CHILDREN'S HEALTH AND ENVIRONMENTAL EXPOSURE RISKS: INFORMATION GAPS, SCIENTIFIC UNCERTAINTY, AND REGULATORY REFORM

WENDY WAGNER AND LYNN BLAIS

The regulatory reform movement has focused the nation's attention on the importance of prioritizing our regulatory agenda to assure that the worst risks are addressed first and that the costs of regulations do not far exceed the benefits they promise to deliver.¹ Virtually all of the regulatory reform activities within the Executive Branch over the past decade involve tools that purport to improve the reliability of the publicly-funded research used for regulation² and the agencies' analytical methods for conducting risk assessments, costbenefit analyses, and peer review.³ Yet these calls for regulatory reform are premised, often implicitly, on the "fact" that there are cogent scientific assessments of environmental risks.

In some important health areas, however, there is effectively no useable base of scientific information from which to develop these quantitative prioritizations and cost-benefit comparisons. Thus, while reformists debate the multiple ways to characterize uncertainty in risk

^{1.} See, e.g., ROBERT W. HAHN, REVIVING REGULATORY REFORM: A GLOBAL PERSPECTIVE 37-39 (2000); John F. Morrall III, A Review of the Record, REGULATION, Nov.-Dec. 1986, at X; Tammy O. Tengs & John Graham, The Opportunity Costs of Haphazard Social Investments in Life-Saving, in RISKS, COSTS AND LIVES SAVED: GETTING BETTER RESULTS FROM REGULATION 172-73 (Robert W. Hahn ed., 1996). See also Richard W. Parker, The Empirical Roots of the "Regulatory Reform" Movement: A Critical Appraisal, 58 ADMIN. L. REV. 359 (2006), for an analysis of these studies and an evaluation of the ongoing debate about their methodologies and usefulness.

^{2.} See, e.g., Data Quality Act, Pub. L. No. 106-554, 114 Stat. 2763A, 153-55 (2001) (passed as a rider to an appropriations bill, § 515 of the Treasury and General Government Appropriations Act for Fiscal Year 2001); Data Access Act, Pub. L. No. 105-277, 112 Stat. 2681, 495 (1998) (passed as a rider to the Omnibus Appropriations Act for Fiscal Year 1999)..

^{3.} See, e.g., Office of Mgmt. & Budget (OMB), Proposed Risk Assessment Bulletin, 71 Fed. Reg. 2600 (proposed Jan. 17, 2006); OMB, Final Information Quality Bulletin for Peer Review, 70 Fed. Reg. 2664 (Jan. 14, 2005); OMB, CIRCULAR A-4, REGULATORY ANALYSIS (2003), available at http://www.whitehouse.gov/omb/circulars/a004/a-4.pdf.

assessment or which discount rates to use in a cost-benefit analysis,⁴ other unstudied and unregulated health risks—which might be just as or even more significant—drop out of the regulatory reformists' radar screen altogether. Data-poor issues cannot be priorities, by definition, in a system that demands numbers as a prerequisite for engaging in the problems.

In this article, we argue that current approaches to setting regulatory and research priorities must find a way to include health risks for which there is little information, except for "snapshots" that suggest potentially serious consequences. Indeed, we argue that the most important set of health risks may be precisely those where there is limited existing regulation and effectively no data or research. Until the system engages in these unknown risks, the health threats will continue without research or regulatory oversight.

Children's health, the subject of the 2006 Duke Environmental Law and Policy Forum symposium, provides a perfect case study, both for underscoring the potential significance of certain unregulated and unmonitored health risks and for developing a method by which these problems can be brought back into regulatory prioritization schemes. The scientific unknowns that surround understanding of environmental exposure risks to children's health are legend.⁵ There is a limited theoretical base for designing studies to test for toxicity to children,⁶ few actual measurements of child exposures to environmental toxins,⁷ and only preliminary research on how children's physiology or development might lead to different expressions of toxicity when compared to adults for any given environmental insult.⁸ Indeed, for certain toxic risks, such as pesticide mixtures and indoor air toxic exposures, the uncertainties and unknowns-both in theory and in simple measurements-are

6. See, e.g., Philip J. Landrigan & Anjali Garg, Chronic Effects of Toxic Environmental Exposures on Children's Health, 40 CLINICAL TOXICOLOGY 449, 453-54 (2002); see also Elaine M. Faustman et al., Mechanisms Underlying Children's Susceptibility to Environmental Toxicants 108 ENVTL. HEALTH PERSP. 13 (Supp. 1, Mar. 2000)

^{4.} See, e.g., Lisa Heinzerling, Discounting Life, 108 YALE L.J. 1911 (1998); Richard L. Revesz, Environmental Regulation, Cost-Benefit Analysis, and the Discounting of Human Lives, 99 COLUM. L. REV. 941 (1999).

^{5.} See, e.g., CHILDREN'S ENVIRONMENTAL HEALTH NETWORK, AN INTRODUCTION TO THE FIELD, available at http://www.cehn.org/WhatisPEH.html (discussing how there are more than 70,000 chemicals in commerce and "[1]ittle is know about the health effects of the majority of these chemicals on children").

^{7.} *See, e.g.*, Landrigan & Anjali, *supra* note 6, at 453 (advocating greater monitoring of environmental health risks to children).

^{8.} See, e.g., Faustman et al., supra note 6, at 13.

overwhelming.⁹ Thus even while scientists make impressive progress in understanding some important risks to children's health, other risks that could be just as dangerous may be largely unexamined and therefore unaddressed.

Not surprisingly, in large part because of this mismatch between the current trajectory of regulatory reform and the looming unknowns that plague some environmental health research, children's health has been strikingly absent from the nation's regulatory agenda, at least in terms of meaningful regulatory requirements.¹⁰ To begin to address this oversight we suggest a new method for identifying and including these under-studied health risks as priorities in the national regulatory agenda. Unlike the quantitative-dependent methods advanced by regulatory reform proponents, our approach does not begin with scientific evidence of harm because that evidence is systematically biased against children's unique or heightened risks. Rather, we adopt a triage-like methodology that identifies regulatory gaps-essentially ways that the current laws systematically underprotect children's health—and match those gaps or "legal blind spots" to available scientific snapshots of ambient concentrations or, where possible, exposure data and adverse health effects. This triangulation of science and law provides an important piece of the puzzle in identifying the worst risks by spotlighting potentially serious problems affecting children's health that are nevertheless ignored by regulators and, because of the resulting limited federal grants, many researchers.

This legal blind spot approach to identifying regulatory priorities has the added benefit of addressing not only regulatory gaps, but also the underlying information gap. Precisely because the problems we identify are not regulated with science-based goals, there is often little corresponding effort by the government to attempt to assess the risks created by these exposures, creating a kind of regulatory inertia "Catch 22." Our triage approach is intended to identify areas where greater regulation could both reduce risks to children's health and generate scientific information to further assess those risks. This science could then form the basis of effective and efficient regulatory responses to these risks, complementing the regulatory reform agenda rather than falling out of its reach.

^{9.} Id.

^{10.} Despite an Executive Order calling attention to the problem, Exec. Order No. 13,045, 3 C.F.R. 157 (1979), there appears to have been little change in many of the Environmental Protection Agency's policies and risk assessments. *See* Faustman et al., *supra* note 6.

We introduce our "legal blind spots" approach to identifying potentially significant environmental risks to children's health in four parts. In the first section, we survey the regulatory reform debate to highlight the dependence of this movement on existing scientific assessments of exposure risks. In the second section we explain why this dependence is likely to leave substantial risks to children's health unaddressed, and outline our method for identifying those children's health risks that are likely to be substantial yet still unregulated. In the third section we apply our methodology to identify several categories of risks that we believe to be the most serious based on this triangulation of gaps in environmental protection and scientific snapshots of insults to children's health. In the final section we offer suggestions for next steps that directly confront and attempt to overcome the vicious circle of ignorance and inattention to potentially worrisome "unknown" risks. Because we know little to nothing about the extent of these risks, it may be difficult to make the problems politically salient. We offer suggestions to address this problem.

I. REGULATORY PRIORITIES AND EXISTING SCIENCE

Scientific assessments of environmental threats to children's health are generated either from the individual research agendas of independent scientists or as a result of information and reporting requirements established or promoted by regulatory programs.¹¹ While scientists likely understand that their own dispersed research projects may not be comprehensive, they may erroneously assume that existing federal regulations provide some comprehensive health protection and establish or support research and reporting programs designed to reveal and evaluate the worst environmental exposure risks. They suppose this, of course, because it constitutes a scientific approach to understanding and controlling environmental risks. But they suppose wrong.

In truth, the legal system regulates health risks based on a multitude of factors, many of which are nonscientific and intrinsically political. These factors often take precedence—by necessity—over scientific assessments of exposure risk.¹² Most notably, regulatory

^{11.} See, e.g., Faustman, supra note 6, at 19.

^{12.} See, e.g., STEPHEN BREYER, BREAKING THE VICIOUS CIRCLE: TOWARD EFFECTIVE RISK REGULATION 33-51(1992), (offering then-Judge Breyer's views of some of the causes of distortions in the regulatory process that may lead to irrational, incomplete, and inefficient regulation of health risks).

coverage is a product of majoritarian politics, which often responds to heightened public concerns that are not rationally correlated to available scientific risk information.¹³ Inversely, the interest group machinations of our political process may result in under-regulation (or the complete absence of regulation) of real and compelling risks that fail to capture the public's attention. Without strong, majoritarian support for regulatory controls, there is often insufficient push to get federal legislation, or at least effective federal legislation, through the special interest-encumbered process.¹⁴

Even when the political process is not relatively encumbered, either by irrational public outcry or disproportionate interest group influence, the response to known and substantial risks may fall short because constraints on the practical design of regulatory programs often mean that some significant health risks will fall through the legal safety net. In the design of an effective regulatory system, the sources of exposure risk must be identifiable and capable of some controls and the proposed controls must be both affordable and enforceable.¹⁵ Thus, the fact that there is an elaborate regulatory infrastructure governing "air toxics" or "pesticides," for example, does not necessarily signify comprehensive health protection. Instead, these elaborate programs may be limited to simply requiring industry to "do their economic best" in limiting pollution, or to label their hazardous products clearly enough to permit safe application (but not to guarantee it). Moreover, some of our most comprehensive regulatory regimes exclude important sources or releases from coverage for a variety of practical or political reasons.¹⁶

It is not surprising, therefore, that scholars have been able to generate data that suggest startling inconsistencies and inefficiencies in this country's regulatory agenda. Over the years, several influential studies have purported to demonstrate that the costs of regulation often far exceed the benefits, that there are more cost-

^{13.} *See id.* at 33-39 (discussing the disconnect between public perceptions of risk and actual risk); Richard H. Pildes & Cass R. Sunstein, *Reinventing the Regulatory State*, 62 U. CHI. L. REV 1, 55-64 (1995) (analyzing public perceptions of risk).

^{14.} See CASS R. SUNSTEIN, LAWS OF FEAR: BEYOND THE PRECAUTIONARY PRINCIPLE (2005) (evaluating in detail the dual phenomena of over-reaction and under-reaction, and the consequences for rational risk regulation); Dan M. Kahan et al., *Fear of Democracy: A Cultural Evaluation of Sunstein on Risk*, 119 HARV. L. REV. 1071 (2006) (critiquing Sunstein's analysis).

^{15.} See, e.g., Corrosion Proof Fittings v. Envtl. Protection Agency, 947 F.2d 1201 (5th Cir. 1991).

^{16.} See generally Lisa Heinzerling, Regulatory Costs of Mythic Proportions, 107 YALE L.J. 1981 (1998).

effective ways to accomplish the same results, and that the system (or lack thereof) for setting regulatory priorities generates bizarre and inexplicable results.¹⁷ In response, legal scholars have proposed a multitude of regulatory reforms. For example, Justice Stephen Breyer proposed the creation of a centralized, powerful, prestigious, politically insulated, and highly specialized group of civil servants charged with the task of rationalizing our risk regulation enterprise.¹⁸ Professors Pildes and Sunstein responded with proposals to rationalize regulatory priority setting without sacrificing democratic participatory values,¹⁹ and Professor Sunstein has recently elaborated on those proposals.²⁰ Similarly, Congress²¹ and the Executive²² have adopted reforms intended to limit the implementation of regulations that cannot be justified in cost-benefit terms, to encourage coordination among agencies in establishing regulatory priorities, and to promote rational regulatory priority setting within and among agencies.

There has been much debate over the accuracy and usefulness of the studies that prompted these reforms²³ and the value of the reforms

21. See generally Unfunded Mandates Reform Act of 1995, Pub. L. No. 104-4, 109 Stat. 48 (codified as amended at 2 U.S.C. §§ 1501-71 (2000)) (requiring that agencies perform quantitative and qualitative cost-benefit analysis before proposing or promulgating any significant rule); Contract with America Advancement Act of 1996, Pub. L. No. 104-121, 110 Stat. 847 (codified as amended at 5 U.S.C. §§ 601, 801-8 (2000)) (providing for congressional review of all major regulations issued by government agencies with the possibility of joint resolution override); Government Performance and Results Act of 1993, Pub. L. No. 103-62, 107 Stat. 285 (codified at 31 U.S.C. §§ 1115-9 (2000)) (requiring agencies to develop multi-year strategic plans along with annual performance plans and reports).

22. The most notable examples are the Executive Orders signed by every President since Jimmy Carter that require some form of centralized oversight of regulatory priority setting and regulatory effectiveness. *See, e.g.*, Exec. Order No. 12,044, 3 C.F.R. 152 (1979) (establishing the Office of Information and Regulatory Affairs (OIRA) within the Office of Management and Budget). In Executive Order No. 12,291, 3 C.F.R. 127 (1982), which replaced Executive Order No. 12,044, President Reagan consolidated and strengthened OIRA's oversight authority. While President George H.W. Bush relied on the Council on Competitiveness (headed by Vice President Dan Quayle) to serve OIRA's centralized regulatory oversight role, both Presidents Clinton and George W. Bush revived OIRA and its focus on rationalizing regulatory decisions. For a review of the historical role of OIRA, see John R. Graham, Paul R. Noe, and Elizabeth L. Branch, *Managing the Regulatory State: The Experience of the Bush Administration*, 33 FORDHAM URB. L.J. 953, 955-65 (2006).

23. See, e.g., FRANK ACKERMAN & LISA HEINZERLING, PRICELESS: ON KNOWING THE COST OF EVERYTHING AND THE VALUE OF NOTHING (2003); Heinzerling, *supra* note 16; Parker, *supra* note 1.

^{17.} See Parker, supra note 1.

^{18.} BREYER, *supra* note 12, 59-63.

^{19.} Pildes and Sunstein, Reinventing the Regulatory State, supra note 13, 86-129.

^{20.} Sunstein, LAWS OF FEAR, supra note 14.

themselves.²⁴ While we tend to agree that the critics of these studies have the better of the argument, it is not our intent to join this debate in this paper. Rather, we offer another critique of the power of these studies to guide effective and efficient regulatory reform, premised on the systematic failure of these studies to realize that existing scientific evidence of exposure risk may not provide the only justification for efficient regulation.

II. THE TRIAGE APPROACH: LEGAL GAPS AND SCARY SNAPSHOTS

It is our contention that existing scientific data, such as the data underlying the reform-generating studies discussed above, can provide only part of the justification for regulatory action in any rational system of setting regulatory priorities.²⁵ This is so because scientific research agendas are not the product of a coherent, rational, efficient process designed to identify the worst environmental exposure risks and comparatively rate the rest.²⁶ Important information gaps exist, and the existence of these gaps undermines the claims by reformists that regulatory priorities should be established to maximize the value of regulations based on existing science.²⁷ Importantly, health and safety laws not only regulate risks, they also provide powerful incentives and often establish the

^{24.} See, e.g., Nicholas Bagley & Richard L. Revesz, *Centralized Oversight of the Regulatory State*, 106 COLUM. L. REV. 1260 (2006) (challenging the conventional view that agencies are prone to inefficient and overzealous regulation and that centralized oversight can remedy these problems).

^{25.} Several legal scholars have persuasively argued that cost-benefit analysis of environmental regulations is inherently flawed because the benefits of such regulations are difficult to quantify and therefore systematically under-valued. Our argument is related to, but distinct from, this point. *See, e.g.*, FRANK ACKERMAN AND HEINZERLING, *supra* note 23, *passim* (2003) (making the case against cost-benefit analysis in environmental, health, and safety regulation); *see also*, Richard W, Parker, *Grading the Government* 20 U. CHI. L. REV. 1343, 1381-1414 (assessing the systematic undervaluation of health and safety benefits in cost-benefit analysis).

^{26.} It is important to note that we are not suggesting that such an agenda setting mechanism should exist. We are simply observing that scientific evidence generated by individuated research agendas and responding to various uncoordinated incentives cannot be counted on to produce comprehensive comparative risk information.

^{27.} Of course, some reformists recognize that efficiency and cost-effectiveness are starting points for regulatory reform, and that value judgments must follow the technocratic scientific evaluation. See e.g., Matthew D. Adler and Eric A. Posner, *Rethinking Cost-Benefit Analysis*, 109 YALE L. REV. 165 (1999) (advocating widespread use of cost-benefit analysis to evaluate competing policies, but recognizing that its conclusions will be only one component of reasoned governmental choice). Our contention is different, however. We argue that there should be room in the scientific assessments for a methodology that begins from premises other than existing scientific information.

mechanisms for generating more information to assist in the regulatory enterprise.²⁸ Legal requirements and associated regulatory programs are responsible for demanding and often financing a great deal of applied research that is then available to scientists and to regulators to fine-tune the regulatory program.²⁹ In the absence of regulation, substantial risks may remain unexamined and large data gaps may develop.³⁰ In this way, legal blind spots become scientific ones. As the D.C. Circuit noted in a slightly different but related setting, "[a]n exemption [to regulation] tends to become indefinite: the problem drops out of sight, into a pool of inertia, unlikely to be recalled in the absence of crisis or a strong political protagonist."³¹

Thus, we propose a triage-type mechanism for identifying regulatory priorities that arises from the premise that what we don't know *can* hurt us. Our approach has three parts. First, we focus exclusively on those regulatory programs that offer no health-based protection for at least one important environmental vector of risks to children's health, either because they exclude this risk entirely or because the protection offered is based on nonscientific factors like technological feasibility. We then confirm that the programs do not provide any requirements or incentives for monitoring and continued data collection.³² Finally, we look for the limited "scientific snapshots" of scientific literature to verify that children could be at heightened risk from these regulatory gaps. When these three factors align, then we have found a blind spot in the protection of children's

^{28.} For example, when the Environmental Protection Agency (EPA) initially adopted its regulation requiring the removal of most of the lead from gasoline in the 1970s, very little was known for certain about the health consequences of lead exposure. Only after a decade of unleaded gasoline use did scientists have sufficient data to demonstrate substantial benefits of the concomitant reductions in blood lead levels, especially in children. *See* Frank Ackerman, Lisa Heinzerling, & Rachel Massey, *Applying Cost-Benefit to Past Decisions: Was Environmental Protection Ever a Good Idea*?, 57 ADMIN. L. REV. 155, 160-71 (2005) (examining the decision to remove lead from gasoline and its consequences).

^{29.} See, e.g., John S. Applegate, *The Government Role in Scientific Research: Who Should Bridge the Data Gap in Chemical Regulation?*, *in* RESCUING SCIENCE FROM POLITICS: REGULATION AND THE DISTORTION OF SCIENTIFIC RESEARCH 261-62 (Wendy Wagner & Rena Steinzor eds., 2006) (applying the supply/demand framework for understanding the interaction between legislation and information production).

^{30.} See, e.g., id. at 257-59 (discussing one data gap).

^{31.} Natural Res. Def. Council, Inc. v. Costle, 568 F.2d 1369, 1382 (D.C. Cir. 1977).

^{32.} Although it is usually the case that where there is no federal science-based regulatory program, there is also no ambient monitoring or system of collecting information about the nature or extent of contamination, that is not always the case. *See generally* Emergency Planning and Community Right-to-Know Act, 42 U.S.C. §§ 11001-50 (2000).

health that we argue deserves further investigation and perhaps regulatory response.

We hasten to add that the risks arising in these legal blind spots are not the only and may not be the most significant environmental threats to children's health. There is strong evidence of other, serious, quantified risks to children's health that remain, notwithstanding the existence of relatively comprehensive regulatory programs.³³ Nevertheless, we believe that the legal blind spots we identify may be of greater urgency because the lack of regulatory oversight means that there are no requirements or incentives for data collection or scientific analysis.

Therein lies the broader implication of our approach. It is our contention that in any attempt to prioritize and rationalize the national regulatory agenda, identifying large gaps in legal protection may be just as important as working from the available scientific information. Because legal protections both respond to and generate scientific evidence of important risks, using existing information to justify legal responses is only half of any logical agenda setting methodology. Our approach provides a crucial missing piece to the regulatory reform puzzle.

III. APPLYING THE TRIAGE APPROACH: POCKETS OF UNDER PROTECTION AND GREAT UNCERTAINTY

Based on an inventory of the federal regulatory programs, we identify three and potentially more areas where regulations do not even pretend to be protective of public health, but where child exposure to risks might be substantial. Consistent with our criteria, these programs not only provide theoretically inadequate protection to most persons, particularly children, living in "hot spot" communities, but they also produce no incentives or requirements for measuring ambient exposures. As a result, the problems tend to fall through the legal and scientific cracks.

A. Air Toxics in "Hot Spot Areas"

The under-regulation of the emission of air toxins and the resulting existence of air toxic hot spots provide a compelling

^{33.} This is particularly true of health risks to children that result from exposure to ozone in the ambient air, a pollutant that is relatively heavily regulated. *See generally* Committee on Environmental Health, *Ambient Air Pollution: Health Hazards to Children*, 114 PEDIATRICS 1699 (2004) (sources omitted).

example of a regulatory gap that may be contributing to serious risks to child health. Air toxins are regulated through technology-based controls.³⁴ This regulatory mechanism ensures only that each major source has done its economic and technological best (as determined by the Environmental Protection Agency (EPA)) to prevent pollution, not that significant risks to public health are averted.³⁵ As a result, industrial sources of air toxins often emit levels of air toxins that far exceed what might be considered "protective" (usually a one in one million standard) based on risk assessments, even when they are in compliance with applicable regulatory standards.³⁶ In some instances, moreover, industrial sources appear to be in violation of even these science-blind, emission control requirements.³⁷ The most common violations result from repeated "upsets" or high bursts of polluting activity that arise from pollution control equipment malfunctions and related causes.³⁸ Scientists studying air quality in Houston, for example, discovered that concentrations of ozone were as much as three times higher than the levels that would be predicted based on the levels of pollution that had been permitted for the area, an observation they attributed to high bursts of pollution (or spikes) in industrial emissions.³⁹ To make matters worse, many large industrial sources are commonly located together in "complexes" or "industrial corridors."⁴⁰ These corridors concentrate large vehicular

36. See generally Victor Flatt, Gasping for Breath: The Administrative Flaws of Federal Hazardous Air Pollution Regulation 10-12 (2006) (draft, on file with authors) (observing this problem and discussing Congress' effort to address it through the residual risk program).

37. See, e.g., Thomas O. McGarity & Karen Sokol, Man-Made Disaster: Texas's Failure to Protect its Citizens from the Perils of the Houston Petrochemical Complex, CPR WHITE PAPER, Sept. 2006, at 8-11, available at http://www.progressiveregulation.org/articles/HPC_605.pdf.

38. See, e.g., id. at 8-9.

40. See, e.g., McGarity & Sokol, supra note 37, at 2-3.

^{34.} See, e.g., 42 U.S.C. § 7521 (2000) (motor vehicle emissions standards).

^{35. 42} U.S.C. § 7412(d) (2000); *but see* 42 U.S.C. § 7412(f) (2000). Although EPA is required to set residual standards for air toxins that present greater than a one in one million cancer risk after technology-based standards have implemented, EPA has moved very slowly on these "residual risk" standards and still sets them on a source-by-source basis. *See, e.g.,* E. Donald Elliott & Alexander Schmandt, Recent Clean Air Act Developments, ALI-ABA Course of Study, February 16-18, 2005, SK058 ALI-ABA 103, 115 (2005) (noting how EPA has proposed residual risk standards for only one source of air toxics– coke oven batteries); *see also infra* text accompanying notes 42-44.

^{39.} See David Allen et al., Accelerated Science Evaluation of Ozone Formation in the Houston-Galveston Area 7-8, 17-18 (Sept. 13, 2001), http://www.utexas.edu/research/ceer/texaqsarchive/accel_science_eval.PDF.

traffic serving the industrial clusters in one area, which contributes to the elevated levels of hazardous air pollutants.⁴¹

Congress anticipated that technology based standards were likely to fall short of the protective measures warranted by the risks of exposure to air toxins, and accordingly required EPA to assess the remaining health risks from air toxins after the technology-based controls were implemented.⁴² Congress also required EPA to devise a strategy for addressing residual health risks, particularly in urban areas.⁴³ Unfortunately, EPA has made little progress addressing air toxins that accumulate additively in "hot spots" that could threaten public health.⁴⁴

Because regulation of air toxins is not yet based on healthprotective goals or measures, the federal regulatory program governing air toxics does not require monitoring of individual toxins either in the ambient air or at the point of their emission from stacks or fugitive sources. As long as the pollution control technology is in place and appears to be functioning, the source is presumed to be in compliance.⁴⁵ Ambient levels are thus effectively irrelevant to the regulatory scheme. Not surprisingly, then, U.S. EPA and most states have given little attention to monitoring the ambient air for air toxics or taking measures to ensure that the public is adequately protected.⁴⁶ Moreover, the ambient data that is collected is of questionable

44. EPA has completed a Report to Congress, published in 1999, that contains "EPA's general framework for assessing risks to public health or the environment." EPA, RESIDUAL RISK REPORT TO CONGRESS ES-12 (March 1999), *available at* http://www.epa.gov/ttn/oarpg/t3/reports/risk_rep.pdf. EPA is also in the process of promulgating emissions standards for individual categories of sources. *See* EPA, Charge to the Environmental Models Subcommittee of the SAB (2006), http://www.epa.gov/ttnatw01/nata/sab_charge7.html (last visited Apr. 11, 2007). In its large air toxics modeling project discussed infra at note 67 and accompanying text, EPA does identify the "next steps" that flow from the assessment. *See* EPA's Use of Results (2006), http://www.epa.gov/ttn/atw/nata/ur.html (last visited Jan. 25, 2007). This explanation does not provide information on EPA's progress on any of the goals, however, and they are not cross-referenced in EPA's Residual risk webpage.

45. See, e.g., 40 C.F.R. § 63.120(a) (2006) (visual inspections are required only once annually for storage vessels); see generally Lynn Blais, Thomas McGarity & Wendy Wagner, ENFORCEMENT AGAINST CONCENTRATIONS OF TOXIC POLLUTION IN TEXAS: A REPORT TO THE TEXAS COMMISSION ON ENVIRONMENTAL QUALITY AND UNITED STATES ENVIRONMENTAL PROTECTION AGENCY 25-26 (Feb. 2003) (on file with authors).

46. See generally McGarity & Wagner, supra note 45.

^{41.} See, e.g., Committee on Environmental Health, supra note 33.

^{42.} See 42 U.S.C. § 7412(d) (2000).

^{43.} See 42 U.S.C. § 7412(k) (2000).

quality, in part because of its inability to detect low quantities of toxins and in part because of imprudent placement of the monitors.⁴⁷

Perhaps even more disturbing, there is often no regulatory infrastructure in place to process ambient air quality data, even when relatively high quality data is available. There are no official federal standards for "safe" levels of ambient air toxins, and "soft" limits are in place only for *some* of the listed federal air toxins.⁴⁸ As of 1998, EPA had provided cancer benchmark concentrations ("safe concentrations" leading to only a one in one million cancer risk) for less than eighty percent of the listed hazardous air pollutants and non-cancer benchmark concentrations for about half.⁴⁹ Moreover, these benchmark concentrations are based on what is needed to protect an average adult (a 154 pound adult who is exposed for seventy years),⁵⁰ not children. There is also insufficient knowledge of svnergistic reactions between multiple air toxics; at best, the risks posed by individual toxins are added together.⁵¹ In some states, however, even this simple calculation is not made. In a study of a "hot spots" of 1,3-butadiene in Scotlandville, Louisiana, for example, the State did not even attempt to account for the additive risks posed by all of the air toxics combined in deciding that no further action was needed, even though the aggregate concentrations of these toxic pollutants were high, particularly in the "strike samples" that take worst case measurements.⁵² See Table 1.

51. See, e.g., id. at 10.

^{47.} See, e.g., Amy D. Kyle NM., Evaluating the Health Significance of Hazardous Air Pollutants Using Monitoring Data, 116 PUB. HEALTH REP. 32, 42 (2001); Tracey J. Woodruff et al., Public Health Implications of 1990 Air Toxics Concentrations across the United States, 106 ENVTL. HEALTH PERSP. 245, 245 (1998); McGarity & Sokol, supra note 37, at 3.

^{48.} See, e.g., EPA's Health Criteria for Air Toxics, *available at* http://www.epa.gov/ttn/atw/ nata/nettables.pdf; Minimum Risk Levels (MRLs) for Hazardous Substances, Agency for Toxic Substances and Disease Registry (ASTDR) (2007), *available at* http://www.atsdr.cdc.gov/ mrls.html; COMMONWEALTH OF PENNSYLVANIA, DEPARTMENT OF ENVIRONMENTAL PROTECTION, POTTSTOWN AREA AIR MONITORING, 9, 21-22 (May 12, 2004), *available at* http://www.dep.state.pa.us/dep/deputate/airwaste/aq/toxics/projects/pottstown/pottstown_report .pdf [hereinafter Pennsylvania Pottstown report].

^{49.} See, e.g., Woodruff et al., supra note 47, at *; Kyle, supra note 47, at 42.

^{50.} See, e.g., Pennsylvania Pottstown Report, supra note 48, at 9.

^{52.} See, e.g., LOUISIANA DEPARTMENT OF ENVIRONMENTAL QUALITY, FINAL AIR MONITORING REPORT FOR THE SOUTH SCOTLANDVILLE AIR TOXICS MONITORING PROJECT 9 (Mar. 22, 2006), available at http://www.deq.louisiana.gov/portal/LinkClick.aspx?link=Air QualityAssessment%2fSouth+scotlandville+final.pdf [hereinafter referred to at the Louisiana Scotlandville Report].

Table 1:

Louisiana Department of Environmental Quality Photochemical Precursor Summary GC Flame Ionization Results

1/27/2006

| Monitoring Site Code | South Scotlandville | otlandville Sample Duration: 25 min Strike Samples | | | | | | | |
|---|---------------------|--|------------------------------|--------------|----------|--|--|--|--|
| Samples Collected: | 72 | | Sample Date Range: 1/10/2005 | - 12/29/2005 | | | | | |
| Estimated Concentrations in parts per billion molar volume (ppbv) | | | | | | | | | |
| Compound | Mean | Max | Compound | Mean | Max | | | | |
| Ethylene | 22.17 | 133.48 | 3-methylhexane | 0.72 | 3.96 | | | | |
| Acetylene | 3.41 | 15.83 | 2,2,4-trimethylpentane | 1.87 | 8.33 | | | | |
| Ethane | 24.57 | 228.23 | n-Heptane | 0.62 | 2.88 | | | | |
| Propylene | 19.07 | 310.49 | Methylcyclohexane | 0.52 | 1.86 | | | | |
| Propane | 214.33 | 5166.60 | 2,3,4-trimethylpentane | 0.61 | 3.36 | | | | |
| Isobutane | 24.60 | 720.24 | Toluene | 3.73 | 14.79 | | | | |
| 1-butene | 2.08 | 20.74 | 2-methylheptane | 0.20 | 0.78 | | | | |
| n-Butane | 44.44 | 300.51 | 3-methylheptane | 0.21 | 0.82 | | | | |
| trans-2-Butene | 2.23 | 19.72 | n-Octane | 0.23 | 0.81 | | | | |
| cis-2-Butene | 1.87 | 13.01 | Ethylbenzene | 0.44 | 1.90 | | | | |
| 2-methylbutane | 24.33 | 145.23 | m/p Xylene | 1.42 | 7.37 | | | | |
| 1-Pentene | 1.58 | 13.01 | Styrene | 0.96 | 9.49 | | | | |
| n-Pentane | 11.13 | 59.48 | o Xylene | 0.56 | 2.69 | | | | |
| Isoprene | 0.69 | 2.70 | n-Nonane | 0.14 | 0.72 | | | | |
| trans-2-Pentene | 3.12 | 22.86 | Cumene | 0.06 | 0.24 | | | | |
| cis-2-Pentene | 1.81 | 12.11 | n-propylbenzene | 0.10 | 0.56 | | | | |
| 2,2-dimethylbutane | 1.20 | 8.10 | m-ethyltoluene | 0.35 | 2.20 | | | | |
| Cyclopentane | 1.22 | 7.05 | p-ethyltoluene | 0.14 | 0.95 | | | | |
| 2,3-dimethylbutane | 1.37 | 5.65 | 1,3,5-trimethylbenzene | 0.19 | 1.14 | | | | |
| 2-methylpentane | 5.30 | 20.72 | o-ethyltoluene | 0.14 | 0.78 | | | | |
| 3-methylpentane | 4.15 | 38.28 | 1,2,4-trimethylbenzene | 0.77 | 3.74 | | | | |
| 1-Hexene | 0.54 | 4.73 | n-Decane | 0.13 | 0.95 | | | | |
| n-Hexane | 6.06 | 116.92 | 1,2,3-trimethylbenzene | 0.15 | 1.05 | | | | |
| Methylcyclopentane | 1.99 | 10.27 | m-diethylbenzene | 0.05 | 0.30 | | | | |
| 2,4-dimethylpentane | 0.46 | 1.89 | p-diethylbenzene | 0.09 | 0.61 | | | | |
| Benzene | 1.87 | 8.83 | n-Undecane | 0.09 | 0.82 | | | | |
| Cyclohexane | 0.81 | 12.56 | 1,3-butadiene | 3.10 | 24.67 | | | | |
| 2-methylhexane | 0.71 | 3.51 | | | | | | | |
| 2,3-dimethylpentane | 0.43 | 2.04 | TNMOC (ppbc) | 1768.56 | 16818.00 | | | | |

The State's failure to consider cumulative concentrations is particularly worrisome since the State adopted protective health levels that were 40 times less protective for benzene (EPA's level is $0.09 \text{ ppb})^{53}$ and 260 times less protective for 1,3-butadiene (EPA's level is 0.016)⁵⁴ than the benchmark levels recommended by EPA.

In most states there is also no ready regulatory response if levels in ambient air exceed "safe" levels. As a technical matter, it can be difficult for a state regulator to isolate the source(s) of the

^{53.} Louisiana's standard is 3.76 ppb, *id*. at 8. EPA's recommended standard is .09 ppb, Kyle et al., *supra* note 47, at 40.

^{54.} Lousiana's standard is .42 ppm, Louisiana Scotlandville Report, *supra* note 52, at 9. EPA's recommended standard is .0016. Kyle et al., *supra* note 47, at 40.

problematic emissions.⁵⁵ Multiple sources, including transportation, can contribute to high concentrations of air toxins in ambient air.⁵⁶ Even more problematic, if the sources appear to be in compliance with their permits (or there is no evidence to the contrary), states may believe they do not have regulatory authority to take action or may lack the political capital to push existing enforcement authorities in creative ways.⁵⁷ In Texas, for example, the State believes it must determine that air toxic concentrations significantly threaten public health before they can demand sources to limit their emissions of a problematic substance below permitted levels.⁵⁸ Indeed, perceived limits in existing regulatory authority may only increase the tendency of U.S. EPA and states to remain ignorant of ambient levels of air toxins in the environment, as well as what those levels might mean in terms of health impacts.

Despite these multiple science-law impediments, a number of states are in the process of developing programs that attempt at least to begin to engage in an analysis of potential health threats through targeted monitoring of air toxins.⁵⁹ An EPA website posts air toxics monitoring data for the states,⁶⁰ although since most states do not have comprehensive monitoring programs the usefulness of this site is limited.⁶¹ Even in states where the monitoring is more extensive, however, regulators appear at least partly hamstrung in taking regulatory action to address high levels of air toxins. In an assessment of the risks of air toxins in Pottstown, Pennsylvania, for example, state regulators discovered high levels of TCE in the ambient air based on stationary monitors positioned in the town and

^{55.} See Louisiana Scotlandville Report, supra note 52, at 8-9.

^{56.} *See, e.g., id.* at 8 (attributing almost half of the high benzene concentrations to mobile sources).

^{57.} This is not always the case, however. In initial research on Scotlandtown, the State found very high levels of 1,3-butadiene that it apparently linked to stationary industrial sources. It took administrative action against fifteen of the sources and required them to engage in fenceline monitoring and the levels decreased (although as mentioned earlier perhaps not to levels that EPA would consider "safe"). Lousiana Scotlandville Report, *supra* note 52, at 6.

^{58.} See, e.g., Blais, McGarity, & Wagner, supra note 45, at 15-26 (describing the limitations in Texas's authority to address high concentrations of urban air toxics).

^{59.} Connecticut, for example, promulgated protective standards for these contaminants in the 1980s and developed a monitoring system in the late 1990s to determine whether there are significant exceedances in the state worthy of further regulatory action. *See, e.g.,* Nescaum, Data Analysis of Air Toxics in Connecticut (Mar. 1, 2005), at 1-1 to 1-3, *available at* http://www.nescaum.org/topics/air-toxics-monitoring.

^{60.} See EPA Database, available at http://www.epa.gov/ttn/atw/wks/fs_monitoring.pdf.

^{61.} See, e.g., McGarity & Sokol, supra note 37, at 3.

at the high school, presenting an excess risk of about 1.6 in 10,000 of cancer to adult residents and, when considering all toxins combined, about a two in 10,000 risk of cancer.⁶² The regulators decided not to take regulatory action because the State Health Department determined that this was not a health risk of significance and because the TCE concentrations had been dropping over two years.⁶³ Curiously absent from the report is any mention of the type of action that the State would have taken against the diverse sources of TCE had it determined that a two in 10,000 risk of cancer to an adult was a risk worthy of concern.⁶⁴ The State of Texas has identified similarly high levels of air toxins in some areas,⁶⁵ yet as mentioned, it seem to believe it lacks the regulatory authority to require sources to reduce pollutants if they are in compliance with their permits.⁶⁶

The scientific snapshots reinforce concerns about excessive risks to children from exposure to toxic air pollutants. The most comprehensive estimate of possible risks comes from an EPA study that modeled emissions data from the Toxic Release Inventory.⁶⁷ EPA's study predicts that in 1996 more than 200 million people in the U.S. face an upper-bound lifetime cancer risk of greater than one in 100,000 and 20 million people face risks greater than one in 100,000 due to their exposure to air toxics.⁶⁸ Other studies produce similarly worrisome projections. One study reaffirms EPA's conclusion that "concentrations of pollutants in many counties… would exceed health benchmarks [of a one in 1 million risk]"⁶⁹ while another study

^{62.} See Pennsylvania Pottsville Report, supra note 48, at 9-10.

^{63.} Id. at 11.

^{64.} Id.

^{65.} *See, e.g.*, McGarity & Sokol, *supra* note 37, at 2; *see also* Dina Capiello, *In Harm's Way*, THE HOUSTON CHRONICLE, January 16, 2005.

^{66.} This is based on the authors' communications with the Texas Council for Environmental Quality. The TCEQ commissioned a study from the authors (and Prof. McGarity) that sought recommendations for how they might respond to the problem of air toxins in the State; although the study was presented in 2002 and finalized in 2003, no action by the State legislature or the TCEQ has been taken.

^{67.} The method for the model is explained by the EPA. EPA, Overview: The 4 Steps, http://www.epa.gov/ttn/atw/nata/4steps.html (last visited Feb. 8, 2007).

^{68.} See EPA, Summary of Results (1996), available at http://www.epa.gov/ttn/atw/ nata/risksum.html. Maps can also be constructed on command to estimate "rough" risk levels and concentrations in counties and states. See, e.g., The Risk Maps, http://www.epa.gov/ ttn/atw/nata/maprisk.html (last visited Feb. 8, 2007).

^{69.} See, e.g., Kyle et al., supra note 47, at 33.

| Table 2. Background concentrations for hazardous air pollutants (HAPs) that exceed benchmark concen- trations in all census tracts | | | | | | | |
|---|--|---|--|--|--|--|--|
| Hazardous air pollutant | Background concentration (µg/m3) | Cancer benchmark concentration (µg/m3) ^a | Ratio of background to benchmark concentration | No. of census tracts with exceedances, disregarding background (%) | | | |
| Bis(2-ethylhexyl) phthalate | 1.6 | 0.25 | 6.4 | 18 (<1) | | | |
| Benzene | 0.48 | 0.12 | 4.0 | 56,000 (92) | | | |
| Carbon tetrachloride | 0.88 | 0.067 | 13 | 1,600 (3) | | | |
| Chloroform | 0.083 | 0.043 | 1.9 | 4,900 (8) | | | |
| Ethylene dibromide | 0.0077 | 0.0045 | 17 | 900(1) | | | |
| Ethylene dichloride | 0.061 | 0.038 | 1.6 | 13,000 (21) | | | |
| Formaldehyde | 0.25 | 0.077 | 3.2 | 57,000 (94) | | | |
| Methyl chloride | 1.2 | 0.56 | 2.2 | 110 (<1) | | | |

summarizes the frequency of predicted exceedances by chemical name. See Table 2.⁷⁰

^aAll cancer benchmarks are for Tier I carcinogens except for bis(2-ethylhexyl) phthalate.

With the help of robust ambient monitoring data in the State of California, researchers found that the risks of cancer in adults were as high as 2-3 in 10,000 for certain areas of the State, an increase that could lead to 8,600 increased lifetime cancer cases.⁷¹ In the State of Minnesota, using a combination of monitoring and modeling, the highest cancer risks were estimated at 2.7 in 100,000 for specific, affected areas, a risk that the researchers concluded was serious enough to warrant further scientific and regulatory attention.⁷²

Because children's lungs continue to develop postnatally, there is reason to believe that they are particularly vulnerable to exposures to air toxins.⁷³ Indeed, the few existing studies of childhood exposure to air pollutants reveal that those children who live in polluted areas with heavy concentrations of particulates have decreased lung function and lung growth.⁷⁴ Some birth defects and infant mortality have even been associated with high particulate concentrations.⁷⁵ "Increased respiratory tract complications in children (e.g., wheezing, chronic productive cough, and asthma hospitalizations) have [also] been associated with residence near areas of high traffic density

75. See id.

^{70.} Table 2 is taken from Woodruff et al., *supra* note 49.

^{71.} Rachel A. Morello-Frosch et al., *Air Toxics and Health Risks in California: The Public Health Implications of Outdoor Concentrations*, 20 RISK ANALYSIS 273, 273 (2000).

^{72.} P.G. Pratt et al., An Assessment of Air Toxics in Minnesota, 108 ENVTL. HEALTH PERSP. 815, 821 (2000).

^{73.} See Committee on Environmental Health, supra note 33, at 1699.

^{74.} See id. at 1701.

(particularly truck traffic). Other investigators have linked various childhood cancers to proximity to traffic.⁷⁶ Unfortunately, however, because there are comparatively few studies on the effects of air toxics exposure on children, the full extent of the public health crisis resulting from air toxics "hot spots" is unknown.

B. Ambient pesticide exposures

A second environmental threat to children's health occurs as a result of the gaps in the regulatory oversight of pesticide use. As a result of the Food Quality Protection Act, substantial public attention has been focused on examining and limiting the residue of pesticides on food and accounting for the particular susceptibility of children in setting these pesticide residue standards.⁷⁷ While the implementation of the Food Quality Protection Act has been disappointing,⁷⁸ the program nevertheless has made some notable progress in limiting children's exposures to pesticides in food.⁷⁹

The actual environmental application and use of these pesticides, however, is not regulated at all at the federal level. While FIFRA mandates federal review and approval of pesticide labeling, and, in theory, compliance with labeling requirements during application,⁸⁰ the actual regulation of the use of pesticides is left to the states.⁸¹ With the exception of some limited school- or application-specific requirements, however, states have not stepped in to fill this regulatory gap.⁸² As a consequence, a great deal of pesticides can be used in unsafe ways and in higher concentrations than expected or needed.⁸³

^{76.} Id. at 1702.

^{77.} See 21 U.S.C. § 321(s) (2000).

^{78.} See Thomas O. McGarity, Politics by Other Means: Law, Science, and Policy in EPA's Implementation of the Food Quality Protection Act, 53 ADMIN. L. REV. 103, 147-202 (2001).

^{79.} See, e.g., U.S. ENVIRONMENTAL PROTECTION AGENCY, IMPLEMENTING THE FOOD QUALITY PROTECTION ACT: PROGRESS REPORT 35 (Aug. 1999), available at http://www.epa.gov/pesticides/regulating/laws/fqpa/fqpareport.pdf [hereinafter FQPA Progress Report].

^{80.} Federal Insecticide, Fungicide, and Rodenticide Act, 7 U.S.C. § 136a (2000).

^{81.} Federal Insecticide, Fungicide, and Rodenticide Act, 7 U.S.C. § 136v(a) (2000).

^{82.} Our focus on environmental exposures sets aside the high incidence of accidental poisonings due to pesticides, which is a concern in and of itself. *See* U.S. ENVIRONMENTAL PROTECTION AGENCY, PESTICIDES AND CHILD SAFETY, http://www.epa.gov/pesticides/ factsheets/childsaf.htm (last visited Feb. 8, 2007).

^{83.} See generally Donald T. Hornstein, Lessons from Federal Pesticide Regulation on the Paradigms and Politics of Environmental Law Reform, 10 YALE J. ON REG. 369, 392-406 (arguing that "pesticide regulation is not... a body of law that addresses in any strategic way the

Even when used as directed, however, there is no guarantee that children or adult family members will be adequately protected from excess risk resulting from exposure to pesticides. Under the U.S. pesticide licensing program, pesticides are restricted or banned only if EPA finds that they ""cause unreasonable adverse effects on the environment,"⁸⁴ a determination that involves a cost-benefit analysis that weighs the benefits of the pesticide against the risks.⁸⁵ In making this calculation, EPA does not require manufacturers to show either the efficacy of the pesticide or the lack of the availability of safer substitutes; pesticides are viewed in isolation and assumed to be efficacious.⁸⁶ Moreover, since many pesticides were on the market when FIFRA was enacted in 1976, older pesticides have received only limited, after-the-fact regulatory oversight, with correspondingly limited toxicity testing to evaluate their safety.⁸⁷ Even for new, more thoroughly tested pesticides, screening for important risks to children is limited.⁸⁸ In their review article on pesticides and children, Landrigan et al. list four deficiencies in current testing requirements for pesticides used by EPA that-even seven years after the article was published—seem to remain largely in place.⁸⁹ First, EPA does not routinely require full-scale neurodevelopmental toxicity testing, even for neurotoxic pesticides.⁹⁰ Although some of these testing

88. "For the first time, EPA is requiring additional studies on pesticides to better understand their effects on children specifically (developmental neurotoxicity, acute and subchronic neurotoxicity). In addition, EPA has developed new tests and risk assessment methods to target the factors unique to infants and children." U.S. ENVIRONMENTAL PROTECTION AGENCY, PROTECTING CHILDREN FROM PESTICIDES (Jan. 2002), http://www.epa.gov/pesticides/factsheets/kidpesticide.htm (last visited Apr. 11, 2007).

89. See Philip J. Landrigan et al., Pesticides and Inner-City Children: Exposures, Risks, and Prevention, 107 ENVTL. HEALTH PERSP. SUPP. 431, 435-36 (1999). Although Landrigan et al.'s article was published in 1999, the dates of the protocols posted on EPA's website indicates that most, if not all, of the protocols Landrigan et al. were criticizing were published in 1998 or earlier. See, e.g., U.S. ENVIRONMENTAL PROTECTION AGENCY, OPPTS SERIES 870 TEST GUIDELINES, available at http://www.epa.gov/opptsfrs/publications/OPPTS_Harmonized/870_Health_Effects_Test_Guidelines/Master/870final.pdf.

90. Landrigan et al., supra note 89, at 435.

underlying prevalence of pesticides in American agriculture, nor is it a body of law designed to minimize pesticide use.").

^{84.} Federal Insecticide, Fungicide, and Rodenticide Act, 7 U.S.C. § 136a(c)(5)(D) (2000).

^{85.} See, Mary Jane Angelo, Embracing Uncertainty, Complexity, and Change: An Ecopragmatic Reinvention of a First-Generation Environmental Law, 33 ECOLOGY L.Q., 105, 161-62 (2006).

^{86.} See id. at 163.

^{87.} See id. at 166-67; see also John S. Applegate, The Perils of Unreasonable Risk: Information, Regulatory Policy, and Toxic Substances Control, 91 COLUM. L. REV. 261, 313-14 (1991).

guidelines technically exist, there is "wide flexibility" in when they are required.⁹¹ Second, most of the tests are performed only on adult animals, even though there is evidence that some toxic effects cannot be assessed on adult animals alone, particularly for neurotic effects.⁹² Third, the testing that is done on infant animals does not follow them over the duration of a lifetime even though there is evidence that some pesticides have degenerative effects that occur later in life.⁹³ Finally, EPA requires only a few tests that explore possible adverse effects of pesticides n the immune system or on their potential to disrupt the endocrine system.⁹⁴

The "scientific snapshots" of potential risks from the use of pesticides, like air toxics, is worrisome. In agricultural areas, pesticide residues in the dust in homes of farmers can be quite high, a fact that is attributed in part to "take home" contributions,⁹⁵ although researchers are still debating the health significance of this finding for children.⁹⁶ Some studies also identify a higher increased risk of a variety of tumors and cancers in families of farm works.⁹⁷

95. See, e.g., Cynthia L. Curl et al., Evaluation of Take-Home Organophosphorus Pesticide Exposure Among Agricultural Workers and Their Children, 110 ENVTL. HEALTH PERSP. 787, 790 (2002); Chensheng Lu et al., Pesticide Exposure of Children in an Agricultural Community: Evidence of Household Proximity to Farmland and Take Home Exposure Pathways, 84 ENVTL. RESEARCH 290, 298 (2000); Nancy J. Simcox et al., Pesticides in Household Dust and Soil: Exposure Pathways for Children of Agricultural Families, 103 ENVTL. HEALTH PERSP. 1126, 1131-32 (1995).

96. Researchers in one study, for example, did not detect higher pesticide concentration residues in the urine of children when the dust levels in their homes were as much as four times higher. Richard A. Fenske et al., *Children's Exposure to Chlorpyrifos and Parathion in an Agricultural Community in Central Washington State*, 110 ENVTL. HEALTH PERSP. 549, 551-52 (2002). In another study researchers did not detect significant correlations between pesticide residues in the home and the incidence of non-Hodgkins lymphoma: the residues were just as high in homes with persons without lymphoma. Mary H. Ward et al., *Proximity to Crops and Residential Exposure to Agricultural Herbicides in Iowa*, 114 ENVTL. HEALTH PERSP. 893, 896-97 (2006).

97. See generally Shelia H. Zahm & Mary H. Ward, Pesticides and Childhood Cancer, 106 ENVTL. HEALTH PERSP. SUPP. 893 (1998). The EPA is currently sponsoring various studies of pesticide exposures of children on the U.S.-Mexico border. These include a study to assess educational methods to reduce exposure, another study to examine methods for measuring neurotoxic effects in small children, and a longitudinal study measuring residues in the residue of children living on the border. See U.S. ENVIRONMENTAL PROTECTION AGENCY, U.S.-MEXICO BORDER 2012 ENVIRONMENTAL HEALTH WORKGROUP, http://www.epa.gov/ehwg/projects_publications.html (last visited Feb. 2, 2007). EPA is also supporting a project measuring "take home" exposure in agricultural areas of Washington. See U.S.

^{91.} Id.

^{92.} Id.

^{93.} Id. at 436.

^{94.} Id.

However, child exposure to pesticides may actually be highest in urban areas, where pesticides are used liberally to control household pests. Pesticide use records reveal that more pesticides are used in urban areas than agricultural areas, and in the urban setting, far more of the use occurs inside rather than outside the home.⁹⁸

Heavy applications of pesticides have been required in inner-city neighborhoods because of the age and poor maintenance of the urban housing stock. The resulting heavy exposure of inner-city children to pesticides is therefore a direct consequence of poverty, overcrowding, and poor housing and must therefore be viewed as yet another manifestation of the environmental injustice that these children suffer.⁹⁹

In one study, researchers found that "[t]he number of gallons of chlorpyrifos [used to control fleas, termites, and roaches] applied in Manhattan exceeded the total number of gallons of all pesticides applied in any other single county."¹⁰⁰ They further found that pesticide use in Harlem not only included high quantities of these two lethal pesticides (chlorphyrifos and organochlorine pesticides), but also illegal "street" pesticides.¹⁰¹ In a study of poor minority women living in or near New York City, "85% of the women report that pest control measures were used in the home during pregnancy" and "at least four pesticides [were detected] in the personal air samples of all women monitored during the third trimester."¹⁰² In fact, the poorer the household, the greater the pest risks and the greater the use of pest control measures.¹⁰³ Another study similarly identified relatively high levels of pesticide exposure to pregnant women in New York City.¹⁰⁴ This worrisome picture of pesticide use is not necessarily

103. Id. at 510, 513.

104. See Gertrud S. Berkowitz, *Exposure to Indoor Pesticides During Pregnancy in a Multiethnic, Urban Cohort*, 111 ENVTL. HEALTH PERSP. 79, 82-83 (2003). In that study, the reports of pesticide use did not correlate with pesticide levels in the urine, although the authors explain why at least the metabolite data may not be indicative of exposure. The study also found that a higher incidence of pesticide use by a household member was correlated with lower

ENVIRONMENTAL PROTECTION AGENCY, PROTECTING CHILDREN, http://www.epa.gov/pesticides/health/children.htm (last visited Feb. 2, 2007).

^{98.} See Landrigan et al., supra note 89, at 431.

^{99.} Id. at 436.

^{100.} Id. at 432.

^{101.} Id. at 432-33.

^{102.} Robin Whyatt et al., *Residential Pesticide Use During Pregnancy Among a Cohort of Urban Minority Women*, 110 ENVTL. HEALTH PERSP. 507, 512 (2002); *see also id.* at 513 (pointing out that only inhalation routes of exposure were considered even though there are other routes of exposure to household pesticides, including unintentional ingestion).

unexpected, but it is disturbing because of the adverse effects that result from uncontrolled pests: The scientific literature suggests a higher incidence of childhood asthma with exposure to cockroaches, for example.¹⁰⁵

As with air toxins, children are much more vulnerable to dangers posed by widespread unregulated use of pesticides than adults. First, the level of exposure to pesticide residues in household dust and other media are typically much higher for children than for adults since pesticides collect in plush toys, carpet, and dust which are not only more likely to be inhaled, but are in fact regularly ingested by children.¹⁰⁶ Children also have much higher biological vulnerability to pesticides than their adult counterparts.¹⁰⁷ Because their metabolic functions are still developing, they are unable to rid their bodies of pesticides in the ways that adults can.¹⁰⁸ Moreover, their development processes, which are more active than adults, can become easily disrupted and the adverse effects can be irreversible.¹⁰⁹ Of greatest concern for children, however, are the wide range of toxic effects, which include impacts on endocrine function, reproductive function, metabolic functions, and cognitive functioning. "There is evidence, for example, that pre- and postnatal [sic] exposures to pesticides increase risk of childhood cancer, and concern has arisen that early exposure to neurotoxic pesticides may increase risk in later life of chronic neurological diseases such as dementia, Parkinson's disease, and amyotrophic lateral sclerosis."¹¹⁰ Exposure to some pesticides during gestation or the early postnatal period reveals adverse neurological development in the children.¹¹¹

However, as the scientists writing the review articles and conducting individual studies readily concede, far more is not known about exposure to pesticides in the environment than is known and there is a compelling need for additional research.¹¹² This blind spot

education or minority status, although all women reported similar levels of pesticide use when the applicator included a third party. *Id.* at 81, tbl. 4.

^{105.} See, e.g., Peyton A. Eggleston et al., The Environment and Asthma in U.S. Inner Cities, 107 ENVTL. HEALTH PERSP. SUPP. 439, 441 (1999) (describing this literature).

^{106.} Landrigan et al., supra note 89, at 433.

^{107.} See generally NATIONAL RESEARCH COUNCIL, PESTICIDES IN THE DIETS OF INFANTS AND CHILDREN (1993).

^{108.} E.g., Landrigan et al., supra note 89, at 434.

^{109.} Id.

^{110.} Id. at 434-35 (citations omitted).

^{111.} E.g., Whyatt et al., supra note 102, at 507 (citing Landrigan et al., supra note 89).

^{112.} See, e.g., id. at 513-14; Zahm & Ward, supra note 97, at 905.

for environmental exposures again provides reason for concern; but, like air toxics, there is simply not enough data available to identify the extent of the risks or to identify the sub-areas or types of pesticide applications of greatest toxicological or exposure concern.

C. Communities with Inadequate Sanitation or Contaminated Drinking Water

Most Americans are aware of the tragic consequences of waterborne illnesses plaguing developing countries.¹¹³ Many, however, would be shocked to realize the number of rural and poor American families that lack potable water supplies or adequate wastewater treatment infrastructure.¹¹⁴ Moreover, a recent study by the NRDC revealed that many American cities have old and dilapidated water treatment infrastructure, resulting in widespread risk of exposure to chemicals and pathogens even among city residents.¹¹⁵ Contaminated drinking water and lack of adequate sanitation are among the direst environmental risks that children can face.

Nonetheless, federal environmental laws do virtually nothing to address health risks to families or small communities that rely on contaminated private wells or failing septic systems. While the regulatory gap is explained in part by federalism based constitutional limits on the reach of federal regulation, the health consequences of the gap can be significant, particularly for children. Inadequate wastewater services—like poorly built or inadequate private septic systems—create risk of exposure to dangerous pathogens like E. coli and the resulting threat of dysentery and other serious diseases.¹¹⁶

^{113.} A recent article in THE NEW YORKER magazine covered the issue extensively. *See* Michael Spector, *The Last Drop*, THE NEW YORKER, Oct. 23, 2006, at 61-71. *See also* Sharon Lafraniere, *Sub-Saharan Africa Lags in Water Cleanup*, N.Y. TIMES, Aug. 27, 2004, at A4.

^{114.} A 2001 EPA survey of drinking water infrastructure needs identified huge numbers of U.S. citizens and residents who lack access to safe drinking water. *See* U.S. ENVIRONMENTAL PROTECTION AGENCY, DRINKING WATER INFRASTRUCTURE NEEDS SURVEY 53-56 (2001), *available at* http://www.epa.gov/safewater/needssurvey/pdfs/2001/report_needssurvey_2001.pdf (estimating that 16 million people in the U.S. get water from sources other than public water supply systems, and that the adequacy of the quantity and quality of this water cannot be comprehensively assessed due to lack of governmental oversight).

^{115.} ERIK OLSON, NATURAL RES. DEF. COUNCIL, WHAT'S ON TAP: GRADING DRINKING WATER IN US CITIES at v-vi (2003), *available at* http://www.nrdc.org/water/drinking/uscities/pdf/whatsontap.pdf.

^{116.} E.g., JANE FRANKENBERGER, PURDUE EXTENSION SAFE WATER OFFICE, E. COLI AND INDIANA LAKES AND STREAMS, http://www.ecn.purdue.edu/SafeWater/watershed/ ecoli.html (last visited Jan. 29, 2007).

Children typically have higher exposure rates than adults because they spend more time outside, close to the contamination.¹¹⁷ At the same time, their immune systems are less developed and they are more susceptible to dehydration from exposure-related illnesses because of their small body size.¹¹⁸ Contaminated drinking water can also cause a wide range of adverse health effects, depending on the type of contamination. While most of the concern is focused on residual contamination from applied toxics, like pesticides and chlorinated compounds, naturally occurring contaminants such as arsenic, nitrates, and even sodium, can cause long-term damage to digestive tracts and heart functioning.¹¹⁹ Again, given their low body weight, their high consumption of drinking water, and their generally heightened vulnerability to toxins because of their developing metabolism, children are likely to be at greater risk from contaminated drinking water than adults.¹²⁰

In a scientific sense, the problems of private well contamination and failed septics are far more straightforward than the problems posed by environmental pesticide exposure or air toxics. Nevertheless, in practice these problems are at least as resistant to reform because of the high costs involved in remediation. For most of these environmental problems, the only recourse is to "upgrade" the septics or centralize the water supply, options which can be extremely costly, particularly for residents in rural areas that have

^{117.} See, e.g., Children's Environmental Health Network, An Introduction to the Field, *supra* note 5 (noting how children's exposures to contamination on land can be higher than adults due to behavioral characteristics of young children which include more time close to the ground, more hand-to-mouth activity, and more time outdoors).

^{118.} U.S. National Library of Medicine, Dehydration, MedlinePlus Medical Encyclopedia (Jun. 13, 2006), http://www.nlm.nih.gov/medlineplus/ency/article/000982.htm

^{119.} See, e.g., MICHAEL H. BRADSHAW & G. MORGAN POWELL, KANSAS STATE UNIVERSITY, SODIUM IN DRINKING WATER (2002), available at http://www.oznet.ksu.edu/ library/h20ql2/MF1094.PDF; see also BERNARD T. NOLAN, U.S. GEOLOGICAL SURV., MOVING FROM MONITORING TO PREDICTION: NATIONAL ASSESSMENT OF NITRATE IN GROUNDWATER (2005), available at http://water.usgs.gov/nawqa/briefing_sheet3.pdf.

^{120.} For example, children under six months old who are fed formula mixed with water contaminated with high levels of nitrates may develop "blue baby syndrome" or methemoglobinemia, which if left untreated can progress rapidly to coma and death. *See* Lynda Knobeloch et al., *Blue Babies and Nitrate-Contaminated Well Water*, 108 ENVTL. HEALTH PERSP. 675, 675-78 (2000). *See also* BRIAN A. COHEN ET AL., ENVTL. WORKING GROUP, JUST ADD WATER (1996), *available at* http://www.ewg.org/reports/JustAddWater/SDWA.html (describing infants' increased vulnerability and higher consumption of fluids relative to their weight).

settled in substandard conditions precisely because of the low cost of living.¹²¹

Since the options for improved health effectively require either extremely expensive upgrades or centralized services, some state and local governments with jurisdiction over these problems actively avoid inspections, particularly in poor communities.¹²² Enforcement of septic requirements in most locales could require violators to install new septic systems.¹²³ With a price tag ranging from several thousand to even ten thousand dollars for remediating a failing septic or contaminated well at a single residence, the price of fixing these health threats may exceed the value of the land and home combined, and in any event often prove beyond the reach of many of the residents who face these risks. Precisely because regulators do tend to turn a blind eye towards the problems, however, there is generally no effort to monitor for the risks nor is there an attempt to provide education about the risks. In at least one community, in fact, the county deliberately avoided monitoring overflowing septic systems and told residents that the systems were safe so that the county sanitarian would not be forced to take enforcement action, which may have required evicting residents from their property if they were unable to correct the problem.¹²⁴

123. See, e.g., 30 TEX. ADMIN. CODE § 285.70 (2007) (mandating that if a septic system is malfunctioning, "the owner shall bring the [system] into compliance by repairing the malfunction" and providing strict and short time limitations for compliance); see also ARK. CODE ANN. § 14-236-104(b) (2007) ("any individual sewage disposal system which is determined by the Division of Sanitarian Services of the Department of Health to be a health hazard or which constitutes a nuisance due to odor or unsightly appearance must conform with the provisions of this chapter and applicable rules and regulations within a reasonable time after notification that the determination has been made").

124. The Environmental Law Clinic at the University of Texas School of Law is assisting a community in which many septic systems are failing due to improper installation, insufficient surface area for the drain field, and an incompatibly high water table. In this community, however, the county sanitation officer regularly declined to test the standing water, because he did not want to initiate enforcement proceedings against residents who could not afford to fix or

^{121.} See Lemos M C, Austin D, Merideth R, Varady R G, 2002, Public - Private Partnerships as Ccatalysts for Community-based Water Infrastructure Development: The Border WaterWorks Program in Texas and New Mexico Colonias, 20 ENV'T & PLAN. C GOV'T & POL'Y 281 (2002).

^{122.} For example, the Texas Office of the Attorney General sued Maverick County in 1993 for its lax enforcement of the water code; the office also worked with Hidalgo County in 1997 for the county's failure to regulate developers under the water code, which had resulted in some new residences having only outhouses. TEXAS ATTORNEY GENERAL, HISTORICAL SKETCH OF TEXAS LAWS RELATED TO COLONIAS REMEDIATION AND PREVENTION (2003), http://www.oag.state.tx.us/border/history.shtml.

The scientific snapshots of existing exposures or adverse consequences associated with inadequate septics or drinking water in the United States reveal that these risks may again be significant. In many states, inadequate and failing septic systems remain a serious problem.¹²⁵ Leaking and pooled sewage from a failing septic systems can lead to significant public health concerns and substantial environmental degradation. Untreated human sewage may contains parasites and pathogens that cause an array of public health problems usually associated with developing countries, such as gastroenteritis, giardiasis, cholera, hepatitis, and dysentery.¹²⁶ Children are more susceptible to the diseases caused by these pathogens and parasites, because they are more likely to play in and around, or even ingest, the water pooling from failed septic systems.

The poor quality of drinking water supplies for many private residences appears still more problematic from a public health standpoint.¹²⁷ Risks from drinking water affect both homeowners who rely on private wells for their water supply and those who are connected to a public water supply system. Well water can be contaminated by natural occurring toxins such as radionuclides and nitrites as well as contaminates applied to the ground that seep into the groundwater, such as pesticides, fertilizers, and untreated sewage from failing septic systems.¹²⁸ While one might hope that the publicly owned water treatment facilities would ensure safe drinking water to those communities that rely on these facilities, in fact the water treatment infrastructure in the United States is old and breaking down, leaving large segments of the population at risk for consistent

replace their septic systems. Interview with Melinda Taylor, Director, Environmental Law Clinic, University of Texas School of Law (April 2, 2007).

^{125.} See, e.g., Sewage Treatment Systems: Ohio's Decentralized Wastewater Infrastructure (Ohio Department of Health) available at http://www.odh.ohio.gov./ASSETS/22E1223A9A9A4 E5B044E91BOA7AF61C/Infrstr%20FS.pdf (estimating the failure rate for septic systems in Ohio at 25% and cataloging the health consequences that flow from this high failure rate); Howard Frumkin, Lawrence Frank, and Richard Jackson, URBAN SPRAWL AND PUBLIC HEALTH: DESIGNING, PLANNING AND BUILDING FOR HEALTHY COMMUNITIES 133 (Island Press 2004) (recognizing the public health problems caused by failing septic systems).

^{126.} See O. BENJAMIN KAPLAN, SEPTIC SYSTEMS HANDBOOK (2d ed. 1991) (detailing the public health hazards of untreated sewage).

^{127.} Dennis M. Sievers & Charles D. Fulhage, *Survey of Rural Wells in Missouri for Pesticides and Nitrate*, GROUND WATER MONITORING REV., Fall 1992, at 142, 148, figs. 4 & 5, tbl. 7(illustrating that 22% of the rural northwestern Missouri wells sampled exceeded the EPA drinking water standard for nitrate, mainly due to chemical fertilizer).

^{128.} See U.S. ENVIRONMENTAL PROTECTION AGENCY, PRIVATE DRINKING WATER WELLS, http://www.epa.gov/safewater/privatewells/health.html (last visited Apr. 11, 2007).

exposure to low grade contaminants and episodic exposure to pathogens at dangerously high levels.¹²⁹

D. Other Legal Gaps with Potentially Significant Risks

There are other worrisome gaps in legal protection that might also engender significant risks to at least pockets of the country's population, which includes children. Because these risks are even more diffused and difficult to isolate, we have even less information available to assess their significance. Nevertheless, since the theme of the article is isolating and spotlighting legal blind spots, it is incumbent on us at least to identify these additional sources of unknown risks to children. The first set of risks arise from home chemicals, which receive little to no federal regulatory oversight, including toys, cleaning supplies, glues used for carpets, and paints (including old, leaded paint). A notoriously weak federal regulatory program not only allows risky products to be marketed, but rarely if ever demands even minimal testing for the products.¹³⁰ While there have been some important advancements-the banning of phthalates in toys, for example or the voluntary agreement by lumber treatment facilities to stop using CCA treated wood-the uncertainties surrounding most products and related household risks creates another large and diverse pocket of uncertain risks to children.¹³¹

A second worrisome source of exposure arises from contaminated land. Most of these exposures occur through ground water or onsite contact, but in some cases they can even involve inhalation.¹³² Currently, land contamination is generally discovered by chance, typically when government regulators are informed of the

^{129.} See What's On Tap? Grading Drinking Water in U.S. Cities (Natural Resources Defense Coucil 2003), *available at* http://www.nrdc.prg/water/drinking/uscities/contents.asp. In 1993, for example, more than 400,000 people were made violently ill by a tiny parasite in their tap water called *Cryptosporidium*. Several thousand were hospitalized and as many as 100 died. *Id.* at 1.

^{130.} See generally John S. Applegate, *The Perils of Unreasonable Risk: Information, Regulatory Policy, and Toxic Substances Control*, 91 COLUM. L. REV. 261, 303-05 (1991) (criticizing TSCA on these grounds).

^{131.} See generally Sarah Bayko, Note, *Reforming the Toxic Substances Control Act to Protect America's Most Precious Resource*, 14 SE. ENVTL. L.J. 245 (2006) (making this argument and promoting a more precautionary approach).

^{132.} See, e.g., U.S. ENVIRONMENTAL PROTECTION AGENCY, DRAFT REPORT ON THE ENVIRONMENT TECHNICAL DOCUMENT at 3-51 (2003), *available at* http://www.epa.gov/indicators/roe/pdf/EPA_Draft_ROE_TD.pdf (discussing inhalation as one of three routes of exposure to contaminated sites).

risks by private parties, such as neighbors or prospective purchasers.¹³³ While there is evidence of risks from land contamination to children and poor communities,¹³⁴ the uncertainties regarding this route of exposure overwhelm what is known, making it particularly difficult to evaluate its significance.

IV. IMPLEMENTING THE TRIAGE APPROACH: NEXT STEPS

Systemic and potentially catastrophic risks to children's health would seem likely to rise to the top of any rational risk priority framework, and to attract attention from the regulatory reform movement. Moreover, proposed regulatory responses to such risks should fare well in the political arena—after all, who would express opposition to safe drinking water for our nation's children?

A. Reforming the Reformers

Ideally, a first step to addressing these potential serious health risks would involve including the "legal blind spot" approach in a new and more comprehensive process for identifying regulatory priorities. The snapshots available on children's health risks suggest that there is good reason for heightened regulatory attention to this under-studied problem. Yet regulatory reformists, given their current quantitativedominated approach to regulatory analysis, have no way of accounting for or including these problems in their prioritization scheme. At the very least, future regulatory reform must explicitly acknowledge—even if it involves bracketing—problems, like the

^{133.} This is the scenario that unfolded in the Beatrice Foods scandal, chronicled in A CIVIL ACTION. *See* JONATHAN HARR, A CIVIL ACTION (1995). Companies such as Beatrice Foods can take such a position provided they do not have "knowledge" of the release of a reportable quantity of a hazardous substance, which is based on a daily rate of leaking that seems incapable of measure because of the passive nature of the release and in any event is likely to below reportable quantities because of the gradual leaching. *See* 42 U.S.C. § 9603(a) (2000) ("Any person in charge of a [vessel or] facility shall, as soon as he has knowledge of any release ... of a hazardous substance from such vessel or facility in quantities equal to or greater than those determined pursuant to section 9602 of this title, immediately notify the National Response Center ... of such release."); Designation of Hazardous Substances, 40 C.F.R. § 302.4 (2003) (listing reportable quantities of various hazardous substances).

^{134.} See, e.g., U.S. ENVIRONMENTAL PROTECTION AGENCY, AMERICA'S CHILDREN AND THE ENVIRONMENT: A FIRST VIEW OF AVAILABLE MEASURES 30-33 (2000), available at http://yosemite.epa.gov/ochp/ochpweb.nsf/content/ACE_Report.htm/\$file/ACE_Report.pdf (summarizing the high percentage of U.S. children living in counties with Superfund sites in 1990 and 2000 and their associated risks); see also ROBERT BULLARD, DUMPING IN DIXIE: RACE, CLASS AND ENVIRONMENTAL QUALITY (3d ed. 2000) (a classic work arguing that inequitable risks are imposed on poor and minority communities, including health risks arising from land contamination).

environmental risks to children, which suffer from inadequate research and deserve added regulation. Simply underscoring the dramatic limitations in and gaps resulting from current datadependent methods of regulatory analysis could go a long way to providing a more comprehensive picture of regulatory and research needs for the future.

But there are several reasons why these risks in their currently sketchy form are unlikely to be addressed at the federal level, and even once identified as potential problems, are unlikely to be addressed with meaningful federal requirements any time soon. The first and most obvious reason is because the risks remain uncharacterized. As any activist understands, political energy is catalyzed and the public's attention captivated by credible and highly salient risks.¹³⁵ General, dispersed, and relatively imprecisely unspecified concerns are not likely to lead to regulatory action.¹³⁶ Indeed, the most worrisome feature about the problems we highlight is that there is a potential for significant health risks to children, but we do not know their extent or severity because we are not monitoring or assessing the problems. The resulting Catch 22-an issue is not salient until the problem is monitored and characterized; but it is unlikely to be monitored until a political body takes actionleaves open the possibility that these problems will become selfperpetuating blind spots. Only voluntary and perhaps privately funded research by scientists might identify credible risks, which in turn can generate more political attention. Until then, the powerful inertia associated with ignorance may keep the issues beneath the political radar.

These problems may also resist political response because they primarily afflict poor communities, a group that is notoriously underrepresented in the political arena.¹³⁷ As Landrigan et al. summarize: "The six million children who live in poverty in inner cities in the United States [face disproportionately high] exposures to lead, air pollution, [pesticides], and hazardous waste sites, as well as [a] disproportionately high prevalence of lead poisoning and incidence of asthma....¹³⁸ Poor families rarely have the extra resources or time to represent their interests and cannot afford to

^{135.} See, e.g., Constance A. Nathanson, Social Movements as Catalysts for Policy Change: The Case of Smoking and Guns, 24 J. HEALTH POL. POL'Y & LAW 421, 442-55, 479 (1999).

^{136.} *Id.* at 422, 442, 446.

^{137.} See generally BULLARD, supra note 134.

^{138.} Landrigan et al., supra note 89, at 436.

contribute to or establish nonprofits to advocate on their behalf. As a result, their needs can drop out of legislative or administrative attempts to ensure adequate health protection. Moreover, even when the particular health and safety concerns of the underprivileged are known to policymakers, many factors may conspire to push these concerns to the back burner.

Finally, because the environmental threats we identify generally concentrate in localized areas, sometimes as small as a residence but rarely larger than a county, their redress is generally left to state or even local governments. However, because the solutions to these problems will typically impose costs on some of the states' most influential interest groups (as pesticide restrictions and air toxic controls would), states might be unable to overcome special interest resistance to protect their citizens. Again, some data indicating that the problems are real and national in scope would be needed to move these issues to the top of the federal agenda, but since this data is unlikely to be generated without regulatory requirements, we see another aspect of the Catch 22.

B. Relying on Universities and Law Schools to Take the Lead

Given the unlikelihood that the triage approach will gain purchase among reformists or within political movements, the next best opportunity for capitalizing on its promise is to encourage universities to incorporate its lessons into their research agendas and practicum offerings. In contrast to nonprofit and private sectors, universities actually receive some unique benefits from engaging in research and representation of poor communities, even when the risks are poorly specified and there is no possibility for direct political or financial benefits as a result of the engagement.

Most universities and particularly law schools are eager for "practice" opportunities, particularly for complex interdisciplinary problems raised by unspecified yet potentially serious health risks. The rapid growth of environmental law clinics in the nation's law schools is a testament to student demand and pedagogical recognition of the value of interdisciplinary, practice-oriented instruction. Universities are recognizing that they benefit from public outreach. State schools are most cognizant of these benefits since they often depend on state legislative support.¹³⁹ Providing evidence that they

^{139.} For example, in a speech to the University of Texas community, the president of the University at the time, President Faulkner, emphasized that the University "must find fresh

are "giving back" to the community can thus be a high priority. Private universities are also slowly responding to criticisms that they have become too insulated and seek ways to contribute their expertise to assist needy communities.

1. The Role of Universities.

The most logical vehicle for a university presence in researching these problems is an interdisciplinary practicum or clinic modeled after, or housed in, a law school clinic. Poor communities that face the unspecified risks highlighted in this paper are perfect clients for such a clinical exercise since addressing their problems will require complex problem-solving skills; considerable client contact; interdisciplinary teamwork; and legal research on local, state, and federal law. In fact, the features of these problems that make them resistant to political action are the same features that make them particularly attractive to universities: they involve complex research questions regarding risk characterization; they afflict unrepresented groups in need of assistance; and they are localized, often in pockets not too far from a university, and hence can be "tackled" by teams of professors and students. The fact that the communities are politically powerless and unable to donate or otherwise support research or advocacy on their behalf also make them more, rather than less, attractive to universities.

Since these unknown risks are spread out across the country and are complex, however, it is also prudent for universities to engage in some collective action or economies of scale, both in order to accomplish more community health protection and also to generate a larger, national picture of the scope of the problems. Ideally, then, universities that decide to address these unspecified environmental threats to children's health would form a larger organization that shared information, collaborated on strategies and approaches, and even attempted to make sure that their work was consistent enough to permit larger extrapolations or generalizations nationwide. This work of addressing the problems at a grander scale would provide yet another pedagogical benefit to the students. Through virtual or realtime conferences, perhaps arranged once or twice a semester, interdisciplinary teams at different law schools or universities could

ways to build public support among state leadership." New President Must Develop Stable Financial Model for UT, Faulkner Says in His Final Address on State of the University, ON CAMPUS, Sep. 30, 2005 (excerpting Faulkner's speech), available at http://www.utexas.edu/opa/ic/oncampus/2005/sept/faulkner.html.

share evidence and develop strategies. We thus recommend not only that universities dedicate themselves more explicitly and actively to these types of problems—particularly since there is no realistic hope that others will otherwise attend to them—but we also recommend a more unified front for law school clinics through collective action and collective organization.

2. The Work of Clinics.

Since rampant uncertainties pull particular, potentially important risks to children's health below the surface so they remain invisible and uninteresting to policymakers and the public at large, the clinics' job is to research these problems to bring them into sharper focus. If there turn out to be significant, unaddressed problems, universities of course cannot fix the problems by themselves; but their role in generating information could prove of pivotal importance in moving off the inertial state of ignorance that deters political action. Clinics can also work closely with individual communities to minimize the extent of their community-specific risks in the short and the longterm.

Specifically, we envision at least four separate, but concurrent roles for a clinic in ensuring better health protection for individual communities and generating more information on the nature and extent of these various, unspecified risks. The first role would involve interacting with the community to better identify the health risks from pesticide applications, air toxics, failed septic systems, or contaminated wells. In some cases, where the risks are significant enough to warrant immediate intervention, this role could also include actively educating the community about how to minimize certain types of risks. Rutgers, for example, is developing an educational videotape for poor urban communities in New Jersey about how to minimize urban exposures to pesticides.¹⁴⁰

Second, the clinic would seek to redress basic gaps in information, which could include working with the community to institute more vigorous monitoring. Since this role might be quite technical, there might be less room for community involvement in designing the information collection systems. Their local knowledge could help point to more specific locations in need of monitoring, like groundwater and air, but the nature of the contaminants, the

^{140.} See, e.g., MARK ROBSON, URBAN RESIDENTIAL IPM STRATEGIES (2006), http://www.epa.gov/oppbppd1/PESP/regional_grants/2003/r2-2003-final.htm .

frequency, and other decisions might need to be made independent from them and ideally could be unified across clinics.

Ultimately, if a community is willing and the science does suggest that a blind spot is serious and has lead to past and continuing harm, a clinic could also work as advocates for the community in the legislature, before a state or local agency, in the city or local government, or through the courts. The clinic's advocacy role would be determined by the community, but again would generate important information not only about the scientific nature of various risks but also about the community's own response to that information. Identifying realistic alternatives and communicating them to the community—for example, the grants and costs of centralizing septic systems versus upgrades versus the status quo—can involve a tremendous amount of research and effort and again provides an invaluable service as well as a tremendous educational experience for graduate students.

Finally, a clinic could work as an advocate at a broader scale on problems that appear to afflict many communities at once. This role would seem the least well defined, but we imagine that a collective national environmental law clinic project could establish a national board of experts (ideally mostly students) who could testify before Congress and work on national and model state legislation, as well as work with the EPA and other federal agencies on key projects. They might also provide more creative national or statewide reports or recommendations for reform based on their work. For example, as information increases about risks from certain types of pesticides in urban areas, as it did for chlorpyrifos and diazinon,¹⁴¹ the clinics could join to lobby EPA and Congress for more expeditious attention to these national risks that are best addressed at the national, licensing stage. This collective advocacy could also fill a nonprofit void in protecting the poor against "hot spots" of environmental risks.

Although it is not even two years old, our experience with an interdisciplinary clinic at the University of Texas Law School formed along the parameters we have outlined gives us some confidence that the model we propose is both realistic and promising. At the University of Texas, our interdisciplinary environmental law clinic dedicates most of its efforts to working with disadvantaged

^{141.} See Jim Morris, EPA Bans Pesticide Dursban, Says Alternatives Available, CNN.COM HEALTH, June 8, 2000, http://archives.cnn.com/2000/HEALTH/06/08/dursban.ban.02/ index.html.

281

communities to improve the quality of private drinking water and wastewater services and to address "hot spots" of air toxic contamination.¹⁴² Although the clinic currently can only manage a few "client" communities at one time, it has made impressive traction in assessing uncharacterized risks to the community and in identifying alternative steps that the community can take to minimize risk.

^{142.} THE UNIVERSITY OF TEXAS SCHOOL OF LAW, CLINICAL PROGRAMS, ENVIRONMENTAL LAW CLINIC, http://www.utexas.edu/law/academics/clinics/environment (last visited Feb. 12, 2007) ("The substantive focus of the clinic in the fall will be: the representation of several low income communities that currently lack access to reliable and sanitary wastewater disposal systems and clean drinking water; improving air quality for citizens in Southeast Houston who suffer from exposure to toxic air pollutants; and several water policy issues.").