BOOK REVIEW

LEGALLY POISONED: HOW THE LAW PUTS US AT RISK FROM TOXICANTS

Carl F. Cranor
Harvard University Press, 2011
328 pages, $35.00
ISBN-10: 0-67-4049705
ISBN-13: 978-0-67-4049703
(hardcover)

THE TOXICITY OF LOW-DOSE CHEMICAL EXPOSURES: A STATUS REPORT AND A PROPOSAL

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For decades great volumes of petrochemical products have been released into the environment without serious regulatory attention, despite signs of health and ecological damage. Carl Cranor's recent book, *Legally Poisoned: How the Law Puts Us at Risk from Toxicants,* is a valuable and timely introduction to this challenge.[1] The book outlines human health risks of low doses

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1. CARL F. CRANOR, LEGALLY POISONED: HOW THE LAW PUTS US AT RISK FROM TOXICANTS (2011) [hereinafter CRANOR, LEGALLY POISONED]. Professor Cranor has long contributed to the understanding of science and the law. See CARL F. CRANOR, REGULATING TOXIC SUBSTANCES—A PHILOSOPHY OF SCIENCE AND THE LAW (1993) [hereinafter CRANOR, REGULATING TOXIC SUBSTANCES] and CARL F. CRANOR, TOXIC TORTS—SCIENCE, LAW, AND THE POSSIBILITY OF JUSTICE (2006) [hereinafter CRANOR, TOXIC TORTS], reviewed in this journal by David S. Caudill, 49 JURIMETRICS J. 519 (2009) (commenting on Cranor's insightful and pragmatic approach to scientific evidence under *Daubert*). Cranor's articles include Carl F. Cranor, *Do You Want to Bet Your Children's Health on Post-Market Harm Principles? An Argument for a Trespass or Permission Model for Regulating Toxicants*, 19 VILL. ENVTL. L.J. 252 (2008) [hereinafter Cranor, *Do You Want to Bet*]; Carl F. Cranor, *A Framework for Assessing Scientific Arguments: Gaps, Relevance and Integrated Evidence*, 15 J.L. & POL'Y 7 (2007); Carl F. Cranor, *Information Generation and Use Under Proposition 65: Model Provisions for Other Postmarket Laws?*, 83 IND. L.J. 609 (2008); Carl F. Cranor & David A. Eastmond, *Scientific Ignorance and Reliable Patterns of Evidence in Toxic Tort Causation: Is There a Need for*

of chemical toxicants, describes the exposures of greatest concern and explains the scientific underpinnings of chemicals regulation.[2] It then summarizes recent breakthroughs in scientific research on chemical toxicity.[3] Cranor's presentation of this work is revelatory, even shocking. It challenges our current tolerance for low-level chemical exposures.

Chemicals regulation in the United States operates on assumptions that date from the 1970s. Since that time, research has shown that environmental factors are important contributors to disease.[4] Toxicology and epidemiology have built a substantial knowledge base on chemicals and human health. New fields, including genetics and epigenetics, are providing data that has substantial implications for the market and for law. For instance, familiar concepts like "the dose makes the poison" are apparently not true for many substances.[5] Some low level chemical exposures in utero and in early childhood are linked to diseases that occur later in life, including some that are widespread and incapacitating. Testing that examines toxic effects one chemical at a time is no longer sufficient because new research shows that additive and synergistic toxic effects may be common. In the field of epigenetics, there are indications that some environmentally induced health effects persist into later generations, without changes in genetic material.

Chemical production and exposures have grown, but even minimal testing for environmental, health, and safety (EHS) effects is not required for most commercial chemicals.[6] As a result, there is a dearth of useful data on chemicals' EHS effects.[7] In *Legally Poisoned*, Cranor proposes a legal model that

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Liability Reform?, 64 LAW & CONTEMP. PROBS. 5 (2001); Carl Cranor, *Scientific Inferences in the Laboratory and the Law*, 95 AM. J. PUB. HEALTH S121 (2005).

2. CRANOR, LEGALLY POISONED, *supra* note 1, chs. 2, 3.

3. *Id.* at ch. 4.

4. Stephen M. Rappaport, *Implications of the Exposome for Exposure Science*, 21 J. EXPOSURE SCI. & ENVTL. EPIDEMIOLOGY 5, 5 (2011). Studies of twins indicate that probably 10% of cancer risks are because of genetic factors. *Id.* at 6. This leaves 90% because of "environmental" factors, but the incomplete data available suggests that conventional pollutants targeted by regulation, including occupational exposures, account for another 7–10% of chronic diseases. *Id.* This means that "environmental" influences beyond those already regulated must be considered (for example, diet, weight, activity levels, smoking and other factors). *Id.*

5. *See infra* Parts II.A, II.B and discussion notes 53, 73.

6. CRANOR, LEGALLY POISONED, *supra* note 1, at 6–8. Part I of this review sketches the history of chemicals regulation.

7. In 1984, the National Research Council estimated that there were 65,725 substances in use that were of possible toxicological concern and that for the vast majority of them, information essential for hazard assessment was lacking: "thousands or even tens of thousands of chemicals are legitimate candidates for toxicity testing related to a variety of health effects." STEERING COMM. ON IDENTIFICATION OF TOXIC AND POTENTIALLY TOXIC CHEMS. FOR CONSIDERATION BY THE NAT'L TOXICOLOGY PROGRAM, NAT'L RESEARCH COUNCIL, TOXICITY TESTING: STRATEGIES TO DETERMINE NEEDS AND PRIORITIES 12, 14 (1984) [hereinafter NRC, TOXICITY TESTING]. Later reports confirm that the situation has not improved. *See* CRANOR, LEGALLY POISONED, *supra* note 1, at 6. *See generally* Cranor, *Do You Want to Bet, supra* note 1, at 278–84 (explaining the information effects of allowing chemical products to be distributed before screening tests are performed); Albert C. Lin, *Deciphering the Chemical Soup: Using Public Nuisance Law to Compel Chemical Testing*, 85 NOTRE DAME L. REV. 955 (2010) (describing the shortfall of tox-

would treat the act of distributing potential toxicants without testing as a common law battery.[8] The simplicity of this proposal is a virtue. It addresses the key obstacle to better chemicals management—lack of data—and also suggests an additional social and analytical frame for considering the issues.

Cranor points out that, in effect, the chemicals sector is running a giant experiment on an unwitting population.[9] He asks: in light of the evidence, is it a good bet to continue with the existing legal scheme?[10] As he has before, Cranor frames the question within our most intense concerns: don't we owe more to our children?[11] His emphasis on fairness and care for children is more than a rhetorical flourish. The science described in *Legally Poisoned* demonstrates the sensitivity of the young to toxicants and presents the specter of toxicity that reaches into the future, affecting unexposed generations.

Part I of this review briefly sketches the history of toxicity research and regulation, drawing in part on Cranor's early chapters. Part II outlines some highlights of the research that Cranor presents in Chapter 4. Part III describes current developments on chemicals regulation and possible legislative reforms. Part IV comments on Cranor's proposal to harness the common law of battery.

I. HOW WE GOT HERE

Trillions of pounds of commercial chemicals are produced and distributed each year throughout the globe.[12] Many are persistent, bioaccumulate in food

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icity data and the relevant legal literature and proposing a novel legal response discussed in Part III *infra*); Mary L. Lyndon*, Information Economics and Chemical Toxicity: Designing Laws to Produce and Use Data,* 87 MICH. L. REV. 1795 (1989) [hereinafter Lyndon, *Information Economics*] (describing market failure because of inadequate understanding of the effects of chemicals and related products); Wendy E. Wagner, *Choosing Ignorance in the Manufacture of Toxic Products*, 82 CORNELL L. REV. 773, 833–53 (1997) (proposing that gradations of testing qualify manufacturers for gradations of liability protection); Wendy E. Wagner, *Commons Ignorance: The Failure of Environmental Law to Produce Needed Information on Health and the Environment*, 53 DUKE L.J. 1619 (2004) (analyzing the causes of a environmental agencies' information dysfunctions).

8. CRANOR, LEGALLY POISONED, *supra* note 1, at 186.

9. *See infra* Part III, for a discussion of the ethical implications of untested exposures.

10. *E.g.*, "When known hazards, such as carcinogens, endocrine disruptors, neurotoxicants, and so on, contaminate a developing fetus, it seems a poor bet merely to *hope* that they will not cause adverse effects." CRANOR, LEGALLY POISONED, *supra* note 1, at 129.

11. CRANOR, LEGALLY POISONED, *supra* note 1, at 130–31, 209; Cranor, *Do You Want to Bet*, *supra* note 1, at 251, 256–76, 311–14.

12. Michael P. Wilson & Megan R. Schwarzman, *Toward a New U.S. Chemicals Policy: Rebuilding the Foundation to Advance New Science, Green Chemistry, and Environmental Health*, 117 ENVTL. HEALTH PERSP. 1202, 1203 (2009) (citing U.S. Environmental Protection Agency data). U.S. companies produced or imported 15 trillion pounds of chemical substances in 2002 (42 billion pounds per day) and 27 trillion in 2005 (74 billion pounds per day), not including fuels, pesticides, pharmaceuticals, or food products. *Id.* Figures vary on the number of chemicals now in use. CRANOR, LEGALLY POISONED, *supra* note 1, at 18. In 1983, OSHA estimated that as many as 575,000 chemical products might be in use, with hundreds of new ones introduced annually. Hazard Communication, 48 Fed. Reg. 53,280, 53,323 (Nov. 25, 1983) (to be codified at 29 C.F.R. 1910.1200). In 1984, the National Research Council (NRC) estimated that there were 65,725 substances in use that were of possible toxicological concern. NRC, TOXICITY TESTING,

chains and are toxic; chemicals with these properties—persistence, bioaccumulation and toxicity (PBT)—can be particularly damaging.[13] These chemicals also travel in the environment.[14] As Cranor describes in Chapter 2, we are exposed to them at the workplace, from contact with consumer products, and by ingesting them in food and water and inhaling them in the air.[15]

Once a substance is released into the environment, it can be difficult to ascertain that an exposure has occurred, let alone identify its source, character and effects. Most low-level exposures are not noticed when they occur; if they have negative effects, these will often appear later. Given this latency and the number of chemicals discharged into the environment, establishing causal links between pollutants and health effects has been very difficult. Chapter 3 of *Legally Poisoned* explains the scientific challenges. Over time, toxicological research has fallen far behind the petrochemical industry's deployment of its products. Of the approximately 75,000 chemical substances in commercial use, a relatively small number have been well characterized for potential toxicity, though many are thought to pose risks.[16]

This state of ignorance is predictable. Testing is costly and the results can be uncertain.[17] The Toxic Substances Control Act (TSCA),[18] enacted in 1976, allows distribution of chemicals until they are shown to be harmful and does not require manufacturers to produce toxicity data. TSCA does not require manufacturers to perform toxicity testing, unless the Environmental Protection Agency (EPA) formally proves that a chemical "may present an unreasonable risk of injury to health or the environment . . . ."[19] Although they are not required to perform research, chemical manufacturers may do so and they are

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supra note 7, at 21. Cranor suggests that about 6,000 chemicals in use pose health concerns. CRANOR, LEGALLY POISONED, *supra* note 1, at 18

13. "Persistence" refers to the length of time the chemical can exist in the environment before being transformed by natural processes, and "bioaccumulation" refers to the process by which organisms may accumulate chemical substances in their bodies. *See* 64 Fed. Reg. 58,666 (Oct. 29, 1999) (to be codified at 40 C.F.R. pt. 372) (lowering reporting thresholds for certain persistent bioaccumulative toxic chemicals); CRANOR, LEGALLY POISONED, *supra* note 1, at 199–200 (describing health significance of persistence in chemicals).

14. *See* Terry Collins, *Toward Sustainable Chemistry*, 291 SCIENCE 48, 48–49 (2001) ("Imagine all of Earth's chemistry as a mail sorter's wall of letter slots in a post office, with the network of compartments extending toward infinity. Each compartment represents a separate chemistry so that, for example, thousands of compartments are associated with stratospheric chemistry or with a human cell. An environmentally mobile persistent pollutant can move from compartment to compartment, sampling a large number and finding those compartments that it can perturb.").

15. CRANOR, LEGALLY POISONED, *supra* note 1, at 1–3, 16–46.

16. *See* Richard Judson et al., *The Toxicity Data Landscape for Environmental Chemicals,* 117 ENVTL. HEALTH PERSP. 685, 685 (2009). *See* sources cited *supra* note 7 and accompanying text.

17. *See* sources cited *supra* note 7 and accompanying text.

18. 15 U.S.C. §§ 2601–92 (2006).

19. § 2603(a)(1)(A)(i). Although manufacturers need not research the effects of their chemicals, they must notify the EPA ninety days before they put a new chemical into the market and must submit to the EPA basic descriptions of the chemical and its physical characteristics. § 2604(a).

required to submit any data they possess that indicates adverse effects. This duty to reveal existing data is difficult to enforce and has often been ignored.[20]

TSCA thus creates a Catch-22 situation, in which the EPA must prove that a chemical is likely to be harmful before it can require testing. In effect, TSCA exempted from any research requirements the 62,000 chemicals that were on the market in 1976 and it imposes only minimal reporting requirements for new chemicals. The EPA has regulated few chemicals, and the original 62,000 are still in use.[21]

In postmarket regulation—that is, when regulation kicks in only after a chemical is on the market—the pressures of the market are arrayed against limits on the product's use. Businesses have strong incentives to resist requirements to produce toxicity data and instead may produce studies aimed at increasing uncertainty about any suspected toxic effects.[22] Regulatory proceedings can then become a tug-of-war over the validity of toxicity data. Bottlenecks develop and agency resources are absorbed in accumulating data on a few problems.[23]

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20. § 2607(e); Judson et al, *supra* note 16, at 687. *See* Marianne Lavelle, *E.P.A.'s Amnesty Has Become a Mixed Blessing: Be Careful What You Wish For*, NAT'L L.J., Feb. 24, 1997, at A1 (under an amnesty program in place from 1991–1996, manufacturers handed in 11,000 old, unpublished studies and adverse reaction reports). In 2008, the EPA settled an enforcement against E.I. DuPont de Nemours and Company (DuPont), levying a fine of $10.25 million, the largest administrative penalty in the EPA's history. *Failure to Report Chemical Risks Can Result in Major Fines: Section 8(e) of the Toxic Substances Control Act*, ENFORCEMENT ALERT (EPA Office of Civil Enforcement, D.C.), Aug. 2008, at 1, *available at* http://www.epa.gov/compliance/resources/newsletters/civil/enfalert/8e-tsca-0807.pdf. DuPont failed to report risk information regarding the synthetic chemical substance perfluorooctanoic acid (PFOA) and related perfluorochemicals. *Id.* "The information withheld by DuPont demonstrated placental transfer of PFOA from human mothers to their babies during pregnancy, and the levels of PFOA found in a newborn and a two-year-old." *Id.* "As a result of this and other information, EPA is investigating the effects of human exposure to PFOA and related perfluorochemicals." *Id.*; CRANOR, LEGALLY POISONED, *supra* note 1, at 37–38, 199–200, 233 (discussing perfluorinated compounds); Bruce J. Berger, *The Trouble with PFOA: Testing, Regulation and Science Concerning Perfluorooctanoic Acid and Implications for Future Litigation*, 76 DEF. COUNS. J. 460 (2009) (describing science and emerging legal responses to widespread PFOA exposure).

21. Wilson & Schwarzman, *supra* note 12, at 1203 (citing U.S. GOV'T ACCOUNTABILITY OFFICE, GAO-05-458, CHEMICAL REGULATION—OPTIONS EXIST TO IMPROVE EPA'S ABILITY TO ASSESS HEALTH RISKS AND MANAGE ITS CHEMICAL REVIEW PROGRAM (2005)).

22. In contrast, an *ex ante* regulatory scheme requires a product's proponent to generate some health and safety information to get the product to market. CRANOR, LEGALLY POISONED, *supra* note 1, at 178–80, describes the ex ante or premarket system that applies to pesticides and pharmaceuticals. The NRC Council's study of testing patterns in 1984 showed the greater performance of ex ante regulation. *See generally* NRC, TOXICITY TESTING, *supra* note 7, at 5, 8, 12 (researching and characterizing toxic chemicals and its effects on human health); David Michaels, *Doubt Is Their Product,* SCI. AM., June 2005, at 96; Paul R. Portney, *Toxic Substance Policy and the Protection of Human Health*, *in* CURRENT ISSUES IN U.S. ENVIRONMENTAL POLICY 105 (Paul R. Portney et al. eds., 1978) (discussing firms' incentives to choose testing procedures most likely to shed favorable light on the substances they wish to market and in some cases to falsify or withhold test data). For further discussion, see sources cited *supra* note 7 and accompanying text.

23. *See* Lyndon, *Information Economics*, *supra* note 7, at 1818–21 (describing information effects of ex post regulatory schemes). Overbroad confidentiality claims also reduce the scope and

Chemicals regulation has gotten more information intensive over time.[24] "Science wars" have plagued agencies with debate over the merits of data supporting any proposed limit,[25] contention over appropriate levels of conservatism in standard setting,[26] and heated rhetoric on the costs of testing and substitution of safer chemicals.[27] How much and what kinds of science are needed to act?[28] The EPA has had to work with a sometimes unmanageable

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flexibility of regulatory processes. *See* Mary L. Lyndon, *Secrecy and Access in an Innovation Intensive Economy: Reordering Information Privileges in Environmental, Health, and Safety Law,* 78 U. COLO. L. REV. 465, 516–21 (2007); Mary L. Lyndon, *Secrecy and Innovation in Tort Law and Regulation*, 23 N.M. L. REV. 1, 22–34 (1993).

24. In the 1980s an information-intensive analytical tool, Quantitative Risk Assessment (QRA), was inserted into the regulatory process, as required screening for regulation. In *Industrial Union Dept., AFL-CIO v. American Petroleum Institute*, 448 U.S. 607, 642–43 (1980), a split Court ruled that the Occupational Safety and Health Administration (OSHA) must make a threshold finding of "significant risk" before regulating benzene exposure in the workplace. This required an attempt at quantification, before protective standards could be issued. *See* Howard Latin, *Good Science, Bad Regulation, and Toxic Risk Assessment*, 5 YALE J. ON REG. 89, 114–18 (1988) ("[T]he conclusion that benzene is a carcinogen may be amply supported by [epidemiological] data that are too indefinite to serve as the basis of a quantitative risk assessment." *Id.* at 116); *see, e.g.*, Adam M. Finkel, *A Second Opinion on an Environmental Misdiagnosis: The Risky Prescription of Breaking the Vicious Cycle*, 3 N.Y.U. ENVTL. L.J. 295, 366–381 (considering reform proposals for the types of quantitative analysis that now dominate federal environmental regulatory policy); Wendy E. Wagner, *The Science Charade in Toxic Risk Regulation*, 95 COLUM. L. REV. 1613, 1617–22 (1995) (stating that in the absence of adequate research, science-based regulation allows agencies to obscure value-laden policy choices with scientific window dressing).

25. *See generally* THOMAS O. MCGARITY ET AL., SOPHISTICATED SABOTAGE: THE INTELLECTUAL GAMES USED TO SUBVERT RESPONSIBLE REGULATION 34–65 (2004) (chronicling the dispute over the validity of environmental science); Thomas O. McGarity, *Our Science Is Sound Science and Their Science Is Junk Science: Science-Based Strategies for Avoiding Accountability and Responsibility for Risk-Producing Products and Activities*, 52 U. KAN. L. REV. 897 (2004) (detailing the efforts of risk-producing industries to shape science to benefit their interests and escape tort liability).

26. *See* Rena Steinzor, *Gridlock and Its Implications: A Progressive Response to the Conservative Agenda for Reforming Environmental Law*, *in* 1 LAW ENVTL. PROTECTION § 3:43 (2012) (outlining reasons for conservative or precautionary environmental standards, including the lack of data on factors that are known to increase effects from pollutants).

27. *See generally* FRANK ACKERMAN, POISONED FOR PENNIES: THE ECONOMICS OF TOXICS AND PRECAUTION (2008) (arguing that traditional cost-benefit analysis has led to some dubious economic results); Frank Ackerman, *The Unbearable Lightness of Regulatory Costs*, 33 FORDHAM URB. L.J. 1071 (2006) (arguing that economic costs from regulations are not overly burdensome); Thomas O. McGarity & Ruth Ruttenberg, *Counting the Cost of Health, Safety, and Environmental Regulation*, 80 TEX. L. REV. 1997 (2002) (challenging the commonly held notion that regulation imposes excessive costs on the private sector).

28. These debates have been long and heated, especially as the integrity of scientific processes has been put in question. *See, e.g.*, Thomas O. McGarity, *Defending Clean Science from Dirty Attacks by Special Interests*, *in* RESCUING SCIENCE FROM POLITICS: REGULATION AND THE DISTORTION OF SCIENTIFIC RESEARCH 24 (Wendy Wagner & Rena Steinzor eds., 2006) (explaining ways that health and environmental science can be manipulated and compromised to undermine legal processes); Holly Doremus, *Scientific and Political Integrity in Environmental Policy*, 86 TEX. L. REV. 1601, 1620–29 (2008) (discussing the role of science in environmental regulation and the challenges of regulation to scientific integrity); Kristin Shrader-Frechette, *Conceptual Analysis and Special-Interest Science: Toxicology and the Case of Edward Calabrese*,

avalanche of apparently conflicting and unrelated data, as protracted disputes over the implications of the available information have delayed assessment.[29] In Chapter 3, Cranor provides an excellent explanation of the science underlying these disputes.[30]

The shortage of useful data on chemical effects has shaped the development of both tort law and regulation.[31] Causation rules and appropriate evidentiary standards in the context of chemical exposures became the subject of extended debate, as the difficulties of proving cause and effect remained a barrier to legal action on chemical exposures.[32] The causal link was conceived

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177 SYNTHESE 449, 453 (2010) (describing industry-funded research purporting to show that low doses of toxins or carcinogens have beneficial effects); Lisa Heinzerling, *Violent Science*, 87 TEX. L. REV. 623 (2009) (reviewing THOMAS O. MCGARITY & WENDY E. WAGNER BENDING SCIENCE: HOW SPECIAL INTERESTS CORRUPT PUBLIC HEALTH RESEARCH (2008)).

29. *See, e.g.*, Wendy E. Wagner, *Administrative Law, Filter Failure, and Information Capture*, 59 DUKE L.J. 1321, 1321 (2010) (stating some legal provisions designed to improve the information base of regulation "inadvertently create incentives for participants to overwhelm" agencies with complex data, reducing the transparency and clarity of agency processes). *See generally* David E. Adelman, *The Art of the Unsolvable: Locating the Vital Center of Science for Environmental Law & Policy*, 37 ENVTL. L. 935 (2007) (describing difficulties of using environmental science that is both general and very complex) [hereinafter Adelman, *The Art of the Unsolvable*]; Adam Babich, *Too Much Science in Environmental Law*, 28 COLUM. J. ENVTL. L. 119 (2003) (arguing that the regulatory system, built on ignorance and obfuscation, cannot handle the amount of science that has been produced); Wagner, *supra* note 24, at 1678–81 (noting that science-based regulation has been slow and costly, while technology-based regulation has a better record of environmental protection).

30. CRANOR, LEGALLY POISONED, *supra* note 1, at 48–80.

31. The Government Accountability Office (GAO) has repeatedly criticized the chemical management system established under TSCA for its information limitations, including the broad confidentiality protections it affords the industry. *E.g.*, U.S. GOV'T ACCOUNTABILITY OFFICE, GAO-05-458, CHEMICAL REGULATION—OPTIONS EXIST TO IMPROVE EPA'S ABILITY TO ASSESS HEALTH RISKS AND MANAGE ITS CHEMICAL REVIEW PROGRAM 34–36 (2005) (concluding, in part, that the current state of EHS knowledge is inadequate); U.S. GOV'T ACCOUNTABILITY OFFICE, GAO-09-428T, CHEMICAL REGULATION—OPTIONS FOR ENHANCING THE EFFECTIVENESS OF THE TOXIC SUBSTANCES CONTROL ACT 7–8 (2009) (concluding that the EPA needs greater authority to require production of toxicity data and share information with states and the public).

32. Confronted with apparent toxicity injuries, law-and-science scholars have struggled to articulate the relationship between science and the law and to reimagine the principles of causation and evidence in tort law and regulation. *See, e.g.*, Steve C. Gold, *The "Reshapement of the False Negative Asymmetry in Toxic Tort Causation*, 37 WM. MITCHELL L. REV. 1507, 1509–10 (2011) (analyzing reasons courts have in practice tended to favor false negative findings on causation although this type of error is more costly than the false positive in the toxic exposure setting). Gold suggests that the RESTATEMENT (THIRD) OF TORTS undermines the position that rules favoring false negatives are simply part of judicial truth seeking. The *Restatement* provides an opportunity to reshape the law toward greater symmetry in tort law causation. RESTATEMENT (THIRD) OF TORTS: LIAB. FOR PHYSICAL AND EMOTIONAL HARM (2010); *see also* CRANOR, REGULATING TOXIC SUBSTANCES, *supra* note 1, 55–70 (describing debate over the challenges that increasing understanding of toxicity presents to evidentiary processes); CRANOR, TOXIC TORTS, *supra* note 1, 205–20 (describing tensions between legal process and toxicological and epidemiological evidence); Adelman, *The Art of the Unsolvable*, *supra* note 29, at 939 (exploring the tradeoffs inherent in acceptance of the limitations of scientific methods). *See, e.g.*, Margaret A. Berger & Lawrence M. Solan, *The Uneasy Relationship Between Science and Law: An Essay and Introduction*, 73 Brook. L. Rev. 847, 848 (2008); Susan Haack, *Irreconcilable Differences? The*

of as a black box,[33] and its mysteries raised deeper questions about the format and the legal functions of science.[34]

When TSCA was enacted, it was thought that private testing would increase.[35] This did not happen, though it is something within the control of the industry.[36] A duty to test products had long been a theme in products liability

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Troubled Marriage of Science and Law, 72 LAW & CONTEMP. PROBS. 1, 7–15 (2009); Susan Haack, *Of Truth, in Science and in Law*, 73 BROOK L. REV. 985, 1004–1008 (2008); Sheila Jasanoff, *Just Evidence: The Limits of Science in the Legal Process*, 34 J.L. MED. & ETHICS 328, 333 (2006). *See generally* Margaret A. Berger & Aaron D. Twerski, *Uncertainty and Informed Choice: Unmasking Daubert*, 104 MICH. L. REV. 257, 282–87 (2005) (describing the negative effects of federal evidentiary rules under the *Daubert* decision and proposing new cause of action for informed choice that would separate the right to existing information on drug toxicity from the burden of proof on harm causation); Troyen A. Brennan, *Causal Chains and Statistical Links: The Role of Scientific Uncertainty in Hazardous Substance Litigation*, 73 CORNELL L. REV. 469 (1988) (describing the difficulty of connecting proof of causation to specific chemical exposure in toxic tort cases); Stephen Breyer, *The Interdependence of Science and Law*, 280 SCIENCE 537 (1998) (stating that since scientific issues and challenges permeate the law, legal proceedings must reflect science that approximately reflects the scientific state of the art); Lisa Heinzerling, *Doubting Daubert*, 14 J.L. & POL'Y 65 (2006) (describing courts' response to *Daubert* decision further reducing admissibility of plaintiffs' evidence in toxics cases); Oliver Houck, *Tales form A Troubled Marriage*, 302 SCIENCE 1926–29 (2003) (discussing the relationship between science and law in regards to environmental policy); Vern R. Walker, *Restoring the Individual Plaintiff to Tort Law by Rejecting "Junk Logic" About Specific Causation*, 56 ALA. L. REV. 381 (2004).

33. Steve C. Gold, *The More We Know, The Less Intelligent We Are?—How Genomic Information Should, And Should Not, Change Toxic Tort Causation Doctrine*, 34 HARV. ENVTL. L. REV. 369, 401–06 (2010) (stating that new sciences explain many toxicity connections without eliminating complexity, so that toxic tort claims will continue to present jurisprudential dilemmas, while also providing ways to improve legal treatment of some causation issues) [hereinafter Gold, *The More We Know*]. Gold describes the use of "black box" imagery in this setting: "Both law and science long perceived the link between toxic exposure and eventual disease as a black box, into which similarly situated people might enter, only to emerge with highly disparate outcomes determined by an invisible, mysterious, and stochastic mechanism within the box." *Id.* at 401; *see* Erica Beecher-Monas, *The Heuristics of Intellectual Due Process: A Primer for Triers of Science*, 75 N.Y.U. L. Rev. 1563, 1591 (2000); David S. Caudill, *Barely Opening, Then Slamming Shut, Science's "Black Box" in Law: A Response to Beecher-Monas's Heuristics*, 23 CARDOZO L. REV. 1795, 1796–97 (2002); Jamie A. Grodsky, *Genomics and Toxic Torts: Dismantling the Risk-Injury Divide*, 59 STAN. L. REV. 1671, 1688–89 n.66 (2007).

34. For instance, how can science become more productive, more flexible and more attuned to what is needed? *See, e.g.*, Adelman, *The Art of the Unsolvable*, *supra* note 29, at 939 (suggesting benchmarks for science to respect the contingencies of environmental problems without lapsing into relativism); Sheila Jasanoff, *Technologies of Humility: Citizen Participation in Governing Science,* 41 MINERVA 223, 230 (2003).

35. Companies such as Monsanto and Allied Chemicals increased their laboratory capacity and a number of companies joined to support the Chemical Industry Institute in conducting tests. *See* WILLIAM DRAYTON, AMERICA'S TOXIC PROTECTION GAP: THE COLLAPSE OF COMPLIANCE WITH THE NATION'S TOXICS LAWS 75 (1984). The expected demand for testing never developed, however, and by 1984 Dr. James Liverman, of Litton-Bionetics, estimated that private testing labs were operating at less than 60% of capacity. *Id.* at 76.

36. In *Beshada v. Johns Manville Products Corp.*, 447 A.2d 539, 549 (N.J. 1982) the Supreme Court of New Jersey denied asbestos manufacturers the "state-of-the-art" defense to strict liability claims. The court reasoned that the industry itself produced the state of the art through its own research and development (R&D) agenda. *Id.* at 548–49. The holding in the case was modified and limited in *Feldman v. Lederle Labs.*, 479 A.2d 374, 386–87 (N.J. 1984). For an overview

law, and was preserved by the Restatement (Third) of Products Liability.[37] Economic and legal analyses of the practical importance and role of testing and proposals for reform abound;[38] some states have enacted laws to close the "data gap."[39] Testing can be done: prominent scientific institutions and chemical management nongovernmental organizations (NGOs) have issued guidelines.[40]

Despite the obstacles, our understanding of chemical effects has grown. Important data resources have been developed that support research and management of chemicals.[41] By the 1990s a consensus was forming that certain categories of chemicals demand attention. These include chemicals that dis-

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of status of the state of the art defense today, see 9 WILLIAM R. GINSBERG & PHILIP WEINBERG, ENVIRONMENTAL LAW AND REGULATION IN NEW YORK § 2:20 State of the Art (2d ed. 2009) and 3 J.D. LEE & BARRY A. LINDAHL, MODERN TORT LAW: LIABILITY AND LITIGATION § 27:110 "State of the Art" Defense (2d ed. 2002).

37. RESTATEMENT (THIRD) OF TORTS: PRODUCTS LIAB. § 2 cmt. m (1997). "[A] seller bears responsibility to perform reasonable testing prior to marketing a product and to discover risks and risk-avoidance measures that such testing would reveal. A seller is charged with knowledge of what reasonable testing would reveal. If testing is not undertaken, or is performed in an inadequate manner, and this failure results in a defect that causes harm, the seller is subject to liability for harm caused by such defect." *Id.*

38. There is an extensive literature on the testing issue that is too large to explore fully here. Early work identifying the problem includes Elinor P. Schroeder & Sidney A. Shapiro, *Responses to Occupational Disease: The Role of Markets, Regulation, and Information*, 72 GEO. L.J. 1231 (1984) (identifying lack of information as a key element in the failure to protect workers from occupational disease). *See also* Howard A. Latin, *Environmental Deregulation and Consumer Decisionmaking Under Uncertainty*, 6 HARV. ENVTL. L. REV. 187 (1982) (emphasizing that sound environmental decision making requires extensive data not produced by an unregulated market); Portney, *supra* note 22, at 138 (proposing to combat perverse information incentives by establishing well-defined testing procedures to be applied by all agencies regulating toxic substances; federal agencies should expedite verification of controversial data; and stiff penalties should be levied for violating protocols or falsifying results).

Other proposals include John S. Applegate, *Bridging the Gap: Balancing the Supply and Demand for Chemical Information*, 86 TEX. L. REV. 1365 (2008); Bradley C. Karkkainen, *Framing Rules: Breaking the Information Bottleneck*, 17 N.Y.U. ENVTL. L. J. 75 (2008); Lin, *supra* note 7, at 1000–12; Lyndon, *Information Economics supra* note 7, at 1836–40 (proposing independent research program funded by tax similar to Superfund to support private-public chemicals research); Wendy E. Wagner, *Choosing Ignorance in the Manufacture of Toxic Products*, 82 CORNELL L. REV. 773, 833–53 (1997) (describing a proposed program that would identify gradations of testing that would qualify manufacturers for gradations of liability protection).

39. Daryl W. Ditz, *The States and the World: Twin Levers for Reform of U.S. Federal Law on Toxic Chemicals*, 8 SUSTAINABLE DEV. L. & POL'Y 27, 28 (2007) (discussing states' and international initiatives to regulate toxic chemicals).

40. *See* Cranor, *Do You Want to Bet*, *supra* note 1, at 305–07 (describing parameters of desirable minimum testing); Lin, *supra* note 7, at 1007–11 (describing testing options including data sets recommended by prominent national and international regulatory bodies).

41. *See generally* Jessica N. Schifano et al., *The Importance of Implementation in Rethinking Chemicals Management Policies: The Toxic Substances Control Act*, 41 ENVTL. L. REP. 10527 (2011) (presenting an historical overview of the EPA's struggles and accomplishments working with TSCA).

play PBT characteristics and endocrine disruptors.[42] In addition, toxicity risks to children began to receive more focused attention.[43]

Recently, the European Union has taken a dramatic lead in chemicals regulation and risk management. The European Union's comprehensive regulatory program, Registration, Evaluation, Authorization and Restriction of Chemical Substances (REACH), reverses the burden of proof for chemical safety in E.U. markets.[44] The basic principle of REACH is "no data, no market:" all chemicals must be registered and studied, or they may not be traded in the European Union.[45] The program requires and supports collaborative research by all companies using the same chemical; when the research is completed each chemical will be ranked on the basis of its toxicity.[46]

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42. *See* discussion *infra* Part II.

43. *See* Valerie Watnick, *Risk Assessment: Obfuscation of Policy Decisions in Pesticide Regulation and the EPA's Dismantling of the Food Quality Protection Act's Safeguards for Children*, 31 ARIZ. ST. L.J. 1315, 1316 (1999).

44. Registration, Evaluation, Authorization and Restriction of Chemical Substances (REACH), EC 1907/2006, establishes a comprehensive set of requirements for chemicals and their safe use. Commission Regulation 1907/2006, of the European Parliament and of the Counsel of 18 December 2006 Concerning the Registration, Evaluation, Authorisation, and Restriction of Chemicals (REACH), 2006 O.J. (L396) 1, 2, 8. Earlier the European Union had moved in this direction in its revised regulation of cosmetics. See Directive 2003/15/EC of the European Parliament and of the Council of 27 February 2003 amending Council directive 76/768/EEC on the approximation of the laws of the Member States relating to cosmetic products (otherwise known as the 7th amendment to the Cosmetics Directive, which came into force on March 1, 2005) published in the Official Journal of the European Union, March 11, 2003. Directive 2003/15, of the European Parliament and of the Council of 27 February 2003 Amending Council Directive 76/768 EEC on the Approximation of the Laws of the Member States Relating to Cosmetic Products, 2003 O.J. (L 66) 26 (calling for collaborative research on alternative testing methods with the goal of avoiding the use of live animals in cosmetic toxicity testing).

45. *REACH: The Registration Process*, HEALTH & SAFETY EXECUTIVE, http://www.hse.gov.uk/reach/regprocess.htm (last visited July 9, 2012).

46. REACH incorporates some innovative design features. *Understanding REACH*, EUROPEAN CHEMICALS AGENCY, http://echa.europa.eu/web/guest/regulations/reach/understanding-reach (last visited Mar. 10, 2012). It establishes an online registry and research coordination system with online platforms called Substance Information Exchange forum (SIEFs) where all registrants for each individual chemical will cooperate to share data and plan any further testing that is needed. *Substance Information Exchange Fora*, EUROPEAN CHEMICALS AGENCY, http://echa .europa.eu/web/guest/regulations/reach/substance-registration/substance-information-exchange-fora (last visited Mar. 6, 2012). The European Chemicals Agency (ECHA) website explains the registration process, including the requirement of participation in each chemical's SIEF to share data. *Id.* SIEFs have no prescribed form and are independently managed by industry. *Id.* The members of each SIEF coordinate preparation of the required data for the ECHA. The ECHA publishes testing proposals or protocols and guidelines on chemicals/testing proposals. *Testing Proposals*, EUROPEAN CHEMICALS AGENCY, http://echa.europa.eu/web/guest/information-on-che micals/testing-proposals;jsessionid=7635865DC25B8FDF24EC7F0785DA189E.live1 (last visited Mar. 6, 2012). Participation in a SIEF may be mediated by a certified "only representative" who assists companies in the participation and protection of their proprietary interests. *See, e.g.*, *REACH Resource Centre*, STEPTOE & JOHNSON, LLP, http://www.steptoe.com/reach (last visited Mar. 26, 2012). The data will be reviewed by ECHA, which will assess the data and decide what risk ranking to assign each chemical; these ratings will be published. *Id*. The system achieves efficiency in testing by reducing duplication and by providing an incentive to remove a chemical from use, before it is listed and more money is spent on it. *Id*.

REACH aims to force change in the industry and it makes the European Union the pacesetter in the global chemicals market.[47] However, the European Union faces an enormous challenge as it begins to require the industry to produce information on more than 30,000 chemicals. Indeed, REACH may face the same resistance to toxicity research that has undermined TSCA.[48] Meanwhile, in the United States, debate on amending TSCA simmers, as the Obama Administration's EPA breaks new ground by interpreting TSCA broadly to support more assertive regulatory action. These developments are discussed below, in Part III.B.

II. NEW RESEARCH RESULTS

Chapter 4 of *Legally Poisoned* describes scientific research that is adding new dimensions to our understanding of chemicals' activity in the environment. Better detection technology and rapidly evolving medical science allow increased attention to a range of health outcomes and to the effects of chemicals on the young.[49] Disease processes have become more transparent as researchers have put genetics and related tools to use. For example, Cranor reports that it is now possible to trace some "chromosomal damage from newborns or young children back to chromosomal translocations in the womb."[50] Using this technique, some studies suggest that most childhood leukemias

47. *See generally* MARK SCHAPIRO, EXPOSED: THE TOXIC CHEMISTRY OF EVERYDAY PRODUCTS (2007) (suggesting that countries that do not follow the European Union's lead will fall behind and become dumping grounds for low quality and hazardous chemicals).

48. Natasha Gilbert, *Data Gaps Threaten Chemical Safety Law—European Companies Are Not Providing Robust Information to Regulators or Alternatives to Animal Experiments*, 475 NATURE 150, 150 (2011) (reporting that a study reviewing 200 dossiers chosen at random from the 3200 filed with the European Union's chemicals regulator, ECHA, shows that many rely on old data and fail to suggest new tests). ECHA's director of regulatory affairs has said, "Industry has not taken full responsibility for the quality of the data." *Id.* (quoting Jukka Malm, Director of Regulatory affairs at ECHA).

49. In the early decades of EHS regulation, occupational studies were the chief source of data about human responses to chemicals. Studies that examine effects on workers, who are generally very healthy (the "healthy worker" effect), are likely to underestimate the adverse effects of exposures on weaker members of the community, including children. *See* CRANOR, TOXIC TORTS, *supra* note 1, at 191. This problem has long been recognized, but environmental toxicology has only recently focused on children's health. *See* Jane A. McElroy, *Environmental Exposures and Child Health: What We Might Learn in the 21st Century from the National Children's Study*, 2 ENVTL. HEALTH INSIGHTS 105, 105–07 (2008). For discussion of the poor fit between current regulatory approaches and our limited information about children's risks, see Michael Schon, *Susceptible Children: Why the EPA's New Risk Assessment Guidelines for Children Fail to Protect America's Future,* 36 ARIZ. ST. L.J. 701, 710–14 (2004); Wendy Wagner & Lynn Blais, *Children's Health and Environmental Exposure Risks: Information Gaps, Scientific Uncertainty, and Regulatory Reform*, 17 DUKE ENVTL. L. & POL'Y F. 249, 252–58 (2007); Valerie Watnick, *Pesticides and Children: Unwitting Participants in Experimentation*, 13 CARDOZO J.L. & GENDER 801, 814–19 (2008); RENA STEINZOR, MOTHER EARTH AND UNCLE SAM: HOW POLLUTION AND HOLLOW GOVERNMENT HURT OUR KIDS, CENTER FOR PROGRESSIVE REFORM, White Paper #801, 1–2, *available at* http://www.cprblog.org/articles/MotherEarthfinalShort.pdf.

50. CRANOR, LEGALLY POISONED, *supra* note 1, at 90.

begin before birth and maternal or perinatal exposures to chemicals or infectious agents are critical factors.[51]

This Part sketches some highlights from Cranor's explanation of this complex material. The discussion is organized under three separate headings, although in actuality the subjects are interwoven. First, long time lapses or latency periods between exposure and symptoms now appear common and may extend from fetal exposures to late-in-life illness. Fetuses and young children are particularly vulnerable and low dose exposures may affect development in ways that are not visible until much later. Second, additive and synergistic effects of chemical substances usually have not been considered in chemical regulation, but research supports the view that they are an important dimension in disease. Third, the study of epigenetics suggests that the cell dynamics that interact with DNA are also active in determining responses to environmental stimuli and that these may be affected by low-level exposures, with effects that carry into later unexposed generations.

The new evidence has substantial implications for tort law and regulation. For instance, regulation has operated on the premise that, except for special cases like carcinogenesis, most chemicals will be safe at some threshold level of exposure. Yet, we know already that the more we learn about some substances, such as lead, the greater the health effects we find at lower doses.[52] It now appears that across a wide range of exposures the dose-response relationship can vary in surprising ways. Responses may be stronger at lower doses or different in nature depending on the dose, the timing and the nature of the affected biological system.[53]

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51. *Id.*

52. *See* CRANOR, LEGALLY POISONED, *supra* note 1, at 26–28, 105–109 (describing life cycle harms from lead exposure); CRANOR, REGULATING TOXIC SUBSTANCES, *supra* note 1, at 15–25 (describing dose-response assessment issues).

53. Classical toxicology has assumed that dose-response relationships are "monotonic" and linear. Monotonic means biological effects move in one direction in response to an increase in dose while linear means the changes in effects are proportional to the changes in doses. Nonlinear monotonic relationships also yield an effect in the same direction, but the size of changes in effects is not proportional to the changes in doses; low doses can yield larger effects than high doses. Nonmonotonic relationships allow decreases in effects at some doses and increases at others and the effects at different levels may be quite different. *See* Linda S. Birnbaum & Paul Jung, *From Endocrine Disruptors to Nanomaterials: Advancing Our Understanding of Environmental Health to Protect Public Health*, 30 HEALTH AFFS. 814, 816 (2011). Birnbaum and Jung point to the implications of these findings: "Tests of effects at some high dose are no longer adequate to determine the full spectrum of response from a given chemical. Many chemicals . . . follow nonmonotonic effect curves, indicating that there may be physiological effects at low doses. What's more, these effects may be different than those observed at high doses." *Id.* at 817.

A. Latency and Early Exposures

In the landmark case, *Sindell v. Abbot Laboratories,*[54] the plaintiffs were young women who were exposed to a synthetic estrogen in utero when their mothers took the pharmaceutical diethylstilbestrol (DES).[55] As they matured, the daughters suffered from reproductive malformations and developed reproductive cancers and related diseases. The defendant companies were found negligent, as they had marketed the drug without sufficient testing.[56] Many of the plaintiffs could not identify the particular company that manufactured the pills their mothers had taken years before and the court adapted the "cause in fact" test to allow DES daughters to sue groups of DES producers, using "market share" as a substitute for a record of each defendant's link to individual plaintiffs.[57]

The *Sindell* plaintiffs' distinctive symptoms were uniquely associated with DES exposure, so they could prove that it was DES that caused their injuries.[58] For a long time, the DES cases appeared to be a rare unhappy accident. The limitations of medical understanding at the time and the strict evidentiary and causation rules of tort law supported the impression that the daughters' disease pattern was an isolated phenomenon. Over time, however, further effects have been chronicled, including increased risk of breast cancer in DES daughters and also in DES mothers.[59] There are indications of effects on DES sons, though the sons' symptoms are less clearly associated with DES exposure.[60]

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54. Sindell v. Abbott Labs., 607 P.2d 924, 926 (1980). *Sindell* started a colloquy about how to handle toxic risks caused by fungible products. *Id.* at 936; *see* Hymowitz v. Eli Lilly & Co., 539 N.E.2d 1069, 1082 (1989) (articulating a public risk version of market share liability).

55. CRANOR, LEGALLY POISONED, *supra* note 1, at 39–40, 86–87. DES is an estrogen that was synthesized in the late 1940s. *Id.* at 39.

56. *Id.* at 86–87. The FDA approved DES for use in pregnancy with minimal testing. Although it was marketed for pregnant women, lab tests had not examined the second generation of lab animals for health effects. DES was widely prescribed to prevent spontaneous abortions and premature deliveries.

57. This theory, crafted for indeterminate defendants and fungible products, was applied in *In re Methyl Tertiary Butyl Ether (MTBE) Products Liability Litigation*, 517 F. Supp. 2d 662, 671–72 (S.D.N.Y 2007).

58. Distinct types of injuries—reproductive cancers and structural abnormalities—were associated with in utero exposure of daughters to DES, providing a clear causal link that facilitated tort litigation. CRANOR, LEGALLY POISONED, *supra* note 1, at 87.

59. *Id.* at 103–04.

60. Tracey I. Batt, *DES Third-Generation Liability: A Proximate Cause,* 18 CARDOZO L. REV. 1217, 1222, 1234–35, 1240–50 (1996) (discussing compensation for DES grandchildren in cases where third generation harm is not due to genetic injury, but is due to birth defects caused by the mother's physical reproductive disabilities caused by DES); Marisa L. Mascaro, *Preconception Tort Liability: Recognizing a Strict Liability Cause of Action for DES Grandchildren*, 17 AM. J.L. & MED. 435, 436–37, 448–55 (1991) (discussing transgenerational DES health effects and cases, including one in which a father alleged his own "*in utero* exposure to DES caused his daughter's cancer"). Like the Agent Orange plaintiffs, DES sons were "indeterminate plaintiffs," as their symptoms were not statistically distinguishable from background rates in the unexposed population. *In re* Agent Orange Prod. Liab. Litig., 597 F. Supp. 740, 740

The DES cases illustrate the ways that fetal exposure may be related to later disease. As the disaster was unfolding, a group of physicians conducted autopsies of 281 female fetuses and neonates; they found vaginal tissue abnormalities (*adenosis* or incomplete development) in about seventy percent of those whose mothers had taken DES, compared with four percent of those whose mothers had not taken it. The *adenosis* was later understood to be a precursor to the abnormalities that emerged when surviving girls matured. The study demonstrated that exposure in utero could cause changes that would become apparent later in life.[61]

The DES epidemic was one of several tragedies that shattered the myth of the protective womb and provided data about fetal sensitivity and latent disease. Another high profile mass exposure was caused by the industrial discharge of methyl mercury into Japan's Minimata Bay between 1953 and 1968 and a similar mass exposure in Iraq in the 1970s.[62] The data gathered from these accidents showed that fetuses were severely affected by doses that caused only mild effects in adults.[63] In utero exposures to the drug thalidomide also caused serious injuries and increased our understanding of the vulnerability of the fetus.[64]

Cranor explains how the risk of disease is heightened when exposures occur in utero. Numerous factors not only increase doses, but also limit the developing body's capacity to defend against them. For instance, mothers share their own body burden of chemicals with the fetus, and fetal exposure may be greater relative to body weight.[65] Compared to adults, children have

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(E.D.N.Y. 1984), *aff'd*, 818 F.2d 145 (2nd Cir. 1987) (discussing structure of indeterminate party cases and legal options for managing them). The Centers for Disease Control website provides a platform for tracking and communicating with DES families. One page specifically addresses the known and suspected health effects for DES sons: *Known Health Effects for DES Sons,* CENTER FOR DISEASE CONTROL, http://www.cdc.gov/des/consumers/about/effects_sons.html (last visited Mar. 1, 2012). The CDC reports consistent research finding for DES sons indicating an increased risk for noncancerous cysts on the testicles and less consistent reports for other reproductive anomalies. *Id.*

61. CRANOR, LEGALLY POISONED, *supra* note 1, at 86–88.

62. *Id.* at 82–84. Between 1953 and 1968, methyl mercury waste that was discharged into Minimata Bay, Japan, entered the food supply of people who ate fish from the Bay. Many adult humans and animals who ate the fish displayed a range of neurological effects.

63. *Id.* at 84–85. Children contaminated in utero displayed symptoms similar to cerebral palsy at a rate ten times that of unexposed children and also suffered mental retardation, inhibited growth, and death. More subtle effects appeared over time. *Id.* at 83.

64. In the United States, the drug was not approved for marketing, but 2 million tablets were distributed without FDA approval and about forty "thalidomide babies" were born in the U.S. *Id.* at 86; *see* Anita Bernstein, *Formed by Thalidomide: Mass Torts as a False Cure for Toxic Exposure*, 97 COLUM. L. REV. 2153 (1997) (reviewing the history of thalidomide events and their effect on the development of toxics law in the United States); *see also* Meredith Wadman, *U.S Lawsuit Extends Thalidomide's Reach—Drug Blamed for a Broader Range of Harmful Effects*, NATURE (Nov. 8, 2011), http://www.nature.com/news/2011/111108/full/479161a.html (reporting that thirteen Americans, suing for effects of thalidomide, say the drug affected more people in the United States than thought and that drug companies have attempted to hide these facts).

65. CRANOR, LEGALLY POISONED, *supra* note 1, at 99–100. Researchers have been identifying the factors that contribute to developmental risk from toxicants, including the ways sub-

limited capacity to defend against chemical exposures.[66] Children also have more years of future life, so diseases that require several critical steps to occur will have time to manifest the full symptoms; early exposure increases the chances that the entire process with be completed and, for example, that a cancer will develop.[67]

The development process itself presents many susceptibilities, as organ systems rapidly grow and change from conception through adolescence. There are critical susceptibilities at different stages of development.[68] The brain and nervous system, for example, are more vulnerable to toxic agents while they are undergoing rapid growth and change. As Cranor explains,

> [T]he brain must grow from a few cells into an extremely complex organ of billions of cells. These must be interconnected, very specialized, and properly located in order to communicate with one another and to function properly. Thus there are numerous opportunities for toxicants to interfere with neurological function during development that do not exist for mature brains or for other organ systems.[69]

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stances penetrate the placenta and contaminate breast milk. *Id.* at 100–01. Methylmercury concentrations in the fetal brain may be five times higher than concentrations in the mother's blood. The high fat content in breast milk may have higher concentrations of fat soluble chemicals, such as polychlorinated biphenyls (PCBs). Lead in a pregnant woman's bones can be released by the same mechanism that releases calcium for the fetus' development; thus a mother's prior lifetime concentration can affect the child. After birth children continue to have higher exposure rates; they have faster metabolisms and inhale more than adults do. *Id.* at 101. Children are more active than adults and also play close to the ground and put things in their mouths. *Id.* at 101–02. For instance, polybrominated diphenyl ether (PBDE) flame-retardants that are released from plastics and furniture, becoming part of household dust, are more likely to be assimilated by children than by adults.

66. *Id.* at 101–02. For instance, the blood-brain barrier, which prevents many agents from entering the developed brain, does not fully develop until six months after birth. *Id.* at 97–98. Also, the development of enzyme-based defenses is incomplete, apparently in some respects until the age of seven. *Id.* Children's skin provides less protection than that of adults, as it is more permeable than that of adults and children have more skin surface area relative to their body weight. The seriousness of these factors depends upon the skin contact exposures individual children experience. *Id.* at 98–99. Newborns' blood protein bonds less easily with toxicants and therefore has less capacity to eliminate them from the body. *Id.* at 99. Neonates and newborns' kidneys function more slowly to eliminate at least some drugs from the body; methylmercury has been shown to have a longer half-life in neonates than in adults. *Id.*

67. *Id.* at 102–03.

68. While not all aspects of children's defenses are weaker than those of adults, the susceptibility of children and fetuses is different from that of adults and needs to be considered specifically. Cranor outlines the stages of development and describes the different organ systems and their particular sensitivities. Different factors can combine to disrupt or change development in ways that are dramatic or subtle, immediate or latent. *Id.* at 89–90. Exposure of eggs in females and sperm in males can lead to transgenerational effects. *Id.* at 90–92.

69. *Id.* at 93.

Some late-life neurological dysfunctions, including Parkinson's disease, may have in utero or postnatal origins.[70] Like the nervous system, the immune system goes through precise sequential phases that are sensitive to disruption by toxicants.[71] The respiratory system and the reproductive system are similarly vulnerable to chemical exposures as they progress through extended developmental sequences.[72]

B. Additive and Synergistic Effects and Endocrine Disruptors

Repeated exposures can cause effects that are quite different from a single chemical hit.[73] Cranor writes that there is a consensus that substances can make additive contributions to disease or dysfunction and that very low concentrations of synthetic substances, when added to background levels of similar substances, can cause adverse outcomes.[74] For example, if there are several natural or synthetic estrogens that attach to similar receptors, the problem increases. Cranor states, "It is even possible that no one synthetic estrogen taken by itself at tiny concentrations may pose problems, even when added to natural background rates. However, when all sources are added together, the

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70. For instance, research suggests that Parkinson's disease may be caused in part by environmental exposures, including some pesticide ingredients. These results are "based on solid rationale and some model systems." *Id.* at 126–28 (quoting Brian K. Barlow et al., *The Gestational Environment and Parkinson's Disease: Evidence for Neurodevelopmental Origins of a Neurodegenerative Disorder*, 23 REPRODUCTIVE TOXICOLOGY 457, 458 (2007)). One model proposes that brain development may be affected by very low levels of exposure, if the timing interferes with the development of neuron reserves needed for lifetime normal functioning. As the individual ages, a slow decline in function results from the early damage. *Id.* at 126–28. Related research on other neurotoxins supports the hypothesis. Pesticides and trichloroethylene, both containing neurotoxins, have been linked with symptoms similar to Parkinson's. For a discussion of Parkinson's and aging boomers, see *id.* at 95, 103. *See also* Samuel M. Goldman et al., *Solvent Exposures and Parkinson Disease Risk in Twins*, ANNALS OF NEUROLOGY, Nov. 14, 2011, *available at* wileyonlinelibrary.com, DOI: 10.1002/ana.22629 (discussing case reports linking solvent exposure to Parkinson's disease supported by an epidemiological study of ninety-nine twin pairs, which found significant increases in PD in twins exposed to one of three solvents).

71. Long-lasting or permanent immune system deficits can be caused by exposure to toxicants at critical stages of development. Autoimmune reactions can also result from early developmental injuries and some patterns of disease are related to immune system deficiencies and may develop because of early influences on the system. Effects may be latent for thirty or more years. For instance, early appearing allergic reactions such as asthma, may be followed over time by ear infections, respiratory infections and even lung cancer in an identifiable pattern. CRANOR, LEGALLY POISONED, *supra* note 1, at 95.

72. *Id.* at 96–97.

73. "Additive" can mean repeated exogenous doses of the same or similar chemicals or the addition of exogenous chemicals to ongoing endogenous biological processes that rely upon precise chemical interactions. Bernard D. Goldstein & Mary Sue Henifin, *2000 Reference Guide on Toxicology*, *in* 4 MICHAEL DORE, LAW OF TOXIC TORTS app. 29K (2011) ("When the effect of multiple agents is that which would be predicted by the sum of the effects of individual agents, it is called an additive effect; when it is greater than this sum, it is known as a synergistic effect . . . ."). Synergisms may be interactions of different chemicals or new impacts on existing conditions created by previous exposures or other factors. *Id.*

74. CRANOR, LEGALLY POISONED, *supra* note 1, at 119.

total estrogens may be more than sufficient to produce adverse outcomes."[75] Consequently, regulation may need to develop a greater capacity to account for multiple environmental influences.[76]

Endocrine disruptors (EDs) are chemicals that can mimic human hormones.[77] Recent research suggests that increased exposure to estrogens, both natural and synthetic, can add to breast cancer risk.[78] EDs are also implicated in developmental disorders of the reproductive system. The National Research Council (NRC) explained in a 1999 report how a low environmental dose of estrogen may affect fetal development:

> No threshold exists for exogenous estrogen, because endogenous estrogens are at a sufficiently high concentration to exceed the threshold for sex determination. . . . If the threshold for response to estrogen has already been exceeded before exposure to an environmental estrogen, the additional load of an environmental estrogen might cause a significant increase in the occupied receptors required for the [sex determination] response.[79]

Thus, when the fetus is already occupied responding to the endogenous estrogens appropriate to its sexual development, environmental exposures may change the path of that development. DES is apparently an example of this phenomenon.[80]

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75. *Id.*

76. *Id.* at 2, 16–19, 20–26 (discussing multiple exposures, biomonitoring, and body burdens).

77. *See* Linda S. Birnbaum, *Endocrine Disrupting Chemicals in Drinking Water: Risks to Human Health and the Environment, Testimony Before the Committee on Energy and Commerce of United States House of Representatives*, U.S. DEP'T OF HEALTH AND HUMAN SERVS. (Feb. 25, 2010), http://www.hhs.gov/asl/testify/2010/02/t20100225a.html. Dr. Birnbaum, the Director of the National Institute of Environmental Health Sciences and the National Toxicology Program, summarizes the development of research on ED mechanisms and effects, including persistent epigenetic effects, and the ongoing federal research in this area. *Id.; see also* Alana Van der Mude, *Endocrine-Disrupting Chemicals: Testing to Protect Future Generations*, 38 B.C. ENVTL. AFF. L. REV. 509, 529–31 (2011) (describing the EPA's slow implementation of the testing requirements of the Food Quality Protection Act (FQPA) and arguing that acceptance of outdated data from manufacturers is arbitrary and capricious under the Administrative Procedure Act and should be challenged); Valerie J. Watnick, *Our Toxics Regulatory System and Why Risk Assessment Does Not Work: Endocrine Disrupting Chemicals as a Case in Point*, 2004 UTAH L. REV. 1305 (2004) (discussing how the current regulatory framework tries to manage tolerable levels of exposure but does not fit the ED harm profile, so screening tests must be mandatory).

78. A large percentage of breast cancers are apparently because of "environmental" influences, a term that includes a wide range of factors such as work place exposures, food contaminants, pharmaceuticals, chemicals in products, air water and soil and physical factors such as radiation. CRANOR, LEGALLY POISONED, *supra* note 1, at 115 (citing two figures: 5% and 33%). Estrogen production and exposure is a major factor, including exposure to one's own estrogen. Women who are childless produce more estrogen over their lifetimes and are consequently have higher risks of breast cancer. Research from hormone replacement therapy supports this view. *Id.* at 116. Other factors exist: twins have more exposure in utero and higher rates. *Id.* at 116.

79. *Id.* at 118 (quoting COMM. ON HORMONALLY ACTIVE AGENTS IN THE ENV'T, NAT'L RESEARCH COUNCIL, HORMONALLY ACTIVE AGENTS IN THE ENVIRONMENT 113–14 (1999)).

80. *Id.* at 116–17.

A number of synthetic estrogens are now widespread in the environment. For example, Bisphenol A (BPA) is produced at a rate of about 2.3 billion pounds annually in the United States.[81] Most people today are continually exposed from various sources and virtually all U.S. residents are contaminated with it.[82] Research suggests a wide range of effects from low doses during development and in adulthood.[83] A committee of the National Toxicology Program (NTP) has expressed concerns about a variety of effects at current exposure levels.[84] BPA is one example of what appears to be a broader phenomenon. Cranor outlines research results for several other kinds of toxicants that are widespread and show troubling effects in recent studies.[85]

While substances like estrogens can attach to particular receptors and have additive toxic effects, they may also add to toxicity in other ways. Several compounds have been shown to interfere with pathways that cause events "upstream" from cells that are adversely affected. An example is reduction of thyroid hormones in pregnant women, which can affect the neurological de-

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81. *Id.* at 117.

82. Virtually everyone in developed countries receives chronic low doses of BPA. *Id.* at 118.

83. Based on animal studies, a panel of scientists has concluded that BPA can contribute to breast cancer and has other adverse effects. The panel found that current body burdens of BPA in adults are "within the range that is predicted to be biologically active in over 95% of the people sampled. *Id.* at 117 (quoting Frederick S. vom Saal et al., *Chapel Hill Bisphenol A Expert Panel Consensus Statement: Integration of Mechanisms, Effects in Animals and Potential to Impact Human Health at Current Levels of Exposure*, 24 REPRODUCTIVE TOXICOLOGY 131, 136 (2007)). BPA's effects may include not only breast cancer, but also "prostate . . . cancer, uro-genital abnormalities in male babies, a decline in semen quality in men, early onset of puberty in girls, metabolic disorder including insulin resistant (type 2) diabetes and obesity, and neurobehavioral problems, such as attention deficit hyperactivity disorder (ADHD)." *Id.* Many of the effects can result from even brief exposures during development and are irreversible. The exposures may act to increase the number of breast cells that are vulnerable to breast cancer, thus increasing the likelihood of the disease. *Id.* at 117.

84. *Id.* at 118.

85. These include petrochemical byproducts used in a variety of processes and products, including PCBs and PBDEs, part of the generic family of halogenated persistent molecules. *Id.* at 109. Some chemicals in the PCB group are persistent in humans and are associated with reproductive, teratogenic, and carcinogenic effects. *Id.* at 109–110. There can be additive toxic effects from exposure to these and other quite different chemicals; the amount of each chemical in the body at any time does not predict its health effects, as additivity may have substantial effects. Some variants within these groups are known especially for their effects on the nervous system and brain. *Id.* at 110–11 (describing incidents in Japan and Taiwan in which rice oil was contaminated and ingested by large numbers of consumers). Some are associated with decreased cognitive functioning in children. *Id.* at 103–05, 110–11.

Gaps in the research on neurotoxicity of PBDEs are because of absence of human studies, but animal evidence suggests cause for serious concern, both about cognitive effects from PBDE exposure and reproductive effects, including both male and female infertility. *Id.* at 112–14. Ninety-seven percent of adults are contaminated with PBDEs and concentrations are doubling every four to six years. *Id.* at 111.

Another class of endocrine disruptor is phthalate esters. *Id.* at 120. Phthalates are used in toys, packaging, pharmaceuticals, medical devices, cosmetics and personal-care products. A wide range of adverse effects appear related to phthalate exposure, including a number of male reproductive diseases and defects. Phthalates are potentially subject to the same additive and synergistic effects discussed above in connection with BPA. *Id.*

velopment of fetuses and newborns.[86] Researchers have also found synergistic effects resulting from the combined actions of chemicals that have antiandrogen effects on the male reproductive system. The separate exposures, each at doses below that which would alone cause an adverse effect, may act jointly through different mechanisms to produce greater effects than would occur from adding doses of a single substance.[87] Given these dynamics, for some chemicals there may not be any safe environmental exposure above zero.[88] In light of these additive and synergistic effects, Cranor cites the conclusion of the NRC, which stated that "the focus in cumulative risk assessment should be on the health outcomes and not the pathways that lead to them, whether defined as mechanisms of action or as modes of action."[89]

C. Epigenetics

Epigenetic mechanisms may explain at least some diseases that have origins in early life.[90] Epigenetics means "outside of genetics" and has been defined as "heritable changes in gene expression that are not due to any alteration in the DNA sequence."[91] Epigenetic mechanisms appear to be important in disease development, though they need not be seen as the only fac-

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86. Various liver enzymes may be triggered by dioxins and dibenzofurans and dioxin-like or non-dioxin-like PCBs at "environmentally relevant doses," that is, at doses that are close to the concentrations humans currently actually receive. *Id.* at 121 (quoting Tracey J. Woodruff et al., *Meeting Report: Moving Upstream—Evaluating Adverse Upstream End Points for Improved Risk Assessment and Decision-Making*, 116 ENVTL. HEALTH PERSP. 1568, 1570 (2008)).

87. *Id.* at 121. Andreas Kortenkamp suggests that "human risk assessment could work on the basis of the rebuttable hypothesis that dose addition [of estrogens] is applicable . . ." and that "joint effects occur *even when all mixture components are present at levels below doses that cause observable effects* . . . [Consequently] the ground is prepared to seriously consider group-wise regulation of EDs [endocrine disruptors]." *Id.* at 118–19 (quoting Andreas Kortenkamp et al., *Low-Level Exposure to Multiple Chemicals: Reason for Human Health Concerns?*, 115 S-1 ENVTL. HEALTH PERSP. 106, 113 (2007) and Andreas Kortenkamp, *Ten Years of Mixing Cocktails: A Review of Combination Effects of Endocrine-Disrupting Chemicals*, 115 S-1 ENVTL. HEALTH PERSP. 98, 104 (2007)).

88. Andreas Kortenkamp et al., *Low-Level Exposure to Multiple Chemicals: Reason for Human Health Concerns?*, 115 S-1 ENVTL. HEALTH PERSP. 106, 106 (2007); Andreas Kortenkamp, *Ten Years of Mixing Cocktails: A Review of Combination Effects of Endocrine-Disrupting Chemicals*, 115 S-1 ENVTL. HEALTH PERSP. 98, 98 (2007).

89. CRANOR, LEGALLY POISONED, *supra* note 1, at 121 (quoting COMMITTEE ON THE HEALTH RISKS OF PHTHALATES, NAT'L RESEARCH COUNCIL, PHTHALATES AND CUMULATIVE RISK ASSESSMENT: THE TASK AHEAD 4 (2008)).

90. *See* Fazal Khan, *Preserving Human Potential as Freedom: A Framework for Regulating Epigenetic Harms*, 20 HEALTH MATRIX 259, 266–76 (2010) (describing current scientific understanding of epigenetics). *See generally* Mark A. Rothstein et al., *The Ghost in Our Genes: Legal and Ethical Implications of Epigenetics*, 19 HEALTH MATRIX 1, 4–24 (2009) (describing current scientific understanding of epigenetics).

91. *See* Khan, *supra* note 90, at 267 (quoting Manel Esteller, *Molecular Origins of Cancer: Epigenetics in Cancer*, 358 NEW ENG. J. MED. 1148, 1148 (2008)). *See also* Romulo M. Brenna et al., *Toward a Human Epigenome*, 38 NATURE GENETICS 1359, 1359 (2006) (defining epigenetics as "the interplay of DNA methylation, histone medications and expression of noncoding RNAs in the regulation of gene expression patterns from early development to adulthood").

tor. Through these mechanisms, environmental influences may cause genes to not express themselves as they should or to express themselves at the wrong time.[92]

Cranor outlines several lines of research that underlie these assertions. One is research on nutritional "mismatch" between in utero nutrition and postnatal nutrition, which may lead to later diabetes and heart disease. Nutritional deprivation of pregnant women followed by adequate nutrition for a child after it is born can result in "metabolic syndrome," a collection of symptoms that include higher than normal body weight, glucose intolerance and a tendency to develop type 2 diabetes.[93] Some studies link chemical exposures to similar symptoms. DES, BPA and other compounds appear to contribute to excessive weight gain and to involve epigenetic mechanisms.[94]

Epigenetic influences may also have transgenerational effects, even though the gene sequence itself is not changed. In experimental settings, DES present in utero increased uterine carcinogens in adulthood and both the epigenetic effect and the tendency to cancer in adulthood were transmitted from one generation to another; similar results have been found with BPA and some pesticides.[95] Recent research suggests that low-level dioxin exposure of males in utero affects the size of the prostate and the changes appear to persist in later generations.[96] Other effects on male development have been shown in laboratory settings.[97]

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92. Epigenetic mechanisms act by modifying structure or function in the character of tissues, organs and systems. CRANOR, LEGALLY POISONED, *supra* note 1, at 122. These influences cause "altered gene expression or altered protein regulation associated with altered cell production and cell differentiation that are involved in the interactions between cell types and the establishment of cell lineages." *Id.* at 122 (quoting Jerrold J. Heindel, *Animal Models for Probing the Developmental Basis of Disease and Dysfunction Paradigm*, 102 BASIC & CLINICAL PHARMACOLOGY & TOXICOLOGY 76, 76 (2008)).

93. *Id.* at 122. Data supporting a developmental factor in these conditions is drawn from the experience of the Dutch population at the end of World War II, when famine was followed by adequate nutrition after the war's end. Children who were inadequately nourished in utero but had adequate diets afterwards were most vulnerable to obesity at age 50. Researchers call the phenomenon the "thrifty phenotype," as the fetuses were apparently preparing for a low calorie diet that did not materialize. They gained more weight than others as children and continued to have this tendency through life. *Id.* at 123. Russian children who were in utero during the famines caused by the siege of Leningrad were born into a world in which the famine continued and these children did not show the same symptoms. *Id.* at 123–24. Similar effects have been seen in animal experiments. *Id.* at 126.

94. Exposure to estrogens can cause estrogen-sensitive gene expression at the wrong time, as when the exposure occurs at a time in which estrogen levels are usually low. *Id.* at 124–25.

95. *Id.* at 124–125.

96. Estrogens increase the size of the prostate in adult mice; these weight increases have not been shown to lead to prostate cancer, but prostate cancer in men often follows an increase in the size of the prostate. *Id.* at 125.

97. For example, pregnant rats exposed to some phthalates have given birth to pups with defective male reproductive tracts, with adult consequences that include reduced sperm counts and testicular tumors. *Id.* at 126. Phthalate exposure has been associated with structural changes in human penises. *Id.* at 126. In utero exposure to a number of compounds, including a fungicide

D. Implications

Cranor characterizes the science as incomplete or "pointillist." However, what is known is very sobering. Chemicals are being loaded into our environment, even though they are linked to a variety of diseases and dysfunctions. Developing organ systems are especially susceptible. Latency periods may span decades and effects may persist through several generations. Cranor draws the strands together at the end of Chapter 4: the research confronts us with "a live possibility that developing fetuses and newborns can be at risk from toxicants contaminating their bodies," a risk that has been demonstrated in humans for numerous substances.[98] He notes with considerable understatement that the situation raises "major questions of justice."[99]

III. DEVELOPMENTS IN SCIENCE AND REGULATION

Steve Gold suggests that as recent breakthroughs have provided "a glimpse inside the black box," it appears that "the better image may be 'a nest of boxes, each with a succession of smaller ones,' with relations within and between them."[100] In spite of the complexity, in some respects our uncertainty is shrinking. We are learning about the biological and ecological systems that channel and respond to toxicants. Scientists are refining the analytical processes for filling in the gaps. Yet the lack of basic test data on chemicals in commerce remains an obstacle. Lack of data hinders regulation and science, and also the market, medicine, and ordinary people who want to be confident that their exposures are reasonably safe. As a society, we have not yet made a commitment to appropriate standards for testing and disclosure about toxicity. This Part sketches some recent advances in the science and technology of toxicity testing and then describes developments in the regulatory and legislative arenas.

A. Advances in Environmental Research Science

We are gaining new understanding of living systems as detection and testing technologies evolve. Genomics has provided a new model for structuring research and has influenced disciplines that are relevant to chemical risk assessment, including exposure science, toxicogenomics[101] and related

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used on grapes and other crops, have led to adverse effects on the male reproductive tract that could be transmitted through the male germ line for at least four generations. *Id.* at 126.

98. *Id.* at 130–31.

99. *Id.*

100. *See* Gold, *The More We Know*, *supra* note 33, at 406 (quoting Margaret R. Spitz & Melissa L. Bondy, *The Evolving Discipline of Molecular Epidemiology of Cancer*, 31 CARCINOGENESIS 127, 132 (2010)). Arguments for reform of causation rules that were based on the impenetrability of the box remain valid. *Id.* at 422.

101. *See, e.g.*, Gary E. Marchant, *Toxicogenomics and Environmental Regulation*, *in* GENOMICS AND ENVIRONMENTAL REGULATION: SCIENCE, ETHICS, AND LAW 11, 11–23 (Richard R. Sharp et al. eds., 2008) (describing expected uses of toxicogenomics, such as providing a fingerprint of various toxicological mechanisms and filling in details of biological mechanisms

fields.[102] These efforts reveal the complexity of environmental influences on health and they promise benefits in the long run. They also face great challenges.[103]

1. *Exposure Science*

Exposure science is moving beyond simply measuring the presence of a pollutant in the air, for example, then extrapolating from this to the amount likely taken into the body. More sophisticated tools now allow a richer understanding of pollution impacts.[104] A particularly useful innovation, biomonitoring, samples the blood of individuals to precisely measure the presence of targeted pollutants.[105] Using biomonitoring, researchers can also distinguish the exogenous inputs, those attributed to pollution, life style, and other outside influences, from endogenous doses, chemicals that the body itself produces. This ability provides greater understanding of chemical dynamics in the body,

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that are now managed in risk assessment by plausible extrapolations). *But see* David E. Adelman, *The False Promise of the Genomics Revolution for Environmental Law*, 29 HARV. ENVTL. L. REV. 117, 117–18 (2005); Gold, *The More We Know*, *supra* note 33, at 401–06.

102. *See, e.g.*, Brenna et al., *supra* note 91, at 1359. *See also* Kellyn S. Betts, *A Study in Balance: How Microbiomes Are Changing the Shape of Environmental Health*, 119 ENVTL. HEALTH PERSP. A 341, A 341 (Aug. 1, 2011), *available at* http://dx.doi.org/10.1289/ehp.119-a340. Betts describes the research investigating the functions and variability of human microbiomes. *Id.* The composition of microbiomes is influenced by host genetics and the environment, diet, and other factors. *Id.* If the composition of a microbiome changes, the range of services it provides its human host also may shift. *Id.* The intestinal microbiome may play a key role in promoting enzymes, which break down substances such as toxic chemicals. *Id.* at A 344.

103. *See* Kenneth Olden, *Genomics in Environmental Health Research—Opportunities and Challenges*, 198 TOXICOLOGY 19, 19 (May 20, 2004).

104. The EPA's website states, "The traditional approach of assessing the risk from a single chemical and a single route of exposure (such as breathing air) is being replaced by methods that attempt to evaluate total risks from exposures; the method requires data on what chemicals are found in the relevant environment, their concentration levels, how they come into contact with humans, how they enter the body, and what they do after that." *Innovative Tools Help EPA Scientists Determine Total Chemical Exposures*, EPA, http://www.epa.gov/sciencematters/august 2011/sheds.htm (last visited Mar. 25, 2012); *see also* DAVID WOOLEY & ELIZABETH MORSS, CLEAN AIR ACT HANDBOOK: A PRACTICAL GUIDE TO COMPLIANCE § 5:31 (21st ed. 2011). In the past, researchers often had to estimate exposure to pollutants relying on questionnaires and also ambient measurements, though these change constantly. If ambient measurements were not available, estimates or measurements of air emissions from major sources, for example, could be run through computer models that estimate dispersion. These processes all entail considerable uncertainty. Further uncertainties arise from the need to extrapolate from the ambient figure to the actual dose to the body after respiration. *See* Latin, *supra* note 24, 114–18 (describing exposure evidence in the benzene case and its poor fit with quantitative risk methodology.).

105. Biomonitoring gives more precise data about exposures but generally does not identify sources of the exposures. CRANOR, LEGALLY POISONED, *supra* note 1, at 20–26; *see* Albert C. Lin, *Beyond Tort: Compensating Victims of Environmental Toxic Injury*, 78 S. CAL. L. REV. 1439, 1470–73 (2005) (new technologies will change tort and regulatory law, as chemical biomonitoring can detect the extremely low levels of chemicals or their metabolites in small samples of human blood, urine, saliva, or tissue); William H. Rodgers, Jr., *Improving Laws, Declining World: The Tort of Contamination*, 38 VAL. U. L. REV. 1249, 1254 (2004) (considering the notion of "normalcy" in a world in which consumers can compare their body burdens of mercury, molybdenum, uranium, and other contaminants with other folks living in other parts of the country).

and it changes the analytical frame. The relevant environment is now the internal one.[106]

Exposure scientists are working on developing the "exposome," an attempt to measure all exposure events from conception to death.[107] The model suggests a top-down regulatory approach that starts with chemicals of concern, rather than starting from discharges into the environment.[108] This view is consistent with the regulatory approach that is partially in place in the United States and is being implemented by the European Union through REACH: developing lists of chemicals of concern using basic testing, conducting further research, and then publicizing the results and placing limits on the use of hazardous substances.[109] It is also consistent with emerging regulatory perspectives that attempt to account for additive and synergistic effects of exposures.[110]

2. *Computational Toxicology*

Conventional toxicology has relied upon in vitro tests to detect cell changes caused by potential toxicants. If mutations occur, the pollutant is a candidate for in vivo or live animal tests designed to reveal signs of carcinogenicity.[111] Cranor provides a detailed and clear explanation of the merits and shortcomings of animal tests in toxicology.[112] As his book also makes clear, the range of potential biological receptors and interactions is enormous, posing an assessment challenge that seems insurmountable using conventional techniques. In 2005, the EPA asked the NRC to assess the options for alternative testing methods. The result was *Toxicity Testing in the 21st Century: A Vision and a Strategy*, published in 2007, which outlines a path forward and recommends a range of new approaches. There are high hopes for automated "high throughput" screening technology, which can process the effects of toxicants

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106. Rappaport, *supra* note 4, at 6; CRANOR, LEGALLY POISONED, *supra* note 1, at 18–19.

107. *The Exposome: A Powerful Approach for Evaluating Environmental Exposures and Their Influences on Human Disease*, EMERGING SCI. FOR ENVTL. HEALTH DECISIONS (the newsletter of the standing committee on use of emerging science for environmental health decisions, Washington, D.C.), June 2010, at 2, *available at* http://nas-sites.org/emergingscience/files/2011/05/03-exposome-newsletter-508.pdf; *see also*, Rappaport, *supra* note 4; Katherine Harmon, *Sequencing the "Exposome": Researchers Take a Cue from Genomics to Decipher Environmental Exposure's Links to Disease*, SCIENTIFIC AM., (Oct. 21, 2010), http://www.scientificamerican.com/article.cfm?id=environmental-exposure; John Howard, *From the Director's Desk—An Expose on Exposomes*, NIOSH E-NEWS (Feb. 2010), http://www.cdc.gov/niosh/enews/enewsV7N10.html (outlining some of the work ahead in developing the exposome).

108. *See* Rappaport, *supra* note 4, at 6–7.

109. *See supra* notes 44–46 and accompanying text.

110. *See supra* Part II.B.

111. Animal research is costly and time consuming, not to mention its terrible price in suffering. Estimates of animals used for research "range from tens of millions to 100 million or more. Between 80 and 800 animals are now needed per drug." Meredith Cohn, *Alternatives to Animal Testing Gaining Ground*, BALTIMORE SUN, Aug. 27, 2010, at 19 (reporting on growing consensus in medical research that animal testing should be limited).

112. CRANOR, LEGALLY POISONED, *supra* note 1, at 67–75.

on a range of biological receptors.[113] The EPA has announced its own plan for testing, based on the NRC report[114] and is joining with several other agencies to develop the technologies and expedite development of new toxicity testing resources.[115]

The effectiveness of these methods is unclear, however; it is too soon to assess their real value.[116] Minimizing animal tests is a consensus goal, but in vitro methods, even very powerful ones, may not capture the biological complexity of toxicity. A central challenge will be to determine the extent to which cell culture and similar non-whole animal tests reflect actual human outcomes.[117] David Adelman points out that, while new computational methods

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113. COMM. ON TOXICITY TESTING AND ASSESSMENT OF ENVTL. AGENTS, NAT'L RESEARCH COUNCIL, TOXICITY TESTING IN THE 21ST CENTURY: A VISION AND A STRATEGY 11 (2007) [hereinafter, NRC, TOXICITY TESTING IN THE 21ST CENTURY], *available at* http://www.nap.edu/openbook.php?record_id=11970&page=R1. *See* Symposium, *Toxicity Testing in the 21st Century*, 29 RISK ANALYSIS 471 (2009). The new research emerges from a confluence of three lines of work: high-throughput automated techniques developed by the pharmaceutical industry that use automated methods to identify the biologic activities of thousands of chemicals that used to be studied in animals; systems biology that provides computational models and laboratory data to describe and understand biologic systems as a whole; and bioinformatics, which applies computational techniques to vast amounts of data to understand how cells and cell systems work. NRC, TOXICITY TESTING IN THE 21ST CENTURY, *supra*. *See generally Computational Toxicology: From Data to Analyses to Applications*, EMERGING SCI. FOR ENVTL. HEALTH DECISIONS (Standing Comm. on Use of Emerging Sci. for Envtl. Health Decisions, D.C.), Nov. 2010, *available at* http://nas-sites.org/emergingscience/files/2011/05/02-comp-tox-newsletter-508.pdf (discussing the scientific aspects of computational toxicology); *Putting Chemicals on a Path to Better Risk Assessment*, 235 SCIENCE 694 (2009) (discussing the use of computers to assess chemical toxicity rather than animal testing).

114. OFFICE OF THE SCI. ADVISOR, U.S. ENVTL. PROT. AGENCY, THE U.S. ENVIRONMENTAL PROTECTION AGENCY'S STRATEGIC PLAN FOR EVALUATING THE TOXICITY OF CHEMICALS (Mar. 2009), *available at* http://www.epa.gov/spc/toxicitytesting/docs/toxtest_strategy_032309.pdf; DORE, *supra* note 73, at app. 29DD; *EPA's Strategic Plan for Evaluation of Chemical Toxicity*, 27 HAZARDOUS WASTE CONSULTANT, no. 5, 2009 at 1, 12 (describing the background and contents of the plan).

115. "EPA's Computational Toxicology Research Program (CompTox) is part of the broader Chemical Safety for Sustainability Research Program" and includes several components. *Computational Toxicology Research Program*, U.S. ENVTL. PROT. AGENCY, http://www.epa.gov/ncct (last visited Mar. 6, 2012). Tox21 is a collaboration between the EPA and other federal agencies that work together to prioritize which chemicals need more extensive toxicological evaluation, develop models that can be used to more effectively predict how chemicals will affect biological responses, and conduct fifty or more ToxCast high-throughput screening assays on this enlarged chemical library every year for the next several years. *Id.* The goal is to be able to provide better data to risk assessors making decisions about protecting human health and environment. *Id.* The system is currently screening over 10,000 chemicals at the NIH Chemical Genomics Center. *Id.*

116. Romualdo Benigni, *Alternative Approaches for the Identification of Carcinogens Are Closer than Usually Thought, but the Present Strategies and Regulations Need to Be Updated*, ALTTOX.ORG, http://alttox.org/ttrc/toxicity-tests/carcinogenicity/way-forward/benigni/ (suggesting that the new approach "tracing molecular perturbations related to specific biochemical pathways with the use of various omics technologies (*in vitro* high throughput assay systems). . . . is still in its infancy, and needs much work and refinement.") (last updated Nov. 29, 2011).

117. NRC, TOXICITY TESTING IN THE 21ST CENTURY, *supra* note 113; Symposium, *Toxicity Testing in the 21st Century*, *supra* note 113.

offer to transform toxicology from a quasi-scientific status to a "true" science based upon detailed understanding of toxicity and precise testing methods, the discovery of new layers of biological complexity, like epigenetics, is likely to delay this dream.[118] Indeed, the prospect of quick and inexpensive testing might distract us from recognizing its shortcomings.[119]

3. *Research Collaboration*

Recognition of the power of information diffusion is leading to greater collaboration in research. Significant biomedical research is being conducted collaboratively by public-private consortia, particularly in genomics and proteomics.[120] There are some examples of voluntary environment-specific collaborations in the United States, but these have had mixed results.[121]

The European Union has developed a large scale public-private partnership to develop alternatives to animal tests for cosmetics. Through a series of directives that phase out the use of animals in cosmetics testing and also

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118. David E. Adelman, *A Cautiously Pessimistic Appraisal of Trends in Toxics Regulation*, 32 WASH. U. J. L. & POL'Y 377, 411–26 (2010).

119. *Id.*; *see also* Joyce S. Tsuji & Michael R. Garry, *Advances in Toxicity Testing Herald Improvements and Challenges for Risk Assessment*, 29 RISK ANALYSIS 490, 490 (2009) (sources of discrepancy between in vitro and in vivo in a recent study appeared to be because of the "likely . . . ability of physiological systems to regulate levels of essential elements . . . despite . . . high reactivity in cell cultures.").

120. The conventional image of the secretive inventor who will lose out if others copy his work is being supplemented by the realization that "giving away" information may build the knowledge base, so that innovation can go faster and arrive at new creative opportunities. *See generally* Rochelle Cooper Dreyfuss, *Collaborative Research: Conflicts on Authorship, Ownership, and Accountability*, 53 VAND. L. REV. 1161 (2000) (proposing a framework for rules that blend traditional intellectual property and transactional approaches to support collaborative research); Rochelle Cooper Dreyfuss, *Does IP Need IP? Accommodating Intellectual Production Outside the Intellectual Property Paradigm*, 31 CARDOZO L. REV. 1437 (2010) (appropriate intellectual property are still important to innovation in many contexts); Rebecca S. Eisenberg & Arti K. Rai, *Harnessing and Sharing the Benefits of State-Sponsored Research: Intellectual Property Rights and Data Sharing in California's Stem Cell Initiative*, 21 BERKELEY TECH. L.J. 1187 (2006) (projects are structured to support sharing of early precompetitive research that eventually will provide a platform for new product development).

121. The EPA's High Production Volume (HPV) Challenge program, organized with the American Chemical Society (ACS) and the nongovernmental organization, Environmental Defense, has made use of voluntary and collaborative principles. *See* David Markell, *An Overview of TSCA, its History and Key Underlying Assumptions, and its Place in Environmental Regulation*, 32 WASH. U. J.L. & POL'Y 333, 356–57 (2010). Under this project, chemical companies have sponsored chemicals and pledged to collect or produce toxicity data. However, the effort has received mixed reviews. *Id.* at 356–60. Some private companies are supporting ToxCast/Tox21. Lee Howard, *Diverse Groups Backing Tox21 Cause: Drug Companies Joining Effort to Reduce Animal Testing, Costs*, THE DAY (Aug. 15, 2010, 3:58 AM), http://www.theday.com/article/20100815/BIZ02/308159876/1019. The EPA and the American Chemical Society (ACS) have an online collaboration to exchange information and encourage green chemistry initiatives. *Green Chemistry Institute*, U.S. ENVTL. PROT. AGENCY, http://www.epa.gov/greenchemistry/pubs/gcinstitute.html (last visited Mar. 8, 2012); *Green Chemistry Resource Exchange*, AM. CHEM. SOC'Y GREEN CHEMISTRY INST., http://www.greenchemex.org/gcex/gc.do?module=search.list (last visited Mar. 3, 2012).

through outreach and coordination with the cosmetics industry, collaborative efforts are producing improved practices in research.[122] Under REACH, all companies that produce or import a listed commercial chemical must register at an online platform designated for that chemical, then work with other registrants to collect and develop information that will determine the chemical's classification. This work includes organizing and assessing existing data and determining what further research should be done. Testing is not voluntary and REACH incorporates an administrative mechanism that will determine the confidentiality status of information. It remains to be seen if REACH will fulfill its promise of toxicity data production.[123]

B. Policy Developments

1. *Administrative Initiatives*

The EPA is attempting to revitalize chemicals regulation by interpreting its own authority under TSCA more broadly. In September 2009, EPA Administrator Lisa Jackson announced plans to strengthen the agency's chemical management program.[124] Jackson said that the EPA would establish a "Chemicals of Concern" list and commence processes that could result in risk reduction regulations under TSCA's section 6.[125] The EPA listed ten chemicals that were being considered for action; it later chose four of these and issued "chemical action plans" for them.[126] This initiative is a distinctive development. Several experts have described the action plans as "almost breathtaking in scope . . . . EPA has never previously announced so many actions under the

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122. *See* Charles Laroche et al., *The European Partnership for Alternative Approaches to Animal Testing*, 14 AATEX J. (SPECIAL ISSUE) 769, 769–73 (2008). For background and discussion see SCHAPIRO, *supra* note 47, at 20–41.

123. *See supra* note 48 and accompanying text.

124. *See* Charles M. Auer et al., *EPA'S Action Plans Signal a New Chapter for TSCA While Informing the Future Legislative Debate on Chemicals*, 40 ENVTL. L. REP. 10243, 10244 (2010).

125. *See Enhancing EPA's Chemical Management Program*, U.S. ENVTL. PROT. AGENCY (Mar. 1, 2012), http://www.epa.gov/oppt/existingchemicals/pubs/enhanchems.html. In 2011, the EPA published a proposed "Discussion Guide" for Identifying Chemicals for Review and Assessment. *Discussion Guide: Background and Discussion Questions for Identifying Priority Chemicals for Review and Assessment*, U.S. ENVTL. PROT. AGENCY (Sept. 6, 2011), http://www.epa.gov/oppt/existingchemicals/pubs/chempridiscguide.html. The agency has held stakeholder meetings to get public input into the criteria it will use to identify chemicals of concern. *See* Richard Denison, *Avoiding Paralysis by Analysis: EPA Proposes a Sensible Approach to Identifying Chemicals of Concern*, ENVTL. DEF. FUND (Sept. 13, 2011), http://blogs.edf.org/nanotechnology/2011/09/13/avoiding-paralysis-by-analysis-epa-proposes-a-sensible-approach-to-identifying-chemicals-of-concern.

126. *Existing Chemicals Action Plans*, U.S. ENVTL. PROT. AGENCY (Mar. 1, 2012), http://www.epa.gov/oppt/existingchemicals/pubs/ecactionpln.html. For instance, for phthalates that pose a concern for development of the male reproductive system, the EPA's plan states that the data warrant initiating TSCA procedures to gather information and possibly a ban of the chemicals. *See* Richard A. Denison, *EPA Deserves an "A for Effort" for Its New Chemical Action Plans*, ENVTL. DEF. FUND (Jan. 4, 2010), http://blogs.edf.org/nanotechnology/2010/01/04/epa-deserves-an-%E2%80%9Ca-for-effort%E2%80%9D-for-its-new-chemical-action-plans; Auer et al., *supra* note 124, at 10244.

Toxic Substances Control Act (TSCA), nor has it ever cited use of §6 so widely."[127] It is not clear how successful the EPA will be, but its plans run parallel to the debate over legislative reform, discussed below, and the two could be mutually reinforcing.

The EPA has also started to improve the availability of useful information about chemicals. For instance, it has increased the types and amounts of data that companies must submit and it requires that the data be submitted electronically and through the Internet.[128] Earlier, the EPA started trimming the expansive confidentiality options that have hampered its operations for so long.[129]

Requiring more information and making it available is a significant line of reform. Toxicity data makes chemicals "visible" in new ways. Disclosure of chemicals' risks opens up the conventional relationship between the agency and regulated firms, engaging the market and public opinion in responses to

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127. Auer et al., *supra* note 124, at 10243. The action plans summarize available hazard, exposure, and use information; outline the risks that each chemical may present; and identify specific steps the EPA is taking to address those concerns. *Id.* The authors point out that the EPA will rely on authority that it has not used before, TSCA §5(b)(4)(A)(i), which authorizes the agency to "'compile and keep current a list of chemical substances . . . which the Administrator finds . . . presents or may present an unreasonable risk of injury to health or the environment.'" *Id.* "The listing requires a rulemaking and a finding that a chemical 'presents or may present an unreasonable risk,' and EPA's announcement emphasizes the 'may present' arm of the findings." *Id.* at 10243–44.

128. On August 2, 2011, the EPA announced that it is increasing the type and amount of information it collects on commercial chemicals from chemical manufacturers. *EPA Publishes Rule to Improve Reporting of Chemical Information*, U.S. ENVTL. PROT. AGENCY (Aug. 2, 2011), http://yosemite.epa.gov/opa/admpress.nsf/0/346B93365E96C25E852578E000542B73. The improved chemical data reporting rule also requires that companies submit the information electronically to the EPA, rather than on paper, and limits the scope of allowable confidentiality claims. *Id.*

129. *See* News Release, EPA, EPA Removes Confidentiality Claims for More than 150 Chemicals / Part of Continuing Effort to Protect Americans' Health by Increasing Access to Chemical Information (June 8, 2011), *available at* http://yosemite.epa.gov/opa/admpress.nsf/bd4379a92ceceeac8525735900400c27/9f7964fcbca3824a852578a900574cea!OpenDocument. Kip Viscusi has suggested that disclosure may lead to better information through an "unraveling" process. If the best firm discloses, others may follow and continue until all the firms have disclosed risks or until a stable equilibrium is established, with some firms still withholding some information. Viscusi suggests that publicity of the conditions in the worst firms is likely to have the opposite effect. W. KIP VISCUSI, RISK BY CHOICE 71–75 (1983). He recommends disseminating information through government mechanisms that operate similarly to right-to-know laws but are amplified by public or independent rating of safety levels. Once measurements are accessible most firms will have incentives to disclose their health and safety performance and to compete for quality in this dimension. *Id.* at 85–87. Wendy Wagner has proposed a "divide and conquer" mechanism to stimulate interfirm rivalries to produce health and safety information. *See* Wendy Wagner, *Using Competition-Based Regulation to Bridge the Toxics Data Gap*, 83 IND. L.J. 629, 640–46 (2008) (arguing that regulation should provide a forum for a firm to show that its product is better in health and environmental effects than its competitors' products). *See* Stephen M. Johnson, *Competition*: *The Next Generation of Environmental Regulation?*, 18 SOUTHEASTERN ENVTL. L.J. 1, 1 (2009) (commenting on Professor Wagner's proposal).

pollution.[130] Information supports a wide variety of nonlegal initiatives including public dialogue on the core goals of chemicals regulation,[131] efforts to support a transition to "green chemistry,"[132] and research to understand and respond to businesses' practical concerns about how to implement environmental practices.[133] There may be a "tipping point," when big market players or firms that are positioned to take advantage of new opportunities in the market see that it is in their interest to improve their toxicity performance.[134] Other firms will then follow if they can.[135]

2. *Legislative Proposals and Debate*

The European Union's enactment of REACH has put pressure on industry to participate in discussions about amending TSCA. The EPA has announced the Obama Administration's TSCA reform principles,[136] and the American

130. *See* Paul R. Kleindorfer & Eric W. Orts, *Informational Regulation of Environmental Risks,* 18 RISK ANALYSIS (No. 2) 155 (1998); Peter S. Menell, *Structuring a Market-Oriented Federal Eco-Information Policy*, 54 MD. L. REV. 1435, 1462–66 (1995).

131. *See* Valerie J. Brown, *Are We on the Same Page? Action Agenda of the National Conversation on Public Health and Chemical Exposures*, 119 ENVTL. HEALTH PERSP, A 484, A 487 (2011), *available at* http://dx.doi.org/10.1289/ehp.119-a484.

132. *See Green Chemistry and Commerce News,* THE GREEN CHEMISTRY & COMMERCE COUNCIL, http://greenchemistryandcommerce.org/eblasts.php?eblastid=10#upcoming (last visited Mar. 1, 2012) (each issue of the newsletter provides current information about upcoming and ongoing GC3 activities, and news about green chemistry and design for environment).

133. *See, e.g.*, Caroline E. Scruggs & Leonard Ortolano, *Creating Safer Consumer Products: The Information Challenges Companies Face*, 14 ENVTL. SCI. & POL'Y 605, 605 (2011) (examining "challenges faced by 20 multinational consumer product companies in obtaining needed information about chemicals used as product inputs, and [describing their] strategies . . . to avoid or reduce hazardous chemicals in their products").

134. Walmart's recent public "green" conversion could have a wider effect on the chemicals market. *See* Rosabeth Moss Kanter, *Wal-Mart's Environmental Game-Changer*, BLOOMBERG L.P. (July 16, 2009, 3:47 PM), http://www.bloomberg.com/apps/Harvardbusiness?sid=H2c7e044be78a5e6d144f9b45ab085921 (reporting on Walmart's announcement of a new environmental labelling program disclosing to consumers the environmental costs of making products sold at Walmart). *See Sustainability Index*, WALMARTSTORES.COM, http://walmartstores.com/sustainability/9292.aspx (last visited Mar. 1, 2012) (explaining its Sustainability Index, "With this initiative, we are helping create a more transparent supply chain, accelerate the adoption of best practices and drive product innovation and ultimately providing our customers with information they need to assess products' sustainability."). More recently, Walmart announced it is banning a controversial flame retardant used in consumer goods, including child car seats, cameras and sofas, and has told manufacturers to provide safer products. *See Walmart Bans Chemicals Believed Harmful*, UNITED PRESS INT'L (Feb. 27, 2011), http://www.upi.com/Top_News/US/2011/02/27/Walmart-bans-chemicals-believed-harmful/UPI-24741298827160.

135. *See* William J. Walsh & Michelle M. Skjoldal, *Sustainability is Driving Toxic Chemicals from Products*, 25 NAT. RESOURCES & ENV'T, Winter 2011, at 16.

136. Lisa Jackson, Administrator, U.S. Envtl. Prot. Agency, Speech at the Commonwealth Club, San Francisco, California (Sept. 29, 2009). *Essential Principles for Reform of Chemicals Management Legislation,* EPA, http://www.epa.gov/oppt/existingchemicals/pubs/principles.html (last visited Mar. 1, 2012) (listing the core principles on the website).

Chemistry Council[137] and Safer Chemicals, Healthy Families, a coalition of environmental NGOs, have announced their own.[138] New versions of earlier TSCA reforms bills were introduced in the Congress in 2011[139] and some hearings have been held.[140]

Under the proposed Safe Chemicals Act of 2011, manufacturers would be required to submit a minimum data set to the EPA for each chemical they produce; the EPA would prioritize chemicals based on their hazard and exposure characteristics; a public database would present the information and confidentiality exceptions would be narrowed; the bill would establish grant programs and research centers to foster safe chemical alternatives.[141] The bill gives the EPA "full authority to request additional information . . . to determine the safety of a chemical. . . . [It] [c]alls on the EPA to categorize chemicals based on risk, and [to] focus resources on evaluating those most likely to cause harm. . . . [And it] [r]equires EPA to take fast action to reduce risk from chemicals that have already been proven dangerous."[142]

While it appears that there is consensus on some important issues, it is not clear how lasting any agreement might be.[143] Big issues are left open and industry is not united or particularly willing to compromise.[144] On some items,

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137. *10 Principles for Modernizing TSCA*, AMERICAN CHEMISTRY COUNCIL, 1, http://www.americanchemistry.com/Policy/Chemical-Safety/TSCA/10-Principles-for-Modernizing-TSCA.pdf (last visited Mar. 3, 2012).

138. *Legislative Update*, SAFER CHEMICALS, HEALTHY FAMILIES, http://www.saferchemicals.org/safe-chemicals-act/ (last visited June 14, 2012). *See* Richard A. Denison, *Ten Essential Elements in TSCA Reform*, 39 ENVTL. L. REP. 10020 (2009).

139. In the House, Representatives Bobby Rush and Henry Waxman introduced the Toxic Chemicals Safety Act of 2010 (H.R. 5820) (TCSA). H.R. 5820, 111th Cong. (2nd Sess. 2010). In the Senate Frank R. Lautenberg released the text of the Safe Chemicals Act of 2010 (S. 3209) (SCA). S. 3209, 111th Cong. (2nd Sess. 2010). Lynn L. Bergeson, Summary and Comparison of House and Senate TSCA Legislative Efforts—Environmental Regulation 2011: Managing in the Face of Rapid Change, 587 PLI/Real 179 (2011).

140. *Sen. Lautenberg Introduces "Safe Chemicals Act of 2011*," FRANK R. LAUTENBERG, http://lautenberg.senate.gov/newsroom/record.cfm?id=332785 (last visited Mar. 6, 2012).

141. *See Senate Bill Would Require Safety Testing for Chemicals*, 29 WESTLAW J. TOXIC TORTS, May 18, 2011, at 11.

142. Press Release, Senator Frank R. Lautenberg, Sen. Lautenberg Introduces "Safe Chemicals Act of 2011" (Apr. 14, 2011), http://lautenberg.senate.gov/newsroom/record.cfm?id=332785; *see also* Richard Denison, *TSCA Reform 2.0, aka, Safe Chemicals Act of 2011: Tastes Great, Less Filling*, ENVTL. DEF. FUND (Apr. 14, 2011), http://blogs.edf.org/nanotechnology/2011/04/14/tsca-reform-2-0-aka-safe-chemicals-act-of-2011-tastes-great-less-filling/.

143. See discussion in *TSCA Reform: The Standard of Safety—Dialogue*, 41 ENVTL. L. REP. 11081 (2011), particularly Richard Denison's summary of apparent consensus [hereinafter *TSCA Reform Dialogue*]. *See also* Leslie Carothers et al., *Key Issues for Reform of TSCA*—Dialogue, 41 ENVTL. L. REP. 10873 (2011) (four experts discuss the status of TSCA reform in the context of the EPA's recent administrative initiatives, state chemicals laws, technological innovations, and international chemicals programs); Schifano et al., *supra* note 41.

144. For instance, see the following statement from Mike Walls, the American Chemistry Council's (ACC) vice president of regulatory and technical affairs, on the EPA's new regulatory initiative under TSCA, sounding firmly rooted in the current stalemate:

> [W]e remain concerned that the EPA chemical action plan process does not include a transparent science-based approach to chemical assessment and chemical management. EPA has a responsi-

debate continues. For instance, under TSCA, there is no mandatory review of existing chemicals and the standard for review is unreasonable risk, with "unreasonable" generally interpreted to mean that the agency should consider costs and the availability of substitutes in determining whether a chemical is safe. The new bills would change these principles, but there is debate over what standard should replace "unreasonable risk."[145] Legislative action might not come soon, and the EPA may be left to soldier on under TSCA.[146] Yet pressure is building from the outside, as other nations and a growing number of states adapt their laws to perform better than current federal law.[147]

> bility to assess the actual risk of a chemical, review the weight of evidence of all scientific studies, and evaluate the specific uses of a chemical, as well as the availability, performance and safety of functional alternatives.

EPA's Chemical Action Plan Process Should Consider Critical Uses for HBCD, AMERICAN CHEMISTRY COUNCIL (Aug. 18, 2010), http://www.americanchemistry.com/Media/PressReleasesTranscripts/ACC-news-releases/EPAs-Chemical-Action-Plan-Process-Should-Consider-Critical-Uses-for-HBCD.html (internal quotation marks omitted). Mr. Walls' statement recites industry's favored "science wars" principles. For information about Mike Walls, see *Michael P. Walls*, AMERICAN CHEMISTRY COUNCIL, http://www.americanchemistry.com/Walls (last visited Mar. 3, 2012). *See also* Richard Denison*, ACC on Safe Chemicals Act of 2011: If You Can't Say Anything Nice . . .,* ENVTL. DEF. FUND (Apr. 15, 2011), http://blogs.edf.org/nanotechnology/2011/04/15/acc-on-safe-chemicals-act-of-2011-if-you-can%E2%80%99t-say-anything-nice-%E2%80%A6/ (reporting that the Consumer Specialty Products Association and DuPont "found some nice things to say" about the 2011 TSCA reform bill; the National Petrochemical & Refiners Association even "acknowledged some improvements"; but, the American Chemistry Council found nothing good to say about it.).

145. Current discussions are focused on the appropriateness of the "reasonable certainty of no harm" standard. This standard controls the EPA's setting of tolerances for "safe" pesticide residues in food under the federal Food, Drug and Cosmetics Act. 21 U.S.C. § 346a(b)(2)(A)(ii) (2006). The "reasonable certainty of no harm" standard was incorporated into the FDCA authority by the 1996 Food Quality Protection Act. Pub. L. No. 104-170, 110 Stat. 1489. The new definition of "safe" in the mandate includes considerations of aggregate risk from all routes of exposure and defines special populations, with explicit provisions about children and vulnerable populations. *Id.* at 21 U.S.C. § 346a(b)(2)(B)(vi); § 346a(b)(2)(C). The question of how to handle aggregate risks, cumulative risks and uncertainty factors are part of the larger debate. *See TSCA Reform Dialogue*, *supra* note 143, at 11081, 11085.

146. *See TSCA Reform Dialogue*, *supra* note 143, at 11085. James V. Aidala states:

> [W]hat happens if there's no TSCA reform over, say, a 10-year period? The smart money is always against amendments in general. Amendments are hard to get through. These proposals are 180-plus pages long; that's not exactly a small package of amendments. I truly don't have an answer to this, just leaving the idea out there that EPA may have to live on the current law—and how do we still meet some of these principles that all the various parties can agree to under current law?

Id. at 11085. Wendy Cleland-Hamnett, of the EPA, adds: "[I]f we don't have TSCA reform—or until we get TSCA reform, is the way I'd like to think of it—how do we improve our work under the current system and the current TSCA? That's something that we are working on very hard here at the Agency." *Id.* at 11088.

147. *See id.* at 11085–86. Richard Denison compares the current the U.S. legislative proposal to the European Union's Registration, Evaluation, Authorization, and Restriction of Chemicals (REACH) regulation and the Canadian Environmental Protection Act, because they are often reference points for the debate on TSCA reform. *See also* Lynn L. Bergeson, *Other Regulatory Developments: Federal and European Union Chemical Management Initiatives in 2010*, *in* ENVIRONMENTAL REGULATION AND COMMERCIAL IMPLICATIONS 2010: HOW THE NEW ADMINISTRATION, CONGRESS AND THE COURTS HAVE CHANGED THE RULES 185 (Philip E.

J. Clarence Davies has remarked that TSCA reform efforts may not have a great impact now, in light of the political climate and the agency's lack of resources to implement change.[148] On the other hand, Davies points out that getting it right is very important because the way we handle the environmental problems of the future will be shaped by TSCA reform. Environmental health concerns from chemicals, nanotechnology and the ecological problem of climate change are caused by the use of valuable products as much as from wastes emitted by facilities, the traditional target of environmental law. These problems are complex and international and require new kinds of risk assessment. TSCA has the advantage over most U.S. environmental laws in that it is product oriented and addresses multimedia exposures.[149]

IV. CHEMICAL EXPOSURE AS A BATTERY

In response to the complexity of toxicity, Cranor points to the simplest legal answer: liability for battery.[150] After surveying the range of current laws and alternatives,[151] he recommends that the distribution of suspect pollutants, without prior research on their toxicity, should be considered the intentional cause of an offensive contact. Cranor's focus on the offensive or "dignitary" version of the tort appropriately places the social meaning of exposures within the toxicity debate. The proposal would harness tort law to create incentives to research and reduce the data gap, harmonizing the industry's obligations with contemporary social, ethical, and legal expectations.[152]

Karmel & Edward L. Strohbehn, Jr. co-chairs, 2010) (surveying and comparing approaches to chemical regulation in the United States and in Europe); Allison Tracy, *New Ways in the Ancient World: Japan and China Advance Their Chemicals Policies,* ENVTL. DEFENSE FUND (Oct. 2, 2011), http://blogs.edf.org/nanotechnology/2011/10/05/new-ways-in-the-ancient-world-japan-and-china-advance-their-chemicals-policies/ (stating that Japan and China "have not allowed themselves to fall behind in chemicals management" and "are expanding their chemicals regulations."). *See generally* Todd Stedeford & Marek Banasik, *International Chemical Control Laws and the Future of Regulatory Testing for Risk Assessment*, 22 GEO. INT'L ENVTL. L. REV. 619 (2010) (describing chemical testing requirements and outlook in the United States, Japan, and the European Union); Richard Denison, *The States—We're in on Chemical Policy Reform in 2011: 30 and Counting*, ENVTL. DEF. FUND (Jan. 19, 2011), http://blogs.edf.org/nanotechnology/2011/01/19/the-states-were-in-on-chemical-policy-reform-in-2011-30-and-counting/ (legislators in 30 states and the District of Columbia introduced or announced plans to introduce bills aimed at reducing the impact of chemicals on public health; the bills differ in scope and content, but all of them address chemicals, products or policy needs that have fallen through the cracks in the 35 years since the Toxic Substances Control Act (TSCA) was enacted).

148. Carothers et al., *supra* note 143, at 10879–81. Davies notes that erosion in agency capacity and budgets has been going on for decades; now in "real-dollar terms, the EPA's budget is about one-half of what it was in the mid-1970s. . . . [and] OSHA's personnel level is about one-half of what it was in the 1980s." *Id.* at 10879–80.

149. *Id.* at 10879–81. Davies suggests that in the future, we will need to consider institutional reforms to address these developments. *Id.* at 10881.

150. CRANOR, LEGALLY POISONED, *supra* note 1, at 132–77.

151. *Id.* at 178–207.

152. Tort law has many practical virtues: it is flexible and can be both broad and precise. Common law courts are already working in tandem with regulatory agencies. *See generally* Wendy Wagner, *When All Else Fails: Regulating Risky Products Through Tort Litigation,* 95

Battery is the foundational tort cause of action. It protects bodily integrity and individual autonomy, creating the essential status and space for social interactions.[153] Indeed, proscribing harmful or offensive physical contacts is a structural prerequisite for a functional society. Is battery law a good fit for toxic exposures? Cranor is not the first scholar to propose battery as an appropriate rule for toxics exposure[154] or for pollution generally.[155] Several other

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GEO. L. J. 693 (2007) (identifying strengths of tort system in relation to regulation of toxic exposures); Thomas O. McGarity, *The Complementary Roles of Common Law Courts and Federal Agencies in Producing and Using Policy-Relevant Scientific Information*, 37 ENVTL. L. 1027, 1029 (2007) (suggesting "the potential for the cooperative sharing of scientific information between regulatory agencies and common law courts," using the regulatory history of PFOA as a case study); Mary L. Lyndon, *Tort Law and Technology*, 12 YALE J. ON REG. 137 (1995) (describing key role that courts play in managing technologies with environmental and health costs, producing and using information that is also valuable to administrative systems).

153. *See* Danuta Mendelson, *Historical Evolution and Modern Implications of Concepts of Consent To, and Refusal of, Medical Treatment in the Law of Trespass*, 17 J. LEGAL MED. 1, 9–12 (1996) (tracing history of trespass function from original protection of the peace to inclusion of individual right to personal integrity).

154. *See, e.g.*, Lynda Collins & Heather McLeod-Kilmurray, *Toxic Battery: A Tort for Our Time?*, 16 TORT L. REV. 131, 132 (2008) (exposing a person to any poorly understood chemical substance meets the definition of harmful or offensive; releasing a chemical that has yet to be characterized or about which so little is known that neither party can prove danger or safety is de facto experimentation, intrudes upon the exposed person's autonomy, and constitutes a harmful or offensive battery); Lisa Heinzerling & Cameron Powers Hoffman, *Tortious Toxics*, 26 WM. & MARY ENVTL. L. & POL'Y REV. 67 (2001) (discussing how the psychological and sociological literature on risk perception shows that the stress effects of enhanced risk support an autonomy- and dignitary-based tort for toxic exposures, whether or not the exposures have led to a physical illness); Kathy Seward Northern, *Battery and Beyond: A Tort Law Response to Environmental Racism*, 21 WM. & MARY ENVTL. L. & POL'Y REV. 485, 565–72 (1997) (relying in part on the risk perception literature to argue for the offensiveness of toxic contacts that are also part of a pattern of racial injustice). *See also* discussion *infra* and text accompanying notes 178–90. *See generally* Christopher J. McAuliffe, *Resurrecting an Old Cause of Action for a New Wrong: Battery as a Toxic Tort*, 20 B. C. ENVTL. AFF. L. REV. 265 (1993) (considering battery in contrast to other tort claims as a remedy for toxic exposure and harm); David B. Ezra, *Smokers Battery: An Antidote to Second-Hand Smoke*, 63 S. CAL. L. REV. 1061, 1063 (1990); Carl B. Meyer, *The Environmental Fate of Toxic Wastes, The Certainty of Harm, Toxic Torts, and Toxic Regulation*, 19 ENVTL. L. 321, 357–63 (1988) (arguing that exposure to toxic waste and other pollution caused by formaldehyde should be treated as a toxic battery); Irene Scharf, *Breathe Deeply: The Tort of Smokers' Battery*, 32 HOUS. L. REV. 615 (1995) (arguing that the manufacture and sale of tobacco products should be actionable under the law of battery).

155. In 1986, Professor David Slawson produced an ambitious and intriguing treatment of the legal options available to address the harms of air pollution and argued for battery and trespass as the best fit. W. David Slawson, *The Right to Protection from Air Pollution*, 59 S. CAL. L. REV. 672, 742 (1986). Slawson extensively described the effects of air pollution on the residents of the Los Angeles area, then proposed a legal strategy that would begin with a class of injured persons making the claim of battery. *Id.* at 762. A court could adjudicate the case by first invoking *Sindell*'s market share doctrine and then finding the State of California obligated to indemnify victims. *Id.* at 747–49. The State would then collect fines from pollution sources and set up a compensation mechanism, modeled on the one Japan established after the Minimata Bay methyl mercury disaster. *Id.* at 680, 794–95. Slawson's proposal is novel, but also pragmatic, harnessing relevant doctrine and available institutional capacity.

Another innovation has been offered by Professor Rodgers who has proposed that there be a "tort of contamination," "defined as interference with the use and enjoyment of ecosystem func-

scholars have also proposed the remedy of liability for chemical exposure without sufficient testing or disclosure.[156] Battery's straightforward response to unwarranted and unwelcome physical contacts is appealing in part because it captures ordinary human responses to pollution. Cranor's interest in the law of battery also springs from its clarity: battery is direct and intense. It is a strong prohibition that places the onus on the batterer: don't do it! Cranor's point is that in analogous settings the law takes essentially similar actions—both harmful and offensive—very seriously.

To prevail in a cause of action for battery, a plaintiff must prove that the defendant committed a voluntary act with the intent to cause a wrongful contact and the contact occurred.[157] The definition of intent includes both purpose-based intent and constructive intent, that is, "knowledge to a substantial

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tions." Rodgers, *supra* note 105, at 1259. While it seems similar to nuisance, Rogers suggests that freeing the pollution and contamination issues from "property" would be an advance because much of the damage and harm from toxic pollution is unrelated to property interests. *See id.* at 1259–61.

156. Others have proposed that liability should follow an exposure caused without prior testing. *See* Lin, *supra* note 7, at 955. Professor Lin describes the evidentiary dynamics of chemical toxicity and surveys the relevant legal literature, including the duty to test and the difficulties that plaintiffs face when seeking to enforce that duty. *Id.* at 963–67. Lin proposes that courts hold that the failure to test is itself a public nuisance because it puts at risk the public health and undermines the society's ability to protect itself. *Id.* at 972–94. Lin has also suggested an administrative mechanism for compensation for toxic exposures. *See* Lin, *supra* note 105 (proposing that major polluters would pay fines at the time pollutants are released, based upon the volume and expected costs in harm to victims, who would receive compensation from the fund formed by the levies on pollution).

See also Berger & Twerski, *supra* note 32, at 282–87 (proposing new cause of action for informed choice that would separate the right to existing information on drug toxicity from the burden of proof on harm causation); Alexandra B. Klass, *Pesticides, Children's Health Policy, and Common Law Tort Claims*, 7 MINN. J. L. SCI. & TECH. 89 (2005) (proposing that where plaintiffs can establish that the defendant failed to conduct reasonably available testing to gather scientific evidence on the issue of causation, the court should shift the burden of proof to the defendant); Thomas O. McGarity, *Proposal for Linking Culpability and Causation to Ensure Corporate Accountability for Toxic Risks*, 26 WM. & MARY ENVTL. L. & POL'Y REV. 1, 44–46, 53–61 (2001) (responding to an earlier proposal by Margaret Berger and outlining a combined administrative-litigative approach that would apply culpability-based assumptions to federal agency assessments of scientific evidence); Margaret A. Berger, *Eliminating General Causation: Notes Towards a New Theory of Justice and Toxic Torts*, 97 COLUM. L. REV. 2117 (1997) (proposing liability based upon defendant's failure "to develop and disclose information . . . needed to assess serious latent risks"). Among other proposals to address the problem are Elizabeth B. Forsyth, Note, *Solving Widespread Toxic Chemical Exposure: A Taxing Job*, 29 VA. ENVTL. L.J. 115, 117 (2011) (proposing a tax "on production of all chemicals relative to each chemical's risk to human health," with the revenues used "to offset the costs to the healthcare system from exposure to chemicals"); Christine H. Kim, Essay, *Piercing the Veil of Toxic Ignorance: Judicial Creation of Scientific Research*, 15 N.Y.U. ENVTL. L.J. 540, 542–43 (2007) (drawing on toxic tort and related literature and proposing that courts require independent research as part of damages in the context of class action litigation). See *supra* note 38 and accompanying text for a discussion of testing program proposals.

157. RESTATEMENT (SECOND) OF TORTS §§ 13, 18.

certainty."[158] The defendant need not have intended that the contact be harmful or offensive; the intent pertains to the contact itself, not its effects.[159] The plaintiff must not have consented to the contact; the recipient of a contact is entitled to choose it with full knowledge of its effects.[160]

Common law claims based upon pollution have not generally been handled under the law of battery. The prospect of developing a battery action for toxic pollution is promising, but it presents issues that cannot be fully considered here.[161] Instead, two ideas will be developed briefly: (1) the legal prob-

158. RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL AND EMOTIONAL HARM § 1 (2010) provides: "[a] person acts with the intent to produce a consequence if: (a) the person acts with the purpose of producing that consequence; or (b) the person acts knowing that the consequence is substantially certain to result." The new Restatement articulates an umbrella rule for intent, meant to be understood in light of the continuing validity of the Second Restatement's substantive provisions. However, Professor Bublick has noted there is an imperfect fit between the Restatement's umbrella rule and the trespassory torts and this will require choices about the development of the relevant torts. Ellen M. Bublick, *A Restatement (Third) of Torts: Liability for Intentional Harm to Persons—Thoughts*, WAKE FOREST L. REV. 1335 (2009). Bublick suggests that reconfiguration of these torts may further extend them, as torts that protect against emotional harms play an important role in defining cultural norms. *Id.* at 1344–45.

These interpretive processes will be important to the development of Cranor's proposal. For example, the new Comment e seems to suggest a limitation on the "substantial certainty" prong of intent that would consider the distance of the plaintiff and the vagueness of the plaintiff's identity from the vantage point of the defendant. Clearly, if this were to become a formal limitation, most chemical exposures would be excluded. However, perhaps more functional considerations would fit better. For instance, Professors Goldberg and Zipursky refer to medical monitoring cases as "recognizing that those who create significant risks of injury have a duty to help mitigate them." John C.P. Goldberg & Benjamin C. Zipursky in *Unrealized Torts*, 88 VA L. REV. 1625, 1718 (2002). Medical monitoring may be an appropriate analogy for the refusal to test in the present context. Similarly, information costs and resources might be useful in devising appropriate explanations of intent in this setting. *See* Henry E. Smith, *Modularity and Morality in the Law of Torts*, 4 J. TORT L. 1 (2011); Wendy E. Wagner, *What's It All About, Cardozo?,* 80 TEX. L. REV. 1577 (2002); *see also* Joseph H. King, *The Torts Restatement's Inchoate Definition of Intent for Battery, and Reflections on the Province of Restatements*, 38 PEPP. L. REV. 623 (2011); Anthony J. Sebok, *Purpose, Belief, and Recklessness: Pruning the* Restatement (Third)*'s Definition of Intent,* 54 VAND. L. REV. 1165 (2001); Kenneth W. Simons, *A Restatement (Third) of Intentional Torts*, 48 ARIZ. L. REV. 1061 (2006).

159. RESTATEMENT (SECOND) OF TORTS § 1613 cmt. c (1965); *id.* § 18(2) (stressing that the section does not endorse negligent infliction of offense).

160. *Id.* at § 13 cmt. d. (absence of consent must be proved by plaintiff as part of case).

161. There is an energetic debate on the nature and function of tort law, including its role in public policy. *See generally* Thomas C. Galligan, Jr., *Deterrence: The Legitimate Function of the Public Tort*, 58 WASH. & LEE L. REV. 1019 (2001) (explaining bases and functions of the public uses of tort law); John C.P. Goldberg & Jonathan Zipursky, *Torts As Wrongs*, 88 TEX. L. REV. 917 (2010) (explaining essentials of civil recourse theory of tort law grounded in private law concepts of duty rather than a public law of incentives, accidents, and social costs); F. Patrick Hubbard, *The Nature and Impact of the "Tort Reform" Movement*, 35 HOFSTRA L. REV. 437 (2006) (describing shift from judicial development of doctrine based on common law reasoning to legislative changes and the role of politics, money, and rhetoric in the struggle over the proper role of tort liability); Michael L. Rustad, *Torts as Public Wrongs*, 38 PEPP. L. REV. 433 (2011) (surveying current tort development and critiquing scholarship that advocates narrower tort focus on the relations between the plaintiff and the defendant rather than public or social concerns); Smith, *supra* note 158, at 1 (stating that tort law's heavy reliance on simple moral norms, which are easy to communicate and self-enforce, is partially explained in terms of information costs). On toxics cases, specifically,

lems that spring from the lack of toxicity data should be understood as procedural and evidentiary issues that are within the courts' ability to manage, and (2) there are good reasons to treat the untested release of suspect chemicals as offensive and actionable. Before considering these points, some threshold concerns are pointed out.

1. *Three Threshold Issues*

First, because battery is one of the "dignitary" torts and protects not only autonomy, but also personal dignity, the offensive battery is often grouped with the "emotional" torts. These are often the site of contention and ambiguity. Debates about the appropriate extent of recovery for emotional and future injury and enhanced risk delve deeply into the nature and role of tort law.[162] In any event, the offensive battery of untested toxic exposure is not only concerned with injury to feelings, but also serves the core tort functions of protecting individuals' health and autonomy and preserving the community's peace and welfare.[163]

Second, battery has most often been applied to individualized fact patterns, usually where there has been direct personal contact between the parties. Applying battery on a large scale in a setting of general indeterminacy of parties—both plaintiffs and defendants—seems to require a leap. Indeed, a likely response to Cranor's proposal will be that "there must be some limits."[164] Yet as injuries scale up, so can tort law. Although individualized narratives can help us parse an appropriate doctrinal response to a problem, when the stories

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see Jeb Barnes, *In Defense of Asbestos Tort Litigation: Rethinking Legal Process Analysis in a World of Uncertainty, Second Bests, and Shared Policy-Making Responsibility*, 34 LAW & SOC. INQUIRY 5 (2009) (considering whether courts should address complex policy issues or defer to other forums; the decision to use common law adjudication to address the asbestos public health problem now seems a reasonable use of judicial power).

162. *See generally* Robert L. Rabin, *Emotional Distress in Tort Law: Themes of Constraint*, 44 WAKE FOREST L. REV. 1197, 1198 (2009) (commenting on the Restatement (Third) of Torts and limitations on recovery for emotional distress as both instrumental and reinforcement of social norms); Goldberg & Zipursky, *supra* note 158, at 1625 (considering implications for tort law and appropriate responses to tort suits brought by plaintiffs who seek compensation for exposures that enhance risk, though they have not suffered any bodily harm or symptoms); Heinzerling & Hoffman, *supra* note 154 (relying on the psychological and sociological literature on risk perception to propose the development of an autonomy- and dignitary-based tort for toxic exposures, whether or not the exposures have led to a physical illness).

163. The offensive battery is functionally tied to harmful battery, protecting autonomy, physical safety and the public peace. *See* Mendelson, *supra* note 153 (describing the evolution of these functions of the tort of battery). The term *offense* and its more narrow applications should not distract us from the fact that offense may be a proxy for a physical harm.

164. *But cf.* Richard L. Abel, *Judges Write the Darndest Things: Judicial Mystification of Limitations on Tort Liability*, 80 TEX. L. REV. 1547, 1549 (2002) (considering unpersuasive reasons given for limiting liability). Professor Wagner examines Abel's analysis and suggests information advantages as a factor courts should consider in this context. Wagner, *supra* note 158.

have been large, the courts have accepted the challenge. In the face of large scale exposures and inadequately researched causal dynamics, insisting on the most ancient forms of the law will result in paralysis. Delay in confronting ongoing injuries may increase their costs. This certainly seems the likely result of delay in this context.

Third, while the offensive battery proposal seems expansive, its duty and breach elements are defined very narrowly and are grounded in established legal requirements. Potential defendants would only have to do something that the law expects anyway and is normal practice in closely related commercial settings: they would have to test their products for suspected risks. The stakes for defendants are not nearly as high as they were, for instance, in the DES cases. Nor would we be looking back in time, asking firms to pay for the losses caused by firms that are now gone. To avoid liability, firms would simply perform and disclose basic testing.

2. *Evidence of Chemicals' Health Effects Is Unfairly Blocked*

Essentially, the contacts we are discussing—exposures to suspected toxicants—are *both harmful and offensive.* Battery law should apply here in its basic form—the proscription against harmful contacts. Pollution qualifies as a harmful contact, as it meets the black letter law definition of harmful battery.[165] We spend a great deal of money and effort trying to reduce pollution and its effects. We know it is harmful; we don't like it; we just haven't thought it was *that* harmful. The significant message of *Legally Poisoned* is that at least some kinds of pollutants appear to be much more harmful than we have supposed.[166]

In toxic exposure cases, the legal system usually has required plaintiffs to prove specific harms from particular pollutants or waste streams. However, the precise nature of the plaintiff's burden is a process question: the law defines the specificity of evidence required and allocates the burden of persuasion.

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165. RESTATEMENT (SECOND) OF TORTS § 15 cmt. a. "Bodily harm is the physical impairment of another's body or physical pain or illness." Comment a states that "[t]here is an impairment of the physical condition of another's body if the structure or function of any part of the body is altered to any extent even though the alteration causes no bodily harm. A contact which causes no bodily harm may be actionable as a violation of a right to freedom form intentional infliction of offensive bodily contacts."

166. For a useful framework for understanding "harm" in this setting, see Albert C. Lin, *The Unifying Role of Harm in Environmental Law,* 2006 WIS. L. REV. 897 (2006). Professor Lin suggests that the harm principle can be seen as "jurisdictional," that is, as a way of identifying a class of conduct that society has the power to regulate. He proposes a definition of harm as "a setback to human interests that community norms have deemed to be significant." *Id.* at 901–02. This is helpful in environmental law, an expansive and dynamic field that touches on so many aspects of life. Toxic exposures, however, are harmful in the more conventional sense of the term as physical intrusions bringing risky and unwanted changes to the body. Lin explores both the unrealized harms of subcellular damage and risk and also the emotional harms due to exposure. *Id.* at 944–68.

The usual allocation in tort law assumes that available evidence can be brought to bear on the issues. Here unusual circumstances, including information and resource asymmetries, put firms in a position to withhold test data and then to argue that no action should be taken, since no harm has been proven. Firms have an unfair but virtually perpetual procedural advantage.

The industry's refusal to perform basic research on suspect chemicals' effects shifts the burden of uncertainty to those with no capacity to bear it; it is "total risk bearing," to use Guido Calabresi's term.[167] Discussing the placement of responsibility for the initial risk of harm from the behavior of others, Calabresi has observed,

> Of course, if victims were chosen to bear *all* risk initially, total atomism would be possible. Such a starting point—essentially a might makes right entitlement—is not *logically* impossible. But no atomistic *laissez-faire* society can, in fact, tolerate it. Total risk bearing by victims would neither avoid injuries cheaply (be efficient) nor result in an acceptable wealth distribution (be fair). At its extreme it would entail no property or bodily integrity.[168]

In Hohfeld's terms, the current system ostensibly provides chemical manufacturers with a power to expose with the correlative of liability.[169] However, in practice it is a power without liability, as the lack of testing and transparency prevents the development of evidence. Liability is suppressed and the entitlement becomes a simple power to expose. With this allocation of evidentiary burdens, the law effectively leaves out of its purview the harms that are caused by chronic exposures, leaving them to be characterized as acceptable risks or uncertain possibilities.[170]

Surely tort law does not contemplate giving a pass to the creation of mass harmful or risky contacts, just because their effects are latent and proving harm after the fact is complex and costly. Instead, numerous legal and ethical principles contemplate that purveyors of suspect substances will test them before exposing others. The common law supports a requirement of testing to

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167. Guido Calabresi, *Torts—The Law of the Mixed Society*, 56 TEX. L. REV. 519, 525 (1978).

168. *Id.*

169. *See generally* Wesley Newcomb Hohfeld, *Fundamental Legal Conceptions as Applied in Judicial Reasoning,* 26 YALE L.J. 710 (1917) (articulating basic jurisprudential architecture of tort law).

170. *See generally* Dayna Nadine Scott, *Confronting Chronic Pollution: A Socio-Legal Analysis of Risk and Precaution*, 46 OSGOODE HALL L.J. 293 (2008) (prevailing regulatory approach misses pollution harms that are caused by continuous, low-dose exposures that are within legally allowed limits). Scott discusses evidence and scholarship concerning the level of environmental health harm that is inherent in contemporary industrial production, a fixed feature of modern economies. *Id.* at 317–19; *see also* Watnick, *supra* note 77, arguing that the true goal of toxics regulation is to regulate toxins so that they present politically acceptable risk, using terminology and standards that imply safety. Watnick examines the assumptions of risk assessment, showing the method's limited goals and inability to consider many exposures such as EDs. *Id.* at 1323–24.

provide a basis for warning[171] and for market choices.[172] A duty to test products has been a theme in tort law.[173] Information access is an important part of public participation in environmental management today.[174]

Cranor's proposal to require testing or suffer liability for offensive battery is functionally related to *res ipsa loquitur,* alternative liability and market share liability. In *Ybarra v. Spangard*,[175] *Summers v. Tice*,[176] and *Sindell*,[177] the courts adjusted plaintiffs' burdens in recognition of the structural evidentiary problems. In those cases there was an elevated likelihood that the plaintiffs were not the cause of the injury and the defendants were positioned to have greater access to evidence than were the plaintiffs. Toxic exposures leave

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171. Risk communication is a strong requirement in the common law. Negligence law imposes a duty to act with reasonable care with respect to third parties. *See* RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL AND EMOTIONAL HARM § 7 (2005) ("An actor ordinarily has a duty to exercise reasonable care when the actor's conduct creates a risk of physical harm."). *Id.* at § 12 (noting an actor's skills or knowledge are "to be taken into account in determining whether the actor has behaved as a reasonably careful person"). There is a duty to warn those who may be affected by one's actions. *Id.* at § 18. Related information entitlements include warranty, fraud, and the law of informed consent. Warranty law requires that information about latent risks be transmitted to buyers. Carl J. Pernicone & Catherine Chen, *Express and Implied Warranties*, 9 N.Y. PRAC. SERIES, ENVIRONMENTAL LAW AND REGULATION IN NEW YORK § 2:9 (2011) (breach of implied warranty is closely related to the "duty to warn" cause of action in negligence and strict product liability). *See, e.g.*, RESTATEMENT (FIRST) OF TORTS § 529 cmt. a (pointing out that a statement containing a half-truth may be as misleading as a statement wholly false; the recipient of the statement is entitled to know the undisclosed facts in so far as they are material).

172. The costs of uncertainty are substantial. For a discussion of the costs of ignorance in this setting, including those identified by George Akerlof, see, e.g., Lyndon, *Information Economics*, *supra* note 7, at 1814–15 (citing George Akerlof, *The Market for "Lemons": Quality Uncertainty and the Market Mechanism*, 84 Q. J. ECON. 488 (1970) (discussing implications of buyers' limited ability to judge the quality of products of varying grades)). The invisibility of chemical toxicity undermines the entire market for chemicals; buyers' inability to screen products removes any incentive for manufacturers to differentiate between toxic and nontoxic products and to screen before production, resulting in a higher overall level of toxicity in products than would result if toxicity were a visible characteristic. Chemical products with lower toxicity are penalized by the presence in the market of some unknown number of toxics. Moreover, so long as the information market remains undeveloped, ignorance of toxicity may be an advantage to a product. New or unstudied chemicals will do better in relation to chemicals that have been shown to have some indication of toxicity. Ignorance will tend to prevail. *Id.*

173. *See* discussion *supra* Part I; *supra* notes 35–40 and accompanying text. *See also* Lin, *supra* note 7, at 963–66 (describing the underpinnings of tort law's duty to test and the ways in which the law is undermined by economic disincentives and enforcement difficulties).

174. *See* Amy Kapczynski, *The Access to Knowledge Mobilization and the New Politics of Intellectual Property*, 117 YALE L.J. 804, 806 (2008); Lea Bishop Shaver, *Defining and Measuring A2K: A Blueprint for an Index of Access to Knowledge,* 4 I/S: J.L. & POL'Y FOR INFO. SOC'Y 235, 237 (2008); Allen L. White, *Why We Need Global Standards for Corporate Disclosure*, 69 LAW & CONTEMP. PROBS., Summer 2006, at 167 (describing development of information function in EHS risk management).

175. Ybarra v. Spangard, 154 P.2d 687, 689–91 (Cal. 1944).

176. Summers v. Tice, 199 P.2d 1, 4 (Cal. 1948).

177. Hymowitz v. Eli Lilly & Co., 539 N.E.2d 1069, 1082 (1989); Sindell v. Abbott Labs, 607 P.2d 924, 936–37 (1980).

plaintiffs unable to prove their injuries, while the means of evaluating exposures are controlled by chemical producers. This is not just a lucky accident for the defendants' side. Their chokehold on the evidence flies in the face of the compelling reasons that they should test, reasons recognized by tort law and in many regulatory contexts.

3. *The Offensiveness of Untested Chemical Exposures*

In battery law, the test for offensiveness is a community standard. The Restatement (Second) of Torts holds that a bodily contact is offensive "if it offends a reasonable sense of personal dignity" and is "one which would offend the ordinary person." Also, it must be "a contact which is unwarranted by the social usages prevalent at the time and place at which it is inflicted."[178] Prosser's hornbook states, "Absent expression to the contrary, consent is assumed to all those ordinary contacts which are customary and reasonably necessary to the common intercourse of life. . . ."[179] May intentionally caused contacts with untested but suspected toxicants be deemed "ordinary contacts"? Cranor persuades readers that they may not. He argues that such contacts violate formal legal and ethical norms. In addition, it seems clear that they violate the standards and expectations of ordinary people today.

Cranor's chief formal arguments for offensiveness are based the ethics of research. He notes that the market for chemicals and the laws that enable it constitute what is, in effect, an enormous experiment that tests chemicals on the general population without their understanding or consent. Marshall Shapo has written extensively about this phenomenon.[180] As a system, the modern

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178. RESTATEMENT (SECOND) OF TORTS § 19 cmt. a, illus. 3 (1965). A bodily contact is offensive if it offends a reasonable sense of personal dignity. Comment a. adds, "In order that a contact be offensive to a reasonable sense of personal dignity, it must be one which would offend the ordinary person and as such one not unduly sensitive as to his personal dignity. It must, therefore, be a contact which is unwarranted by the social usages prevalent at the time and place at which it is inflicted." Illustration 3 provides this example: A, who is suffering from a contagious skin disease, touches B's hands, thus putting B in reasonable apprehension of contagion. This is an offensive touching of B.

179. PROSSER AND KEETON ON THE LAW OF TORTS 42 (W. Page Keeton et al. eds., 5th ed. 1984). Examples are given of these ordinary contacts: "a tap on the shoulder to attract attention, a friendly grasp of the arm, or a casual jostling to make a passage."

180. *See* MARSHALL S. SHAPO, A NATION OF GUINEA PIGS (1979); MARSHALL S. SHAPO, EXPERIMENTING WITH THE CONSUMER: THE MASS TESTING OF RISKY PRODUCTS ON THE AMERICAN PUBLIC (2009). The books provide a detailed history and analysis, including the more notorious cases of pharmaceutical and related trials and marketing without sufficient prior medical research. This predicament has long been understood. Commenting on an earlier and stricter proposed version of TSCA, the EPA's Deputy Administrator, John R. Quarles, Jr., in testimony before a House Subcommittee, declared:

> [T]he Nation's population and environment provide testing grounds for determining the effects a toxic substance has on human or environmental health. The authority contemplated by the Toxic Substances Control Act would establish requirements for testing substances believed to pose an unreasonable risk before they are dispersed by various means throughout the environment and are difficult, if not impossible, to control.

commercial chemical enterprise relies upon practices that violate the norms of medical research.[181] Cranor cites as precedent the case of *Mink v. University of Chicago*, in which doctors at the University of Chicago administered DES to women without revealing that they were part of an experiment.[182] The court found that the defendants had committed an offensive battery. Are there meaningful distinctions between medical experimentation and commercial distribution of untested suspect chemicals? The latter is conducted on a much larger scale and with less apparent interest in the health outcomes.

Research using children is especially problematic. Cranor invokes children—their vulnerability and their entitlement to protection—in support of creating a stronger legal regime.[183] At the very least it is offensive, Cranor

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Toxic Substances Control Act: Hearings on H.R. 7229, H.R. 7548, and H.R. 7664 Before the Subcomm. on Consumer Protection and Finance of the Comm. on Interstate and Foreign Commerce, 94th Cong., 1st Sess. 213 (1975) (statement of John R. Quarles, Jr., Deputy Administrator, EPA). See Collins & McLeod-Kilmurray, *supra* note 154, at 134–37, for a consideration of the importance of autonomy and medical research norms and their relevance to toxic battery. *See also* Watnick, *supra* note 49, at 814–22 (noting that in light of failure to fully implement the FQPA, regulation must become more precautionary and the public should be educated about the dangers that pesticides pose to humans and particularly to children).

181. CRANOR, LEGALLY POISONED, *supra* note 1, at 180–82. Full discussion of this point is beyond the scope of this review, but a sampling of articles addressing the ethical issues in medical research suggests that some of the controversies in that field may be instructive in the present context. *See generally* Robert Gatter, *Walking the Talk of Trust in Human Subjects Research: The Challenge of Regulating Financial Conflicts of Interest*, 52 EMORY L.J. 327 (2003) (medical research financial conflicts of interest are subject to scrutiny that may be inadequate); Barbara A. Noah, *Bioethical Malpractice: Risk and Responsibility in Human Research*, 7 J. HEALTH CARE L. 7 POL'Y 175 (2004) (proposing that as increasing research volumes leads to pressures on institutional review boards and more injuries, expanded liability may be appropriate and serve as catalyst for reform); Lars Noah, *Coerced Participation in Clinical Trials: Conscripting Human Research Subjects*, 62 ADMIN. L. REV. 329 (2010) (development of pressures and quid pro quo systems to induce participation in medical trials); L. Song Richardson, *When Human Experimentation is Criminal,* 99 J. CRIM. L. & CRIMINOLOGY 89, 101–7 (2009) (researchers' status and the perceived social benefits of their research insulate them from penalties that would otherwise penalize actions that violate the bodily integrity and autonomy of research subjects); Richard S. Saver, *Medical Research and Intangible Harm*, 74 U. CIN. L. REV. 941 (2006) (discussing if greater recognition should be given to intangible harms of research subjects, including effects on personal dignity, opportunistic conduct in research situation, and abandonment of the research and its emotional effects).

182. Mink v. Univ. of Chicago et al, 460 F. Supp. 713 (N.D. Illinois, 1978). In *Mink* the court found that the administration of DES to the plaintiffs as part of a planned experiment conducted by the defendants was clearly intentional. The plaintiffs needed to show only the intent to bring about the contact; intent to harm was not required. The act of administering the drug supplied the contact with the plaintiffs' persons. The court found that "the administration of a drug without the patient's knowledge comports with the meaning of offensive contact."*Id.* at 717–18. *See generally* Tania Cruz & Eric K. Yamamoto, *A Tribute to Patsy Takemoto Mink*, 4 ASIAN-PAC. L. & POL'Y J. 569 (2003) (recounting the life of Patsy Mink, the first woman of color in Congress, who in 1951 had unknowingly been part of the DES experiment and later channeled her anguish over possible harm to her daughter into the class action suit against the University of Chicago and the Eli Lilly Company).

183. CRANOR, LEGALLY POISONED, *supra* note 1, at 186–90.

argues, to subject children and future generations to unknown risks. Cranor assesses the chemical exposure phenomenon in light of the ethical principles on research that emerged from the Nuremberg trials.[184]

What about offensiveness as a personal experience? If it is tortious to seriously insult or startle someone, it would seem an even greater transgression of social boundaries to inflict a silent, invisible bodily contact with a possible toxicant, leaving those "touched" to await whatever may come. The common law invokes the actual lived experience of toxic exposure as a source of governance, adding substance to the flat numerical risk estimates that regulatory experts produce. Expression of emotional, psychological and moral responses to risks and harms are foreign to official risk management. EHS regulation establishes a dimension of instrumental analysis that dispenses with or sublimates ordinary human values.[185] Hazard, risk, uncertainty, statistical lives, monetization of death, injury and natural resource damage are useful conceptual tools. Yet some of these same words, when used in ordinary language, carry substantial emotional weight.[186] As technical concepts they are func-

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184. *Id*. at 180–82. *See also* Larry I. Palmer, *Paying for Suffering: The Problem of Human Experimentation*, 56 MD. L. REV. 604, 623 (1997) (rejecting the premise that liability is sufficient response to medical research abuses in the context of the Tuskegee study and human radiation experiments).

185. *See generally* Christopher H. Schroeder, *In the Regulation of Manmade Carcinogens, if Feasibility is the Answer, What is the Question?*, 88 MICH. L. REV. 1483 (1990) (reviewing FRANK B. CROSS, ENVIRONMENTALLY INDUCED CANCER AND THE LAW) (considering technological feasibility and competing frameworks for toxics regulation). Of the modern administrative state, Schroeder suggests that "[t]he crucial move here is to divide policymaking into two distinct parts, the value-laden and the value-free, and subsequently to treat the first as a 'political' and the second as a 'scientific' activity. . . . This strategy proves to be incoherent." *Id.* at 1491; *see also* LANGDON WINNER, THE WHALE AND THE REACTOR: A SEARCH FOR LIMITS IN AN AGE OF HIGH TECHNOLOGY, 142–54 (1986) (discussing the emergence of risk assessment as a technocratic discourse and its separation from the larger culture).

186. There is a growing literature on emotions and cognitive psychology and the law. This work is relevant to the subject of this review, but adequate consideration of its implications is not possible in this review. *See, e.g.*, Dan M. Kahan et al., *Who Fears the HPV Vaccine, Who Doesn't, and Why? An Experimental Study of the Mechanisms of Cultural Cognition*, 34 LAW & HUM. BEHAV. 501 (2010) (applying cultural cognition thesis that holds that individuals form risk perceptions that reflect their commitments to contested views of the good society); Molly J. Walker Wilson, *Adaptive Responses to Risk and the Irrationally Emotional Public*, 54 ST. LOUIS U. L.J. 1297, 1298–1303 (2010) (social science research shows affect-based responses are properly viewed as expressions of values and preferences, providing a legitimate basis for risk policy that should be expanded through greater solicitation of public views of risks); Molly J. Walker Wilson, *Cultural Understandings of Risk and the Tyranny of the Experts*, 90 OR. L. REV. 113 (2011). *See generally* Susan A. Bandes, *Emotions, Values and the Construction of Risk*, 156 U. PA. L. REV. PENNUMBRA 421 (2008) (discussing emotion and reasoning), *available at* http://pennumbra.com/responses/03-2008/Bandes.pdf; Susan A. Bandes, *Foreword to Symposium on Emotions in Context: Exploring the Interaction Between Emotions and Legal Institutions*, 33 VT. L. REV. 387 (2009) (explaining that the goal of the symposium is to inquire how emotion interacts with legal reason); Susan A. Bandes, *Repellent Crimes and Rational Deliberation: Emotion and the Death Penalty*, 33 VT. L. REV. 489 (2009) (contributing to the symposium a discussion of misconceptions about emotion and rationality); Dan M. Kahan, *Two Conceptions of Emotion in Risk Regulation*, 156 U. PENN. L. REV. 741 (2008) (discussing the role emotions can and should

tional, but they can distract or even mislead. Statistical lives represent real lives.[187] Choices made without full information still risk harm to others.[188]

Lay responses to chemical exposures are an ordinary part of that social life. The person who is concerned about exposure may react with anxiety, fear, and anger, dismay and helplessness. Lisa Heinzerling and Cameron Powers Hamilton have drawn on sociological literature that studies how ordinary people experience the prospect of dangers, and they report that substantial individual and community damage results.[189] This research shows that experts and lay people perceive risks differently, with lay risk perceptions and attitudes characterized by greater stress and fear when the risk is associated with "dread risk factors," including that the risk is not controllable, is not equitable, may be catastrophic, poses a high risk to future generations, is not easily reduced, increases over time and is involuntary.[190] These factors are all present in low-level exposures to suspected toxicants.

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play in law and policy decision making); Govind C. Persad, *Risk, Everyday Intuitions, and the Institutional Value of Tort Law*, 62 STAN. L. REV. 1445 (May 2010) (comparing tort versus non-tort approaches to mass harm); Michael Stocker, *Some Questions about Emotions and Risk Evaluation*, 156 U. PA. L. REV. PENNUMBRA 412, 415–16 (2008) (assessing and building upon Professor Kahan's positions on cultural cognition and related aspects of emotions and risk); Cass R. Sunstein, *Some Effects of Moral Indignation on Law*, 33 VT. L. REV. 405, 416–17 (2009) (exploring the cognitive and emotional bases of indignation and related feelings that provide moral foundation for legal processes and outcomes).

187. Liza Heinzerling, *The Rights of Statistical People*, 24 HARV. ENVTL. L. REV. 189, 189–90 (2000) (stating the "basic kind of right—the right to be protected from physical harm caused by other people . . . is denied to those whose lives are framed in statistical terms").

188. *See generally*, Liza Heinzerling, *Knowing Killing and Environmental Law*, 14 N.Y.U. ENVTL. L. J. 521 (2006) (moral commitment against knowing killing should play a role in decisions about environmental problems, including cost-benefit analysis). *But see* James A. Hobbs, *Pollution and the Doctrine of Double Effect: A Reply to Heinzerling*, 15 N.Y.U. ENVTL. L. J. 362, 365 (2007) (disputing validity of Heinzerling's argument).

189. *See generally* Heinzerling & Hoffman, *supra* note 154 (relying on the psychological and sociological literature on risk perception and stress to propose the development of an autonomy- and dignitary-based tort for toxic exposures, whether or not the exposures have led to a physical illness). Similarly, discussing pharmaceutical exposures and tort law, including the scope of the "dignitary" harms, Margaret Berger and Aaron Twerski describe the anguish of choice deprivation suffered by Bendectin mothers and others who were not entitled to information about the risks of birth defects, unless they could prove the lack of it made them choose this procedure and that it caused their harms. Berger & Twerski, *supra* note 32, at 282–87 (proposing new cause of action for informed choice that would separate the right to existing information on drug toxicity from the burden of proof on harm causation).

190. *See* Paul Slovic, *Perception of Risk*, 236 Science, Apr. 17, 1987, at 283–84 (experts and lay people assess risks differently, with lay assessments reflecting legitimate concerns that are typically omitted from expert risks assessments). Experts' perceptions are not correlated with these factors. *Id.* at 283; *see also* Robin Gregory & Robert Mendelsohn, *Perceived Risk, Dread and Benefits,* 13 RISK ANALYSIS 259, 262–63 (1993) (in research following earlier work by Slovic and others, high dread ratings were associated with greater impacts on future generations).

The risk perception literature presents a framework for identifying the characteristics of pollution that are offensive to people. It is clear that most people want to be able to respond effectively to risks and they reject actions that disable them from protecting and caring for their children and grandchildren.[191] Exposures that violate these normal expectations are offensive in the common law sense of the term.

Distribution of suspected toxic chemicals has proceeded to date as if it were an ordinary commercial behavior, subject to the usual legal constraints on market participation. In fact, however, there have been very few constraints. The full range of the environmental and normative shortcomings of the current system adds up to a basis for mobilizing tort doctrine to address this problem. Carl Cranor's proposal would require an appropriate level of accountability for chemical producers. This accountability is what the law already requires for pharmaceuticals and pesticides and what the European Union is now imposing on all commercial chemicals in its market. Recognizing a common law obligation to test for toxicity would normalize the duties of U.S. chemical manufacturers.

Exposure to toxicants is pervasive today. Indeed, as Professor Rodgers has pointed out, contamination is now the norm.[192] Our expectation that low levels of pollution will not affect us is undermined by the research that Cranor outlines. Fazal Khan has written of epigenetic risks that they, "generally implicate the underlying fairness and justice of our social contract."[193] In Chapter 6 of *Legally Poisoned*, Cranor asks, what kind of world do we want to create? Numerous environmental scholars have written on pollution as a violation of

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191. Berger & Twersky, *supra* note 32, at 257–58, quote from the deposition of Elizabeth Mekdeci, the first plaintiff to bring an action against Merrell Dow claiming that the Bendectin she had taken in the first trimester of pregnancy caused her child to be born with limb reduction:

> I feel like there were certainly enough [adverse reactions of limb reduction in children born after their mothers had taken Bendectin to alleviate symptoms of nausea] reported, given our bad reporting system . . . to have warranted some kind of acknowledgment of this on the labeling and to physicians. I think I should have had the choice to make up my mind whether I wanted to take this drug based on the fact of what you had in your files and what the FDA had. . . . [I]t's not fair to you to have this knowledge, whether or not you have established in your minds this causal relationship, and not share it with the medical community and with the public who is going to be consuming this stuff.

Mekdeci Dep. at 568–70, Mekdeci v. Merrell Nat'l Labs., Div. of Richardson-Merrell, Inc. (M.D. Fla. filed Jan. 20, 1978), *aff'd* 711 F.2d 1510 (11th Cir. 1983).

192. Rodgers discusses contamination as a new norm, Rodgers, *Improving Laws, Declining World*, *supra* note 105, at 1256, while Scott, *Confronting Chronic Pollution*, *supra* note 170, at 318–20, describes the ways that environmental justice activists describe polluted communities as "'sacrifice zones.'" Both are logical outcomes of the law's permission to expose everyone to chemicals without prior testing.

193. Khan, *supra* note 90, at 260. Khan argues that the capabilities approach of Amartya Sen and Martha Nussbaum provide the best framework for understanding the social meaning of epigenetic harms. *Id.* at 294–99; 322–23.

justice between generations and also justice across society today.[194] Cranor has done a service in presenting this complex and important issue so accessibly in *Legally Poisoned*.

194. Environmental justice issues related to toxic exposures are well explained by Scott, *supra* note 170 and Northern, *supra* note 154. For a discussion on intergenerational justice see Neil H. Buchanan, *What Kind of Environment Do We Owe Future Generations?*, 15 LEWIS & CLARK L. REV. 339, 342 (2011). *See generally* Alyson C. Flournoy, *Protecting a Natural Resource Legacy While Promoting Resilience: Can It Be Done?*, 87 NEB. L. REV. 1008 (2009) (discussing the merits of a proposed National Environmental Legacy Act in providing long term environmental protection); Holly Doremus, *Constitutive Law and Environmental Policy*, 22 STAN. ENVTL. L.J. 295, 299 (2003) (exploring the ways law shapes technologies, institutions, communities, relationships, and values and suggesting that attention to these effects can provide concrete form to persistent vague concerns for the future); Lisa Heinzerling, *Environmental Law and the Present Future*, 87 GEO. L.J. 2025 (1999) (examining the current direct effects of pollution on human health); Edith Brown Weiss, *Our Rights and Obligations to Future Generations for the Environment*, 84 AM. J. INT'L L. 198, 202 (1990) (outlining principles that form the basis of "intergenerational obligations and rights, or planetary rights and obligations, that are held by each generation . . . . as part of the intertemporal entity of human society").