens*, OTA-BP-FI-42, 17 (Washington, D.C.: U.S. Government Printing Office, Nov. 1987) (discussing time line for animal studies). The Author and Karl Kronebusch of the Office of Technology Assessment also personally interviewed risks assessors at the U.S. EPA Air

commercialization, and the products cause adverse developmental effects in children, these affected children will continue to be born, exposed to toxicants for a minimum of seven years (and probably much longer). This could easily be the case with PBDEs. They have been in commerce since the early 1970s without testing before commercialization (despite similarities to PCBs), are in high concentrations in children's and adults' bodies, and now appear to present quite serious risks to children and perhaps to adults. Post-market laws do not prevent substantial exposures and any harm they might cause. At some time in the future there might be shorter-term tests that can accurately identify developmental toxicants, but until then, agencies will need to rely on the testing technologies that are currently available, despite their shortcomings. When pre-market or post-market laws fail to catch risks before harm occurs to the public, the workforce, or the environment, tort or personal injury law offers the possibility of correcting the mistakes for particular individuals who seek redress.¹⁴⁵

B. Harm-Based Tort (Personal Injury) Law

Tort law seeks to set matters right for a victim, typically by providing post-injury compensation sufficient to restore the injured person to the condition in which he or she would have been had the injury not occurred in the first place. In addition, the effects of *general deterrence* (the general threat of tort actions) or *deterrence by example* (any deterrence resulting from successful legal actions against others for harmful behavior or products) can include the modification of risky behavior and products prior to any further harm occurring. The extent of tort law deterrence is unclear, reflecting differing views in applicable literature.

Tort law addresses potential toxicants after the fact of an injury to a plaintiff, and virtually all legal causes of action must show evidence of some kind of harm.¹⁴⁶ In tort law, a plaintiff, the person claiming injury from a toxic substance, typically must show that a particular defendant's substance more likely than not can cause the kind of injury from which the plaintiff suffers and more likely than

Toxics Program in Washington, D.C. at the U.S. EPA main office who reported such long time-lines for testing April 1986.

145. See CRANOR, *supra* note 141, at 355-57 (putting tort laws in context with pre-market laws). See generally KEETON, *infra* note 185.

146. There are a few causes of action that do not explicitly require a showing of harm in order to justify a remedy, such as being at an increased risk of serious disease, medical monitoring, or reasonable fear of serious disease, such as cancer.

not caused plaintiff's injuries.¹⁴⁷ Thus, any "regulation" of toxicants by tort law (e.g., reductions in exposures, product reformulations or removal from the market) is post-market, harm-based, and post-injury at least for those harmed. The extent of deterrence that litigation provides is not clear, since individual companies could choose to respond quite differently to adverse legal decisions affecting other companies. Although tort law should allow for non-human scientific evidence of various kinds to show the potential human toxicity of products since scientists typically rely upon such evidence, there is even greater pressure by defendants and some judges to have plaintiffs provide evidence of harm based upon human studies.¹⁴⁸ To the extent that defense efforts are successful or judges require such showings, before a plaintiff can "put matters right" with a defendant whose products are believed to be harmful to the plaintiff, *other persons* must have already been harmed.¹⁴⁹ Tort law need not make such demands, but it is in defendants' interests to press for them and some judges require this before scientific experts are permitted to testify.¹⁵⁰

147. See *Donaldson v. Cent. Ill. Pub. Serv. Co.*, 767 N.E.2d 314 (2002) (showing that not all jurisdictions insist on this rigid legal distinction).

148. See CRANOR, *supra* note 141, at 224 (emphasizing court's demand for human data as necessary foundation for expert testimony); see also *In re Agent Orange Prod. Liab. Litig.*, 611 F. Supp. 1223, 1231 (E.D.N.Y. 1985); *Lynch v. Merrell-National Lab., Div. of Richardson-Merrell, Inc.*, 830 F.2d 1190 (1st Cir. 1987); *Brock v. Merrell Dow Pharm., Inc.*, 874 F.2d 307, 312 (5th Cir. 1989), *cert. denied*, 494 U.S. 1046 (1990); *Richardson v. Richardson-Merrell, Inc.*, 857 F.2d 823, 825, 831 n.59 (D.C. Cir. 1988), *cert. denied*, 493 U.S. 882 (1989); *Renaud v. Martin Marietta Corp.*, 749 F. Supp. 1545 (D. Colo. 1990); *Carroll v. Litton Sys., Inc.*, No. B-C88-253, 1990 WL 312969, at *47 (W.D.N.C. Oct. 29, 1990); *Thomas v. Hoffman-La-Roche, Inc.*, 731 F. Supp. 224 (N.D. Miss. 1989), *aff'd on other grounds*, 949 F.2d 806 (5th Cir. 1992); *Chambers v. Exxon Corp.*, 81 F. Supp. 2d 661 (M.D. La. 2000).

149. TONY HONORÉ, *THE MORALITY OF TORT LAW – QUESTIONS AND ANSWERS, PHILOSOPHICAL FOUNDATIONS OF TORT LAW* 79 (David G. Owen ed., Clarendon Press 1995) (characterizing aims and rationales of tort law).

150. See CRANOR, *supra* note 141, at 237-38 (arguing against requirement in tort law that other people must have suffered similar injuries); see also *In re Agent Orange Prod. Liab. Litig.*, 611 F. Supp. 1223, 1231 (E.D.N.Y. 1985); *Lynch v. Merrell-National Lab., Div. of Richardson-Merrell, Inc.*, 830 F.2d 1190 (1st Cir. 1987); *Brock v. Merrell Dow Pharm., Inc.*, 874 F.2d 307, 312 (5th Cir. 1989), *cert. denied*, 494 U.S. 1046 (1990); *Richardson v. Richardson-Merrell, Inc.*, 857 F.2d 823, 825, 831 n.59 (D.C. Cir. 1988), *cert. denied*, 493 U.S. 882 (1989); *Renaud v. Martin Marietta Corp.*, 749 F. Supp. 1545 (D. Colo. 1990); *Carroll v. Litton Sys., Inc.*, No. B-C88-253, 1990 WL 312969, at *47 (W.D.N.C. Oct. 29, 1990); *Thomas v. Hoffman-La-Roche, Inc.*, 731 F. Supp. 224 (N.D. Miss. 1989), *aff'd on other grounds*, 949 F.2d 806 (5th Cir. 1992); *Chambers v. Exxon Corp.*, 81 F. Supp. 2d 661 (M.D. La. 2000).

C. Ignorance of the Toxicity Properties of Products

The existing legal structure is not adequate to identify toxic substances early and before commercialization, making it more difficult to prevent diseases and adverse effects of toxic substances especially for children. In 1984, when the NRC sought to determine the current knowledge base regarding manufactured chemical substances in commerce, the results were disturbing. There were:

- 12,860 substances produced in volumes exceeding one million pounds per year; 78 percent of which had *no* toxicity information available, while eleven percent had minimal toxicity information (but insufficient for a complete risk assessment) [post-market];
- 13,911 chemicals produced in volumes of less than one million pounds; 76 percent with no toxicity data, twelve percent had minimal toxicity information) [post-market];
- 21,752 chemicals production volume unknown; there was no data on 82 percent and had minimal toxicity data.
- 8,627 food additives, 46 percent had no toxicity data, 34 percent had some toxicity information (but below the minimal level) and one percent had minimal toxicity information [some post-market];
- 1,815 drugs; twenty-five percent had no toxicity data, 36 percent had some toxicity data (but below the minimal level) and three percent had minimal toxicity information;
- 3,410 cosmetics; 56 percent had no toxicity data, eighteen percent had some toxicity data (but below the minimal level) and ten percent had minimal toxicity information [post-market];
- 3,350 pesticides; 36 percent had no toxicity data, twenty-six percent had some toxicity data (but below the minimal level) and two percent had minimal toxicity information.¹⁵¹

The vast majority of substances are subject to post-market regulation and the consequence has been *substantial ignorance about their*

151. See NAT'L RESEARCH COUNCIL, *supra* note 126, at 12 (providing statistics on ability to conduct health-hazard assessment of various substances). The data presented here comes from TOXICITY TESTING, but the presentation and organization are the Author's.

toxicity. On average, as near as one can estimate for eighty to ninety percent of substances subject to post-market laws, there is *no toxicity data* for about seventy percent of them.

The ignorance of drugs, pesticides, and new food additives, all chemicals subject to pre-market testing and approval laws, was not as serious as the ignorance of substances regulated under post-market statutes. Even the above statistic, however, is not especially impressive given that pre-market testing is required for some food and drug products because they had been grandfathered by federal regulation and were not subject to procedural testing laws. In the early 1990s, some members of the original NRC committee were asked to consider updating the NRC report, but insufficient changes in the data did not warrant an update.¹⁵² In 1998, there remained substantial knowledge-gaps for about seventy-five percent of approximately three thousand substances produced in the highest volume when the EPA entered into a voluntary agreement with producers to close the knowledge-gaps.¹⁵³ Major chemical companies sponsored about 1,900 of about 2,800 substances for which there was agreement to provide toxicity data, but about ten percent of the substances eligible for sponsorship remain without sponsors. Two and one-half years after the deadline for industry to submit final datasets, about one-fifth have not provided initial submissions and of those that have, one-third have not submitted final datasets.¹⁵⁴ Public release of the substances data from EPA has been delayed eighteen months.¹⁵⁵

The 3,000 substances produced in the highest volume might have been the most worrisome, but the United States Congress Office of Technology Assessment (OTA) found that there are an additional one thousand to twelve thousand substances for which extensive toxicological information would be important but was not

152. Conversation with John C. Bailor & Eula Bingham, (2002) (reporting discussion with NRC concerning possible update to NRC report).

153. Env'tl. Prot. Agency et al., *EPA, EDF, CMA Agree on Testing Program Targeting 2,800 Chemicals*, 37 ENVTL. HEALTH LETTER 193, 193 (Oct. 1998) (detailing agreement with Chemical Manufacturers Association for volunteering toxic products for testing purposes).

154. RICHARD DENNISON, PH.D., ENVTL. DEF., *HIGH HOPES, LOW MARKS: A FINAL REPORT CARD ON THE HIGH PRODUCTION VOLUME CHEMICAL CHALLENGE 3* (July 2007), http://www.environmentaldefense.org/documents/6653_HighHopesLowMarks.pdf (discussing final assessment for HPV Challenge, which was established by EPA and negotiated with manufacturers of HPV chemicals).

155. *Id.* at 3 (discussing that data sets were delivered to EPA more than eighteen months later than deadline for public release).

available.¹⁵⁶ These results suggest that any risks resulting from the products of a free enterprise system are beyond the understanding and control of the legal institutions designed to protect the public.¹⁵⁷ The post-market harm-based or risk-of-harm-based legal structure is failing to produce toxicity data about the products subject to its jurisdiction so that adverse effects can be prevented. There is evidence that the ignorance of the toxic properties of substances is not accidental. For example, Margaret Berger has pointed out that a corporation subject to regulation or a tort action

did not test its product adequately initially, failed to impart information when potential problems emerged, and did not undertake further research in response to adverse information. It appears that the corporations took virtually no steps to determine or minimize the possibility of harm until their hands were forced, *usually by litigation*. Only after extensive and expensive discovery have documents and witnesses come to light that showed the corporations' awareness of potential problems.¹⁵⁸

156. See OFFICE OF TECH. ASSESSMENT, CONGRESS OF THE U.S., OTA-BP-ENV-166, SCREENING AND TESTING CHEMICALS IN COMMERCE I (1995) (explaining importance of additional toxicological information). There are "some 15,000 chemicals that are produced in significant volumes, with approximately 3 - 4,000 produced in excess of 1,000,000 lbs/year." *Id.* Subtracting that 3,000 produced in excess of 1 million pounds leaves up to 12,000 produced in "significant volumes" about which there appears to be some concern. *Id.*

157. See POUL HARREMOËS ET AL., LATE LESSONS FROM EARLY WARNINGS: THE PRECAUTIONARY PRINCIPLE 1896-2000 11, 168-69 (Poul Harremoës et al. eds., European Env'tl. Agency 2001). There have also been *high profile harms to health and the environment* done by DDT, chlorofluorocarbons, PCBs, lead, mercury, cadmium, nickel, benzene, asbestos, and other toxicants, as well as poor disposal practices. There is *little monitoring/surveying of the current state of the environment and public health*. There are some *reporting requirements* as part of the Food, Drug and Cosmetic Act, (requiring reports on adverse drug and vaccine reactions) and as part of the Toxic Substances Control Act and the Occupational Safety and Health Act (requiring medical record-keeping and reporting of toxicity effects). There may be a few other such requirements, but the efforts appear insufficient to provide a systematic picture. Often, *long-term effects are even less well-known* than short-term adverse effects. There appears to be *little or no understanding of the life cycle of products*. There also appears to be *little sensitivity or response to credible warnings of serious adverse effects*. In LATE LESSONS FROM EARLY WARNINGS: THE PRECAUTIONARY PRINCIPLE 1896-2000, the European Environmental Agency notes that in the past there have often been credible early warnings of adverse effects on human health or the environment that went unheeded; there were no (or slow) legal or other social responses. Finally, even once a problem has been identified, there are often slow procedures to remove substances or reduce their risks. See *id.*

158. Margaret A. Berger, *Eliminating General Causation: Notes Towards a New Theory of Justice and Toxic Torts*, 97 COLUM. L. REV. 2117, 2135 (1997) (emphasis added) (discussing flaws in toxic tort litigation). Berger cites in particular studies of Agent Orange, asbestos, Bendectin, breast implants, the Dalkon Shield, thalidomide, tobacco, MER/29 (a cholesterol-reducing drug that caused cata-

Berger's concern has been echoed by others.¹⁵⁹

This, however, should not come as a surprise. The regulatory and tort law structures provide implicit incentives for manufacturers to refrain from testing their products. If they do conduct tests, they may be tempted to design the tests so that they do not detect adverse effects.¹⁶⁰ Additionally, self-interested considerations may prevent the full report of research findings, even when adverse effects are identified. Testing will only invite legal problems; tests showing even minimal adverse effects will invite inquiries from regulatory agencies or suits by tort lawyers. There are examples of these responses in the last thirty years, some within the last year.¹⁶¹

Each year the National Toxicology Program authorizes only a small number of animal tests on potentially cancerous substances.¹⁶² It is unclear the extent to which testing for other kinds of toxicity is authorized. Post-market testing, assessment and follow

racts), alachlor, atrazine, formaldehyde, and perchloroethylene. *See id.*; *see also* BARRY I. CASTLEMAN, REGULATIONS AFFECTING USE OF CARCINOGENS, CANCER CAUSING CHEMICALS 78 (N. Irving Sax ed., Van Nostrand Reinhold 1981) (discussing flaws in toxic tort litigation with respect to carcinogenic materials such as asbestos); David E. Lilienfeld, *The Silence: The Asbestos Industry and Early Occupational Cancer Research – A Case Study*, 81 AM. J. PUB. HEALTH 791, 791–98 (1991) (discussing flaws in toxic tort regulation with respect to asbestos); David Michaels, *Waiting For The Body Count: Corporate Decision Making and Bladder Cancer in the U.S. Dye Industry*, 2 MED. ANTHROPOLOGY Q. 215, 215–27 (1988) (discussing flaws in toxic tort litigation in context of cancer causing dyes); Donald R. Mattison & John E. Craighead, *Reproductive System*, in PATHOLOGY EVTL. & OCCUPATIONAL DISEASE 559–72 (John E. Craighead ed., Mosby 1995) (discussing flaws in toxic tort litigation in American occupational environment). In some instances, even responsible firms may also fall prey to ambiguity and difficulty in interpreting data and scientific studies.

159. *See e.g.*, Carl F. Cranor & David A. Eastmond, *Scientific Ignorance and Reliable Patterns of Evidence in Toxic Tort Causation: Is There a Need for Liability Reform?*, 64 LAW & CONTEMP. PROBS. 5 (2001) (discussing fact that plaintiffs are placed in position where they must expend large sums of money to prove they were exposed to dangerous chemicals, when chemicals toxicity could have been studied and determined before public exposure).

160. *See* CRANOR, *supra* note 141, at 357 (discussing market incentives that keep companies from fully investigating toxic effects of chemicals before they are placed in stream of commerce); *see also* John C. Bailar, III, *How to Distort the Scientific Record Without Actually Lying: Truth, and the Arts of Science*, 11 EUR. J. ONCOLOGY, 217, 218 (2006) (describing paths of manipulation that can be used to “lie with statistics” and other techniques to distort toxicity of products).

161. Alex Berenson, *Follow-Up Study on Vioxx Safety is Disputed*, N. Y. TIMES, May 13, 2006, available at <http://www.nytimes.com/2006/05/13/business/13merck.html?n=top/Reference/Times%20Topics/Subjects/L/Liability%20For%20Products&pagewanted=all#> (reporting that Merck interpretation of study showing patients who ceased taking Vioxx after one year were at no significant risk for heart attacks was “narrowly correct, but misleading”).

162. Conversation with Ronald Melnick, Senior Toxicologist and Director of Special Programs, Environmental Toxicology Program, National Institute of Environmental Health Services (Oct. 25, 2002) (explaining how few substances are tested for toxicity each year on animals).

up of suspect substances lags far behind the production of new substances. If federal or state agencies have not assessed the toxicity of substances, it is unlikely that others have tested the substances or that there is sufficient public information about the products.

There are numerous and substantial problems with post-market laws. The public will be protected only if government agencies act (or successful tort suits lead to the removal or reduction of the cause of harm), but they must act in legally difficult circumstances. The agency has the burden to change legal relationships. It must meet that burden and withstand review by an appellate court, which has become increasingly difficult in recent years.¹⁶³ New scientific studies needed to support legal action take up to seven years for animal studies, and possibly much longer for human studies to allow for the induction and latency periods of disease (e.g., much longer for developmental effects that take time to manifest themselves, such as learning disabilities).¹⁶⁴ Human studies take much longer for developmental effects, such as learning disabilities, to manifest themselves. Regulated parties subject to administrative proceedings often possess tests on products and exposure data, especially in occupational settings, allowing them to substantially affect whether data are revealed and influence a regulatory outcome. Moreover, legal action is delayed by claims that more evidence is needed, that the science presented is not sufficiently sound, and that conclusions must be asserted with greater certainty.¹⁶⁵ Further research into scientific conclusions can delay regulation or court decisions for years or sometimes decades.¹⁶⁶

163. Cranor, *supra* note 136, at 77-78 (discussing agency challenge of enforcing and litigating post-market tort and toxicity laws). Moreover, regulatory action has become increasingly data and evidence intensive as regulated industries increasingly challenge legal action and their challenges are upheld by federal courts. *See id.* This feature of the regulatory world means that because of the data intensive nature of regulation, extensive documentation of the legal record, protective regulations often take years or even decades before exposures are reduced to safe or safer levels. *See id.*

164. KENNETH J. ROTHMAN & SANDER GREENLAND, Types of Epidemiologic Studies, in MODERN EPIDEMIOLOGY 67, 68-70 (Kenneth J. Rothman & Sander Greenland eds., Lippincott Williams & Wilkins 1998) (discussing strengths and weaknesses of different kinds of epidemiological studies).

165. *See* CRANOR, *supra* note 141, at 200-04 (recognizing different methods that firms in regulated industries adopt or utilize to challenge accuracy and relevance of negative evidence in court).

166. *See* CRANOR, *supra* note 144, at 121 (highlighting detrimental effect of delays in regulation); *see also* Sara M. Hoover et al., *Improving the Regulation of Carcinogens by Expediting Cancer Potency Estimation*, 15 RISK ANALYSIS 267, 267-80 (Apr. 1995).

Any exposure of developing fetuses and children to toxic substances may well be greater than those for adults, as they currently appear to be for PBDEs. If agencies are maneuvered into requiring human studies to show risks of harm, they could utilize cohort epidemiological designs to study children already known to have been exposed to a toxicant prenatally or neonatally and follow them for sufficient time to permit subtle adverse effects to manifest. Case-control studies could also be used to study children with subtle learning disability problems to determine what exposures or lifestyle factors are associated with the adverse effects. Neither cohort nor case-control studies are easy to use in order to detect subtle effects arising from developmental or early childhood exposures. Human epidemiological studies are generally insensitive and are quite limited in detecting subtle differences between healthy and adversely affected children. Currently, there is a national children's cohort study aimed at identifying factors that contribute to adverse developmental effects.¹⁶⁷ Even if the studies are able to detect toxicants' adverse effects, many children, *de facto* serving as research subjects, will suffer the developmental effects in the process of establishing that exposure causes *harm*.

If animal studies are utilized, to the extent that efforts to reduce exposures to carcinogens are typical, regulated parties will argue that animal studies do not indicate potential human harm or do not accurately measure exposures that will pose harm to humans. Regulated parties will focus on the differences between humans and animals, arguing that few reliable inferences can be made from animals to humans. In the case of developmental toxicants, critics of testing will argue that there are developmental differences between humans and different animals, such as rats, mice, guinea pigs, or rabbits.¹⁶⁸ Selecting an appropriate model for study is therefore challenging. Despite this, there appears to be substantial positive predictive value in animal studies for human harm (75 percent to one hundred percent).¹⁶⁹ This degree of accuracy

167. Grandjean & Landrigan, *supra* note 25, at 9 (discussing national children's cohort study).

168. See SCHARDEIN, *supra* note 23, at 40 (indicating that, "animal studies cannot predict effects in humans with absolute certainty"). Suitably designed studies give some indication of risk, but they do not guarantee safety to the fetus. *Id.*

169. *Id.* (using empirical tests to indicate scientific value and accuracy from animal testing). Positive predictive value in this case appears to be the number of animal studies that correctly predict human harms divided by the number of positive predictions, both those that correctly predict harm and those that mistaken predict harm (Correct predictions from animals/(correct predictions + incorrect predictions)).

seems defensible for public health protections, because it is better to correctly identify between 75 and 100 percent of the developmental toxicants by means of animal studies than to wait for slow, science-intensive, insensitive human epidemiological studies to try to identify them after the fact of exposure.

Thus, our mammalian relatives provide practical models on which to conduct actual experiments with toxicants (humans are unacceptable research subjects). Moreover, as in other areas there are substantial similarities in mammalian biology in order to provide insights and evidence for likely human harm. Those wishing to restrict studies of developmental toxicology to humans will have to live with the consequences of harm to their fellow citizens and their children while long-term, insensitive epidemiological studies are conducted on people in an attempt to detect subtle (and not-so-subtle) adverse effects. If these studies are even scientifically plausible, some diseases may not manifest themselves until middle or old age, and generations of cohorts will have been exposed for which such scientific and legal policies would have to answer.¹⁷⁰

Is there a better way to go, so we do not have to rely upon time-consuming, corroborative science with imprecise, insensitive studies in order to prevent our children from suffering harm? If products cause adverse effects, the harm will not be halted until exposures are reduced sufficiently to eliminate the harm and to satisfy the law and appellate courts in question. For developing children, many years of exposed cohorts would suffer adverse effects before exposures causing harm could be reduced or eliminated. While there are some legal reforms that would assist in addressing these issues, this Article presents two alternative moral conceptualizations of these issues that will lead to different legal approaches.¹⁷¹

V. AUTONOMOUS CHOICES IN USING LIFESTYLE DRUGS

We can begin to appreciate some of the rights citizens have or should have over aspects of their life in the regulation of toxicants by considering a proposal by Margaret Berger and Aaron Twersky for the reform of part of tort law, otherwise a bastion of harm-based, post-market "regulation" of toxicants. They recommend reforming part of tort law, calling attention to the rights citizens

170. Schwartz & Stewart, *supra* note 35, at 671 (citing concern that lead currently in citizens' bodies will cause future detrimental health effects).

171. See generally Carl F. Cranor, *Some Legal Implications of the Precautionary Principle: Improving Information Generation and Legal Protections*, EUR. J. ONCOLOGY 31, 41-44 (2003) (proposing various legal reforms to provide monitoring mechanisms for exposure effects and disease).

should have over drugs that they voluntarily take for lifestyle purposes, e.g., such as Viagra to enhance sexual potency. "Lifestyle drugs," drugs that are not medically necessary to one's health, are drugs that enhance one's life in various ways. These authors have argued that

the time has come for courts to recognize the right of patients to informed choice about risks associated with the use of a drug, a right that does not require plaintiffs to prove that the toxic agent was the cause of the plaintiff's harm. To do so we shall suggest a new paradigm for this informed choice cause of action that protects the right of patient autonomy, yet does not impose liability for the full extent of damages as would be the case when a plaintiff is able to prove causation. Absent recognition of a right predicated on informed choice, plaintiffs will be deprived of vital information necessary to make critical decisions regarding lifestyle drugs and pharmaceutical manufacturers will have little incentive to discover and warn about uncertain risks. With causation standing as a barrier to recovery, defendants will sit back confident that liability is highly unlikely to attach to conduct that is admittedly negligent.¹⁷²

Thus, Berger and Twersky argue that tort law imposes a duty on a manufacturer to warn consumers against risks posed by a drug that are uncovered by research after products are in commerce so that individuals can make informed decisions before taking the drug. This duty is breached under current law when a risk is of sufficient consequence that reasonable persons would seek to be warned against such risks.¹⁷³

The Learned Hand risk-utility test requires that an actor take precautions to warn against even remote risks when the gravity of the foreseeable harm is great. That there be a causal nexus between the defendant's wrongful conduct and the harm suffered is a principle deeply ingrained in tort jurisprudence and we do not question that hoary maxim. However, in the context of toxic tort cases, to re-

172. Margaret A. Berger & Aaron D. Twersky, *Uncertainty and Informed Choice: Unmasking Daubert*, 104 MICH. L. REV. 257, 259 (2005) (arguing that citizens must be given accurate information about toxicants in products in order to make informed autonomous consumer choices).

173. *Id.* at 267-68 (discussing breach of legal duty).

quire that the plaintiff actually demonstrate that the toxic agent caused the plaintiff's harm flies in the face of the well-recognized right of a patient to make an autonomous decision as to whether she wishes to expose herself to even an uncertain risk. The *assault on autonomy* is especially egregious in the case of lifestyle drugs where the drug has little therapeutic value. In such cases one can predict with a high level of confidence that a patient informed of the potential risk would almost certainly have opted against taking the drug and subjecting herself to the risk.¹⁷⁴

Implicit in the Berger/Twersky proposal is an individual's right to make autonomous decisions about risks associated with lifestyle drugs. This idea of autonomy is deeply embedded in our moral, legal and civic culture. American society insists on patients being informed so they can make autonomous choices when undergoing medical procedures, operations, and clinical trials. Informed consent has been part of medical malpractice law for more than three decades,¹⁷⁵ and part of medical testing guidelines for at least sixty years.¹⁷⁶ Moreover, courts have endorsed an informed choice cause of action in products liability law:

In *Davis v. Wyeth Laboratories, Inc.*, [399 F.2d 121 (9th Cir. 1968)] the defendant manufacturer sold polio vaccine without warning of the risk that one person in a million would contract polio from taking the vaccine. The court held that the manufacturer had a duty to warn the consumer of the risks involved and that the failure to meet this duty rendered the drug unfit and unreasonably dan-

174. *Id.* at 268 (emphasis added) (presenting therapeutic damages for lifestyle damages). See generally *United States v. Carroll Towing Co.*, 159 F.2d 169 (2d Cir. 1947); ARNO C. BECHT & FRANK W. MILLER, *THE TEST OF FACTUAL CAUSATION IN NEGLIGENCE AND STRICT LIABILITY CASES* (1961); DAN B. DOBBS, *THE LAW OF TORTS* 166-69 (West Group 2000); Wex S. Malone, *Ruminations on Cause-in-Fact*, 9 STAN. L. REV. 60 (1956); Richard W. Wright, *Causation in Tort Law*, 73 CAL. L. REV. 1735 (1985).

175. See generally TOM L. BEAUCHAMP & LEROY WALTERS, *CONTEMPORARY ISSUES IN BIOETHICS* 139-55 (Tom L. Beauchamp & LeRoy Walters eds., Thomson 6th ed. 2003) (discussing history and practicability of informed consent in medical malpractice law).

176. See *id.* at 354-57. See also Library of Congress, *Trial of War Criminals Before the Nuernberg Military Tribunals Under Control Council No. 10.* (Oct. 1946-April 1949), http://www.loc.gov/rr/frd/Military_Law/NTs_war-criminals.html; World Medical Association, *Declaration of Helsinki: Ethical Principles for Medical Research Involving Human Subjects* (Sept. 10, 2004), <http://www.wma.net/e/policy/b3.htm> (identifying informed consent principles within text of international codes and declarations).

gerous within the meaning of [§] 402A of the Restatement (Second) of Torts.¹⁷⁷

In addition, courts have recognized a cause of action for battery when medical experiments on DES were conducted on patients without their knowledge.¹⁷⁸ In order “to state a cause of action for battery, the plaintiffs must allege intentional acts by the defendants resulting in offensive contact with the plaintiffs’ person, and the lack of consent to the defendants’ conduct [Moreover,] the actor must ‘intend to cause the other, directly or indirectly, to come in contact with a foreign substance in a manner which the other will reasonably regard as offensive.’”¹⁷⁹ The court found that “the administration of a drug without the patient’s knowledge comports with the meaning of offensive contact. Had the drug been administered by means of a hypodermic needle, the element of physical contact would clearly be sufficient. We believe that causing the patient to physically ingest a pill is indistinguishable in principle.”¹⁸⁰

Finally, tort law recognizes similar assaults on autonomy by granting dignitary damages “for assault, battery and false imprisonment without regard to whether the plaintiff suffered physical harm.”¹⁸¹ The need for informed consent and the importance of personal autonomy has been much more widely utilized in moral philosophic discussions since its rise to prominence in the medical ethics context and, one might even say, since John Stuart Mill wrote about the importance of autonomy in *On Liberty* in 1859.¹⁸²

Berger and Twersky recommend reduced compensation for violation of informed consent for risks from lifestyle drugs, presumably because full-fledged *harms* may not have occurred, but are still violations of rights. Compensation for violation of informed consent is recognition that a *wrong* has occurred, but perhaps not yet a physical *harm* or not as serious a harm as when a risk fully material-

177. Berger & Twersky, *supra* note 172, at 273 (citing case law to emphasize the importance of informed consent, even in extremely low risk circumstances).

178. *Mink v. Univ. of Chi.*, 460 F. Supp. 713, 718 (1978) (furthering importance of informed consent in cases where medicine was used on patients without their knowledge).

179. *Id.* (quoting in part the Restatement (Second) of Torts section 18, Comment (c) at 31 (1965)) (detailing informed consent and intent requirements necessary to hold firm accountable in torts).

180. *Id.* (defining offensive contact in medical profession).

181. Berger & Twersky, *supra* note 172, at 282 (discussing dignitary tort damages); see generally DAN B. DOBBS, *THE LAW OF TORTS* 42 (West Group 2000).

182. See generally JOHN STUART MILL, *ON LIBERTY* (Curran v. Shields ed., Prentice Hall PTR 1956) (discussing the importance of autonomy in moral philosophy).

izes.¹⁸³ Thus, citizens should have a cause of action and compensation for a violation of informed consent and should receive higher compensation according to the degree of harm caused.

Autonomous choices as articulated by Berger and Twersky presuppose: (1) that the person has moral and legal rights in this area of their lives; (2) that they have or should have more specific moral and legal authority to exercise this control over lifestyle drugs that they voluntarily consume; and (3) that the person should have knowledge of risks from such drugs in order properly to exercise their rights in this respect. Berger and Twersky's paper, strongly reinforced by the court's decision in *Mink*, provides a useful comparison with the inevitable entry of chemicals in citizens' bodies.

VI. APPROACHES TO ADDRESSING INVASIONS OF MANUFACTURED CHEMICAL SUBSTANCES

A. Use of Legal Presumptions

Given the scientific case for the increased chances of developmental harm to children, one legal strategy to prevent increased chances of developmental harm to children is that scientific evidence supports the legal presumption that *in utero* and perinatal exposures to substances have the potential to cause harm during development. Consequently, manufacturing firms would have the burden of proof, as opposed to government agencies, and would have to show, on the basis of animal and other non-human evidence, that the exposures would not pose risks of harm to developing children before the products entered commerce or the environment or in order to permit existing substances in commerce to remain. This approach would be based on the idea of a legislatively created *legal presumption*.

Legislative bodies and courts often create presumptions. For instance, in criminal law Congress created a presumption that a person arrested in the United States in possession of marijuana or

183. See Berger & Twersky, *supra* note 172, at 285 (comparing invasion and negligent infliction of emotional distress). In explaining their view, Berger and Twersky state, "[w]e thus advocate a cause of action for negligent infliction of emotional distress when plaintiff is deprived of an informed choice about material risk even if the causation of the actual physical injury cannot be established with the certainty demanded by traditional causation norms. We would expect that the greater the materiality of the risk, the greater the damages assessed against the defendant. And we would also expect that greater damages would be assessed if it were found that a defendant acted in bad faith in refusing to reveal material risk information. The sense of betrayal and hurt suffered by a plaintiff deprived of meaningful choice cannot be divorced from the conduct of the defendant who was responsible for the deprivation." *Id.*

heroin “unless explained to the jury’s satisfaction, ‘shall be deemed sufficient evidence to authorize conviction’ for smuggling, . . . buying, selling, or facilitating the transportation, concealment, or sale of the drug, knowing that it had been illegally imported.”¹⁸⁴ Congress therefore created the presumption that such a person is a drug smuggler and dealer, not a mere purchaser of drugs for his or her own use. Thus, the penalties would be much greater for importing and dealing than for mere purchase for personal use. If such presumptions can be used for the criminal law where there is a much greater concern for individual rights, they could also be utilized to protect our children with respect to toxicants.

If there were a presumption that chemical substances could enter the uterus and potentially pose risks to the fetus, companies would have to provide evidence, not mere assertions, to rebut it. If a presumption were used for manufactured products, however, it might be criticized as non-scientific, simply because it would be legislatively created, despite the powerful scientific case that manufactured chemical products invade and expose fetuses *in utero* and neonatally. This need not be a fatal flaw, and may seem somewhat awkward, but legislatures have this authority. There are two additional legal models, however, that are preferable because they better capture moral concerns.

One strategy is based on the idea of trespass, or more broadly, interference with one’s rights over one’s person and possessions. The other strategy is based on jurisdictions placing reasonable conditions on permissions for companies to create chemical products for commercial products. Similar to a “permission” model, and much like the REACH legislation in Europe, if there is no safety data about products for developing children or adults, companies may not market their products and expose citizens. The slogan is no data, no market. The remaining sections of this Article will discuss some of the implications of the trespass view and then the permission view.

184. *Turner v. United States*, 396 U.S. 398, 404 (1970) (citing 21 U.S.C. § 176(a) (2006)) (stating presumption existence).

B. The Trespass Model

1. *Background*

The doctrine of trespass provides much of the historical background of torts,¹⁸⁵ but what remains in contemporary tort laws as trespass largely concerns real property:

Historically, the requirements for recovery for trespass on land under the common law action of trespass were an invasion (a) which interfered with the right of exclusive possession of the land, and (b) which was a direct result of some act committed by the defendant An interference with this exclusive possessory interest brought about in a direct way from an act committed by the defendant was regarded legally as actionable. This was so even though the invasion caused no harm and even though the defendant was not at fault in causing the invasion The most important of the trespass rules to survive [from historical doctrine] was that which imposed liability for invasions of property which were neither intended nor negligent. The defendant was not liable so long as he had done no voluntary act, as where he was carried onto the plaintiff's land by others against his will.¹⁸⁶

Moreover, a trespass could be sustained "without proof of any actual damage. From every direct entry upon the soil of another, 'the law infers some damage; if nothing more, the treading down grass or herbage.'"¹⁸⁷ "The action was directed at the vindication of the *legal right*"¹⁸⁸ In addition, "one who trespasses upon the land of another incurs the risk of liability for any bodily harm which is caused to the possessor of the land or to members of the household by any conduct of the trespasser during the continuance of the trespass."¹⁸⁹

More importantly an invasion of the right to possession results if one enters the property without "authorization," without paying for it, or without permission.¹⁹⁰ These terms are important because it is clear that one who has the right to exclusive possession of prop-

185. See W. PAGE KEETON ET AL., PROSSER & KEETON ON TORTS 28-30 (W. Page Keeton ed., West Publ'g Co. 5th ed. 1984) (discussing historical place of trespass in tort law).

186. *Id.* at 67-68 (discussing trespass).

187. *Id.* at 75 (explaining trespass).

188. *Id.* (emphasis added) (discussing legal rights).

189. *Id.* at 76 (discussing bodily harm and trespass).

190. See KEETON ET AL., *supra* note 185, at 70-71 (detailing aspects of trespass).

erty has the important associated *right to choose who can be on it or the right to authorize the presence of others on it or the right to license them to be there*.

The trespass doctrine principally concerns real property, but some case law extends that right to persons.¹⁹¹ Trespass encompasses not only the deposition of persons or things, but also of molecules and particles on another's land.¹⁹² More importantly, however, the idea of trespass rests on a deeper consideration, related to the rights over aspects of one's life and the right to authorize others to enter those protected areas. For example, Arthur Ripstein's following narrative suggests that harmless trespass into your house is a wrong:

I let myself into your home, using burglary tools that do no damage to your locks, and take a nap in your bed. I make sure everything is clean. I bring hypoallergenic and lint-free p[a]jamas and a hairnet. I put my own sheets and pillowcase down over yours. I do not weigh very much, so the wear and tear on your mattress is nonexistent. By any ordinary understanding of harm, I do you no harm You[r] objection is to my deed, my trespass against your home, not to its effects.¹⁹³

Other violations of a person's right to authorize entry onto one's property or to authorize intrusions on one's person include: (1) unknown fluoridation of your teeth at the dentist's office even though you are philosophically opposed to it; (2) a doctor's unauthorized touching a patient in an intimate place while he or she is sedated; and (3) harmless medical experiments performed on unconscious patients that leave no trace, do no obvious harm, and are without permission.¹⁹⁴

To add to this point, consider the following hypothetical. Suppose you dispose of some trichloroethylene (TCE) from home ex-

191. *Sullivan v. Dunham*, 55 N.E. 923, 926-27 (N.Y. 1900) (extending trespassing liability to personal injury caused by debris from blast on one's own property).

192. See *Martin v. Reynolds Metals Co.*, 342 P.2d 790 (Or. 1959) (holding manufacturing operation which caused fluoride gases and particulates to become airborne and settle upon owner's land constituted direct trespass); *Borland v. Sanders Lead Company, Inc.*, 369 So.2d 523 (Ala. 1979) (characterizing dangerous accumulation of lead particulates and sulfide deposits as trespass).

193. See Arthur Ripstein, *Beyond the Harm Principle* 34 PHIL. & PUB. AFF. 215, 218 (2006) (discussing harmless trespass as trespass nonetheless).

194. *Id.* at 227 (comparing to harmless invasions).

periments in a hot tub without permission.¹⁹⁵ TCE is a carcinogen, but diluting small amounts in a hot tub reduces any risk of harm. Have you done anything *wrong*? You did not *harm* those who were exposed to TCE in the hot tub, or perhaps even pose a *risk of harm*. But the TCE invaded, or “trespassed” on the hot tub owner’s property and perhaps on those who used the hot tub. It was present on the property and therefore exposed others to the chemical without authorization. Following the reasoning in *Mink*, the disposal of TCE could be thought of as a battery, exposing the hot tub users to a chemical without permission just as Patsy Mink was exposed to DES without permission.¹⁹⁶

Similarly, if a doctor conducts medical experiments on patients by exposing them to molecules without their permission as the University of Chicago did to Mink and others, the doctor has trespassed on or battered and wronged them. Finally, consider another example, more like human invasions of chemical substances. Suppose a chemist has an unknown substance. The chemist does not know whether it is toxic or not, a poison or not, or totally benign; it might even be beneficial if it enters your body. If the chemist puts this substance into another’s drink or into the environment knowing it will enter his or her body without informing the person and asking permission, has the chemist not wronged the victim by simply putting it into his or her drink?

This Article does not propose principles for the criminal law, as Ripstein does. Nor does it propose a principle for the tort law. Instead it suggests a model by analogy with trespass to guide regulatory policy in governing toxic exposures. The above examples contain a common thread: a person is *wronged* even though no harm is done because the pertinent invasion violated a right without proper authorization by the person invaded, either a specific or a more general authorization. Ripstein has argued that an *unauthorized* invasion of citizens’ bodies or property by others is a wrong analogous to a trespass.¹⁹⁷ This Article suggests by examples that invasion by potentially toxic substances is analogous to a trespass or even a battery. Morally, citizens should regard invasion of their bodies with-

195. See Raymond Neutra, *Risk Assessment*, 237 Sci. 235 (Jul. 1987) (detailing experiment).

196. See *Mink*, *supra* note 178, at 718 (discussing action under theory of battery taken by women given diethylstilbestrol against university and manufacturer of drug).

197. See Ripstein, *supra* note 195, at 228, 233-36, 241-42 (explaining how various unauthorized invasions of persons or their property somewhat resembles trespass).

out permission by humanly created substances as a trespass. The deeper point is the idea of a person having considerable rights or authority, or as Ripstein puts it, “sovereignty” over her life and property.¹⁹⁸ The invasion of a person’s sovereignty or authority by invading her person or her property is a trespass into *areas of her life over which she has or should have sovereignty* or substantial authority.¹⁹⁹ Thus, he argues for a sovereignty principle, noting that “the only legitimate restrictions on conduct are those that secure the mutual independence of free persons from each other.”²⁰⁰ This Article does not follow out such a general idea, but it may hold promise for the view suggested here.

When citizens have an explicit right to either accept or reject exposure to substances such as lifestyle drugs, as Berger and Twersky argue, they should be informed of any risks that have emerged since the drug was approved, so that they have a realistic opportunity to exercise that right.²⁰¹ Any rights citizens have or should have over their bodies and the drugs they take is empty unless they possess the requisite knowledge of the risks involved. Thus, knowl-

198. *Id.* at 229-45 (discussing concept of sovereignty and some of its implication).

199. *See id.* at 231, 233-36, 241-42 (showing various ways wrongs consist in violations of areas of life over which one should have authority).

200. *Id.* at 229 (discussing restrictions on conduct). He adds to this view as follows:

You are independent if *you are the one who decides* what ends you will use your powers to pursue, *as opposed to having someone else decide for you* . . . You remain independent if nobody else gets to tell you what to do. . . [Moreover, the interest in independence is] a distinctive aspect of your status as a person, entitled to set your own purposes, and not required to act as an instrument for the pursuit of anyone else’s purposes . . . Each person is free to use his or her own powers to set and pursue his or her own purposes, consistent with the freedom of others to use their powers to set their purposes. *Id.* at 231 (Emphasis added).

[In short] the sovereignty principle says that each person is entitled to use his or her own powers as he or she sees fit, consistent with the ability of others to do the same. The consistency is achieved through the joint ideas of non-interference and voluntary cooperation. Nobody is allowed to use or damage another person’s means without their permission. If everyone forbears from doing these things, each person is independent of all the others. . . . Wrongdoing takes the form of domination. *Id.* at 233.

[P]art of being free to use your powers to set and pursue your *own* purposes is *having a veto on the purposes you will pursue*. . . . When I usurp your powers, I violate your sovereignty precisely *because I deprive you of that veto*. *Id.* at 234-35 (emphasis added).

201. *See* Berger & Twersky, *supra* note 172, at 259 (explaining risks emerging from research into Bendectin and Parlodel, two “lifestyle” drugs, but whose risks were not sufficient to support tort cause of action for harm, but were risks patients would likely want to know before ingesting).

edge of the risks from products is a condition of a credible exercise of their rights.

Berger and Twersky's issue in torts is not the problem that many citizens face concerning protections from involuntary chemical invasions. The CDC's biomonitoring program and the Faroe Islands Conference on the human health effects of developmental exposure to environmental toxicants together have identified the extent of involuntary, uninformed invasions, as well as risks from invasions to fetuses, developing children, and, of course, adults. Citizens are generally not aware of, nor do they have individual control over chemical invasions in the same way they have choices to ingest lifestyle drugs. Citizens could make political decisions in the future about how the law should address such substances, if they understood the issues.

This Writer does not want to be an alarmist about the chemical invasions, but it is a matter of serious concern. Some substances pose no risks or harm, but others do. Some substances enter our bodies through the ingestion of foods and water. Many of these substances contain nutrients the body needs. Others are naturally occurring substances that might enter our bodies through ingestion or through inhalation. Some substances, however, might enter citizens' bodies as a result of their living near industrial activities that concentrate naturally occurring substances that pose risks.²⁰² Some substances in our bodies are not naturally occurring substances at all, but are rather industrial chemicals created for specific purposes, such as endocrine mimicking chemicals in plastic bottles or other products, organochlorine pesticides, flame retardants (PBDEs) in furniture, beds and computers, and so on. Still other substances may be the by-products or contaminants of other manufacturing processes such as smelting, but invade our bodies and pose substantial potential risks, such as arsenic, dioxins or polycyclic aromatic hydrocarbons. Indeed, considerable care is needed to craft an effective substance invasion policy because they come from so many different sources. Accordingly, the following section will focus on possible policy responses to these concerns.

2. *Policy Proposals*

What might be a morally appropriate policy for humanly created substances that invade our bodies or trespass on us? Industrial chemicals invade even if at present citizens strongly would prefer to

202. Examples of this would include smelters that concentrate metals such as arsenic, cadmium or mercury.

prevent this. If citizens cannot make individual decisions about and *control* whether industrial chemicals enter their bodies, how should they think about the *involuntary invasion* of potentially toxic substances.

This problem bears some similarities and some dissimilarities to trespass. If someone harmlessly trespasses on property, although the land owner might prefer to control access, he or she is not always able to exclude trespassers by posting warnings, creating enclosures to resist entry, or locking doors. Nonetheless, the trespass is a wrong precisely because *he/she did not authorize it*. That is, a trespass occurred even though it is difficult physically to prevent it. In this respect trespass on a person's property resembles chemical entries into our bodies.

The creation of new substances and their release when they are *known to invade human bodies* are also deliberate acts akin to trespass on property.²⁰³ That is, someone knowingly created the substance, and given the tiny size of molecules, knew or was practically certain that these substances would invariably invade human bodies. Moreover, the deliberate creation and knowledge that a product will invade resembles the intentionality needed for trespass.²⁰⁴ Consequently, it is plausible to argue that the creation of chemical substances known to invade bodies is an instance of "an intent to be [in the body] where the trespass allegedly occurred," analogous to the presuppositions of trespass (or even battery) in tort law.²⁰⁵

An ideal policy, of course, would be no invasion without explicit permission; that is the moral basis of human medical experimentation and the informed consent doctrine that goes along with it,²⁰⁶ as well as the moral basis of battery and trespass on property.²⁰⁷ This, however, is impractical because citizens do not have the ability to choose or not choose whether individual chemicals invade on a particular occasion, although they could collectively address this politically. Moreover, the CDC biomonitoring shows that nearly, but not quite all substances that are in the air, our food, our

203. New substances might also include naturally occurring substances that are in much greater concentration than they typically are in nature.

204. See KEETON ET AL., *supra* note 185, at 73 (describing level of intent needed for trespass). "The intent required as a basis for liability as a trespasser is simply an intent to be at the place on the land where the trespass allegedly occurred." *Id.*

205. *Id.* (discussing intent associated with trespass).

206. See *Mink v. Univ. of Chi.*, 460 F. Supp. 713, 716-17 (1978) (discussing traditional and modern view of informed consent as well as the two theories of liability for unauthorized medical treatment; battery or assault and negligence).

207. This Author has a paper in progress on this topic.

water, some things we touch, will enter human bodies, including the placenta. None of these entries can generally be prevented. By contrast, citizens' choices can prevent the entry of vaccines, lifestyle drugs, and medicines.

To sharpen the issue, consider a hypothetical that bridges some of the differences between involuntary chemical invasion of industrial chemicals and Berger and Twersky's examples concerning lifestyle drugs. Imagine a world in which persons had individual control over the entry of chemical substances into their bodies. Suppose we had an invisible, partially chemically impermeable cloak, similar to the children's book character Harry Potter's invisibility cloak, that blocked the invasion of many chemical substances.²⁰⁸ Suppose we could program it to exclude substances, but this operation was fairly difficult to carry out. Suppose also that it permitted the entry of all chemical substances unless we programmed it to exclude individual substances. If we had this kind of limited and somewhat burdensome individual control, it would suggest a strategy toward inevitable invaders. In the imaginary cloak world, would we not want to know which environmental chemicals would permeate our cloak and what their properties and risks would be? In our actual world, would we not want to know which created chemical substances and highly concentrated naturally occurring (harmful) ones that would inevitably invade were toxic and posed risks of harm, potentially disrupting our biological functioning?

Unfortunately, we do not have such cloaks. Nonetheless, this hypothetical suggests that it is reasonable to require product manufacturers to conduct certain tests on their products, so that the public and regulatory agencies will have knowledge about the substances and their risks. We might imagine a kind of surrogate "Harry Potter test." What knowledge would we require about substances to justify programming our chemical cloak to preclude invaders?

The preceding discussion suggests a second best policy. The *community* could permit by legislation the creation of products that invariably would trespass, but only if testing evidence provided reasonable assurance that there would not be significant risks from the invasions and a public agency certified the test results and approved entry into commerce. Such legislation, in effect a social contract, would permit companies to place their products in commerce, but only on condition that they conducted specified tests, to be

208. J. K. ROWLING, *HARRY POTTER AND THE DEATHLY HALLOWS* (Bloomsbury 2007) (describing invisibility cloak).

sketched below, to determine whether substances will a) invade; and b) pose risks or harms to humans. Based on the tests a public agency would then review the results and permit them into commerce if they posed no significant risks. Children, newborns, and developing fetuses are especially vulnerable and extremely important to protect. Consequently there would need to be testing specifically designed for their protection.

Failure to test for potential risks is analogous to medical experiments on humans without proper preparation and assurances of safety of the experiment and it is just as serious. It might even be more serious since medical experiments are typically conducted to determine beneficial health effects, but industrial chemical products are not created with beneficial health effects in mind for those they might invade.

Test results will vary; some invaders will be innocuous, others will be quickly and innocuously expelled through breathing or other waste-removal procedures. They will neither stay long nor metabolize into harmful by-products during their stay. For such substances, no further testing may be needed.

Not all substances that enter our bodies are easily addressed, however, and some substances inevitably will remain. Some will accumulate and some that accumulate will pose risks, but some may not. Some that remain or accumulate will cause harms to adults and some that accumulate may not cause harm to adults, but could harm developing children, such as low-level lead, PCB or pesticide exposures. In addition, some substances that are quickly expelled may be metabolized or otherwise broken down into harmful by-products before they are expelled.²⁰⁹

Similar tests should also be provided to discover whether substances can invade the womb or breast milk, and, if they can, then additional tests should be conducted to discover whether they cause risks or harms to human developmental processes. In each case without any testing for invasion, any entry into the womb or breast milk would be unauthorized.

Recall the trespass analogy which suggests the testing strategy. One wrong of a trespass is an *unauthorized invasion or violation of a right*. By analogy there should be *no presumption of moral innocence* for the invasion.²¹⁰ Although the primary wrong in the trespass

209. Such as polycyclic aromatic hydrocarbon's (PAH's).

210. PROSSER & KEETON ON TORTS suggests that in trespass, a cause of action "could be maintained without proof of any actual damage. KEETON ET AL., *supra* note 185, at 75. From every direct entry upon the soil of another, 'the law infers

analogy is the unauthorized invasion, there are secondary (and potentially just as serious) wrongs when invasions create risks of harm or actually contribute to harm. Consequently, those who create and distribute compounds that *invade our bodies* should be required to engage in *appropriate testing to help ensure against other wrongs (risks or harms)*, secondary to the invasion. Commercialization and public release of products could be deemed appropriately authorized if there was appropriate testing for entry into human or mammalian bodies, and subsequent testing revealed no significant risks of harm to adults, fetuses, neonates or developing children, and an impartial government agency had reviewed the tests and authorized entry into the market (or for existing chemicals, authorized their remaining in the market).²¹¹

When substances test positive for risks or harms, this would provide a basis of further regulation to prevent the serious secondary wrongs before substances could enter the market if they were new or to prevent their remaining there, if they were already present. Moreover, if products are permitted into commerce resulting in exposures, there should be close monitoring of them to catch

some damage; if nothing more, the treading down or grass or herbage.'” Keeton, *supra* note 185, at 75 (discussing the wrong of trespass and minor role of harm). On the harm principle, roughly, there is a presumption of moral innocence *unless* there is harm or risk of harm. The harm or risk of harm is the *primary wrong*. *Id.*

211. More specifically as examples for testing, agencies could reasonably require firms to address at least the following issues about which science knows before receiving agency approval to enter or remain in commerce:

- Whether when companies’ products invade, are the kinds of substances that, although they may be expelled relatively quickly, are like PAHs that may be metabolized into more toxic compounds, such as epoxides, during their elimination.
- Whether, when they invade they create precursor conditions that could develop into future harm or create conditions for future harm. For example, very low levels of benzene suppress the immune system; childhood exposures to ozone “restructure the lungs.” Both invasions may lead to harm in the future.
- Whether when they invade, they are likely to *remain* in our bodies. For example, are they lipophilic or otherwise likely to attach to receptors or tissues in our bodies and potentially pose problems, e.g., as do DDT, PCB’s, chlorinated or halogenated compounds of higher molecular weight—PBDEs, PBBEs, as well as some of the metals, e.g., mercury, lead and cadmium?
- Whether when they invade, their presence over time is likely to cause further injury. For example, cadmium’s harm to the respiratory system is a function of time and level of exposure.
- Whether with accumulation, they are likely to pose *risks* or contribute to *harm immediately or later in life as do many persistent bioaccumulating substances*.

This Author owes some of these examples to David A. Eastmond, Chair Environmental Toxicology, personal communication, November 15, 2007.

any further risks or harms as early as possible.²¹² Additionally, there should be legal provisions authorizing relatively quick removal if secondary risks or harms appear.²¹³

C. The REACH “Permission” Model.

Another alternative for addressing products that pose risks to developing fetuses, newborns and children is a “permission” model, similar to the recently passed REACH legislation in Europe.²¹⁴ REACH is the acronym for the Registration, Evaluation, Authorization and Restriction of Chemicals.²¹⁵ This complex piece of legislation requires the registration and testing of some 30,000 new and existing chemicals in order to determine any risks or harms from them.²¹⁶ It contains a pre-market testing and approval law for new chemicals, but it also requires similar testing and review of existing substances so both new and existing substances are subject to the same requirements.²¹⁷

The European Community has made the judgment that chemical products will not be permitted to be sold in the European Union (EU) unless there is appropriate testing and assurances of safety of the products sold therein. In a nutshell, the splendidly simple epigram that captures this law is no (safety) data, no market. If firms do not provide appropriate safety about their products, they will not have permission to sell or distribute products in the EU.

The “European Union is aiming to achieve that, by 2020, chemicals are produced and used in ways that lead to the minimization of significant adverse effects on human health and the environment.”²¹⁸ It seeks to “ensure that substances of high concern are eventually replaced by less dangerous substances or technologies where suitable economically and technically viable alternatives are available.”²¹⁹ Responsibility for testing and ensuring the safety of

212. See Cranor, *supra* note 171, at 41-46 (discussing need to monitor substances for toxic effects and to have reasonably quick means of removing them from commerce in order to best protect ourselves).

213. *Id.* at 38 (discussing advantages and disadvantages of pre-market and post-market statutes).

214. See generally Registration, Evaluation, Authorization and Restriction of Chemicals (REACH), No. 1907 (2006) (U.K.) (stating general provisions of regulation).

215. *Id.* (explaining acronym).

216. *Id.* (setting forth requirements of REACH legislation).

217. *Id.* (explaining in detail precise requirements).

218. *Id.* at Art. I, ¶ 4 (discussing European Union’s goals).

219. See Registration, Evaluation, Authorization and Restriction of Chemicals (REACH), No. 1907 (2006) (U.K.) at art. I, ¶ 12 (discussing purpose).

the products rests with the manufacturers and importers.²²⁰ And the required testing and assurance of safety will be based on the quantity of the product produced.²²¹ EU authorization to place products on the market should occur “only if the risks arising from their use are adequately controlled, where this is possible, or the use can be justified for socio-economic reasons and no suitable alternatives are available, which are economically and technically viable.”²²² The “restriction provisions” of REACH “should allow the manufacturing, placing on the market and use of substances presenting risks that need to be addressed, to be made subject to total or partial bans or other restrictions, based on an assessment of those risks.”²²³

The number and generic types of tests needed for a specific substance are based on the number of tons of production volume as a surrogate for exposure.²²⁴ Moreover, the tests are cumulative: any test required for small production volume also applies to larger production volumes. For example, under REACH, developmental toxicants products produced in the highest volume would need a reproductive/developmental toxicity screen in one species, a developmental study, a two-generation reproductive study, and a long-

220. *Id.* at art. I, ¶ 18-19 (discussing who should be held legally responsible for harm caused by substances). “Responsibility for the management of the risks of substances should lie with the natural or legal persons that manufacture, import, place on the market or use these substances.” *Id.* at art. I, ¶ 18.

221. *See id.* at art. I, ¶ 28-29 (discussing safety and obligations of researchers to develop testing requirements based on quantities of substances).

222. *Id.* at art. I, ¶ 22 (discussing use of substances based on socio-economic reasons).

223. *Id.* at art. I, ¶ 23 (detailing aspects of REACH).

224. Approximate and high end numbers of tests needed for each level of production are indicated in Table 1.

Table 1
Cumulative Developmental Tests Under REACH Based on Production Volume

	>1 ton per year	>10 tons per year	>100 tons per year	>1000 tons per year
Physical-chemical data	14	14	17	17
Environmental fate	1	4	10	13
Ecotoxicology data	2	4	12	16
Mammalian toxicity-related data	6	14	18	24

Summary taken from a presentation by David A. Eastmond, Chair, Department of Environmental Toxicology, University of California, Riverside (Nov. 2007) (summarizing types of tests under the REACH regulation).

term (greater than twelve months) toxicity study, a study of reproductive toxicity, a two-generation study, and a carcinogenicity study. Whether this battery of tests will work to identify substances that would cause sub-clinical developmental effects or risks depends upon the tests that are required. Some knowledgeable observers of the REACH legislation are skeptical about this, but the details of the program will be importantly determinative.²²⁵

With regard to testing the EU specifies some particular tests that firms will need to utilize in order to provide reasonable assurance of no significant developmental risks from toxicants.²²⁶ The advantage of this is that the government agencies would choose the tests to be followed and have some assurance that the chosen tests were sufficiently protective for their purposes. REACH does permit firms to present evidence of alternate testing protocols, if it can be shown that they better serve the purposes or are more sensitive and so on.²²⁷

REACH shares with the trespass model the idea of testing products before they come into market, or testing those already in the market, in order to determine whether or not they pose any risks to adults, developing fetuses, newborns or somewhat older children. In both cases an impartial government agency must find the test acceptable, review the products for safety, and then grant or deny their entry into (or continuation in) the market. In some respects REACH is simpler than a trespass model because it rests on the idea of permission; although the idea of appropriate permissions has a role in both cases.

Under REACH companies do not have a *right* to manufacture, sell, or distribute any goods in the EU that they think are appropriate; their products may enter the market only by permission of the EU, and that permission is conditional upon testing products and ensuring their safety to the satisfaction of an agency.²²⁸ This makes clear that access to a market is not a liberty right or a claim right, but a permission granted by a country, provided some further con-

225. Conversation with Philippe Grandjean, University of Southern Denmark and Harvard University, at the Faroe Islands Conference on Prenatal Programming and Toxicity, May 24, 2007.

226. See Registration, Evaluation, Authorization and Restriction of Chemicals (REACH), No. 1907 (2006) (U.K.) at L 396/339, L 396/352-53, L 396/364-66 (providing information on tests specified by EU).

227. See *id.* at art. I, ¶ 14 (describing alternate testing protocols).

228. See *id.* at art. I, ¶ 19-21 (stating that REACH permits substances to enter market only after their safety has been ensured by various testing).

ditions are satisfied concerning the safety of the products to be marketed.

As noted above, under post-market laws, it is as if firms have a legal right to market products in the United States unless and until the products cause harms or risks of harm. The permission model treats access to markets not as a right, but as a privilege, a much different moral and legal relationship between a country and firms seeking to do business within it, whether they are domestic or foreign firms.

D. Choosing between the Trespass and REACH Models

REACH has some attractive features. It is splendidly simple—if there is no safety data for developmental and other adverse health effects, company may not sell in the EU market. It reminds citizens that those who create and sell products have *no right* to do so unless they can assure the citizenry of the products' safety. Participation in the market is conditioned on doing something for the citizenry other than providing products—testing them to ensure health protections.

The “trespass” model has other attractive features. It reminds citizens of their substantial moral and legal authority over their lives in a variety of dimensions, not just property. And, it reminds them how their bodies can be invaded and that they have or should have proper authority over them. It also reminds them of important relations between citizens—one citizen may not cause humanly created substances to invade the bodies of others without appropriate assurance of the safety of the product that enters other citizens' bodies.

The trespass analogy tends to model what the science is revealing and reinforces the moral and legal rights on which citizens should insist. Do substances invade human bodies, the womb or breast milk to which children are exposed? If there is invasion, does it cause harm or pose risks? It also appears to resonate with citizens—there is some anecdotal evidence and news accounts that when citizens learn about the presence of industrial chemicals in their bodies, substances whose presence they have not authorized; they feel “surprised,” “concerned,” “angry,” or perhaps simply wronged.²²⁹ They might well feel as if they are experimental subjects without having given their permission for the experiment.

229. Examples of such substances would include mercury compounds, PCB's, polybrominated diphenyl ethers (flame retardants), organochlorine pesticides, phthalates and bisphenol A (plasticizing agents), or perchlorate (a component of

VII. FINAL POINTS ON THE BET

The title of this Article, "Do you want to Bet Your Children's Health on Post-market Harm Principles?" asks something of a rhetorical question, given the scientific evidence and arguments presented above. The arguments of this Article, however, rest on some quite significant claims, all of which appear to be correct. If one wishes to bet his or her children's health on one of these claims not being true, those would be poor investments of money.

Should one bet that there will be no more contamination of our bodies by manmade chemicals? This would be a bad bet to make, since it seems quite plausible that such contamination will continue except for the largest polymers.

Is one willing to bet that mothers will not share their chemical burden *in utero* or through nursing? Again, this would be a bad bet as a matter of basic biology. It seems highly likely that many chemical substances in women's bodies will pass through the placenta into the womb and then later through the breast milk to developing infants. Some substances in mothers' bodies will not enter the womb because of size, electric charge, and so on, but many will. Perhaps not all substances present in mothers' bodies will enter breast milk and expose their developing children, and not all will follow the "calcium stream," but some will.

Is one willing to bet that children will not be more susceptible than adults, more exposed on a body weight basis, or have fewer defenses against chemical assaults than adults? Should one bet that developing organ systems will not be as susceptible to toxic substances as they have in the past? These are very poor bets; there is no reason to believe that children's basic biology will change radically in the near future or that we have bred a race of new humans with super organ systems that will not be susceptible to toxicants.

Should one bet that there will be no other developmental toxicants than those known to date? It is more difficult to know the answer to this question, but again it appears to be a poor bet to assume that all developmental toxicants are known and under-

rocket fuel). See e.g., Dave Newbart, *Are You Being Poisoned? Five Volunteers Tested by Environment Illinois Were Surprised to Find Out Their Bodies Contained Toxic Chemicals — All from Using Everyday Products*, CHICAGO SUN-TIMES, Nov. 9, 2007 (describing some results of biomonitoring on five volunteers); Robert C. Pollack, *Study Shows Woman has Traces of 17 Toxic Chemicals in System*, SHORE LINE TIMES.COM, Nov. 13, 2007, http://www.zwire.com/site/news.cfm?newsid=19020618&BRD=1630&PAG=461&dept_id=7736&rft=6 (providing example of public concern); ENVTL. DEF., *supra* note 12, at 1 (indicating people from all walks of life are contaminated by mixture of toxic chemicals).

stood. It would also be an especially poor bet to count on post-market laws catching toxicants before they cause harm. Quite the contrary, these problems are just coming to light and it appears highly likely that the more scientists look into the issues, the more developmental toxicants they are likely to find. As scientists develop the tools to identify more subtle adverse developmental effects, the more they are likely to find. This has occurred with such diverse substances as lead, mercury, DDT, various plastics and endocrine disrupters. Moreover, as noted in section III (F) PBDEs are a class of lipophilic, bioaccumulating substances that almost certainly will cause the same kinds of problems that PCBs do.²³⁰ Post-market laws are simply not up to the task of preventing harms from PBDEs or other developmental toxicants.

Is it a good bet that research has discovered all the substances that can cause developmental problems at *low concentration levels*, that no other substances will trigger cancer early as other substances have? This, of course, is much more difficult to know. As research progresses in this area it appears that subtle adverse effects are being discovered at quite low levels. This is certainly true of lead, for which there appears to be no lowest level of toxicity *in utero*. How many substances are sufficiently similar to lead for which there is no lowest adverse effect level or only an extremely low adverse effect level for developing fetuses and neonates? The general biological principles that developing fetuses and newborns are much more susceptible than adults does not make such a bet a promising one.

Some might want to bet that mammalian research models will have little biological relevance to humans. This is certainly a point that manufacturers of products will argue, despite the fact that they and other companies utilize the same kinds of studies in their own research. Nonetheless, experimental mammalian studies are the primary means by which scientists come to understand the toxicity of substances. They are genuine scientific experiments and avoid the need to conduct unethical experiments and chemical trespass on humans. Moreover, biologists and toxicologists will continue utilizing such studies for the foreseeable future; for the most part it will be the best science available until superior short-term tests are designed. Finally, without relying on animal studies and other non-

230. Hooper & McDonald, *supra* note 94, at 387 (discussing similarity of PBDEs to PCBs). "PBDE toxicology resembles that of the PCBs. Some of the persistent and bioaccumulative PBDE congeners seem likely to cause cancer and thyroid and/or neurodevelopmental toxicity, based on available PBDE toxicology data and on structure-activity relationships with PCBs" *Id.*

human tests, as a society we will put at risk many decades of cohorts of children when we do not use experimental studies in order to detect adverse effects in a timely manner.

Does the citizenry wish to bet that post-market laws that seem so ineffective in addressing toxic risks to children will suddenly become strikingly efficacious in this regard? This bet seems a non-starter. Post-market laws have substantial scientific and legal burdens of proof for agencies to overcome and too many incentives for regulated companies to delay and frustrate testing, science and regulation. Business as usual seems not only risky, but probably harmful to our children.

VIII. CONCLUSION

Under existing laws governing chemical substances citizens are in large measure guinea pigs or experimental subjects. They are *guinea pigs* to the extent that current laws permit the vast majority of substances into commerce without significant testing (with drugs, pesticides and new food additives partial exceptions), and then wait for any risks or harms to be revealed as a result of human exposures or non-human testing, which are typically slow to be conducted. Perforce, citizens are experimental subjects in that *de facto* they become one of the main testing grounds for the toxicity of products, but without having authorized or consented to the exposures. Moreover, to the degree that actual practice requires that human harm must be scientifically demonstrated before regulatory action is justified and exposures reduced or eliminated, citizens are guinea pigs in a more robust sense.

Citizens are also the *subjects* of manufacturers' choices because it is the *manufacturers' decisions*, not citizens' choices, that determine whether citizens are invaded by chemical compounds for which adequate testing has not occurred. Of course, it will be impossible or impractical for citizens to become full sovereigns over their bodies for each individual invasion where chemical substances are concerned because there is no biologically practical way to exclude them. Invasion is even worse when firms' decide to send chemicals into the environment and inevitably into our bodies without understanding what their trespass can do to us.

We can move some way toward gaining greater civic control over chemical exposures and greater individual rights over our bodies and our lives by requiring no trespass without testing for both invasion and risks, or no permission to send products into our commercial markets (or permit them to remain there) without safety

data and government approval. There should be no invasion of existing or newly created compounds or greatly concentrated natural compounds from manufacturing processes without testing for what they will do after invasion. Testing will help remove uncertainty about their properties and help ensure that the primary wrong of invasions do not carry with them secondary wrongs of harm or risks of harm that can exacerbate the initial wrong of trespass. If invasions pose harm or risks of harm, the substances should then be the subjects of regulation to try to prevent the secondary wrongs. Were the citizenry to take such steps through legislation it would make progress in going from guinea pigs or subjects to sovereigns over their lives where environmental chemicals and any potential risks associated with them are concerned.