JODY A. ROBERTS AND NANCY LANGSTON

toxic bodies/toxic environments: an interdisciplinary FORUM

NEW TECHNOLOGIES AND METHODS for the detection of toxins, particularly endocrine disruptors, have drawn increasing attention to the pervasive and persistent presence of synthetic chemicals in our lives. Some of these tests, such as biomonitoring and body-burden analyses, highlight that we not only experience our environment in obvious ways, but that we also are united with it at the molecular level. Trace chemicals found in the air, water, and soil are now being detected *within* us. The very chemical composition of our bodies is being altered in ways that reflect the transformations of our everyday environments.

Chemicals occupy a position along the border between the "natural" and "cultural" worlds. Industrial chemicals, in particular, prove difficult to categorize. They are artifacts of an industrial society brought into being within a highly specific cultural infrastructure. And yet they increasingly occupy a part of the natural world—and as persistent chemicals, many of them will continue to be a part of the world far into the future, beyond the point of remembering their origins as artificial or synthetic.

These landscapes, which now contain the various molecular traces of the industrialized world, are not simply environments that can be avoided—as we might once have tried with "contaminated" spaces like those around Chernobyl. These spaces are occupied by people, among others. They are landscapes of life, and therefore "landscapes of exposure."[1] Gregg Mitman, Michelle Murphy, and Christopher Sellers's collection, *Landscapes of Exposure: Knowledge and Illness in Modern Environment,* brought together the disparate threads of knowledge-

Jody A. Roberts and Nancy Langston, "Toxic Bodies/Toxic Environments: An Interdisciplinary Forum," *Environmental History* 13 (October 2008): 629-635.

making practices; knowledge in and of environments; and perceptions of health, illness, and disease. The collection emphasized the need to grapple with scale, materiality, and uncertainty—concepts that provide the bedrock for much of what follows in this forum.

In March 2007, the American Society for Environmental History (with funding from the National Science Foundation) brought together environmental scientists, historians of science, science studies scholars, and environmental historians to discuss the new chemical bodies of the twenty-first century. The workshop participants, many of whom contributed to *Landscapes of Exposure*, addressed the uncertainty that surrounds the fact that organisms of all types, kinds, and geographies—including but certainly not limited to humans—find themselves composed of a cadre of chemicals heretofore unknown to the planet. The problems of toxins in the environment are now inseparable from the issue of toxins in us.

This special forum in *Environmental History* continues that lively discussion. In these brief reflection essays, sciences studies scholars, historians of science, and environmental historians provide perspective on the failures of existing toxicological frameworks. While disciplines, topics, and actors differ, there is a surprising amount of cohesion among these works. Four main themes emerge: the uncertainty of knowledge, the place of knowledge production, the politics of dealing with environments and bodies, and the historical roots of current toxicological frameworks. For at least the last three decades, historians and social scientists have worked to uncover the ways in which scientific knowledge is constructed. But in dealing with the crisscrossing issues of environmental pollution, human and nonhuman exposure, and toxicity, the problem is not necessarily with what we know, but with all that remains unknown. We don't often think about this shadow space to our collected knowledge—at least not in those terms. We speak, instead, of uncertainty. Uncertainty implies an aspect of failure: we tried to understand, but certainty unfortunately eludes us. The concept of uncertainty, then, raises questions about the politics of neutrality.

Sometimes the difference between what we know and don't know is simply a matter of asking the right questions. As Michael Egan recounts in his stories on the presence of mercury in the environment and in bodies, mercury and its more potent organic version, methylmercury, were already widespread in much of the industrial world long before anyone ever bothered to look. While countries such as the United States claimed that they did not have a mercury pollution problem, authorities found themselves making an abrupt about-face after actually looking for it in the nation's waterways. What had seemed like an isolated incident in Minamata, Japan, suddenly became one case in a larger, more widespread public health dilemma.

When a regulatory agency identifies scientific uncertainty about the effects of a particular chemical, the typical response is to call for "more research." Yet as Scott Frickel argues in his essay, that strategy has clear political outcomes. What if we spoke not of uncertainty, but instead of "knowledge gaps" or "lost knowledge," as Scott Frickel does in his essay "On Missing New Orleans"—how

would this change our perspective on what it is we know and don't know about our environments, our bodies, and the transformations each are undergoing?

Frickel's essay explores the ways in which ignorance and nonknowledge are actively produced in post-Katrina New Orleans. Frickel highlights the ways in which the tools used to examine the construction of knowledge also can be used to probe the knowledge gaps that coexist alongside the vast expanses of what we claim to know. By transitioning from uncertainty to the "institutionalization of ignorance," Frickel reinserts politics back into the picture, and the collective "we" can no longer hide behind the veil of uncertainty. Making the nonproduction of knowledge visible and hence political allows us to explore the ways in which information is kept out of reach by the efforts made not to produce it in the first place.[2]

Another important theme that highlights the ways in which land and bodies are united echoes the work that historians of environments and sciences have pursued for years: the transitions between labs and fields as sites for investigation.[3] Labs are neat, they are clean, and they are orderly. The field, the lab's antithesis, is messy, complicated, and chaotic. Understanding how the cultivated practical knowledge of the laboratory interacts with the dynamic space of the outside world requires an appreciation for the ways in which the artifacts and knowledges that link these places circulate.[4]

In Linda Nash's essay, the circulating artifacts are the bodies of the farm workers. Moving across fields and spaces, their bodies resist the reduction that lab-based science requires for understanding toxicity. Precisely because their bodies circulate, they also accumulate—traces of the specific chemicals applied in each field find their way into the farm workers' bodies. As Fritz Davis points out in his essay, modern toxicology has struggled with calculating effects of multiple chemical exposures. Experimental researchers find it far simpler to focus on the dose-response mechanism for one chemical at a time. But bodies in the field don't have just one chemical in them at a time; they are multiply exposed. Accounting for the synergistic effects of these chemicals presents a challenge to the lab, but mirrors reality in nature.

The Union Carbide methylisocyanate release in Bhopal, India, in 1984 that killed thousands in the first few hours and potentially scores of thousands more since then highlighted the ways in which bodies, environments, and the politics of the local and global can merge at a single point in time and space. The event is widely acknowledged as the worst industrial disaster in history. And yet, as the residents of Institute, West Virginia—where Bhopal's sister plant was located—as well as those of fence line communities throughout the United States quickly realized, releases don't have to emerge out of a single moment to be catastrophic. A slow constant poisoning of the air, water, and soil can lead to the same outcome, but without the dramatic effect. These realizations led to the creation of so-called "right to know" legislation—the right to know what's being produced next door, what's being released, and what to do when the alarm at the plant sounds.

Right-to-know legislation is premised on the idea that indeed someone does know, and ought to be required to share that knowledge. But as Barbara Allen

points out in her essay, it's easy to subvert right to know by simply preventing anyone from knowing at all. That is, the nonproduction of knowledge works to disrupt the fundamental flows of information that provide the basis for regulatory oversight (at least in the United States). She notes two ways in particular that this occurs: through the active nonproduction of knowledge related to exposure and health, and through the removal of archival sources, particularly the recent attempt by the U.S. Environmental Protection Agency (EPA) to close libraries containing several decades worth of environmental health information.[5]

The attempted closing of the EPA libraries presents a more obvious example of the destruction of knowledge—one that should alarm historians and activists alike. The libraries are an essential tool for understanding what we know as well as what we don't know and for motivating action on the part of communities and governments alike (as Allen, Egan, Frickel, and Nash all highlight). Advocacy efforts by the American Society for Environmental History, the Society for Environmental Journalists, and the American Library Association helped persuade Congress to order a halt to the library closures in December 2007. The protection not only of right to know but the ability to know is what links environmental history with ecology, public health workers, citizen groups, and regulators alike. Increased public awareness of exposures has always been a driving impetus in changing and modifying regulatory policies.

Perhaps the most important contribution, and one most intimately tied to the readers of this journal, involves our efforts to tease out the origins of the problems now emerging that have once again highlighted the intimate links between bodies and their environments. Understanding the historical contingencies that have made the present moment what it is allows us to think creatively about how things could have been, and can be different. While all of the contributors to this forum have historical roots, three in particular present very different perspectives on the construction of the modern landscape of exposure.

The chemical plasticizer bisphenol-A (or BPA) has begun to enter the common vernacular. But as Sarah Vogel demonstrates in her essay, our knowledge of the toxicity of the chemical has a long, if underappreciated history. Chemists working furiously in the wake of World War II sought to capitalize and profit from the widespread acceptance of synthetics into our lives. Plastics would be the modern marvel of the twentieth century. At the same time, the chemistry behind plastics progressed with little understanding of the consequences. Drawing on the reflections of the sixteenth century proto-toxicologist Paracelsus, plastics chemists believed that the "dose makes the poison." But the story of BPA is a challenge to that notion, and to that history. Vogel notes that even in its early years, BPA already exhibited the properties that have now made it the center of scientific and regulatory controversy. Teasing apart these intertwined histories is creating a space for moving from the "dose makes the poison" to the "timing makes the poison," which in turn is opening up possibilities for new mergers of toxicology and regulation.

While Vogel's essay traces the development of a specific compound through the changing terrain of toxicology, Nash and Frederick Davis look back even further, casting their gaze onto fields of study that have come to dominate the toxicological terrain that unites bodies and environments. Nash argues that modern concepts about health and environment emerged from the germ-theory of health and bacteriology, while in his essay, Davis sees the history running through pharmacology (which in any case would help us link back to Paracelsus). Davis argues that toxicology developed from pharmacology and chemistry, newly empowered in the wake of the world wars and armed with new understandings about chemicals and their designed and accidental effects on organisms of all types. Nash's history offers us a narrative that helps to explain why causal mechanisms have remained so important in current toxicology, despite the fact that they are almost always elusive. Both Nash and Davis contribute to a genealogy of contemporary toxicological sciences, whether the focus is on cancer in mice or cancer in humans; pesticides in plants or in the bodies of farm laborers.

While the essays in this forum cover much new territory, the overlap is not always neat. Tensions exist, for instance, in the ways in which the authors view the historical roots of our current situation. Where Davis sees a linear progression from wartime chemistry to toxicology, Nash sees a convergence of theories and practices giving rise to a situation where only certain types of knowledge are possible. The differences may seem trivial at first, but they have serious implications if we view untangling the past as an important precursor for creating the future.

Indeed, understanding how these current problems will unfold into the future leads to further friction. Take, for instance, the essays by Frickel, Michelle Murphy, and Arthur Daemmrich. All three discuss the possible implications of more widespread use and adoption of biomonitoring data, but they see the possible outcomes of the creation of this information in quite different ways. For Frickel, concerned about nonknowledge production as much as knowledge production, biomonitoring might potentially be used to further highlight environmental injustices and the links among landscapes, exposures, and health. But this will only happen if the tool is used properly: that is, if users ask the right questions and ensure the flow of information to those who can use it.

Murphy explores the possible "molecularization of life" involved in biomonitoring, questioning the individualization of risks that follow efforts to detail synthetic chemicals inside individual bodies. With the emergence of what she calls a chemical regime of living, what new molecular relations might be formed? Biomonitoring has the potential to lead to social change, but it also has the potential to further privatize risk and lead to "boutique" medicine for the privileged.

Daemmrich has a radically different view of the future of biomonitoring. For Daemmrich, as biomonitoring studies highlight the extent of chemical contamination in the public writ large, risk will become a concept increasingly associated with the masses, generally, and with affluence, more particularly. That

is, if exposure increases with consumption of everyday products, then does it follow that those who consume more are at greater risk? Daemmrich refers to this as a democratization of risk since the chemical traces can now be found in nearly everyone. If true, what will this situation mean for environmental justice advocates whose primary argument has always been one of unevenly distributed risk? Or, as Frickel responds, is this a red herring, distracting us from the lived experience of those still positioned along the fence lines of the industrial refineries? The outcome is unclear, but it is safe to say that the relationships between environment, health, justice, and power are being redefined as we come to a different understanding of our chemical selves in this chemical world.

THE ESSAYS IN THIS FORUM speak many languages, but with common voice they call upon historians to provide their skills in uncovering a history of the present. While the history itself needs to be told, there is a much larger, more important need to bring these stories to light. Nash sums it up best in the conclusion of her essay: "Rather than hoping that increased scientific knowledge will—like some deus ex machina—reveal the solutions to contemporary problems, we might instead insist upon the need to consider more critically the cultural models and historical assumptions that guide contemporary regulatory policy." Our hope is that this forum will help catalyze such an effort by enrolling historians in a project that at once elucidates the past while working to reconfigure the future.

Jody A. Roberts *is Environmental History and Policy program manager at the Chemical Heritage Foundation's Center for Contemporary History and Policy in Philadelphia.* ***Nancy Langston*** *is professor in the Nelson Institute for Environmental Studies and the Department of Forest and Wildlife Ecology at the University of Wisconsin-Madison. She has recently completed an environmental history of endocrine disruptors, titled* Toxic Bodies: Endocrine Disruptors and the Lessons of History*, which is forthcoming from Yale.*

NOTES

The authors thank the National Science Foundation for its generous support of both the workshop and this forum, the workshop participants and forum authors for their hard work, ASEH Executive Director Lisa Mighetto, for her logistical support, the editor of *Environmental History*, Mark Cioc, and the external reviewers for their thoughtful comments.

1. See Gregg Mitman, Michelle Murphy, and Christopher Sellers, eds., *Landscapes of Exposure: Knowledge and Illness in Modern Environments, Osiris*, vol. 19 (Chicago: University of Chicago Press, 2004).
2. A similar call was made by Peter Rogers, a Harvard University professor of environmental engineering, when a series of articles by the Associated Press drew attention to the presence of pharmaceutical agents in the nation's tap water. "I think

the government and utilities are quite right to be very skittish about telling people their results. People will claim it's causing all sorts of problems. If I were a water utility, I would stop those measurements right away because if you measure something, it will get out, and people will overreact. I can just imagine a whole slew of law suits." See, for instance, "Water providers, researchers rarely release full test results," *USA Today,* March 10, 2008.

3. See, for example, Robert Kohler, *Landscapes and Labscapes: Exploring the Lab-field Border in Biology* (Chicago: University of Chicago Press, 2002).
4. Bruno Latour, *Pandora's Hope: Essays on the Reality of Science Studies* (Cambridge: Harvard University Press, 1999), ch. 2.
5. For background on the EPA library closures and the ASEH resolution opposing these closures, see http://www.aseh.net/resources/advocacy/epa_resolution. For updates on Congressional action, see http://www.aseh.net/resources/advocacy/congress-epa.

MICHAEL EGAN

FORUM
toxic knowledge: a mercurial fugue in three parts

IN A PROVOCATIVE DISCUSSION on the nature and historicity of scientific knowledge, Bruno Latour asks: "Where were microbes before Pasteur?" He concludes: "*after* 1864 airborne germs were there all along," which presents the historian with an interesting portal into the history of scientific knowledge and its relationship with environmental politics. The history of toxic environments is largely reactionary in nature: the framing of new environmental standards comes in response to the discovery of hazards and those standards are frequently revised as new information becomes available. Reactionary history and the changing contexts of awareness of toxic hazards are suggestive of what Latour called the "historicity" of scientific knowledge: "History not only passes but transforms."[1] Scientific discoveries alter our reading of the past. Drawing on a similar epistemological trope, this essay surveys the histories of knowing and unknowing surrounding a series of confusions related to the discovery of mercury contamination in rivers and lakes in the northern hemisphere between the 1960s and 1980s. In so doing, it offers an index toward thinking historically about toxic bodies and toxic environments.

Chemistry is the science of material change and the scientific knowledge developed to understand these changes offers an opportunity for environmental historians to tell stories about stories about nature. Chemical knowledge has been pivotal in human interactions with nature, and the accounts that follow rely

Michael Egan, "Toxic Knowledge: A Mercurial Fugue in Three Parts," *Environmental History* 13 (October 2008): 636-642.

heavily on the polity of a constructed scientific knowledge. In addition to the prospect of chemistry offering insight into constructions of science as they relate to environmental history, however, one might also recognize the material significance of chemicals to environmental narratives. Studying landscapes in which various natural and synthetic chemicals come together to form insalubrious settings for organic beings also presents new opportunities for considering nonhuman agency in our environmental histories. In all three snapshots of toxic environments that I present in discussing mercury pollution, mercury's chemical make-up undergoes changes that are only partly influenced by human activities. Weaving together narratives of chemical knowledge and toxic environments, then, offers ways of complicating our environmental histories.

The most common form of mercury poisoning involves methylmercury, an organic mercury compound that accumulates in humans and animals and acts as a highly dangerous neurotoxin. The issue that plagued the scientists from Sweden, Canada, and the United States, whose research comprises the main thrust of this work, was that methylmercury was appearing in freshwater systems where it did not belong. The absence of a scientific rationale for methylmercury's presence in the places it was being discovered mystified researchers until a number of breakthroughs in understanding resolved their confusion and painted a troubling canvas of the complexity and severity of the global mercury pollution problem.

In the early 1950s, Swedish conservationists observed a marked reduction in the populations of seed-eating birds while also encountering more bird carcasses around the countryside, which were found to contain staggering amounts of mercury. By 1960, predatory birds also were found to have elevated levels of mercury in their systems. The high mercury content ultimately was traced to the use of mercury in agricultural fungicides and the treatment of seedgrain.[2] During the investigations into the source of mercury in birds, scientists began considering the repercussions if mercury used in agriculture should find its way into freshwater systems. According to one account on the Swedish response to mercury pollution, "not much imagination was needed to realize the potential hazard to human health of the mercury in fish."[3] In 1964, teams of scientists began taking samples of fish from several bodies of freshwater in Sweden.[4] In short order, they found alarmingly high levels of methylmercury, the quantities of which indicated that they could not be attributed solely to the mercury treatment of seedgrain.[5]

In Sweden, scientists knew that mercury was emitted into rivers and lakes from three industrial sources. The paper mills used phenylmercury to prohibit the formation of mucus in the paper machines; the pulp industry also used phenylmercury to protect wet mechanical wood pulp from mould fungi; and the chloralkali industry emitted ionic mercury in its electrolysis wastewater. These inorganic forms of mercury were relatively nontoxic, unlike the methylmercury that had been discharged into the waters near Minamata in the early 1950s. And yet, methylmercury—responsible for the devastating cases of mercury poisoning

in Japan—was prevalent throughout Swedish river systems. In 1966, acting on suspicion rather than evidence, several different Swedish researchers proposed that methylmercury was somehow created by bacterial action. Microbial activity in the mud on lake bottoms, they posited, could methylate inorganic and metallic mercury. The following year, Sören Jensen and Arne Jernelöv confirmed this hypothesis, showing that bacteria methylated mercury in anaerobic (oxygen-free) ecosystems, but they could not explain how.[6] By 1968, another team of scientists found that microorganisms in the sediment of river and lake bottoms metabolized inorganic and metallic mercury, and excreted them as methylmercury.[7] While the scientific work constituted an important breakthrough in understanding mercury's characteristics in the environment, the repercussions of the discovery were devastating. No matter what its form—or however benign—when entering the ecosystem, mercury now constituted a serious threat to human health. If biological systems could convert less harmful mercury compounds into a harmful, lipid-soluble form—methylmercury—then mercury use in industry posed grave and long-term health problems.

IN JUNE 1968, A CONFERENCE on the toxicity of persistent pesticides was convened at the University of Rochester. Among those invited were several of the scientists whose research had illuminated the severity of the mercury problem in Sweden. As they presented their results on mercury contamination in Swedish waters, American participants tried to identify the sources of Swedish exceptionalism. Why had Sweden suffered from such a calamitous pollution problem when other countries—and especially the United States—had not? When one biologist, Thomas Clarkson of the University of Rochester, hypothesized that mercury compounds might have been used in Sweden longer than they had in other countries, Alf Johnels of the National Museum of Natural History in Stockholm corrected him, stating that Sweden had copied American industrial mercury practices. Mercury compounds in chloralkali production had been used for longer and in significantly greater proportions in the United States.[8] Indeed, where Sweden lost almost 20,000 kgs of mercury to the environment in 1967, American industry and agriculture lost an estimated 600,000-650,000 kgs.[9] So what was it, then? The northern climate, perhaps? The geography? Some speculated that Sweden's archipelagoes kept water from circulating. Indeed, some present made the connection between the Swedish incident and the massive mercury poisoning at Minamata in Japan, also a geography of protected sea waters. Gently, the Swedes and some of the more concerned American scientists suggested the real distinction between Sweden and the United States stemmed from the fact that Sweden actually was looking for mercury.[10]

Sure enough, in 1969, shortly after the Rochester conference, the Canadian Department of Fisheries and Forestry banned commercial fishing catches from a number of lakes and rivers in Manitoba. More than a million pounds of fish that contained mercury in quantities of 5 to 10 ppm—ten to twenty times the government-ordained action level—were confiscated and destroyed.[11] Then, in March 1970, Norvald Fimreite, a zoology student at the University of Western

Ontario, reported to the Canadian Department of Fisheries and Forestry that he had found 7.09 ppm mercury in pickerel from waters that fed Lake Erie. Fimreite's discovery prompted rapid action from the Canadian government, which identified chloralkali plants as the source and forced them to eliminate mercury from their operations. In addition, the government banned the taking of fish—sport or commercial—in the area, and threatened polluters with legal action. All this within a month of Fimreite's letter. In *The Closing Circle*, the biologist Barry Commoner remarked that Fimreite undoubtedly held "the world record ... for the fastest, one-man, large-scale ecological action."[12]

Ultimately, the reason mercury contamination had not become a serious ecological problem in the North American context had everything to do with the fact that nobody was looking for it, which raises some interesting questions surrounding the sociology of scientific knowledge and its role in defining toxic knowledge and ecological problems. ("Where were microbes before Pasteur?") What the Swedish and Canadian lessons taught was that when organic pollutants enriched river systems, the nutrients fed aquatic plants and microbes would thrive and methylate more mercury. One of the major environmental projects of the late 1960s and early 1970s was the reduction and—in many cases—elimination of mercury from industrial production. While Canada and the United States set mercury limits at 500 ppb in the aftermath of Swedish contamination and at the outbreak of their own nightmares, by 1976, the World Health Organization had determined that 200 ppb might serve as a better threshold for the concentration of methylmercury required to yield the classic symptoms of Minamata disease. That number was reduced by a factor of ten in 1990 to 20 ppb.[13]

IT WAS DURING AND AFTER THIS cleanup that another methylmercury mystery presented itself. In 1975, a team headed by University of New Mexico biologist Loren Potter sought to establish baseline levels of mercury in predatory fish in the Lake Powell reservoir in order to predict the effects of future industrial and recreational developments. Lake Powell was a Bureau of Reclamation storage and hydroelectric generation reservoir, initially impounded in 1963. It served as a good test site, the subsequent article argued, because it was "a new reservoir remote from major man-caused pollution sources."[14] Their findings revealed disproportionately high levels of mercury at the top of the artificial lake's food chain. Walleye taken from Lake Powell averaged 427 ppb mercury in their axial muscle, and bass averaged 314 ppb. In the aftermath of the Swedish alarm, both Canada and the United States had set acceptable limits of 500 ppb mercury in commercial and recreational fish consumption. The predatory fish in Lake Powell were just under that limit, but Potter and his colleagues were astonished to discover that the mercury levels were that high; given the absence of industrial pollutants, the mercury levels should have been substantially lower. "Due to bioamplification, mercury concentrations of nonacceptable amounts by FDA standards are being approached in the higher trophic levels," they warned. "If a mercury content above 500 ppb is confirmed as common to the muscle of large game fish, mercury levels could become a significant factor in the management

of the Lake Powell fishery."[15]

The mercury content in Lake Powell was not an isolated incident. In 1977, environmental engineers identified high levels of methylmercury in largemouth bass in three new reservoirs on the Savannah River and its tributaries in western South Carolina.[16] Similar discoveries were made in Finland and in northern Quebec, Manitoba, and Labrador, all in sites with little or no industrial pollution.[17] Following on from the Swedish studies, Frank D'Itri, a water chemist at Michigan State University, had proposed that the high volatility of mercury explained the contamination of fish located far from industrial mercury emissions. Mercury's volatility—its tendency to pass into a gaseous state—suggested its ability to travel significant distances by air.[18] This is what made mercury such a serious environmental hazard; its capacity to travel in the air and pollute not just local waters, but distant waters as well. But while methylmercury was accumulating in disproportionate quantities in human-made hydroelectric reservoirs, adjacent, unimpounded lakes did not show concomitant signs of increased mercury burden. If distant sources of pollution—coal-fired power generators, pulp and paper mills, and chloralkali manufacturers—were the cause of methylmercury deposits as D'Itri had posited, then why were they concentrating in new reservoirs and not elsewhere to the same extent?

In the early 1980s, Canadian researchers demonstrated that methylmercury bioaccumulation in nonpolluted reservoirs was not the product of distant industrial activity, but resulted from microbial activity on flooded, decaying organic matter that contained inorganic mercury.[19] Rising water levels in new reservoirs enveloped naturally occurring mercury present in the terrestrial environment and also flooded vegetation and soils rich in organic carbon. Their decomposition created the conditions through which the microbial methylation of inorganic mercury was fueled.[20] Just as in the Swedish example, the discovery of inorganic mercury in the environment stressed its hazardous potential when methylated. But unlike the Swedish example, in the instance surrounding impounded lakes, natural mercury deposits interacted with artificially flooded reservoirs to introduce methylmercury; humans had not introduced mercury into the ecosystem. This discovery prompted the continuing study of mercury methylation in shallow marshes and natural wetlands where the flooding of vegetation occurs without human influence.

IN CONCLUSION, some comments or observations, which aim to situate the history of toxic environments more firmly within the purview of environmental historians and their historiographies. The short of it is this: beyond drawing on themes like health, pollution, and the hubris of new and ambitious technologies, histories of toxicological sciences and politics provide environmental historians with an interesting opportunity to engage with themes of natural agency in heretofore unexamined ways. A seductive, intellectual paradox exists in the nature of the methylation of mercury in hydroelectric reservoirs; the *containment* of water in the reservoirs resulted in the *release* of another natural phenomenon. Mercury's transition from elemental isolation to unwelcome ecological

integration offers an intriguing blend of human and natural partnerships of the sort that make environmental history an important avenue for historical and environmental inquiry. On the one hand, my accounts of the release of methylmercury belong to a long and well-documented history of the tragedy of unintended toxic consequences spurred by technology and visions of progress. On the other, they offer an interesting opportunity to engage with themes of natural agency in heretofore underexamined ways. Mercury has a nature. Its transmutation into the toxic methylmercury when it "communicates" with microbes in polluted water systems and in hydroelectric reservoirs occurs at a curious intersection between human and nonhuman activity. The construction of hydroelectric reservoirs provides the context for this communication, but the creation of methylmercury is a distinctly nonhuman occurrence that alters human and ecological landscapes. In mercury's transformation into and release as a toxic vapor, nature suggests an agency that palpably shapes how mercury and humanity mix.

Michael Egan *is assistant professor in the Department of History at McMaster University. He is the author of* Barry Commoner and the Science of Survival: The Remaking of American Environmentalism *(MIT, 2007).*

NOTES

1. Bruno Latour, *Pandora's Hope: Essays on the Reality of Science Studies* (Cambridge: Harvard University Press, 1999), 145, 173 (my emphasis), 306. See also Scott Kirsch, "Harold Knapp and the Geography of Normal Controversy: Radioiodine in the Historical Environment," *Osiris* 19 (2004): 167-81.
2. For an overview, see Lennart J. Lundqvist, *The Case of Mercury Pollution in Sweden: Scientific Information and Public Response* (Stockholm: Swedish Natural Science Research Council, 1974), 12-25. See also Katherine and Peter Montague, *Mercury* (San Francisco: Sierra Club Books, 1971), 34-37; and Patricia A. D'Itri and Frank M. D'Itri, *Mercury Contamination: A Human Tragedy* (New York: John Wiley & Sons, 1977), 29-34.
3. Lundqvist, *The Case of Mercury Pollution in Sweden*, 29.
4. Ibid., 26.
5. T. Westermark, *Kvicksilverfrågan i Sverige* (Stockholm: Ministry of Agriculture, 1965), 25. See also Gunnel Westöö, "Methylmercury Compounds in Fish: Identification and Determination," *Acta Chemica Scandinavica* 20 (1966): 2131-37.
6. Sören Jensen and Arne Jernelöv, "Biosyntes av Metylkvicksilver I," *Biocidinformation* 10 (May 1967): 4-5. See also Jensen and Jernelöv, "Biosyntes av Metylkvicksilver II," *Biocidinformation* 14 (February 1968): 3-5. Jensen and Jernelöv published their findings in English in *Nature* in 1969. See Jensen and Jernelöv, "Biological Methylation of Mercury in Aquatic Organisms," *Nature* 223 (August 16, 1969): 753-54.
7. J. M. Wood et al., "Synthesis of Methyl-mercury Compounds by Extracts of a Methanogenic Bacterium," *Nature* 220 (October 12, 1968): 173-74.
8. Morton W. Miller and George G. Berg, eds., *Chemical Fallout: Current Research on Persistent Pesticides* (Springfield, IL: Charles C. Thomas Publishers, 1969), 91.
9. A. Hanson, "Man-made Source of Mercury," in *Mercury in Man's Environment* (Ottawa: Royal Society of Canada, 1971); and R. L. Mitchell, "Mercury Consumption Survey, April

10, 1969," The Chlorine Institute, 1969.
10. Miller and Berg, eds., *Chemical Fallout*, 90-93.
11. E. G. Bligh, "Mercury and the Contamination of Freshwater Fish" (Winnipeg: Fisheries Research Board of Canada, Manuscript Report # 1088, 1970); and G. Wobeser et al., "Mercury Concentration in Tissues of Fish from the Saskatchewan River," *Journal of the Fisheries Research Board of Canada* 27 (1970): 830-34.
12. Barry Commoner, *The Closing Circle* (New York: Alfred A. Knopf, 1971), 202.
13. World Health Organization, *Environmental Health Criteria 1: Mercury* (Geneva: World Health Organization, 1976).
14. Loren Potter et al., "Mercury Levels in Lake Powell: Bioamplification of Mercury in a Man-made Desert Reservoir," *Environmental Science and Technology* 9 (January 1975): 41-46, quotation on 41.
15. Potter et al., "Mercury Levels in Lake Powell," 44.
16. A. R. Abernathy and P. M. Cumbie, "Mercury Accumulation by Largemouth Bass (*Micropterus salmoides*) in Recently Impounded Reservoirs," *Bulletin of Environmental Contamination and Toxicology* 17 (1977): 595-602.
17. For Finland, see Martin Lodenius et al., "Accumulation of Mercury in Fish and Man from Reservoirs in Northern Finland," *Water, Air, and Soil Pollution* 19 (1983); 237-46; M. Verta et al., "Causes of Increased Mercury Levels in Finnish Reservoirs," Publications of the Water Research Institute, National Board of Waters, Finland 65 (1986), 44-58; and P. Porvari, "Development of Fish Mercury Concentrations in Finnish Reservoirs from 1979–1994," *Science of the Total Environment* 213 (1998): 279-90. For Canada, see R. A. Bodaly et al., "Increases in Fish Mercury Levels in Lakes Flooded by the Churchill River Diversion, Northern Manitoba," *The Canadian Journal of Fisheries and Aquatic Sciences* 41 (1984): 682-91; R. Boucher et al., "Teneur en mercure des poissons des réservoirs La Grande 2 et Opinaca (Québec, Canada) avant et après la mise en eau," *Revue Française des Sciences de l'Eau* 4 (1985): 193-206; and W. J. Bruce and K. D. Spencer, "Mercury Levels in Labrador Fish, 1977-78," *Canadian Industry Report of Fisheries and Aquatic Sciences* 111 (1979): 1-12.
18. Frank D'Itri, *The Environmental Mercury Problem* (Cleveland: The Chemical Rubber Company Press, 1972), 89-90.
19. Bodaly et al., "Increases in Fish Mercury." See also R. E. Hecky et al., "Increased Methylmercury Contamination in Fish in Newly Formed Freshwater Reservoirs," in *Advances in Mercury Toxicology*, ed. T. Suzuki et al. (New York: Plenum Press, 1991), 33-52.
20. B. D. Hall et al., "Impacts of Reservoir Creation on the Biogeochemical Cycling of Methyl Mercury and Total Mercury in Boreal Upland Forests," *Ecosystems* 8 (2005): 248-66.

SCOTT FRICKEL

FORUM
on missing new orleans: lost knowledge and knowledge gaps in an urban hazardscape

AS A RULE historians, philosophers, and sociologists of scientific knowledge study knowledge *making*; seldom do scholars study the nonproduction of knowledge or the creation of knowledge gaps. Yet scientific work involves the interplay of these two countervailing processes, and answers to questions concerning what kinds of scientific knowledge get made by who, where, and for what purposes hinge also on "undone science" and the consequent institutionalization of ignorance.[1] This forum on "Toxic Environments/Toxic Bodies" provides an opportunity to reflect on those dynamics as they shape what we know and don't know about environmental toxins and public health, and what historians and others who study the past can do to recover those missing pieces.

Rather than cast my comments in terms of what we know or are beginning to know—concerning, for example, the effects of environmentally prevalent synthetic compounds such as bisphenol-A on human reproductive systems and development, or the ways that biomonitoring and body burden studies are helping to mobilize environmental health activists—I will focus on what we don't know and why that might be. My points of departure are two recently completed studies of the hazardscape in New Orleans prior to and following the landfall of hurricanes Katrina and Rita in 2005.[2] Both studies examine the problem of urban soil contamination, but from different angles. The first investigates how knowledge about remnant industrial contaminants in the city may have become hidden and effectively lost over time; the second investigates knowledge gaps resulting from the U.S.

Scott Frickel, "On Missing New Orleans: Lost Knowledge and Knowledge Gaps in an Urban Hazardscape," *Environmental History* 13 (October 2008): 643-650.

Environmental Protection Agency's (EPA) post-hurricane environmental hazard assessment. I want to use the findings from these studies to consider how lost knowledge and knowledge gaps are related spatially and how these two ways of missing New Orleans can inform a deeper appreciation of—and concern for—the historical nonproduction of environmental knowledge.

TAKING OUR CUE from research by environmental historians and historical geographers on the accumulation and disposal of "relict industrial waste," the first study investigates the conversion of industrial lands to other commercial and noncommercial uses between 1955 and 2006.[3] Working with a total of 215 former industrial sites that we identified from mid-twentieth century manufacturing directories, we selected ninety-two at random and then conducted site surveys on each of those lots to assess patterns of contemporary land use.[4] Our goal was to better understand the nature and spatial distribution of former industrial facilities throughout the city and to learn what those sites had more recently become.

New Orleans is an old port city with a long history of manufacturing, but it has never been a center for heavy industrial production. So it is not surprising that most of the sites in our historical sample had been relatively small operations averaging between ten and seventy employees that had clustered mainly, although not exclusively, along the city's water, road, and rail corridors. Still, these small manufactories likely packed a hefty environmental punch. Much of the city's industry after mid-century was tied to the region's oil and natural gas resources, with the timing of that development coming on the heels of an oil boom that crested in the late 1970s. Over half of the facilities in our sample either refined petroleum or processed petroleum into chemical or plastic products; the rest supplied the oil fields with marine transportation equipment and pipeline hardware or furnished the accompanying construction boom with concrete and other building materials. Based on information contained in the Historical Hazardous Substance Data Base, it is probable that dozens of persistent environmental contaminants may have been used for these and associated industrial activities during the past fifty years.[5] Given the vagaries of hazardous waste disposal regulation and enforcement in New Orleans, many of those contaminants were likely to have been buried, dumped, injected, spilled, rinsed, or otherwise come to inhabit an unknown number of these sites.[6]

When we visited these former industrial sites in the summer of 2006, we found more than a quarter (28.6 percent) to be occupied still by manufacturing establishments and another 12 percent to be abandoned lots. Totaling just over 40 percent of our sample, currently operating industrial facilities and derelict "brownfields" are the two endpoints that have attracted the most attention among academics studying urban industrial hazards and environmental justice.[7] Yet well over half of our sample sites (59.4 percent) had converted to various nonindustrial uses, be they commercial sites such as restaurants or grocery stores; public and quasi-public uses such as parks, public housing, or churches; or private residences. These are not the endpoints that research on brownfields and environmental justice typically capture, in large part because places like playgrounds or restaurants tend

not to be listed on federal and state hazard inventories, nor do they tend to appear hazardous to people living nearby. Yet nonindustrial uses together represent the dominant conversion pattern among the sites in our sample.

These former industrial sites, and whatever contaminants that may remain behind, represent lost knowledge of various sorts: community knowledge about daily life in and around the facilities and about the people who worked inside or played nearby; managerial knowledge about the social organization of industrial production in those places; technical knowledge about the materials used there, how they were transformed and where they went; geographic knowledge about the spatial distribution of those now-relict wastes; and not least, chemical knowledge about how substances change over time and in interaction with air, soil, water, and living organisms (see Egan, pp. 636-642 in this forum).

While it is rarely missed, lost knowledge matters. Because people (and bureaucracies) tend to make decisions on the basis of what they know, rather than what they do not know, lost knowledge limits the possibilities of social action. It is in this sense that lost knowledge can forestall even well-meaning efforts to understand, for example, the relationship between environmental hazards and public health in New Orleans, as our second study illustrates.

ON AUGUST 29, 2005, overtopping and breeches in levee walls produced by hurricane Katrina's storm surge inundated over 80 percent of the City of New Orleans with an estimated 114 billion gallons of corrosive salt water containing a complex mixture of chemicals, metals, and biological pathogens. The flood soaked parts of the city for six weeks, creating a nightmarish risk scenario and inspiring an unprecedented response from federal agencies tasked with assessing the storm's ecological and human impacts.

The official hazard assessment that followed the flood was an organizationally massive undertaking. Led by the EPA, it spanned an entire year and involved collaborations with a dozen or so other federal, state, and local agencies and departments. In metropolitan New Orleans alone, assessment efforts produced some 400,000 analytical tests, examining about two thousand sediment and soil samples for the presence of two hundred toxic substances.[8] The final report based policy recommendations on those analytical results, noting that "the sediments left behind by the flooding from the hurricanes are not expected to cause adverse health impacts to individuals returning to New Orleans."[9] Inferring environmental risk—or in this case its relative absence—from what is known about contaminants and their spatial concentration, is standard regulatory procedure.

We have been a bit less orthodox in our approach, employing the same EPA data to investigate what remains unknown. Using GIS mapping techniques to measure the spatial density of 951 sampling locations, we identified several large contiguous areas within the New Orleans floodprint where no soil or sediment samples were collected or tests conducted.[10] We view these areas as "spatial knowledge gaps" to indicate the formal nonproduction of spatially relevant knowledge. By linking city blocks located within these gaps to socio-demographic data from the U.S. Census, we found that nearly a fifth of the pre-storm population whose neighborhoods

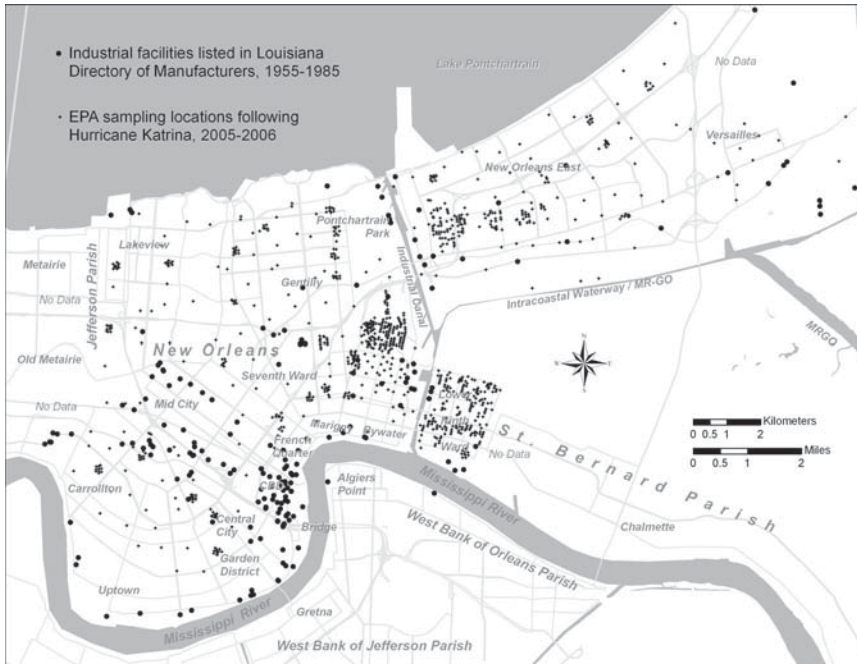
flooded live in these gaps (n = 65,962; 18.5 percent). While most are African American (n = 47,310; 71.7 percent), the racial/ethnic and socioeconomic characteristics of knowledge gap populations are roughly proportionate to the composition of the pre-Katrina population whose homes and neighborhoods flooded.[11] Where the lost knowledge described earlier resulted from a combination of general historical processes including deindustrialization, suburbanization, white out-migration, and urban redevelopment, these spatial knowledge gaps are social products of a different sort—the (presumably) unintended consequences of bureaucratized regulatory science.

CREATED AT DIFFERENT TIMES and resulting from different processes, lost knowledge and knowledge gaps are nevertheless related, at least in spatial terms. Map 1 shows all 215 former industrial site locations from our first study and all 951 EPA sediment and soil sampling locations examined in our second study. While there are exceptions, the general lack of spatial correspondence between potentially contaminated land and knowledge about contamination is clear: historical industrial sites—those urban parcels most likely to contain legacy contaminants—are located precisely where regulatory science undertaken in the name of public health did not occur. By my rough but conservative estimate, hazards assessment sampling occurred within several blocks of fewer than twenty historical industrial sites, representing less than 10 percent of total.

This striking disconnect is explained in part by EPA's institutional mandate following the hurricanes to assess environmental risk in *flooded residential areas*—a mandate that ostensibly relieves the agency of the responsibility of assessing neighborhoods outside the floodprint and nonresidential areas inside the floodprint. Our data from these two studies show that the organization of knowledge production within EPA closely followed this framework. Whether that plan is in the best interest of public health seems debatable, resting as it does on a logic that only loosely conforms to recent history.

Industry in New Orleans historically has concentrated along the Mississippi River and since the 1930s also along the industrial canal that links the river to Lake Pontchartrain. Levees bordering both waterways were at elevations sufficient to protect those facilities from Katrina's flood. Even so, if one were searching for relict wastes in New Orleans, these former and contemporary industrial areas would be among the obvious places to look.[12] At the same time, now that the city's relatively high ground has become prime real estate for redevelopment, incentives to not find legacy contaminants may be easier to come by.[13] By contrast, industrial sites at lower elevations located along interstate and rail lines in the city's interior also were systematically avoided in the hazard assessment process, even though these areas did experience catastrophic flooding. And while these interior neighborhoods are industrial, they are not exclusively so. As I noted earlier, prior to Katrina tens of thousands of New Orleanians were living near existing or former industrial facilities in areas that are best characterized by mixed residential, commercial, and industrial use. These neighborhoods gained new significance in the spring of 2007 when the Mayor's

Map 1. Locations of Historical Industrial Sites and EPA Sampling Points, New Orleans.



EPA sampling points are derived from analysis of test results data for Orleans Parish; data available online at http://www.epa.gov/katrina/testresults/index.html. Industrial site data are taken from the *Directory of Louisiana Manufacturers* for 1955, 1965, 1975, and 1985.

Industry in New Orleans historically has concentrated along the Mississippi River.

Office of Recovery Management announced that it would target some of these same areas for redevelopment investments because the economic and cultural diversity in such mixed-use neighborhoods was seen as offering a way to anchor recovery efforts.[14] So not only were these interior industrial areas populated before the storm, today they are on their way to becoming centers of concentrated repopulation.

NEW ORLEANS TRADES ON ITS UNIQUENESS,but the result of our research in that one-of-a-kind city leads me to three observations of a more general sort about the relationships entwining toxins, knowledge politics, and environmental history. The first is that despite the global flows of hazardous chemicals across the planet, pollutants still concentrate in some bodies, communities, and environments more than others. Louisiana, for example, is not only a top producer, but also a net importer of other states' hazardous waste.[15] Structural inequalities such as this matter, especially when posed against currently fashionable arguments by scholars such as Ulrich Beck, whose now famous admonition that "poverty is hierarchic, smog is democratic" implies that wealth no longer can purchase freedom from technologically introduced environmental risks.[16] Nikolas Rose's arguments concerning the "molecularization of life" is another example with similar implications (see Murphy, pp. 695-703 in this forum). I am wary of the tendency for

such narratives to universalize technoscientific power and its consequences in ways that mask deepening environmental and social inequalities and that mystify the political and economic forces that drive the global production and planetary spread of synthetic chemicals. The products of technoscience are everywhere, but not evenly or randomly so. Tragically evoking the wilderness that William Cronon has so provocatively sought to humanize, the broken streets and abandoned houses of New Orleans offer a muted plea for tying our stories about people, pollution, and power to experienced moments and lived places.[17]

My second observation is that our knowledge about pollution—what it is, where it is, and how it affects us when it becomes part of us—is also highly uneven. The approximately two hundred chemicals tested in New Orleans sediment represents less than a quarter of 1 percent of the more than 82,000 substances listed in EPA's chemical inventory—about the same proportion of chemicals for which EPA is reported to possess "complete health data."[18] These ratios illustrate just how "unworkable" traditional approaches to chemical assessment continue to be (see Nash, pp. 651-658 in this forum) and should compound concern for the uneven hazard assessment strategies that created spatial knowledge gaps in New Orleans. While they may not be planned, knowledge voids like these do not simply just happen. Lost knowledge and knowledge gaps are the results of historical processes and take particular social forms that historians of environmental knowledge are particularly well positioned to recover but that will require protracted collective political effort to repair.

I suspect such discontinuities in the systems that preserve historical knowledge and produce bureaucratic knowledge are common, so my third observation is that mostly, it seems, we don't know. As our research shows, ignorance overlaps, entwines, and accumulates. The absences it creates are complex. We should acknowledge that much at least. Doing so would mean acting on evidence that the regulatory regimes we've entrusted to protect us and our communities ignore our genuine ignorance about the dynamic states of our "toxic environments/toxic bodies."[19] In this context Arthur Daemmrich's suggestion that biomonitoring may usher in newly democratized forms of risk assessment and chemical regulation seems to me naively optimistic. Instead, as the work of several other contributors to this forum variously attest, ways of not knowing have become deeply programmed into the epistemic machinery that gives us conventional toxicology, analytical chemistry, risk analysis, and public health.[20] In New Orleans, political and civic action—or inaction—propelled by these and other forms of institutionalized ignorance may come at the high price of missing an unprecedented opportunity.

All cities are contaminated. But unlike most city dwellers elsewhere, citizens and government officials in New Orleans are uniquely positioned to address soil contamination problems on a scale and in a manner few can claim to have had. The hurricanes and flooding of 2005 destroyed or severely damaged hundreds of thousands of homes and businesses. These, along with tens of thousands of less severely damaged buildings and most of the city's public spaces—parks, golf courses, schools, and street medians—may eventually be renovated, rebuilt, redeveloped, or converted into green space. In short, the recovery process now haltingly underway

presents the city, the state of Louisiana, and the nation with a historic opportunity to enhance public health and environmental well-being by making an urban ecosystem less toxic. Whether this opportunity is seized, missed, or muddled through will depend on finding the resources, political will, and collective civic effort to craft and implement more ecologically sustainable solutions. If we are smart about it, those solutions will embody a healthy respect for what remains unknown—not because more knowledge is inevitably better, but because it is difficult to imagine how less ignorance would be worse.

Scott Frickel *is assistant professor of Sociology at Washington State University, where he studies environmental hazards, expert knowledge, and politics. He is the author of* Chemical Consequences: Environmental Mutagens, Scientist Activism and the Rise of Genetic Toxicology *(Rutgers, 2004), which received the Robert K. Merton Award from the Section on Science, Knowledge and Technology of the American Sociological Association, and is coeditor (with Kelly Moore) of* The New Political Sociology of Science: Institutions, Networks, and Power *(Wisconsin, 2006).*

NOTES

Resources for this research were provided through Tulane/Xavier Center for Bioenvironmental Research in New Orleans and by a grant from the Social Sciences Research Council. Richard Campanella, Jim Elliott, and Bess Vincent collaborated on the original research summarized in this essay. Lauren Richter created the map. I thank them all for their contributions, and I thank the forum editors for helpful comments.

1. Essays in this forum by Michael Egan ("Toxic Knowledge: A Mercurial Fugue in Three Parts," 636-42), Linda Nash("Purity and Danger: Historical Reflections on the Regulation of Environmental Pollutants," 651-58), and Barbara Allen ("Environmental Health and Missing Information," 659-66) pursue related themes. On the concept of undone science, see David J. Hess, *Alternative Pathways in Science and Industry: Activism, Innovation, and the Environment in an Era of Globalization* (Cambridge: MIT Press, 2007). On the cultural production of ignorance, see Robert N. Proctor and Londa Schiebinger, eds. *Agnotology: The Making and Unmaking of Ignorance* (Palo Alto, CA: Stanford University Press, 2008).
2. See Susan L. Cutter, *American Hazardscapes: The Regionalization of Hazards and Disasters* (Washington, DC: Joseph Henry Press, 2001).
3. Scott Frickel and James R. Elliott, "Tracking Industrial Land Use Conversions: A New Approach for Studying Relict Waste and Urban Development," in *Organization and Environment* (in press). On relict industrial waste, see Craig E. Colten, "Historical Hazards: The Geography of Relict Industrial Wastes." *Professional Geographer* 42 (1990): 143-56.
4. The sample was drawn from a population of establishments in six major industrial groups—chemicals, petroleum, plastics, primary metals, stone/glass/clay, and transportation equipment. Detailed methodological information is provided in the original study.
5. Craig E. Colten, *Historical Hazardous Substance Data Base* (Springfield: Illinois State Museum, 1992).

6. See Craig E. Colten, *An Unnatural Metropolis: Wresting New Orleans from Nature* (Baton Rouge: Louisiana State University Press, 2005).
7. For overviews see, respectively, Nancy Green Leigh and Sarah L. Coffin, "How Many Brownfields Are There? Building an Industrial Legacy Database," *Journal of Urban Technology* 7 (2000): 1-18; and Robert J. Brulle and David N. Pellow, "Environmental Justice: Human Health and Environmental Inequalities," *Annual Review of Public Health* 27 (2006): 103-24.
8. The EPA test data are available at http://www.epa.gov/katrina/testresults/index.html. Metropolitan New Orleans includes contiguously populated portions of Jefferson, Orleans, Plaquemines, and St. Bernard parishes. The analysis summarized here is based on data from Orleans Parish (City of New Orleans) only.
9. "Summary Results of Sediment Sampling Conducted by the Environmental Protection Agency in response to Hurricanes Katrina and Rita" Environmental Protection Agency (August 17, 2006). Available at http://www.epa.gov/katrina/testresults/sediments/summary.html.
10. Scott Frickel and M. Bess Vincent, "Katrina's Contamination: Regulatory Knowledge Gaps in the Making an Unmaking of Environmental Contention," Chapter submitted for inclusion in *Risk, Recovery, and Repatriation after Katrina*, ed. Rachel Dowty and Barbara Allen.
11. On the socio-demographic characteristics of the Katrina flood victims in New Orleans, see Richard Campanella, "An Ethnic Geography of New Orleans," *Journal of American History* (2007): 704-15.
12. The other types of places to look in New Orleans are Superfund sites and landfills See chapter 4 in Colten, *Historical Hazardous Substance Data Base,* note 5.
13. Richard Campanella, "Geography, Philosophy, and the Build/No Build Line," *Technology in Society* 29 (2007): 169-72.
14. City of New Orleans, Mayors Office of Communications, "City Announces First 17 Target Recovery Zones: Areas Will Attract Investment, Residents to Key Resources." Press release, March 29, 2007. Available at: http://www.cityofno.com/portal.aspx?portal=1&load=~/PortalModules/ViewPressRelease.ascx&itemid=3813.
15. See J. Timmons Roberts and Melissa M. Toffolon-Weiss, *Chronicles from the Environmental Justice Frontline* (New York: Cambridge University Press, 2001), 16-22.
16. Ulrich Beck, *Risk Society: Toward a New Modernity,* (London: Sage, 1992), 36.
17. William Cronon, "The Trouble with Wilderness: Or, Getting Back to the Wrong Nature," *Uncommon Ground: Rethinking the Human Place in Nature,*ed. William Cronon (New York, Norton, 1996), 69-90.
18. United States Government Accounting Office, *Chemical Regulation: Options Exist to Improve EPA's Ability to Assess Health Risks and Manage its Chemical Review Program* (Washington, DC: United States Government Accounting Office, 2005). Available online at http://www.gao.gov/new.items/d05458.pdf.
19. On the ignorance of ignorance, see Jerome R. Ravetz, "The Sin of Science: Ignorance of Ignorance," *Knowledge: Creation, Diffusion, Utilization* (December, 1993): 157-65.
20. On "epistemic machinery," see Karen Knorr Cetina, *Epistemic Cultures: How the Sciences Make Knowledge* (Cambridge: Harvard University Press, 1999).

LINDA NASH

FORUM
purity and danger: historical reflections on the regulation of environmental pollutants

FOR THOSE WHO STUDY current environmental problems, it is easy to assume that the history of those problems, however interesting, is not essential or even relevant to their solution. Yet as we debate how to respond to contemporary dilemmas, we might consider the knowledge and the vocabulary with which we conduct the argument. Debates over chemicals and their regulation are, at root, debates about the relationship between bodies and their environments. Embedded within these arguments are assumptions about the nature of both bodies and the spaces within which those bodies dwell: It is history that allows us to understand how we came to perceive health, disease, and environmental pollution in the ways that we do. So what are the contexts that gave rise to modern forms of environmental regulation in the United States? And what historical assumptions about bodies and disease do we reproduce in current discussions of regulation and chemical pollutants?

Two different intellectual contexts underlie twentieth-century environmental standards. The first is what we might call the germ-theory theory of the environment. In this view, the natural environment is intrinsically healthful, while the healthy body is a pure body, a body free from disease-causing agents. Nonhuman landscapes are not harmful in themselves, though they may be periodically traversed by dangerous pathogens and chemicals.[1] This perspective is widely familiar today, but it is, historically speaking, a relatively recent and uneven development. It has its roots in the rise of bacteriology in the latter

Linda Nash, "Purity and Danger: Historical Reflections on the Regulation of Environmental Pollutants," *Environmental History* 13 (October 2008): 651-658.

nineteenth century, although it reached its apotheosis only in the early 1900s as sanitarians and doctors undertook vigorous campaigns against germs, insects, and unhygienic behaviors. What preceded those developments was something quite different. In earlier periods, bodies were widely understood as porous and open to their environment. For both physicians and lay people, "health" signified not a pure body but a body that was in balance with its surroundings. Natural environments could be either healthy or unhealthy; and a given locale might be healthy for some bodies and dangerous for others, or healthy in some seasons and sickly in others. Environments seeped into and shaped human bodies; however, natural environments were not presumed to be healthful, and a healthy place was not necessarily a clean or uncontaminated landscape. Health was not a matter of keeping pathogens out of the body but of ensuring the proper interaction between a body and its surroundings.[2]

These earlier ideas gave way among public health professionals at the turn of the last century as they came to understand the microbial sources of many major illnesses. Germ theory located the cause of disease not in the broader environment but within specific pathogens. Disease became bounded, and environments far less relevant. For a new generation of medical professionals the insights of bacteriology were nothing less than revolutionary. As Hibbert Winslow Hill, one of the leading popularizers of germ theory and the "New Public Health," wrote in 1916: "We do not fear or dread anything from our skins out. Nothing outside can hurt us until it gets into us. ... Only from our skins in can anything harm us; and this is why we have turned from regarding the environment and doctoring it, to regarding ourselves and keeping ourselves diseaseless."[3]

At the same moment that Hill was writing, the U.S. Public Health Service (PHS) was struggling to devise the first national water quality standards. The PHS's principal concern lay with typhoid, a disease often spread through drinking water, and the agency responded by establishing a committee in 1913 to devise the nation's first water quality standards. Surgeon General Rupert Blue invoked the language of environmental purity when he announced "the necessity for a federal *standard of purity* for drinking water" [emphasis added]. The need to keep pathogenic organisms out of the body necessitated keeping those same organisms out of water supplies.[4]

Purity, however, proved an elusive concept. What emerged from the deliberations of the PHS's Drinking Water Committee was a series of compromises reflecting the fact that natural water sources were unavoidably impure. And moreover, even if a definitive line could be drawn between purity and impurity, there were not sufficient technical tools to do so. Given the limited enforcement powers of the PHS, committee members understood it would be at best useless and at worst counterproductive to propose a quantitative standard that large numbers of water suppliers could not meet; in the end their approach was highly pragmatic. They recognized that their proposed standards were a compromise and would require modification. Nonetheless, the assumption of environmental purity had been institutionalized: waters that met the standards were deemed "pure," while those that did not were labeled "contaminated."[5]

While the first water quality regulations turned on questions of infectious disease and environmental purity, the first air quality regulations had a starkly different history. Efforts to regulate air quality emerged at roughly the same historical moment but from a different location. Unlike attempts to protect water consumers from environmental impurities, the first air quality standards emerged from the effort to limit occupational disease among factory workers. The assumptions that governed both bodies and environments inside the factory were quite different from those that applied outside. Whereas water quality regulations sought to restore the natural environment to a presumed prior and benign condition in order to maintain the integrity of the (pure) body, standards for air quality began with the assumption that the factory environment was unhealthful and that the processes of industrial labor inevitably left their mark upon workers' bodies.

That bodies suffered under conditions of labor was axiomatic in the nineteenth century. Under the law of contract, the worker's body was conceived as a form of capital that could be invested in return for wages. Under this logic, a diminishment in bodily capital was not a problem in itself; rather, it was a rational trade-off for money capital. Although it was widely recognized that certain occupations had high rates of injury or illness, the reigning legal assumption was that workers were capable of assessing those risks and demanding higher, compensatory wages in return.[6] In other words, whereas outside the factory walls disease signaled abnormality and impurity, within those walls illness as well as injury were conceptualized as more or less normal.

Workers did not passively accept this condition, but the prevailing legal and cultural system worked strongly against any effort to de-normalize occupational injury and disease at the time. In a labor-relations system that one historian has characterized as feudal, American workers had little power to rectify poor working conditions or to receive just compensation for their injuries: employer liability was limited; the law held employees culpable for their own and others' injuries; and workers who protested faced the very real prospect of demotion or dismissal.[7]

As the Progressive Movement brought public health to new prominence, however, it also brought attention to the problem of occupational disease and injury. In their effort to improve the lives of American workers, social reformers marshaled medical science to bolster their case. In particular, both researchers and reformers pointed to recent physiologic studies of fatigue as evidence that injuries could be prevented and as justification for shorter working days. Researchers pointed to quantitative measurements of muscular fatigue and corresponding metabolic changes to argue that worker efficiency decreased dramatically while injury rates increased after a certain point.[8] When confronted with the problem of hazardous substances in the workplace, both researchers and managers would turn again to physiology to understand the response of workers' bodies. Over the course of the 1920s, industrial hygienists would develop a new set of techniques—what we know now as modern toxicology—to quantify chemical exposures and to correlate those exposures with both physiologic variables and obvious signs of disease. Growing out of a collaboration between

university scientists and corporations, the questions that shaped the field of industrial physiology and toxicology revolved less around general ideas of "health" than around questions of labor efficiency and worker productivity.

Understandings of the body's relationship to its environment forged in this occupational context shared a conceptual similarity with those developed in bacteriology—that is, the new toxicologists reproduced the exceedingly narrow definition of both causality and disease enshrined by the recent dominance of germ theory. Chemicals were conceptualized as akin to microbes, as singular agents that were capable of inducing a specific disease once they entered the body. What mattered was not the broader environment but the specific chemical exposure.[9]

Furthermore, assumptions about what constituted "disease" within the field were subtly (and sometimes not so subtly) shaped by both the desire to make labor more efficient and the assumption that workers were inherently recalcitrant and dissembling. In the toxicological model, only those conditions that could be linked quantitatively to a specific chemical exposure (e.g., a measurable level of airborne lead dust in the workplace) and a known physiological effect (e.g., elevated levels of lead in the bloodstream) could constitute chemically induced disease. Felt conditions of illness that could not be diagnosed in the laboratory were dismissed, while diagnosed conditions that could not be traced to a specific chemical exposure were attributed to other causes.[10]

But assumptions about the body forged in experimental physiology and occupational health differed from those common in bacteriology in at least one important respect: physiologists viewed the human body less as a container subject to contamination than as a self-regulating system that sought, in Harvard physiologist's Walter Cannon's famed term, a condition of "homeostasis." Physiology emphasized the body's ability to achieve a condition of stability and balance, an echo of nineteenth-century environmental medicine but with a crucial difference. In experimental physiology the focus was less on the external factors that might push a body out of balance than on the body's inherent ability to balance itself. Drawing on the concept of self-regulation, the new industrial toxicologists would develop the concept of biologic thresholds—that is, the assertion that there is always a level of exposure below which the body can absorb and adjust to pollutants without sustaining permanent harm. Derived from laboratory studies of animals that had been dosed with measured amounts of chemicals, these biologic thresholds then became the basis for "threshold limit values" (TLVs; later "maximum contaminant level goals")—that is, the level of chemical concentration below which no biological effects were believed to occur. By developing TLVs for a variety of chemical compounds, modern toxicology normalized the problem of low-level chemical exposures, at least within the factory. Consequently, for these professionals (and their industrial patrons) the concept of environmental purity had no role: that both environments and bodies absorbed industrial chemicals was not, in itself, a problem.[11]

Regulators are seldom innovators. As industrial substances migrated out of the factory and into the broader environment, policymakers drew on existing

regulatory frameworks. The two professions that held relevant knowledge and experience were public health doctors and their sanitary engineering colleagues on the one hand, and industrial toxicologists on the other.[12] Both groups would bring their professional techniques and assumptions to the new environmental problems in the decades after World War II. The regulation of chemical pollution still reflects this uneasy mixture. On the one hand, the emphasis of twentieth-century bacteriologists and sanitarians on bodily purity and their belief that disease could be localized in particular pathogens have cultivated an unrealistic public expectation that environments are—or at least should be—external to health. As a consequence, the prevailing assumption has been that environments can be manipulated toward other ends without seriously considering how those manipulations will ramify in bodies. By separating bodies from environments and health from landscapes, germ theory helped underwrite ever greater environmental change without consideration for possible health effects. In those cases where environments do affect health, there is an expectation that the offending environment can and should be purified. Yet as body-burden testing has so clearly revealed, what is released into the environment—be it the typhoid bacillus or PCBs—will find its way into the bodies that reside in that environment (see Daemmrich, pp. 684-694 in this forum). Moreover, environmental arrangements affect health in multiple and complex ways that the germ model cannot account for. Chemicals interact with one another. A body's past exposures and history conditions its response to new exposures. Chemicals that are benign under one set of conditions may be highly toxic under different conditions. Health and disease are affected not only by specific chemical and biological exposures but by the arrangement of cities, the thickness of the ozone layer, and the larger biological and social community within which an individual resides. Contrary to the assumptions of early public health reformers, bodies cannot be walled off from their surroundings.[13]

Whereas the bacteriological model of bodies and environments generated expectations that were at once unrealistic and inadequate for the broader issue of environmental regulation, the assumptions that have structured modern toxicology have proven equally—though differently—problematic. Most obviously, the assumption that workers are able to make rational assessments of risk and demand appropriate compensation for risky labor has always been a shibboleth for all but the most privileged, while it has not even theoretical applicability to those outside the workplace who come into contact with industrial chemicals (see also Daemmrich, pp. 684-694in this forum). Similarly, the insistence that there exist clear lines between "safe" and "hazardous" levels of exposure was not simply a "fact" established by scientific investigation; the concept of identifiable thresholds included within it the assumptions of early twentieth-century physiology as well as the social realities and relations of power that governed industrial labor in that period. Not only were TLVs subject to corporate influence, later research soon challenged the scientific basis for the concept of a no-effect level, especially in the case of chemical mutagens.[14]

But the limits of the toxicological approach extend beyond the adequacy of particular chemical standards. When toxicology moved outside the factory, it brought with it the assumption that industrial chemicals are a normal part of the environment and that the only relevant question to ask was at what level. In the 1920s, the reliance on safe concentration levels and TLVs had absolved researchers and regulators of the need to consider how the environment of the factory should or should not be organized; that was left to corporate managers.[15] Today, even though the task of assessing the effects of tens of thousands of chemicals on large and highly diverse populations has proven unworkable (to say the least), toxicology holds out the hope that it will be possible. In effect, toxicology reproduces the world in the image of the early twentieth-century factory.

CURRENT REGULATORY APPROACHES are a product of this history. They assume that the broader environment is irrelevant to health unless proven otherwise; and the mechanisms for proving the relevance of the environment rely on models of disease and exposure derived from early bacteriology and toxicology. Consequently, the only aspects of the environment that can be regulated in the name of health are those that can approximate bacteria—in other words, specific chemicals or pathogens that can be demonstrated to cause, in the narrowest of senses, specific diseases. At the same time, the assumptions that underwrite our reliance on chemical-by-chemical standards are derived from early twentieth-century physiology and the power-laden environment of the early industrial factory.

The historical contexts of environmental regulation should warn us against the easy assumption that what we need most is "better science" or less uncertainty. Science emerges in specific contexts and responds to a host of historical and social factors: its perceived social utility, the interests of capital, existing techniques and technologies, the needs and desires of practicing scientists. And while particular disciplines may supersede and even challenge those contexts, they never fully escape them. Scientists and regulators necessarily build on what has come before.

How have these historical frameworks structured the response to subsequent environmental health threats? When organic pesticides were introduced into American agriculture during the late 1940s and 1950s and farmworkers began falling ill in the fields, regulators turned to toxicology, hoping to link worker illnesses to specific exposures of single chemicals. But they found that workers fell ill even when toxicology predicted that they should not. Farmworkers who traveled from field to field had far more complex and unknowable exposure histories than factory workers, and their past exposures increased their sensitivity to future exposures. Moreover, what mattered was not simply the pesticides but the environmental conditions under which they were encountered. In the agricultural environment—where weather and crop conditions were changeable and multiple applications of multiple pesticides were the norm—it was not practicable to monitor for safe levels and enforce TLVs. Thus regulators

were forced to make a host of assumptions about how application rates, weather conditions, picking styles, and crop types might or might not affect the toxicity of specific fields. Safe concentration levels could not be ascertained in this environment, and yet the toxicological approach pushed regulators to promulgate just such levels.[16] Not surprisingly, the problem of pesticide poisoning among farmworkers persists even today.

Or consider the more recent issue of food-borne illness. In one of the largest outbreaks, consumers fell ill after eating bagged spinach tainted with a deadly variant of the e. coli bacteria whose source was traced to feces from a nearby dairy farm. If we define the cause of these illnesses narrowly, as emanating from a single pathogen, then the solution is to rid environments of that pathogen. It should come as no surprise that current proposals to control e. coli outbreaks focus on sterilizing agricultural environments and increased testing of the food supply.[17] A different approach might consider the health effects of industrialized cattle ranching, centralized food processing, or the marketing of bagged salad, which fosters the growth of bacteria. How we understand the cause of e. coli poisoning will determine how we respond to it.

In the wake of all we have learned about the ecology of infectious disease, the complex factors that condition chemical toxicity, and the influence of social and environmental conditions on health, does our best hope for regulating the relationship between bodies and environments still lie in the chemical-by-chemical, germ-by-germ approach? Perhaps, but it is a question worth asking. Rather than calling somewhat blindly for more science, we might ask whether a regulatory system cobbled together out of two distinct branches of early twentieth-century medicine can still serve our needs in the twenty-first century. Rather than wringing our hands over the problem of scientific "uncertainty," we might grant that the modernist hope for perfect knowledge will always be unfulfilled. Rather than hoping that increased scientific knowledge will—like some deux ex machina—reveal the solutions to contemporary environmental problems, we might instead insist upon the need to consider more critically the cultural models and historical assumptions that guide contemporary regulatory policy.

Linda Nash *is associate professor of history at the University of Washington. She is the author of* Inescapable Ecologies: A History of Environment, Disease, and Knowledge *(California, 2006). Her current project,* Engineering a Modern World*, is a cultural and environmental history of technical knowledge production and postwar development practice viewed through the experience of American water engineers.*

NOTES

1. Michael Worboys, *Spreading Germs: Disease Theories and Medical Practice in Britain, 1865-1900* (Cambridge: Cambridge University Press, 2000), 285.
2. Conevery Bolton Valencius, *The Health of the Country: How American Settlers Understood Themselves and Their Land* (New York: Basic Books, 2002).

3. Herbert Winslow Hill, *The New Public Health* (New York: MacMillan Company, 1916), 8.
4. Patrick Gurian, and Joel A. Tarr, "The First Federal Drinking Water Standards and Their Evolution: A History from 1914 to 1974," in *Improving Regulation: Cases in Environment, Health, and Safety*, ed. Paul S. Fischbeck and R. Scott Farrow (Washington, DC: Resources for the Future, 2001), 43-69, 53.
5. Ibid. A similar logic underlay the regulation of processed foods, which began with the Pure Food and Drug Act of 1906. Suzanne Rebecca White, "Chemistry and Controversy: Regulating the Use of Chemicals in Foods, 1883-1959" (PhD diss., Emory University, 1994).
6. Steve Sturdy, "The Industrial Body," in *Medicine in the Twentieth Century*, ed. Roger Cooter and John Pickstone (Amsterdam: Harwood Academic Publishers, 2000), 217-34.
7. Ibid.; Karen Orren, *Belated Feudalism: Labor, the Law and Liberal Development in the United States* (New York: Cambridge University Press, 1991).
8. Alan Derickson, "Physiological Science and Scientific Management in the Progressive Era: Frederic S. Lee and the Committee on Industrial Fatigue," *Business History Review* 68 (1994): 483-514; Richard Gillespie, "Industrial Fatigue and the Discipline of Physiology," in *Physiology in the American Context, 1850-1940*, ed. Gerald L. Geison (Bethesda, MD: American Physiological Society, 1987), 237-62.
9. Christopher Sellers, *Hazards of the Job: From Industrial Disease to Environmental Health Science* (Chapel Hill: University of North Carolina Press, 1997), 141-86.
10. Sellers, *Hazards of the Job*, 159-69. On the ways in which toxicology produces invisibility, see also Michelle Murphy, *Sick Building Syndrome and the Problem of Uncertainty: Environmental Politics, Technoscience, and Women Workers* (Durham, NC: Duke University Press, 2006), esp. 84-92.
11. Steve Sturdy, "Biology as Social Theory: John Scott Haldane and Physiological Regulation," *British Journal of the History of Science* 21 (1988): 315-40; Sellers, *Hazards of the Job*, 175-83. Also Sarah A. Vogel, "From 'The Dose Makes the Poison' to 'The Timing Makes the Poison': Conceptualizing Risk in the Synthetic Age," *Environmental History* 13 (October 2008): 667-73.
12. In fact, by the 1960s, both groups had begun to identify themselves as "environmental" professionals—e.g., as environmental health specialists or environmental engineers.
13. See also Vogel, "From 'The Dose Makes the Poison' to 'The Timing Makes the Poison.'" Since the 1920s, the field of disease ecology has also challenged assumptions about environmental purity with respect to infectious disease and has cast the successes of twentieth-century disease prevention efforts in a more provisional light. A.J. McMichael, *Human Frontiers, Environments, and Disease: Past Patterns, Uncertain Futures* (Cambridge: Cambridge University Press, 2001).
14. S. A. Roach and S. M. Rappaport, "But They Are Not Thresholds: A Critical Analysis of the Documentation of Threshold Limit Values," *American Journal of Industrial Medicine* 17 (199): 717-53; Barry Castleman and Grace Ziem," Corporate Influence on Threshold Limit Values," *American Journal of Industrial Medicine* 13 (1988): 531-59.
15. Sellers, *Hazards of the Job*, 175-76.
16. Linda Nash, *Inescapable Ecologies: A History of Environment, Disease, and Knowledge* (Berkeley and Los Angeles: University of California Press, 2006), 139-69.
17. Monterey County Resource Conservation District, *Reconciling Food Safety and Environmental Protection: A Literature Review*, prepared by Diana Stuart, October 2006; Michael Pollan, "The Vegetable Industrial Complex," *New York Times Magazine*, October 15, 2006.

BARBARA ALLEN

FORUM
environment, health and missing information

THE WORKSHOP ON "TOXIC BODIES" highlighted an emerging concern with pervasive and persistent environmental toxins at low doses, such as endocrine disruptors. While the study of these chemicals is relatively new, the study of the effects of toxic chemicals on the human body is not. From lead exposure to workplace carcinogens, historians have long documented the many health hazards of increasing industrialization. This research has relied on a variety of sources for information on public health, such as medical archives and government data —all of them important to understanding the historic record of the effects of industrial toxins. My concern for future historians is with the more recent suppression of environmental health data, a trend that is growing in the United States and possibly elsewhere. What data and information on today's chemical exposures will be available in archives and from government sources in the future and what will be missing?

My past work on environmental justice and human health has uncovered some disturbing regional and national trends in the transparency of science and the reliability and availability of public health data. These disturbing trends can be broadly placed in two categories: undone science or knowledge gaps, and suppression or secrecy in science. The first trend, undone science, is the consequence of the framing of scientific studies to either intentionally avoid specific areas or questions (see Frickel, pp. 643-650 in this forum) or to answer only the questions of experts, government regulators, or corporate advisers. This often results in not addressing and not answering the questions citizens have related to their environment and health.[1] For example, citizens who live near

Barbara Allen, "Environment, Health, and Missing Information," *Environmental History* 13 (October 2008): 659-666.

numerous hazardous industries want to know the synergistic effect of a variety of toxins on their bodies, over time, and in small doses. What these communities actually get is quite different—at best, results based on animal laboratory studies of one chemical at a time with regulations based on a chemical-by-chemical standard (see Nash, pp. 651-658 in this forum). Thus the science needed to answer the complexity of their questions remains undone.

The notion of knowledge gaps is another way to frame the issue, as Scott Frickel, in this workshop has demonstrated. He questions why the Environmental Protection Agency (EPA) chose not to do soil samples in certain predominantly African-American neighborhoods in post-Katrina New Orleans, leaving the displaced residents without potentially important health information. This "uneven" science, whether due to intentional ignorance or the cost of producing citizen-relevant science, does not bode well for environmental health in a time of increasing global industrial production and concern about newer risks from endocrine disruptors, whose effects on the human body occur even at miniscule doses.

Suppression and secrecy is the second unfortunate trend in the creation and circulation of environmental health information. It is this category of "missing data" that I will focus on. That environmental health knowledge, data, and science exist (i.e., are collected for public health reasons or as part of occupational health studies) but are buried for questionable reasons, will create a knowledge gap for future researchers. Notably, the first advocacy position taken by the American Society for Environmental History (ASEH) was a resolution opposing the closing of many EPA libraries. In 2006, the EPA suddenly closed the Office of Prevention, Pollution and Toxic Substances Library, the only one of its libraries specializing in research on the health effects of toxic chemicals. Soon after that EPA libraries in Chicago, Dallas, and Kansas City were closed, and more closures were sure to follow. The EPA cited low usage of its libraries as the reason for their closure. These libraries contained over 50,000 primary source documents not available elsewhere and received over 134,000 research requests a year from EPA staff alone, not including the public and outside scientists. Fortunately, in 2007 as a result of pressure from Congress, the EPA placed a moratorium on further closures and for fiscal year 2008, Congress included additional funding for reopening the dormant libraries. According to the reasoning the ASEH advanced for its advocacy position, "the retention of historical memory—the archiving of knowledge and documents that would otherwise be lost forever—is among the defining attributes of a civilized community." The society position background paper concluded that, "not everything should have to pass a cost-benefit test to be protected."[2]

My work on the history of the chemical industry and expert-activists in Louisiana uncovered another version of the problem of missing information. In this case the hiding of scientific data appears to be prevalent, particularly with research data that involves the effects of chemical hazards on human health and residential environments. This occurs when there is a fear of industry regulation, liability claims, or pressure for possible corporate responsibility. There are two common ways that information is legally obscured. The first is the "homeland security" excuse and the second is the use and misuse of the "right to privacy."

PROBLEMS WITH THE RIGHT-TO-KNOW

WITHIN A YEAR after 9/11, the chemical companies together with the Bush administration were busy instituting new security measures. In the name of increased safety, thousands of pages of formerly public information disappeared from websites, including data on chemical facilities, the hazards stored and produced, as well as evacuation maps for accidents.[3] For years, activists and community members have used these "risk management plan" (RMP) websites, along with TRI data, to alert residents to the chemical dangers in their community. This information also has been used to pressure plants into adopting cleaner practices, sometimes through lawsuits and regulatory changes. In addition, data from these sites were important for community planning purposes, for activities such as locating new schools and playgrounds as well as immediate information for first responders, who, in some areas, are citizen-volunteers. Furthermore, this tendency to hide information from the public has grown since 9/11. A few years later in 2006, both houses of Congress finally passed an appropriations bill for 2007 requiring the Department of Homeland Security (DHS) to propose rules requiring high-risk chemical facilities to perform security self-assessments and remedies. The chemical companies fully embraced the new secrecy ethos but lobbied heavily to make sure that the regulations would be determined by industry and be voluntary.

In the meantime some states having many noxious facilities located near urban areas, such as New Jersey, passed enforceable plant security practices. New Jersey officials deemed that 43 of their 140 chemical plants were subject to the state's new Toxic Catastrophe Prevention Act, thus mandated to consider the adoption of "inherently safer technologies" (ISTs). There had been numerous attempts to pass national legislation on chemical security and safety, predominantly by legislators from large urban areas. The types of provisions they were looking for included: 1) the use of ISTs where feasible; 2) enhanced site security at plants; 3) workers having a voice in safety/security and whistleblower protection; and, 4) allowing stronger state laws, if passed, to supercede federal laws.[4] Nowhere in these proposals is hiding information from the public ever mentioned.

In late 2007 the House, following the Senate, passed an appropriations bill for the DHS requiring the establishment of interim rules for high-risk facilities. The bill had none of the provisions previously advocated in earlier attempts and instead endorsed a "business as usual" approach. DHS was directed to ask that chemical facilities assess their own security risks and propose solutions. Elected officials from New Jersey were particularly dismayed because this weaker law displaced the stronger regulations the state already had in place.

Another industry-driven effort to avert terrorism was to make sites more able to "detect, deter, and delay" potential intruders.[5] While, in principle, this appears to be a sound idea, in practice, it has snared some unlikely people. While "suspicious activity" around chemical plants was always reportable long before 9/11 under various local, state, and national statutes, after 9/11 new standards of "suspicious activity" were adopted at some plants. In March 2005, Willie Fontenot,

a community liaison officer with the Louisiana state attorney general's office, was escorting a group of college students on a tour of "cancer alley," the term that many locals use for the chemical corridor between New Orleans and Baton Rouge. They were interested in environmental justice issues facing communities that shared the fence line with industry. While walking and photographing in a community being bought out by ExxonMobile, the group was suddenly detained by a group of plant security officers. Fontenot was removed from his position and forced to take early retirement. From my own observations, the level of harassment of student groups and researchers in "cancer alley" has increased under the guise of national security regulations.[6]

This new cloak of secrecy that industry adopted responding to post-9/11 threats has arguably decreased the safety of residents living and working near noxious facilities.[7] Conveniently, pesky environmental activists and concerned citizens now have less knowledge with which to pressure plants into responsible, cleaner practices. With the passage of the 2007 bill, it appears that DHS is willing to let industries determine their own responses to security, even at the expense of openness and the availability of public information.

With the guidance of industry, the right-to-know is becoming the need-to-know, a problem not only for current residents, but also for future historians of industry, the environment, and public health. That public information on locations and types of hazards stored at chemical facilities was removed from websites and that academic groups are now forbidden to closely examine sites of environmental justice claims diminishes the amount of archival and documentary materials that will be available in the future. Repeating the argument that the ASEH made opposing the closure of EPA libraries is instructive: "Trying to piece together responsibility for PCB contamination at a Superfund site, for example would be impossible without such records. For communities of color struggling to understand asthma and air contamination, for epidemiologists searching for patterns of mercury deposition and disease incidence, for historians seeking to learn about restoration of streams after the Clean Water Act—the holdings of the EPA libraries are quite simply irreplaceable."[8]

PROBLEMS WITH THE RIGHT-TO-PRIVACY

THE RIGHT-TO-PRIVACY, to most Americans, is sacrosanct. In a country where health insurance is expensive and/or difficult to obtain, the protection of one's records against corporate voyeurs could mean the difference between obtaining coverage or going bankrupt from hospital costs. Fears of employer discrimination or stigmas attached to certain illnesses reinforce people's insistence on their health data security. In 1996, the Health Insurance Portability and Accountability Act (HIPAA) was passed by Congress, primarily to address the "portability" of electronic records by health care providers and insurers. Privacy legislation was added in 2003, including strict guidelines for the use and disclosure of a person's medical record with legal ramifications, both civil and criminal, for noncompliance.[9]

While strong privacy laws appear to protect individual rights, they form a barrier to some public health research. Cancer surveillance, for example, began early in the twentieth century, with Connecticut being the first state to establish a cancer registry in the 1930s. As of 2006, cancer data are contributed by forty-three states to a national cancer incidence report, which represents 92 percent of the U.S. population.[10] Cancer surveillance, however, is one area that has felt the unintended consequences of HIPAA. Only a few sections of HIPAA are directed at research, and those try to balance privacy and data access. Because the privacy act requires written authorization for use of medical records from living persons, researchers must obtain waivers from a privacy board to gain access. Exceptions to obtaining written authorization include: 1) if the health information is de-identified (name, address, social security number, etc.); 2) if the research is about people who have died; or 3) if information is required by law for public health activities.[11]

Despite attempts at balance, "early anecdotes suggest that IRBs are being conservative in their interpretation of HIPAA, erring on the side of privacy … [and] HIPAA does not preempt state regulations so there is no national standard," making multi-state research difficult.[12] For environmental health researchers, including historians of public health, the application of privacy rules to cancer and other types of health data can mean the inability to do both larger epidemiological studies as well as analyze illness clusters and links between illness and proximity to hazardous sites.[13] Minnesota enacted strict medical confidentiality standards in 1997, requiring that patients give written permission for each study for which their records are used, and the consequences are instructive. In one multi-state federally sanctioned study on seizures induced by pain medication, only 19 percent of Minnesotans gave permission, whereas ascertainment in the other states was 93 percent.[14]

In my research on "cancer alley," I discovered an astounding fact: neither the citizens nor the state could say for sure if there really was a higher incidence of cancer in people living near industry. The state agency mandated to collect cancer data, the Louisiana Tumor Registry (LTR) claimed that there was no evidence of such a correlation: however, there was also no evidence that there was not a correlation. The data had been collected and aggregated into large multiparish areas (equivalent to several counties) over five-year periods rendering it useless to show health effects in smaller locale-based frames.

A group of citizens and medical researchers sued the LTR in the late 1990s asking for the release of cancer data in one-year intervals, by zip code, and including rare cancers such as pediatric cancer. While the LTR eventually complied with the request to publish cancer data in one-year intervals, they fought the release of data by zip code and the release of data on some rare cancers. Their argument against the release of zip code data was that it would be misused and misinterpreted. They claimed: "the entire scientific community of cancer research professionals regard zip code-specific cancer incidence data as statistically and clinically meaningless."[15] Furthermore, they asserted that if the data was released in such small geographic units, for some rare cancers the incidence could be "one," thus violating a person's right-to-privacy.

The medical researchers in the lawsuit were particularly interested in pediatric cancer as anecdotal evidence suggested it was quite high and potentially linked to environmental hazards. Because there are sixteen classification types of pediatric cancer, to delete all instances of "one" could effectively miss sixteen cancers in a year in a geographic area. In 2004, the citizens and researchers eventually won a partial victory as the data was released to them by parish and for all cancers, including rare cancers, but not by the smaller unit of zip code.[16] The bottom line—the right-to-privacy had been used to deny access to medical data that could show links between environmental pollutants and human health.

About a month after the Environmental History panel on "Toxic Bodies," I attended a conference on "New Chemical Bodies" at the Chemical Heritage Foundation (CHF) in Philadelphia. The event was focused on "biomonitoring, body burden, and the uncertain threat of endocrine disruptors" and featured some of the top scientists and social scientists working in the field today.[17] Interestingly, the most outspoken advocate of stronger right-to-privacy regulations with regard to biomonitoring was one of the representatives of the chemical industry.

In 2004 a Cancer Surveillance and Information Summit was held to address the concerns of professionals and allied groups regarding the future of cancer data and public health. Their recommendations included both the standardization of data collection and better legal access to individually identifiable data for public health purposes.[18] This allows for easier retrospective research such as combing a hospitals' archive looking for patterns of illness or treatment.[19]

One of the primary themes of cancer surveillance literature is the balancing of individual privacy with public health research needs. Among the recommendations is the development of standards for protection of personal information including policies regarding who can use the data and for what purpose. There are eighteen personal identifiers in surveillance data, and not all information is needed by researchers, depending on the scope and needs of their investigations. Digital records enable registries and agencies to strip nonessential data from patient records while complying with researcher requests. This requires both standardization in reporting as well as standardization of technical systems, which, to date, has not happened because data collection and dissemination still is driven predominantly by state standards. Improving standardization and accessibility also would ensure that this kind of data would be available to environmental and public health historians in the future as they try to understand this period of rapid industrialization and expansion of chemical hazards.

CONCLUSION

THERE ARE THREE concluding issues that I would like to address regarding the intersection of environmental history and missing information. The first is the importance of protecting data currently held by government agencies and other public organizations and ensuring open public access to that information. The ASEH has taken an advocacy position about this in the past, and with regard to the closing of the EPA's libraries, continued vigilance may be necessary to ensure these kinds of materials are available in the future.

The second issue is that environmental historians need to know what data is absent, suppressed, or hidden as well as what is readily available in archives and other places. Two books come to mind as exemplary in their use of creative sources and/or hard-to-obtain materials. Craig Colten and Peter Skinner's *The Road to Love Canal: Managing Industrial Waste Before EPA,* is an excellent example of scholarship on environmental hazards. Prior to writing the book, Colten had spent a decade investigating the historical geography of industrial waste for government agencies and had served as an expert witness in over twenty-five legal cases helping reconstruct the "state of knowledge" about hazardous materials and contamination. According to the authors, the historical records to which they had access had: "frequently been confined to court cases about specific sites, and consequently there had been no overarching review of the testimony or technical literature of the day. The fragmented delivery of countless witnesses and the volumes of reports and articles collected created a unique opportunity to consider the broad landscape of toxic waste management from a historian's perspective."[20] With unprotected corporate records, public documents, and archival and library sources, Colten was (with Skinner, a scientist) able to assemble enough information to produce the history of the United States's toxic legacy.

The other book, Gerald Markowitz and David Rosner's *Deceit and Denial: The Deadly Politics of Industrial Pollution*, focused specifically on the history of environmental health in relation to the lead and chloro-chemical (plastics) industries. As historical experts, they had been asked by a number of law firms to review an enormous number of primary documents obtained though legal discovery motions, including those of the Manufacturing Chemists' Association and its member companies, and the Lead Industries Association and its member companies. They were given no restrictions on what they could later publish from the materials and their findings of corporate environmental data suppression and misinformation were shocking and revealing.[21] According to Rosner and Markowitz, occupational hazards eventually become environmental problems and thus provide a good starting point for histories examining the relationship among toxins, pollution, and industrial development. That it took numerous lawsuits against corporations to uncover information about the industrial transgressions that they write about is unfortunate. That it might take legal action for future historians to uncover government-mandated chemical risk management plans, taxpayer-funded public health data, or private health data on deceased citizens will make such research projects enormously time-consuming and expensive.

Last, the third issue, and the one I would like to end with, is the need for environmental historians to work with affected communities to determine what kinds of records have been generated (from lay studies to corporate documents made public via lawsuits) that are important to protect for future scholars and future community leaders. I have no easy answer for these dilemmas, only a desire to caution academic researchers about the possibility of these important past and current knowledge gaps and to point to strategies that might help to ensure a more robust archive of publically available information in the future.

Barbara Allen *is associate professor and Director of the Graduate Program in Science and Technology at Virginia Tech's Northern Virginia Center. Her past research and current book was on the dynamics of regulation, science, and environmental justice in Louisiana's chemical corridor.*

NOTES

1. For a longer discussion of "undone science," see David J. Hess, *Alternative Pathways in Science and Industry* (Cambridge: MIT Press, 2007), 22-24.
2. See "EPA Background Document" on the ASEH website at http://www.aseh.net/resources/advocacy/epa_background. Full text of "ASEH Resolution Opposing EPA Library Closures," can be found at http://www.aseh.net/resources/advocacy/epa_resolution.
3. Alan Elsner, "Bush Expands Government Secrecy, Arouses Critics," Reuters, September 3, 2002, from http://www.fas.org/sgp/news/2002/09/re090302.html.
4. Frank Lautenberg, "Senators Lautenberg, Obama and Menendez Introduce Sweeping Bill to Tighten Security at Chemical Plants," *U.S. Senate*, March 30, 2006.
5. Chris Schneidmiller, "Terrorist Threat to U.S. Chemical Facilities Can Be Lowered, Not Eliminated, Officials Say," *Global Security Newswire*, September 7, 2005, from http://www.nti.org/d_newswire/issues/2005_9_7.html#16FC149B.
6. Mark Schliefstein, "Activists' Ally Snared in Security Net," (New Orleans) *Times Picayune,* April 5, 2005.
7. Sean Moulton, "Different Opinion on Chemical Security," *OMB Watch*, September 16, 2002.
8. ASEH, "EPA Background Document."
9. Robert A. Hiatt, "HIPAA: The End of Epidemiology, or a New Social Contract?" *Epidemiology* 14 (November 2003): 637-39.
10. Dennis Deapen, "Cancer Surveillance and Information: Balancing Public Health with Privacy and Confidentiality Concerns (United States)," *Cancer Causes Control* 17 (2006): 640.
11. Robert A. Hiatt, "The Future of Cancer Surveillance," *Cancer Causes Control* 17 (2006): 638.
12. Ibid., 638
13. Deapen, "Cancer Surveillance and Information," 633-37.
14. David L. Wheeler, "Is the Loss of Personal Privacy the Price of Medical Progress?" *Chronicle of Higher Education*, September 17, 1999.
15. Taylor, Porter, Brooks & Phillips, L.L.P., *Brief Filed on Behalf of Defendant-Appellee, Board of Supervisors of Louisiana State University and A&M College*, Docket No. 2003-CA-0079, 14-15, 2003.
16. For a complete discussion of the lawsuit of the citizens vs. the LTR see B.L. Allen, "The Problem with Epidemiology Data in Assessing Environmental Health Impacts of Toxic Sites," in *Environmental Exposure and Health,* M. M. Aral et al. (Billerica, MA: Computational Mechanics, Inc., 2005), 467-75.
17. See Jody A. Roberts, "New Chemical Bodies," in *Studies in Sustainability* (Philadelphia: Chemical Heritage Foundation, 2008).
18.. Hiatt, "The Future of Cancer Surveillance," 643-44.
19. David L. Wheeler, "Is the Loss of Personal Privacy the Price of Medical Progress?" *The Chronicle of Higher Education*, September 17, 1999.
20. Craig E. Colten and Peter N. Skinner, *The Road to Love Canal: Managing Industrial Waste Before EPA* (Austin: University of Texas Press, 1996), 4.
21. Gerald Markowitz and David Rosner, *Deceit and Denial: The Deadly Politics of Industrial Pollution* (Berkeley and Los Angeles: University of California Press, 2002), xii-xviii.

SARAH A. VOGEL

FORUM
from 'the dose makes the poison' to 'the timing makes the poison':
CONCEPTUALIZING RISK IN THE SYNTHETIC AGE

FOR SCIENTISTS AT GENERAL ELECTRIC in the late 1950s, the future was plastics. The GE scientists dreamed that scientific understanding of carbon-based (or organic) compounds, specifically polymers such as carbohydrates and proteins, would one day culminate in the creation of the ultimate machine—the "synthetic man." Though at the time only a dream, the rapid development and commercial success of synthetic polymers, notably plastics in the 1950s, fed such grandiose visions of the future. Today General Electric's dream of the "synthetic man," although not actualized as the science fictional character "The Terminator," has nonetheless become a reality. Traces of compounds used to make plastics, as well as pesticides and other industrial chemicals, have been detected in human blood, breast milk, placental tissue, amniotic fluid, and fat. We have literally merged with our material environment to become synthetic humans, a reality discussed and examined in this issue's forum essays by Michelle Murphy and Linda Nash. Determining the biological impact of our new synthetic bodies and the necessary political response to such a transformation informs a contentious contemporary debate about the meaning of chemical risk and toxicity.

This essay begins with a general overview of how chemical risks were conceptualized in regulatory law and articulated within the discipline of toxicology over the past half century. I then describe how research on endocrine disruptors beginning in the early 1990s challenged the meaning of risk and safety by providing empirical evidence of biological effects at extremely low doses of exposure—levels often long presumed to be safe. By challenging the safety of low-

Sarah A. Vogel, "From 'the Dose Makes the Poison' to 'the Timing Makes the Poison': Conceptualizing Risk in the Synthetic Age," *Environmental History* 13 (October 2008): 667-673.

dose exposures, endocrine disruptor research undermined the long-standing regulatory approach to protecting the public's health, which upheld the notion that above all else, the dose makes the poison, and exposure at low levels was safe.

RISK AS RELATIVE

FROM THE DEBATES over the safety of leaded gasoline beginning in the 1920s and pesticide residues on foods beginning in the 1950s to the institutionalization of risk assessment as the Environmental Protection Agency's (EPA) regulatory policy under President Ronald Reagan, the prevailing assumption about chemical toxicity was that the risks of disease or injury were relative to the amount of exposure. Put another way, any chemical would have some probability greater than zero of causing an adverse health effect. Conversely, no risk is absolute. In order to reduce risk, exposure to a given hazard must be minimized. A simple equation resulted: risk = hazard x exposure. As Arthur Daemmrich describes in his essay in this forum (pp. 685-695), the development of new methods and technologies for quantifying exposure inside the body (i.e. biomonitoring), meant risk equaled the hazard x presence.

In either equation, the logic only holds true given that the hazard remains constant, and for the hazard to remain constant, it must be considered absolutely necessary and therefore, exposure to it deemed unavoidable. Indeed, this narrative on risk was used to legitimize the proliferation of industrial chemicals throughout the twentieth century. As Jerry Markowitz and David Rosner detail in *Deceit and Denial,* in the mid-1920s the lead, oil, and automotive industries defended the introduction of lead into gasoline by arguing that it was necessary for American economic progress and that all technological development required some form of risk.[1]

This trope of the inevitability of chemical risks and the necessity of hazards for economic progress structured the central narrative of public relations campaigns of the chemical industry throughout the twentieth century. In the early 1970s, the Society of the Plastics Industry, the industry trade association, launched the public relations campaign, "Plastics Not Pollution." The campaign extolled the ecological and safety features of plastics at time when their pollution and toxicity were under considerable scrutiny.[2] Thirty years later, in response to a contemporary debate over the safety of endocrine-disrupting chemicals, such as bisphenol A, phthalates and atrazine, the chemical trade association, the American Chemistry Council, launched its "essential$_2$" campaign. This most recent campaign promoted the chemical industry as a necessary and fundamental component to a healthy economy, society, and environment.[3]

The legal conceptualization of risk as relative to the amount of exposure to a given chemical was formally articulated in the 1958 Federal Food, Drug, and Cosmetics Act, which regulated the "safe" exposure to chemicals in foods, including pesticides, color additives, and plastics (due to their ability to leach into food when used as wraps and containers). Prior to this Act, the food laws of 1906 and 1938 considered dangerous products or poisons as hazards per se,

regardless of any evaluation of safety, and, theoretically, restricted any poison from entering food. This per se standard was reversed in 1958 based on the logic that many of the new industrial chemicals in use, in particular the pesticide DDT, increasingly detected in cow's milk, were "necessary in production or unavoidable."[4] Such compounds, some lawmakers and FDA officials argued, couldn't be excluded per se; as such, safety standards were necessary to regulate their use. With the passage of the 1958 law, the regulation of chemicals in foods shifted from the per se rule to the de minimus standard that inscribed into law the notion that chemical risks were a function not of the hazard itself, but dependent upon the exposure. Safety, in turn, could be achieved not by questioning the hazard per se, but by minimizing the exposure.

The exception to this rule was the Delaney Amendment to the 1958 law, which established a zero tolerance standard for chemical carcinogens, effectively upholding the per se standard.[5] Industry trade groups and lawyers fought for decades to shift the interpretation of the clause from a per se rule, which effectively supported banning carcinogens, to the de minimus standard, which provided for the setting of safety standards for such chemicals. In 1962, the Delaney clause was effectively watered down by the DES Proviso, an amendment to the 1958 food law, which permitted the use of a carcinogen, diethylstilbestrol (DES), a drug used to increase livestock meat production, provided that no detectable amount of the chemical was found in the edible tissue. Risk, according to this proviso, was tied to quantifiable detection.[6]

During the mid-1970s, the Monsanto Chemical Company, with support from the major industry trade associations, successfully blocked efforts by the FDA to implement the Delaney clause for carcinogens in food by successful providing evidence that the migration of suspected chemicals from plastics into food, including vinyl chloride and acrylonitrile, fell below the detection limit. Additional efforts to block the restriction of carcinogens involved linking risk to the quantitative presence of a chemical *and* empirical evidence of adverse biological effects at the detection limit. In 1980 the Supreme Court upheld this interpretation of risk when it ruled in favor of the American Petroleum Institute and overturned the Occupational Safety and Health Administration's (OSHA) low safety standard for benzene. The American Petroleum Institute successfully argued that OSHA failed to provide quantitative evidence of adverse effects at the new, very low standard.[7] In 1996, the Delaney clause was finally removed from the law as it affected the regulation of pesticides in food with the passage of the Food Quality Protection Act that put in place a lowered tolerance limit for pesticides.[8]

THE DOSE MAKES THE POISON

TOXICOLOGY AS A SCIENTIFIC DISCIPLINE evolved in response to rising demands for regulations and litigation in the early part of the twentieth century. In contrast to industrial hygiene of the late nineteenth century and early twentieth century, toxicology shifted the study of toxic chemicals from the workplace to the laboratory where controlled experiments on animals were used to evaluate

the toxic effects of chemical exposure.[9] Experimental, animal-based research expanded to meet growing demands for safety standards in the workplace and consumer products, particularly with the establishment of the Occupational Safety and Health Administration and the Environmental Protection Agency in 1970. Safety standards such as permissible exposure levels, used in the occupational setting, and reference doses or safety standards for food additives or water pollutants, permit some small detectable levels of a hazard based on the fundamental toxicological principle, the dose makes the poison.

This founding principle is most frequently attributed to Paracelsus, a physician and alchemist of the eighteenth century. Paracelsus described the study of chemicals as a process of drawing distinctions between their therapeutic and toxic properties through experimentation. He contended that the difference between these two properties is often but not always "indistinguishable except by dose."[10] Determining the exposure level where the toxic response begins and ends represents the domain of regulatory toxicology.

Toxicological experiments used to derive regulatory safety standards throughout the twentieth century typically exposed adult animals to very high doses of a chemical to determine the lowest level at which a toxic effect occurs or preferably the level at which no toxic effect is seen—what's referred to as the lowest observed adverse effect level (LOAEL) or no observed adverse effect level (NOAEL). An additional "safety" or "uncertainty" factor of 100- or-1,000 fold is then added to the LOAEL or NOAEL to account for differences in human responses, vulnerable populations, and general uncertainty about the relationship between the dose and the response below this level. Exposures below the safety level are generally considered safe for humans, although rather than using empirical evidence of harm or lack of harm at low levels, varying predictive models of the dose-response relationship at levels below the LOAEL or NOAEL are employed to assess the risks of low-dose exposure.[11] Over the past fifteen years, however, studies measuring the effects of very low-dose exposures to pesticides, herbicides and industrial chemicals—levels presumed to be safe—began filling in the black box of low-dose effects.

CHALLENGES TO RISK AND TOXICOLOGY

IN 1991, AT THE INVITATION of Theo Colborn, an interdisciplinary group of researchers—wildlife biologists, experimental endocrinologists and molecular biologists—gathered together for several days at the Wingspread Conference Center in Racine, Wisconsin. The objective of this meeting was to discuss an inchoate body of research on the reproductive and developmental effects of chemicals capable of interacting with the hormone systems of laboratory animals, wildlife, and humans. The consensus statement produced from the workshop, what became known as the Wingspread Statement, declared with certainty that "a large number of man-made chemicals that have been released into the environment, as well as a few natural ones, have the potential to disrupt the endocrine system of animals, including humans." The effects of developmental exposure to such chemical compounds, collectively referred to as endocrine

disruptors, extended beyond cancer to include reproductive, immunological, behavioral and neurological abnormalities and diseases.[12]

Endocrine disruption quickly became a controversial scientific theory and a politically salient idea because it challenged the traditional boundaries of toxicology and risk assessment—not to mention the presumed safety of dozens of economically valuable chemicals. The theory argues that chemicals capable of interacting with the hormone system can elicit long-term and multigenerational effects at minute levels of exposure— levels detected in the ambient environment and inside human bodies.[13] This understanding challenged the presumption that unavoidable environmental exposure, long considered to be a necessary trade-off for the economic benefits chemicals afforded society, was safe.

Since the early 1990s, a growing number of studies have examined the long-term effects of exposure to hormonally active pesticides, flame retardants, plasticizers, and other industrial chemicals including bisphenol A and polychlorinated biphenyls, during critical periods of development—pregnancy and early in post-natal life. As Howard Bern, a prominent DES researcher since the 1970s and participant at the Wingspread workshop, wrote in 1992, the "fetus is fragile" because the communication systems directing development are laid down early in the organization of the living organism.[14] As opposed to examining the overt toxic effects of high doses, (i.e., death, malformation and/or cancer in adult animals), researchers working with endocrine disruptors began to investigate how low-dose, developmental exposures affect different hormonally sensitive tissues and organs of the reproductive, immune, metabolic, or neurological systems. Further, in the past five years, researchers have begun to examine how these low-dose functional changes or disruptions to major communication systems manifest as chronic disease (such as polycystic ovarian disease, endometriosis, and breast and prostate cancer) as the exposed animal's age.

Within this research framework, the timing of exposure and the timing of the observation of effect are critical. Since the 1970s, epidemiological and laboratory research on the reproductive effects of DES exposure during fetal development supported the supposition that timing of exposure is essential in determining the health effect. For example, the daughters of women who took DES during their pregnancies from the 1940s to the early 1970s developed rare vaginal cancers and reproductive problems in adult life, not the mothers themselves.[15] By emphasizing the importance of timing of exposure, the endocrine disruption thesis reframes the "dose makes the poison" principle as the "timing makes the poison."[16]

Since the early 1990s efforts to undermine research on endocrine disruptors as "junk science" or inappropriate for assessing human health risks has effectively shrouded the emergent field in uncertainty so as to control and minimize the political implications of this knowledge.[17] But despite efforts to block or undermine the endocrine disruptor thesis, research has snowballed over the past fifteen years, opening up new understandings of the interaction among genetic expression, chemical exposure and disease development. Recent research on endocrine disrupting chemicals suggests that these compounds alter heritable

gene expression through nonmutating, epigenetic mechanisms, such as DNA methylation—chemical modification that alters the expression of DNA without causing a change in the sequencing.[18] If small amounts of chemicals—levels present in the body and environment—can alter gene expression and disrupt tissue organization, which leads to increase disease susceptibility later in life, how do we begin to reframe the meaning of chemical risk and safety?

CONCLUSION

THE VAST PROLIFERATION of synthetic chemicals, particularly since the 1950s, was accepted, politically and legally, in the United States based on the assumption that chemical exposure was necessary for economic progress, but the risks of polluted bodies, water, air, and food could be minimized by reducing the exposure level—dilution as the solution to pollution. As scientific researchers began exploring the effects of endocrine-disrupting chemicals at very low, environmentally relevant levels in the 1990s, new understandings about the ability of synthetic compounds to turn on and off genes and alter the development of tissues and communication systems in the body fundamentally undermined the long held logic of safety and revealed that there is more to defining risk than the dose.

Such low dose work does not suggest an endocrine disruptor is hazard per se, as the Delaney clause sought to do for carcinogens, but rather is contingent on dose level (with lower doses sometimes having greater effects than higher doses) *and* the timing of exposure and effect.[19] If the timing makes the poison and low-dose exposure can manifest in long-term health problems then the discipline of toxicology and regulatory safety standards have failed to protect the public's health and a new paradigm of risk is desperately needed. By tracing the political, scientific, and physical construction of the synthetic human, historians of health and environment contribute to this process of re-imaging risk. They do so not simply by breaking down the straw man of the nature-culture or human-environment dichotomy, but by offering critical insight into the production of this binary, which informed the seeming inevitability of the petrochemical age and the synthetic human.

Sarah A. Vogel *received her PhD from Columbia University. Her dissertation, "The Politics of Plastics: The Political, Economic and Scientific History of Bisphenol A," examines the uneven development of legal and scientific understanding s of what constitutes a "safe" industrial chemical and a health risk from the 1950s to the present . She also holds a Master ofPublic Health and Master of Environmental Management from Yale University. She is the 2008-2009 Haas Fellow at the Chemical Heritage Foundation.*

NOTES

1. Jerry Markowitz and David Rosner, *Deceit and Denial: The Deadly Politics of Industrial Pollution* (Berkeley and Los Angeles: University of California Press), 26-27.

2. "Plastics Newsfront," *Plastics World*, September 1976, 9.
3. American Chemistry Council's "essential2" website: http://www.essential2.com/.
4. House Hearings before a Subcommittee of the Committee on Interstate and Foreign Commerce House of Representatives, *Federal Food, Drug, and Cosmetic Act (Chemical Additives in Food) on H.R. 4475, H.R. 7605, 7606, 8748, 7607, 7764, 8271, 8275*.84th Congress, 2nd Session. Statement of George Larrick, February 14, 1956.
5. John Wargo, *Our Children's Toxic Legacy: How Science and Law Fail to Protect Us from Pesticides* (New Haven: Yale University Press, 1996), 106-07.
6. Ibid.
7. Ibid.; Markowitz and Rosner, *Deceit and Denial,* 225-26.
8. The Food Quality Protection Act put in place a single standard for all pesticide residues on food (raw or processed) and provided for an additional safety factor for infants and children.
9. Chris Sellers refers to this transition as pax toxicologica in Christopher C. Sellers, *Hazards of the Job: From Industrial Disease to Environmental Health Science* (Durham: University of North Carolina Press, 1997).
10. Mary O. Amdur and John Doull, eds., *Casaret and Doull's Toxicology: the Basic Science of Poisons*, 5th ed. (New York: McGraw-Hill Health Professions Division, 1996), 5.
11. Robert Proctor, *Cancer Wars: How Politics Shapes What We Know and Don't Know about Cancer* (New York: Basic Books, 1995), ch. 7, 171.
12. Theo Colborn and Coralie Clement, "Chemically-induced Alterations in Sexual and Functional Development—The Wildlife/Human Connection," *Advances in Modern Environmental Toxicology* 21 (Princeton, N.J.: Princeton Scientific Publishing. Co., 1992).
13. W. V. Welshons et al., "Large Effects from Small Exposures. I. Mechanisms for Endocrine-Disrupting Chemicals with Estrogenic Activity," *Environmental Health Perspectives* 111 (2003).
14. Howard A. Bern, "The Fragile Fetus," in *Chemically-induced Alterations in Sexual and Functional Development: The Human/Wildlife Connection*, ed. Theo Colborn and Coralie Clement, Advances in Modern Environmental Toxicology, vol. 21 (Princeton, NJ: Princeton Scientific Publishing Co., 1992).
15. A. L. Herbst, H. Ulfelder, and D.C. Poskanzer, "Adenocarcinoma of the Vagina: Association of Maternal Stilbestrol Therapy with Tumor Appearance in Young Women," *New England Journal of Medicine* 284 (1971); Arthur L. Herbst and Howard Alan Bern, *Developmental Effects of Diethylstilbestrol (DES) in Pregnancy* (New York: Thieme-Stratton, 1981).
16. Philippe Grandjean et al., "The Faroes Statement: Human Health Effects of Developmental Exposure to Chemicals in Our Environment," *Basic and Clinical Pharmacology and Toxicology* (2007).
17. Steve Milloy, publisher of the website "Junk Science," "junkscience.com," called endocrine disruption as discussed in the popular book by Theo Colburn et al., *Our Stolen Future: Are We Threatening Our Fertility, Intelligence, and Survival?—A Scientific Detective Story* (New York: Plume, 1996), "junk science." See, http://www.junkscience.com/news/stolen.html.
18. D. Crews and J. A. McLachlan, "Epigenetics, Evolution, Endocrine Disruption, Health, and Disease," *Endocrinology* 147 (2006): S4-10.
19. This is referred to as a nonmonotonic dose response. See W. V. Welshons et al., "Large Effects from Small Exposures. I. Mechanisms for Endocrine-disrupting Chemicals with Estrogenic Activity," *Endocrinology* 111 (2003): S56-S69.

FREDERICK ROWE DAVIS

FORUM
unraveling the complexities of joint toxicity of multiple chemicals at the tox lab and the fda

> When the public protests, confronted with some obvious evidence of damaging results of pesticide applications, it is fed little tranquilizing pills of half truth. We urgently need an end to these false assurances, to the sugar coating of unpalatable facts. *It is the public that is being asked to assume the risks that the insect controllers calculate.* The public must decide whether it wishes to continue on the present road, and it can do so only when in full possession of the facts. In the words of Jean Rostand, "The obligation to endure gives us the right to know."[1]
>
> —Rachel Carson, *Silent Spring*, 1962

IN *SILENT SPRING*, Rachel Carson presented environmental risk to members of the public and urged them to consider their risk tolerance. In the statement above and throughout *Silent Spring*, Carson called upon the public to evaluate evidence and assess threats to environmental and public health. Carson's argument drew upon her exhaustive review of the scientific and medical literature that addressed ecological and human health effects of synthetic insecticides. To clarify her indictment of the chemical industry and federal agricultural and public health programs, Carson dramatized scientific and medical findings and personified them. As much as *Silent Spring* was about toxicity and lethal doses, it was about the victims of poisonings: American robins, bald eagles, Atlantic salmon, farm workers, and children.

Frederick Rowe Davis, "Unraveling the Complexities of Joint Toxicity of Multiple Chemicals at the Tox Lab and the FDA," *Environmental History* 13 (October 2008): 674-683.

In part, Carson blamed the lack of knowledge regarding ecological and health effects on overspecialization: "There is still very limited awareness of the nature of the threat. This is an era of specialists, each of whom sees his own problem and is unaware of or intolerant of the larger frame into which it fits."[2] Ironically, it was a new specialization that provided the tools to assess the novel risks: environmental toxicology. Despite her criticism of overspecialization, Carson wrote and interpreted the language of toxicology and environmental risk: "acute and chronic toxicity," "LD_{50}," "parts per million," "carcinogenicity," "reproductive effects." These phrases and the concepts they represent came to dominate the study and regulation of environmental risks such as synthetic insecticides.

My research traces the genesis of environmental toxicology and environmental risk in the United States. Since *Silent Spring*, toxicology and environmental risk have become the dominant paradigms for how scientists assess threats to the health of humans, wildlife, and ecosystems. Along with my initial objective of tracing the development of environmental risk, ancillary questions arose: Who were the scientists who developed the theory and particularly the practice of toxicology? What were their institutional affiliations and what groups supported their research? How did the methods of toxicology develop? What was the role of laboratory animals in toxicity studies? When did scientists become concerned with the impact of insecticides on wildlife? How did toxicology evolve as a distinct discipline? To what extent did scientists interact and cooperate with scientists at other agencies, universities, and corporations? How did toxicology and policy interact in the evolution of environmental laws? Many of these questions are directly related to the expansion of federally sponsored research at universities in the aftermath of World War II.

A growing group of scholars, some of whom contributed to this forum, examines the historical roots of toxicology. Linda Nash identifies two sources of environmental toxicology: bacteriology (broadly conceived) and industrial toxicology, neither of which provides a complete understanding of environmental risk. Nash argues: "Farm workers who traveled from field to field had far more complex and unknowable exposure histories than factory workers, and their past exposures increased their sensitivity to future exposures." (See Nash, p. 656, in this forum). In his foundational study, *Our Children's Toxic Legacy*, John Wargo broadens this claim: "In this study I have chosen to explore the significance of childhood exposure to a complex mixture of compounds permitted to exist as contaminants of human and animal foods and drinking water. We have been regularly exposed to an ever-changing mixture of these compounds in our diets, homes, schools, playgrounds and athletic fields, workplaces, and hospitals."[3] Sarah Vogel questions the dedication of toxicologists to dose response curves, particularly with respect to low dose exposures. Research into endocrine disruptors suggests that timing of exposure is equally if not more important than the dose. Thus, Vogel suggests that "The timing makes the poison." (See Vogel, pp. 667-673, in this forum.) In a recent study of diethylstilbestrol, Nancy Langston noted that researchers were puzzled when they discovered that lower exposures to DES seemed to have more toxic effects.[4] Each of these scholars questions the

utility of toxicology in understanding environmental risk, especially when applied to multiple chemical exposures and low dose exposures. Early toxicologists struggled to develop effective tools to assess the risks presented by these complex (yet common) problems in toxicology.

The historical origins of environmental toxicology can be traced to the science of pharmacology. Toxicologists, many of whom initially studied pharmacology, accepted the fundamental aphorism of toxicology, attributed to Paracelsus: "The dose makes the poison." This widely accepted translation of Paracelsus's statement has been questioned, and scholars have presented the following statement as an alternative that is closer to Paracelsus' original intent: "Solely the dose determines that a thing is not a poison." The dynamic between risk and benefit inherent to pharmacology permeates the science of toxicology also.

For evidence of the origins of toxicology in pharmacology, I look to two sources: the University of Chicago Toxicity Laboratory, which was formed with contracts from the Chemical Warfare Service during World War II, and the Division of Pharmacology at the Food and Drug Administration (FDA), which oversaw the analysis of new chemical insecticides during and after the war. Both institutions developed methodologies that came to define the science of toxicology, but in light of issues raised at the workshop, I am particularly interested in the efforts at Tox Lab to develop adequate measures of joint toxicity of multiple chemicals.

JOINT TOXICITY AND THE TOX LAB

WITH THE INTENSIFICATION of World War II, the National Defense Research Council (NDRC) contracted the University of Chicago to establish a facility capable of evaluating the toxicity of chemical agents for the Chemical Warfare Research Division of the Office of Scientific Research and Development. In doing so, the military officials hoped to avoid the crippling injuries inflicted on American troops by chemical warfare during World War I. One of the main reasons the NDRC selected Chicago was that the university possessed an unused smokestack at an old powerhouse which could be used to ventilate the laboratory. Along with extensive laboratory space for researchers, the Toxicity Laboratory contained facilities for animals ranging from monkeys and mice down to cockroaches and silkworms. In addition, Chicago had emerged as a center for research on the development of the atomic bomb. Finally, E. M. K. Geiling, recognized for his research on the toxicity of diethylene glycol, was an ideal unifying force for the project.[5]

Drawing on their roots in pharmacology, Geiling and his colleague Graham Chen first explored the subject of joint toxicity in the examination of several new antimalarial drugs. With the considerable development of antimalarial drugs during and immediately following World War II, some physicians began to experiment with combinations of different drugs with the expectation that they might be more effective than individual drugs in the cure of disease. Chen and Geiling sought to determine the joint toxicity in the host as well as the efficacy of various combinations of atabrine, quinine, hydroxyethylapocupreine, pamaquine, and pentaquine in mice.

For guidance regarding dosage mortality relationships, they turned to the research of Chester I. Bliss, a biologist and statistician who had developed for individual drugs rigorous biostatistical approaches to dose mortality curves and the LD_{50} ("Lethal Dose 50" or the dose that is lethal for 50 percent of an experimental population). In addition, Bliss devised statistical methods for the evaluation of joint toxicities. The subject was of interest to Bliss on theoretical grounds and for practical reasons, particularly with respect to new insecticides: "In the search for new insecticides combined poisons offer many possibilities, but criteria are needed for separating mixtures in which the combined ingredients possess an enhanced toxicity from others in which they act independently since the former group provides the more promising field of investigation." Bliss cited a study of the toxicity of rotenone-pyrethrin sprays in which the authors did not find evidence of synergism while another researcher utilized the same original data and discovered definite evidence of synergism.[6]

To resolve such confusion, Bliss defined three kinds of joint toxic action in which the percentage mortality was employed as the measure of response. In the case of "independent joint action," the poisons or drugs acted independently and had different modes of mortality. Susceptibility of an organism to one component might or might not be correlated with susceptibility to the other. Quantitatively, the toxicity of the mixture could be predicted from the dosage-mortality curve for each constituent applied alone and the correlation in susceptibility to the two poisons. Bliss employed the term "Similar Joint Action" for poisons or drugs that produced similar but independent effects such that one component could be substituted at a constant proportion for the other. Individual susceptibility would be completely correlated or parallel. Quantitative calculation of the toxicity of compounds with similar joint action could be predicted directly from the toxicities of the constituents as long as their relative proportion was known. Finally, and perhaps most significantly, Bliss delineated "Synergistic Action" in which the effectiveness or toxicity of a chemical mixture could not be assessed from that of the individual components, but rather depended upon knowledge of the chemicals' joint toxicity when used in different proportions. Synergistic action had the most serious implications for pharmacology and toxicology, because one component exacerbated or diminished the effect of the other.[7] As Bliss predicted, his research and methodologies had wide application in the development and applications of drugs, insecticides, and other chemical mixtures.

For their part, Chen and Geiling directly applied Bliss' definitions and methods to antimalarial drugs. Atabrine and quinine, for example, acted in an independent and similar manner, as did quinine and hydroxyethylapocupreine. However, the combinations of quinine and pamaquine as well as quinine and pentaquine were much more toxic than predicted from their individual toxicities, which was a clear case of synergism in the two combinations. The dosage mortality curves looked like those for different drugs, rather than the summation of the curves for the individual drugs. Chen and Geiling explained the joint toxicity of atabrine and quinine by suggesting a common site of action, but they were at a loss to explain the synergism between quinine and pamaquine.[8] Speculatively, Chen and Geiling

suggested that the joint toxicity might result from an effect on an enzymatic process essential for life. Emphasizing acute toxicity, their paper mentioned chronic toxicity only in passing, but this important distinction was often overlooked in the early toxicology and pharmacological literature. Joint toxicity would become an issue of central importance in the study and legislation of pesticides, particularly the organophosphates.

JOINT TOXICITY, POTENTIATION, AND OP CHEMICALS

THE ORGANIC PHOSPHATE INSECTICIDES (now organophosphates or OPs), demanded novel toxicological techniques and strategies, as had DDT and the chlorinated hydrocarbons. The Tox Lab responded to this considerable need. In particular, Kenneth DuBois recognized the major toxicological effects of the organic phosphates: cholinesterase inhibition. DuBois and his research group developed toxicological profiles for many of the new insecticides. In addition to DuBois's research, Arnold Lehman at the FDA compared the risks of the OPs to other insecticides like the chlorinated hydrocarbons. Like DuBois, Lehman constructed hierarchies of toxicity for the new chemicals. In general, the OP insecticides had a greater acute toxicity (due to cholinesterase inhibition), but considerably reduced chronic toxicity in comparison with the chlorinated hydrocarbons. One possible exception to this developing rule was malathion, or so American Cyanamid and scientists associated with it argued.

A team of FDA pharmacologists led by John P. Frawley analyzed the additive toxicity resulting from simultaneous administration of two anticholinesterase compounds, which was essentially a study of joint toxicity. After reviewing the rather sparse literature on the toxicity of OP insecticides, Frawley and his colleagues criticized previous studies for focusing on exposure to a single compounds rather than exposures to multiple compounds.[9] Individuals could be exposed to two OPs inadvertently through occupational exposure and even normal daily consumption patterns.[10]

Frawley and his team chose two OPs, EPN and malathion, because they were each less toxic than other OPs. First they determined the acute toxicity (LD_{50}) of each chemical for rats and dogs and then they established the toxicity of the two chemicals in combination. In dogs, EPN and malathion administered simultaneously caused up to 50 times the potentiation (additive toxic effects) of separate exposures. And they noted potentiation in rats as well. From these findings, Frawley and his team concluded that the hazard associated with chemical and drug combinations could not necessarily be evaluated from the toxicity of the individual compounds.[11] The FDA group also investigated the joint toxicity of malathion and EPN combined in several ratios, to house flies, using a housefly bioassay, but found no indication of potentiation. This finding suggested that potentiation involved complex chemical reactions between the two OPs and the biological system.

At the Tox Lab, Kenneth DuBois also addressed the potentiation of OPs. He reasoned that the simplest method for detecting potentiation by acute toxicity

tests would be to administer half of the LD_{50} of each of two OPs. If mortality due to the combination of the two compounds was additive (50 percent) or less than additive, no potentiation had occurred. DuBois used this approach to test for potentiation in several OPs and found that most showed additive or less than additive acute toxic effects. This meant that the combination of half of the LD_{50} of the two chemicals produced a toxic effect that was equal to or less than the full LD_{50} dose for either chemical. DuBois anticipated these results when the compounds had the same mode of action, parallel dosage-mortality responses, and a similar time of onset of toxic effects. From the results of the tests of acute toxicity, it became clear to DuBois that he had to clarify the mechanism of toxicity for each OP involved in potentiation to fully explore subacute effects. Such research revealed that some agents inhibited hydrolytic detoxification reactions. DuBois thought this discovery was potentially valuable for basic research into normal metabolism, but it left unresolved the implications for food residues and occupational exposures.[12] He noted, "Our present knowledge of the problem of potentiation of the toxicity of organophosphates does not provide an answer to the question of whether or not this effect constitutes a health hazard in connection with consumption of contaminated food."[13]

TOXICOLOGY FOR THE PUBLIC

AS THE 1950s PROGRESSED, scientists and regulators developed a deeper understanding of the risks related to insecticides. To the known risks posed by the heavy metal insecticides such as Paris Green and lead arsenate, scientists added cholinesterase inhibition. Potentiation significantly complicated the study of toxicology of insecticides and other chemicals. By the end of the decade, scientists and the public were raising questions regarding DDT and its long-term effects on wildlife. Moreover, Rachel Carson had begun work on a book she tentatively titled *Man against Nature* which would assess the threats posed by the new insecticides, including the organic phosphates. At the same time, scientists from the Tox Lab at the University of Chicago and the Division of Pharmacology at FDA were beginning to organize a separate discipline of toxicology.

The year 1958 marked several important new developments in evolution of toxicology into an academic discipline, but consolidation of the field among government and academic researchers had little impact on popular conceptions of changes in the natural world. Important insights could be gleaned from careful study of the toxicological literature, as we have seen. Nevertheless, several science writers simultaneously took up the subject of environmental contamination by pesticides, and it was these authors who educated the public, most notably Rachel Carson.

In *Silent Spring*, Carson established a hierarchy of insecticides. She first addressed the chlorinated hydrocarbons, starting with DDT, and progressively described other chemicals in the class, including chlordane, heptachlor, dieldrin, aldrin, and endrin. Carson wove details about their toxicity to mammals, birds,

and fish into her descriptions of the chlorinated hydrocarbons. In just a few pages, Carson introduced concepts such as bioaccumulation, lipofelicity (the bonding of chemicals to fats), passage of chemicals from mother to offspring via breast milk, food residues, and liver toxicity even at the residual levels found in food.[14] Nevertheless, Carson did not believe that chlorinated hydrocarbons posed the greatest threat to humans and wildlife: she had yet to address the OPs.

Carson left no doubt where OPs stood in the hierarchy of insecticides: "The second major group of insecticides, the alkyl or organic phosphates, are among the most poisonous chemicals in the world. The chief and most obvious hazard attending their use is that of acute poisoning of people applying the sprays or accidentally coming in contact with drifting spray, with vegetation coated by it, or with a discarded container."[15] Carson went on to describe the ironic development of the OPs as nerve gases during World War II and the incidental discovery of insecticidal properties; but it is her powerful description of the major effect of the OPs on organisms that sets her account apart from previous reports: "The organic phosphorous insecticides act on the living organism in a peculiar way. They have the ability to destroy enzymes—enzymes that perform necessary functions in the body. Their target is the nervous system, whether the victim is an insect or a warm-blooded animal."[16]

Aware that her subject demanded precision, she described the normal function of the central nervous system and the deleterious effects of excess acetylcholine, including tremors, muscular spasms, convulsions, and death.[17] But the body provided for this contingency: "A protective enzyme called cholinesterase is at hand to destroy the transmitting chemical once it is no longer needed. By this means a precise balance is struck and the body never builds up a dangerous amount of acetylcholine. But on contact with the organic phosphorus insecticides, the protective enzyme is destroyed, and as the quantity of the enzyme is reduced that of the transmitting chemical builds up. In this effect, the organic phosphorus compounds resemble the alkaloid poison muscarine found in a poisonous mushroom, the fly amanita."[18]

This elegant description of cholinesterase inhibition is both vivid and technically precise. Carson elucidated the relation between the symptomology of cholinesterase inhibition and the normal function of the nervous system, in a way that made clear the risk OP insecticides such as parathion posed to humans: "Repeated exposures may lower the cholinesterase level until an individual reaches the brink of acute poisoning, a brink over which he may be pushed by a very small additional exposure. For this reason it is considered important to make periodic examinations of the blood of spray operators and others regularly exposed."[19]

But what was the risk to people who were not exposed on a regular basis? Carson answered this question with additional data showing that seven million pounds of parathion was applied in the United States and the amount used on California farms alone could "provide a lethal dose for 5 to 10 times the whole world's population."[20] What saved the people of the world was the rate at which the organic phosphorous chemicals decomposed. They broke down into harmless

components rapidly in comparison to the chlorinated hydrocarbons, and their residues did not remain as long, yet even relatively small quantities remaining posed a real threat: "The grove had been sprayed with parathion some two and a half weeks earlier; the residues that reduced [eleven out of thirty men picking oranges] to retching, half-blind, semi-conscious misery were sixteen to nineteen days old."[21] Carson noted that similar residues had been found in orange peels six months after the trees had been treated with standard doses.

Not even malathion, the OP insecticide with the lowest toxicity, escaped Carson's perceptive analysis. Malathion, according to Carson, was almost as familiar to the public as DDT. It was used in gardens, household insecticides, and mosquito spraying. Carson revealed that nearly a million acres of Florida communities had been sprayed with malathion in an attempt to control the Mediterranean fruit fly. She questioned the assumption of many people the they could use malathion freely and without harm: "Malathion is 'safe' only because the mammalian liver, an organ with extraordinary protective powers, renders it relatively harmless. The detoxification is accomplished by one of the enzymes of the liver. If, however, something destroys this enzyme or interferes with its action, the person exposed to malathion receives the full force of the poison."[22] Citing research on potentiation by the FDA and Kenneth DuBois, Carson explained that the synergy between two OP chemicals could significantly exacerbate the effects of either or both.[23] Moreover, Carson cited evidence that potentiation was not limited to the OPs. Parathion and malathion intensified the toxicity of certain muscle relaxants and others (malathion included) dramatically increased the effect of barbiturates.

Carson stressed that the advantages that OPs possessed over the chlorinated hydrocarbons, such as rapid decomposition, were significantly offset by the dangers of cholinesterase inhibition and potentiation. Her remarks on the acute toxicity of the various pesticides were only a preamble to her larger case: namely the long-term risks of pesticides (particularly the chlorinated hydrocarbons) to landscapes, wildlife, and humans. In the remainder of *Silent Spring*, the organic phosphate insecticides recede to the background. Although Carson thoroughly documented and dramatized the lingering damage to soil, water, flora, and fauna associated with chlorinated hydrocarbons, her research revealed few such problems with the organic phosphates. Her one example of the effects of OPs on wildlife was typically dramatic. In an attempt to control flocks of blackbirds that fed on cornfields, a group of farmers engaged a spray plane to spray a river bottomland with parathion. More than 65,000 Red-winged blackbirds (*Agelaius phoenicus*) and European starlings (*Sturnus vulgaris*) died, and Carson wondered how many other animals perished from the acute effects of this universally toxic substance. Had rabbits, raccoons, and opossums succumbed as well? Carson was most concerned, however, about unintended effects on humans, workers, and children.[24]

Most of *Silent Spring* focused on the more subtle chronic effects of chlorinated hydrocarbons. Were any such effects tied to organic phosphate insecticides? To support her claim that there might be, Carson recounted the case of "ginger

paralysis," a condition brought about when people consumed "Jamaican ginger" as an alternative to the more expensive medicinal products substituted for liquor during Prohibition. The fake ginger contained triorthocresyl phosphate, which Carson noted destroyed cholinesterase in the same way that parathion did. More than 15,000 people were permanently crippled by a paralysis of their legs accompanied by destruction of nerve sheaths and the degeneration of spinal cord cells. Carson compared the effects of OP poisonings to ginger paralysis. Even malathion had induced muscular weakness in chickens and, just as in ginger paralysis, the sheaths of the sciatic and spinal nerves were destroyed. Carson even found evidence that regular exposure to organic phosphate insecticides might induce mental disease.[25] It is clear that Carson believed that the OPs posed an equivalent, if not greater, risk to wildlife and humans than the chlorinated hydrocarbons.

The current concern regarding joint toxicity of multiple insecticides has deep roots. As toxicology emerged from pharmacology, scientists at the Tox Lab determined the toxicities of antimalarial drugs in combination. Kenneth DuBois extended these investigations to include the OP insecticides and the FDA pharmacologists contributed to the understanding of potentiation. In *Silent Spring*, Rachel Carson animated the research conducted at the Tox Lab and the FDA to indicate the considerable risks posed by OP insecticides, while noting their widespread use in the United States. Ironically, when DDT was banned in 1972, by all accounts a major victory in the American environmental movement, many farmers turned increasingly to OPs to continue the never-ending battle against agricultural pests. By studying the emergence of toxicology from pharmacology, historians can disentangle the tightly woven strands of risk and benefit.

Frederick Rowe Davis *is associate professor of history at Florida State University, where he teaches the history of science and medicine and environmental history. He is the author of* The Man Who Saved Sea Turtles : Archie Carr and the Origins of Conservation Biology *(Oxford, 2007).*

NOTES

1. Rachel Carson, *Silent Spring* (Boston: Houghton Mifflin Company, 1962), 13, emphasis added.
2. Ibid.
3. John Wargo, *Our Children's Toxic Legacy: How Science and Law Fail to Protect Us from Pesticides* (New Haven: Yale University Press, 1996).
4. Nancy Langston, "The Retreat from Precaution: Diethylstilbestrol (DES), Endocrine Distruptors, and Environmental Health," *Environmental History* 13 (January 2008): 41-65.
5. See James Harvey Young, "Sulfanilamide and Diethylene Glycol," in *Chemistry and Modern Society: Historical Essays in Honor of Aaron J. Ihde*, ed. John Parascandola and James C. Whorton (Washington, DC: American Chemical Society, 1983).
6. Cited in C. I. Bliss, "The Toxicity of Poisons Applied Jointly," *Annals of Applied Biology* 26 (1939): 586.

7. C. I. Bliss, "The Toxicity of Poisons Applied Jointly," *Annals of Applied Biology* 26 (1939): 585-587.
8. Graham Chen and E. M. K. Geiling, "The Acute Joint Toxicity of Atabrine, Quinine, Hydroxyethylapocupreine, Pamaquine and Pentaquine," *Journal of Pharmacology and Experimental Therapeutics* 91 (1947): 138-39.
9. John P. Frawley et al., "Marked Potentiation in Mammalian Toxicity from Simultaneous Administration of Two Anticholinesterase Aompounds," *Journal of Pharmacology and Experimental Therapeutics* 121 (1957): 96.
10. For examples, see Pete Daniel, *Toxic Drift: Pesticides and Health in the Post-World War II South* (Baton Rouge: Louisiana State University Press in association with Smithsonian Institution, Washington, DC, 2005); andLinda Lorraine Nash, *Inescapable Ecologies: A History of Environment, Disease, and Knowledge* (Berkeley and Los Angeles: University of California Press, 2006).
11. Ibid., 106.
12. Kenneth P. DuBois, "Potentiation of the Toxicity of Insecticidal Organic Phosphates," *A.M.A. Archives of Industrial Health* 18 (1958a): 490-96.
13. Ibid., 495.
14. Carson, *Silent Spring*, 26-27.
15. Ibid., 27-28.
16. Ibid., 28.
17. Ibid., 28-29.
18. Ibid., 29.
19. Ibid.
20. Ibid., 30.
21. Ibid.
22. Ibid., 31.
23. Ibid., 31-32.
24. Ibid., 126-27.
24. Ibid., 196-98.

ARTHUR DAEMMRICH

FORUM
risk frameworks and biomonitoring:
DISTRIBUTED REGULATION OF SYNTHETIC CHEMICALS IN HUMANS

ENVISION BRIEFLY TWO hypothetical individuals. The first is a chemical engineer who works directly in the synthesis of compounds added to plastics to give them desired properties such as flexibility and hardness. By some analyses, this person has taken on voluntary risks related to the workplace; furthermore this person is protected by federal and state regulations governing chemical exposure and worker safety.[1] The second is a vegan environmentalist who works in a natural foods store and enjoys hiking in national parks. This person makes lifestyle choices to avoid exposure to synthetic chemicals and may reasonably assume that federal laws regulating the introduction of new chemicals ensure safety under normal conditions. Yet when their blood is tested, they have similar levels—measured at parts per billion—of compounds known to cause harm at much higher doses. How should they interpret this finding? Is it possible that people with such different exposure to chemicals have similar "body burdens"?[2] Because of the minute quantities of materials that all of us absorb through regular encounters with synthetics, this scenario of equivalent measures in two otherwise different people can occur. Whether drinking from plastic bottles while enjoying remote vistas, working at a chemical plant, or engaged in one of thousands of routine daily activities, we are exposed to trace amounts of industrial compounds that make their way into our bodies and environmental systems.

Arthur Daemmrich, "Risk Frameworks and Biomonitoring: Distributed Regulation of Synthetic Chemicals in Humans," *Environmental History* 13 (October 2008): 684-694.

Over the past century, and at an accelerated rate in recent years, new analytical tools have made it possible to identify substances at a part per million, billion, or even trillion (the equivalent of finding a single grain of sand in an Olympic-sized swimming pool). In the United States, the Centers for Disease Control and Prevention (CDC) now regularly survey the population for the presence of 275 specific chemicals. In addition to the CDC surveys, biomonitoring studies carried out by academic scientists and by a variety of environmental NGOs are generating significant new data about the presence of chemicals in the U.S. population and eventually will measure changes in chemical presence over time. These efforts to measure and track chemicals pose a complex challenge to a regulatory system that historically was focused on controlling the physical location of hazardous compounds and measured risk based on a calculus of hazard and exposure.[3]

This essay argues that new technologies for measuring synthetic chemicals in humans, innovations in the field of toxicology, and increased mobilization by environmental nongovernment organizations (NGOs) regarding chemical exposure are combining to undermine risk frameworks and regulatory systems built up over the past quarter-century. In effect, the very definition of risk used in regulatory decisions is at play as a result of biomonitoring's emerging measures of chemicals in bodies at parts per billion or below. I argue that a two-part shift is underway associated with biomonitoring: first, from regulating chemicals by physical location to regulating them by chemical reaction; second, from defining risk as a function of exposure to chemicals to basing it on the presence of compounds in the body. Regulating chemicals by location relied on clear delineations between industry and government, with central control and oversight by federal agencies producing visible environmental and human health benefits. Regulating chemicals by reaction will involve distributed controls and require greater cooperation among interested parties to define standards, carry out biomonitoring, and make policy decisions that draw on test results. Drawing on the historical trajectory of changing regulatory methods, this essay advances a regulatory framework that involves collaborative testing programs and information sharing among industry, NGOs, and government agencies.

REGULATION BY LOCATION

EVER SINCE THE CHEMICAL INDUSTRY'S origins in mining and extraction, though with greater intensity once synthetic dyes fostered international growth in the late nineteenth century, efforts at controlling human and environmental exposure have focused on location. Materials in the wrong place, such as leftovers from potash and soda manufacture or synthetic dyes coloring rivers purple were considered pollutants. Materials in the right place were productive elements of an industrial age that promised greater prosperity and quality of life.[4]

As the first legal and regulatory interventions emerged to control the disposal of byproducts of manufacturing, physical location proved critical. Over the course of the latter third of the twentieth century, the EPA, industry, and academic scientists developed sophisticated models for how pollutants travel in air and water or migrate through soil. Disputes, such as over the Love Canal disaster,

have hinged in part on testing for the physical presence of hazardous compounds. Resolution in cases of accidental releases or discovery of buried toxins involved moving people or physically removing the pollutant, frequently at high costs.[5]

Beginning in the 1960s, however, a combination of new laboratory and field studies shifted attention to the finding that chemicals in the environment could harm wildlife and cause cancer in humans.[6] Much of the subsequent political mobilization around carcinogens sought to develop control measures on pollution, whether from a smokestack or a waste stream. Some studies also began tracking human health effects of chemicals found in consumer products and packaging, including studies of toxicity an carcinogenicity.

The central legislative act for regulating chemical safety in the United States, the Toxic Substances Control Act (TSCA), was passed by Congress in 1976. Written at a time of concern with cancer and its relationship to environmental factors, the legislation listed and banned known or suspected carcinogens, including vinyl chloride, asbestos, and PCBs.[7] Unlike other federal environmental regulatory statutes, such as the Clean Air Act or Clean Water Act, TSCA required manufacturers to characterize the risks posed by new chemicals before they could be introduced to commerce. As implemented, this provision required firms to submit a pre-manufacture notification (PMN) ninety days before producing or importing a new chemical substance. Yet in the period between 1979 and 2002, some two-thirds of PMNs filed with the agency failed to provide complete data on physical properties or environmental impacts. As a result, the agency largely regulated compounds based on structure-activity relationships in which potential effects of a new chemical are estimated based on the known characteristics and effects of structurally similar molecules. More recently, voluntary initiatives such as the high production volume testing program have begun to fill in gaps for basic information on chemicals in commerce.[8]

TSCA's sections concerning the regulation of existing chemicals called for the EPA to balance the economic and social benefits derived from the use of a chemical against its risks. The agency was to regulate those chemicals that presented an "unreasonable" risk of harm to human health or the environment.[9] Since Congress did not specifically define "unreasonable" risk, the agency found itself caught in extensive and costly cycles of litigation and delay as environmental groups and manufacturers interpreted the benefit-risk balance differently. Since 1976, only six existing substances or chemical groups have been banned. Other substances in widespread commercial use for decades, including phthalates, bisphenol-A, and brominated fire retardants remain embroiled in disputes over exposure, effects on humans and wildlife, and the economic consequences of regulation.[10]

Facing a multidecade dilemma of both incomplete data on synthetic materials and scientific uncertainty about how to design risk assessments that did not "unreasonably" take chemicals out of commerce, EPA's regulatory approach became dominated by exposure assessment. Officials focused on manufacturing sites, transportation pathways, and (less frequently) people living in close proximity to manufacturers. The combination of occupational health and safety

laws with TSCA meant that most monitoring for health impacts from exposure was carried out on plant workers. As Michael Egan points out elsewhere in this forum (pp. 636-642), once compounds are in the environment and interact with biological systems, tracing their origins can become nearly impossible.

Identifying hazards through place-based analysis and regulating pollution through end-of-pipe controls are fundamentally linked to defining risk as hazard times exposure (see Sarah Vogel's essay, pp. 667-673 in this forum, for more on the development of the formula, risk = hazard x exposure). With the hazards posed by pollution often visible or odorous, wealthier people physically separated themselves from manufacturing sites.[11] A prevailing assumption, seemingly borne out by public health surveys, was that avoiding direct contact with pollutants would reduce cancer rates and extend healthy lifespan. Under this regulatory framework, government agencies regulated "point-sources" of pollution and forced environmental cleanups in order to ensure safe physical locations for humans and wildlife.

REGULATION BY CHEMICAL REACTION

THE PROLIFERATION OF BIOMONITORING studies since the early-1990s is challenging key aspects of prevailing methods for calculating risk and controlling chemicals. Two kinds of studies have emerged in recent years, with broad surveys by the government tracking chemicals across the entire population and narrower studies by academics and NGOs focusing on specific compounds or subpopulations.[12] Together, these tests are indicative of a shift underway from pollution in air, water, or soil to measuring chemicals in bodies. They also suggest that regulatory systems based on central government control over the location of chemicals will need reform to achieve human and environmental health goals based on interactions among chemicals and body systems.

The origins of what today has become known as biomonitoring can be dated back to public health studies of lead levels among inhabitants of Baltimore, Boston, and other northeast cities starting in the 1890s.[13] During the following century, testing people's blood, teeth, tissue, or fat for the presence of specific compounds was undertaken in an episodic manner. Nevertheless, biomonitoring studies affected policy debates in areas as diverse as nuclear testing and worker safety.

In the late 1950s, Barry Commoner and the Committee for Nuclear Information initiated a study that eventually collected over 60,000 children's teeth and measured strontium-90 absorption. Results suggested that background levels of the radioactive material had increased one-hundred-fold after 1948, providing a powerful rationale for the ban on atmospheric nuclear weapons testing passed in 1963.[14]

In a second example, the publication of studies linking vinyl chloride (a precursor to PVC) to cancer in laboratory animals and identification of VC-related cancers in employees at a B.F. Goodrich plant in Louisville sparked a controversy regarding the compound in the early 1970s.[15] In the wake of a contentious exposure

standard-setting process by the Occupational Safety and Health Administration (OSHA), firms began to closely monitor workers exposed to vinyl chloride and other compounds by collecting urine and blood samples.

As a result of these and other controversies, government agencies ranging from the Nuclear Regulatory Commission to OSHA became aware of new analytical techniques for identifying chemicals in humans. In the mid-1990s,the CDC began to include data on lead and compounds found in cigarette smoke in the National Health and Nutrition Examination Survey (NHANES) it had administered since 1971. After 2001, the CDC NHANES report incorporated biomonitoring studies, initially measuring twenty-seven chemicals.[16] Phthalates—compounds that act as plasticizers in many consumer products—were found at unexpectedly high levels and sparked interest among NGOs in the United States and Europe. Media reports soon drew attention to the fact that we all contain these chemicals.[17]

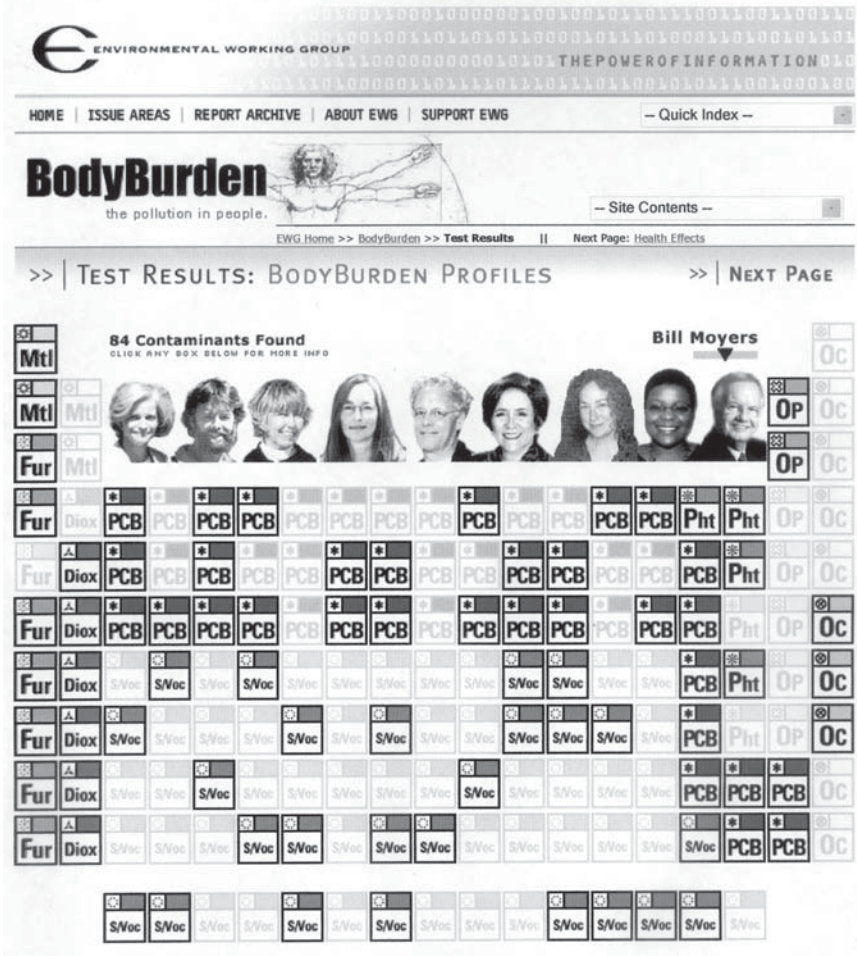
CDC released a second *National Report on Human Exposure to Environmental Chemicals*, in February 2004.[18] Covering 116 chemicals measured in blood and urine from a sample of 3,500 people, the survey targeted a number of specific populations, including African-Americans, whites of lower economic class, pregnant women, and people over 60 years old. These populations were chosen partly in response to concerns expressed by environmentalists and public health experts that data available to EPA rarely includes populations other than plant workers. Likewise, CDC's selection of chemicals to monitor was based on several criteria, including suspected health consequences of exposure and direct lobbying by environmentalists and public health experts.

The 2005 CDC report covered 148 chemicals based on a sample of 2,400 people from across the United States.[19] The data showed both encouraging and potentially alarming trends. Among the former, the pesticides aldrin, endrin, and dieldrin were undetected or only present at very low levels and the percentage of children with elevated blood lead levels declined significantly from previous surveys. Phthalates were measured with greater sensitivity, though CDC noted that there is little basis yet to judge health effects at the detected levels, stating "just because people have an environmental chemical in their blood or urine does not mean that the chemical causes disease."[20] Other compounds present in people included polycyclic aromatic hydrocarbons and a variety of dioxins and furans. A fourth national report scheduled for release in late 2008 will cover 275 chemicals; CDC also has begun issuing interim reports on specific compounds.

Largely independent of government-sponsored surveys, NGOs and academic centers have collected data on synthetic chemicals in humans through their own biomonitoring studies. Sampled populations and reporting styles have varied considerably, ranging from politicians to babies, and from peer-reviewed publication to interactive websites. A common thread to the NGO studies is the goal of using results as the basis for mobilizing public concern and political action.

In perhaps the best-known series of nongovernmental biomonitoring surveys, the Environmental Working Group (EWG) began to test adults and children for

Figure 1. Body Burden Profile.



The EWG "Body Burden" website visually portrayed testing results for a small group of people in a style similar to the periodic table of the elements. This image features chemicals found in Bill Moyers, a journalist, commentator, and critic of the chemical industry.

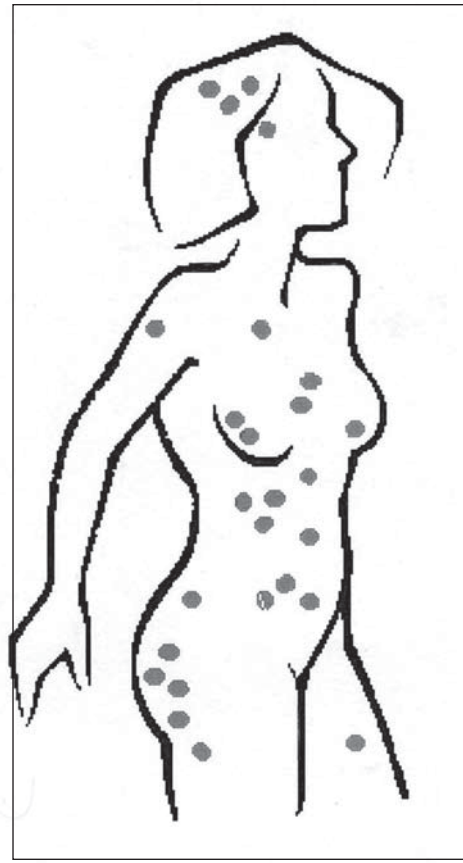
the presence of synthetic chemicals in 2002. An initial study carried out with Mount Sinai School of Medicine included twelve well-known scientists and media personalities. EWG found an average of 91 industrial compounds in each person, with a total of 167 different chemicals across the group. Results were then displayed on the web in an interactive format reminiscent of the periodic table showing the variants of PCBs, furans, and other organic compounds in each participant's blood.[21] More recently, EWG shifted attention to infants as an at-risk population with a study measuring the transfer of industrial chemicals from mothers to their daughters through the placenta or breast milk. Concerned that "a substantial portion of the chemical burden inherited at birth by the daughters in this study will last for decades; some will last a lifetime," EWG recommended

that consumers should avoid food, packaging, and household items containing six specific compounds, ranging from lead to brominated flame retardants.[22]

Similarly, the World Wide Fund for Nature tested blood samples from 156 Europeans in 2003, including fourteen ministers of health and environment. Findings showed that all participants contained polychlorinated biphenyls, organochlorine pesticides, and other compounds. Results were published in a report titled "Bad Blood."[23] Reporting the results, Dr. Vyvyan Howard of the University of Liverpool stated, "exposure is universal."[24]

A second type of NGO study more explicitly seeks to promote an environment free from chemical exposure. For example, the "Coming Clean Network" website shifts the reader's attention from well-known cancer sites such as a woman's breast to risks posed by chemicals located elsewhere in the body. The organization presents case studies of ex-posure and makes suggestions for how to avoid contact with toxins.[25] Likewise, a 2004 report by the Pesticide Action Network (PAN) featured cover imagery of children at play and a mother and child facing the threat of unwelcome chemical exposure from industrialized agriculture.[26]

Figure 2. Coming Clean.



The Coming Clean Network's portrayal of a body at risk suggests that potential toxins are distributed widely, not just concentrated in one place.

Third, a number of interactive websites give estimates of people's exposure to synthetic chemicals and their consequent "body burden" based on questions about lifestyle, including the use of cosmetics and cleaning agents.[27] Combining play with education, the World Wildlife Fund hosts an asteroids-type video game called "Toxic Blaster" in which visitors pilot a spaceship that shoots toxins, first in a polar bear, then a whale, and ultimately in a human. Screens pop up between levels that invite participants to "take action now" by learning more about chemicals and by contacting congressional representatives.[28]

Unlike the CDC's population survey and high level of data aggregation, NGO-

Figure 3. Toxic Blaster.

The toxic blaster site seeks to use a video game to mobilize political action regarding chemicals in wildlife and humans.

sponsored studies personalize biomonitoring data and couple results to suggestions for how individuals can take action in the absence of government regulation of compounds such as phthalates, bisphenol-A, and others. In a recent example of their influence, Nalgene, the manufacturer of polycarbonate drinking bottles popular with outdoor enthusiasts, announced the phase-out of bisphenol-A, while expert commissions and regulators in the United States, Canada, and European Union came to different conclusions about designating it a toxic substance.[29]

Many of the biomonitoring studies are not comparable, making it difficult to analyze a specific chemical across different tests. Likewise, at present no coordinated priority-setting is taking place among the groups carrying out studies to select among the 15,000 chemicals in commerce or the 80,000 chemical substances listed on the EPA's complete inventory. Diverse national surveys, population surveys, studies of specific sub-populations, and even tests of small influential groups pose a significant challenge to traditional regulatory analysis.

As biomonitoring expands its reach, it will impact the risk calculus employed in regulatory decision-making. From measuring risk as a product of hazard and exposure, risk is shifting to be defined as hazard times the amount of a compound measured in the body. Theoretically, large-scale testing programs underway to collect and publish hazard data on chemicals in commerce could be coupled to biomonitoring results to give far more detailed risk measures than were previously possible. This will require both additional testing and a more distributed and coordinated regulatory approach.

DEMOCRATIZING RISK

ADVANCES IN ANALYTICAL CHEMISTRY, increased mobilization by environmental NGOs, and the emergence of biomonitoring studies are contributing to an emerging form of risk democratization. As we learn about the ways in which trace amounts of chemicals enter our bodies, wealthier people no longer escape exposure as they hoped to in the past by moving to less industrialized landscapes. While workers in developing countries who disassemble discarded electronics clearly are exposed to more flame retardants and heavy metals than those who use computers in office buildings, there are no obvious routes to avoid any measurable exposure. Biomonitoring thus challenges the concept that specific, identifiable, and manageable parts of technical, engineering, and industrial systems are the key loci of risk. Now consumer culture itself acts as an exposure pathway and risk conduit (a concept developed further in Michelle Murphy's essay in this volume).

We live in an era in which consumer products, electronic devices, food packaging, and building materials are manufactured by the chemical industry. NGOs have endeavored in recent years to make the underlying substances, supply chains, and exposure pathways more visible to the public, in part through biomonitoring studies. For regulators, this information poses challenges to ways of calculating risk and ensuring the safety of chemicals.

At the same time, over the last two decades command-and-control regulation reached its political limits and deregulatory initiatives were advanced in the United States. For a variety of testing initiatives and reporting programs, ranging from high-production volume chemicals to the toxic release inventory, the EPA acts less like a traditional regulator than as a forum hosting results that are then debated and used by industry and NGOs. Effective regulation based on biomonitoring studies will require industry, NGOs, and the EPA to resolve what will count as appropriate tests in this area. Currently we are relying—for better or worse—on industry to self-regulate under watchful and critical NGO observation.

Biomonitoring would benefit from a central data depository that would standardize and electronically post results. In this new regulatory model, the EPA would host data and ensure that test results meet basic standards. Unlike the current division among agencies or divisions responsible for insecticides, food packaging, cosmetics, and other products, a central biomonitoring data set is needed to make meaningful comparisons, measure risk levels, and understand exposure pathways. The initial data set could be built out of CDC and other national surveys, but to be effective and inclusive, results from industry studies and NGO surveys of targeted communities should also be included. Over time it would become clear whether levels are declining or increasing, epidemiologists and other public health scientists could draw on the data sets for research, and the public could view results in a single location.

The social and political roles of government, citizen groups, and industry shifted significantly over the past three decades. Government agencies used to issue standards and rules; now they sometimes coordinate voluntary testing programs. NGOs used to ask the government to regulate more; now they directly

negotiate with or confront the private sector, often with extensive scientific data in hand.[30] In the case of toxics release inventories, they largely rely on firms to supply data to EPA. In biomonitoring studies, NGOs play an active role in developing the data. Citizens used to assume certain relationships with the government and industry, but consumers have a status based more on financial power than voting. Corporations have taken on certain attributes previously thought to belong to nation-states, but have not felt compelled to institute structured forms of representation beyond shareholder (proxy) voting. Resolving these dilemmas and stabilizing the shifting roles of industry, government, and NGOs are critical to the future of chemicals regulation.

A distributed system for testing and hosting data holds the potential for greater transparency and improved availability of information about chemicals in consumer products than has been possible under classic regulatory structures. This neoliberal model should not preclude the possibility of more traditional rule-making or standard-setting in cases where concerns emerge under the new risk model. But it does offer a means to overcome stasis in an area of high uncertainty like biomonitoring at present.

Arthur Daemmrich *is assistant professor at Harvard Business School.* *The author thanks Nancy Langston and Jody Roberts for their detailed review of an earlier version of this paper and other forum participants and Alastair Iles for feedback on the ideas presented here.*

NOTES

1. Chauncey Starr, "Social Benefit versus Technological Risk: What is Our Society Willing to Pay for Safety?" *Science* 165 (1969): 1232-38; Baruch Fischoff et al., "How Safe is Safe Enough? A Psychometric Study of Attitudes toward Technological Risks and Benefits," *Policy Sciences* 9 (1978): 127-52.
2. Environmental NGOs popularized the term "body burden" to signal a total synthetic chemical load borne by humans. See: www.chemicalbodyburden.org.
3. Scholars have long noted the influence of values, ethics, and political culture on regulatory definitions of risk. See: Mary Douglas and Aaron Wildavsky, *Risk and Culture: An Essay on the Selection of Technological and Environmental Dangers* (Berkeley and Los Angeles: University of California Press, 1982); Ronald Brickman et al., *Controlling Chemicals: The Politics of Regulation in Europe and the United States* (Ithaca, NY: Cornell University Press, 1985), 187-217; Sheila Jasanoff, *Risk Management and Political Culture* (New York: Russell Sage Foundation, 1986), 25-32, 69-77.
4. John K. Smith, "Turning Silk Purses into Sows' Ears: Environmental History and the Chemical Industry," *Enterprise and Society* 1 (2000): 785-812.
5. Adeline Levine, *Love Canal: Science, Politics, and People* (Lexington, MA: Lexington Books, 1982); Marc Landy et al., *The Environmental Protection Agency: Asking the Wrong Questions* (New York: Oxford University Press, 1990).
6. Rachel Carson, *Silent Spring* (Boston: Houghton Mifflin, 1962), 15-51.
7. H.R. Rep. No. 94-1241, at 4.
8. www.epa.gov/hpv/index.htm.
9. Library of Congress, Environment and Natural Resources Policy Division, *Legislative*

History of the Toxic Substances Control Act (Washington, DC: U.S. Government Printing Office, 1976).

10. McKenna Long & Aldridge LLP, *TSCA Handbook*, 4th Ed. (Lanham, MD: Scarecrow Press, 2005), 7-12; Theo Colborn et al., *Our Stolen Future* (New York: Penguin, 1997), 210-30.
11. This issue came to the fore in the environmental justice movement of the 1990s: see Phil Brown, *Toxic Exposures: Contested Illnesses and the Environmental Health Movement* (New York: Columbia University Press, 2007); and Robert Bullard, *Dumping in Dixie: Race, Class, and Environmental Quality* (Boulder, CO: Westview Press, 1990).
12. Ken Sexton et al., "Human Biomonitoring of Environmental Chemicals," *American Scientist* 92 (2004): 38-45; Alastair Iles, "Identifying Environmental Health Risks in Consumer Products: Non-Governmental Organizations and Civic Epistemology," *Public Understanding of Science* 16 (2007): 371-91.
13. Christian Warren, *Brush With Death: A Social History of Lead Poisoning* (Baltimore: Johns Hopkins University Press, 2000); Gerald Markovitz and David Rosner, *Deceit and Denial: The Deadly Politics of Industrial Pollution* (Berkeley: University of California Press, 2002).
14. Michael Egan, *Barry Commoner and the Science of Survival* (Cambridge: MIT Press, 2007).
15. Joseph Badaracco, *Loading the Dice: A Five-Country Study of Vinyl Chloride Regulation* (Boston: HBS Press, 1985), 5-18.
16. Centers for Disease Control and Prevention, *First National Report on Human Exposure to Environmental Chemicals* (Atlanta: CDC, 2001).
17. For representative examples, see Peter Waldman, "Common Industrial Chemicals in Tiny Doses Raise Health Issue," *Wall Street Journal* (25 July 2005): A1, A12; and David Duncan, "Pollution Within," *National Geographic* (October 2006): 116-43.
18. Centers for Disease Control and Prevention, *Second National Report on Human Exposure to Environmental Chemicals* (Atlanta: CDC, 2004).
19. Centers for Disease Control and Prevention, *Third National Report on Human Exposure to Environmental Chemicals.* (Atlanta: CDC, 2005).
20. CDC, *Third National Report*, Executive Summary, 2.
21. The site subsequently was taken down, although the EWG has extensive information at www.ewg.org/sites/humantoxome.
22. Environmental Working Group, "Across Generations" (May 10, 2006), 2. See also Jane Houlihan et al., *BodyBurden: The Pollution in Newborns* (Washington, DC: EWG, 2005).
23. World Wildlife Fund, *Bad Blood? A Survey of Chemicals in the Blood of European Ministers* (Brussels: WWF, 2004).
24. "EU Citizens are Walking Chemical Cocktails," EUobserver.com (August 6, 2003).
25. See: www.chemicalbodyburden.org.
26. Kristin Schafer et al., *Chemical Trespass: Chemicals in Our Bodies and Corporate Responsibility* (San Francisco: Pesticide Action Network, 2004).
27. See: Inside Bay Area, "A Body's Burden: Our Chemical Legacy," extras.insidebayarea.com/bodyburden/bodyburden.html.
28. http://assets.panda.org/custom/games/toxicblaster.
29. Ian Austen, "Bottle Maker to Stop Using Plastic Linked to Health Concerns," *New York Times* online (18 April 2008).
30. Sheila Jasanoff, "NGOs and the Environment: From Knowledge to Action," *Third World Quarterly* 18 (1997): 579-94; Debora Spar, and Lane LaMure, "The Power of Activism: Assessing the Impact of NGOs on Global Business," *California Management Review* 45 (2003): 78-101.

MICHELLE MURPHY

FORUM
chemical regimes of living

IN CANADA, THE COUNTRY in which I work and live, the increased price of oil, itself linked to the war in Iraq, has recently made the extraction of oil from the "Tar Sands" of Alberta economically profitable. At this same historical conjuncture, Canada has elected its first prime minister from Alberta, a province characterized by an almost Texan neoliberalism and minimal environmental regulation. The extraction of oil from the Tar Sands is not only an energy and water intensive process, it is also profoundly polluting.

Meanwhile, the residents of the small town of Fort Chipewyan, many of whom are members of the Athabasca Chipewyan First Nation, recently have been disturbed by the rise of rare cancers in their community. Their local doctor responded with alarm and sent a report to Health Canada, a federal department, requesting an investigation. After some media attention, Health Canada's Alberta arm complied, concluding there was no unusual incidence of cancer, and then proceeded to file a formal complaint against the troublemaking doctor with the Alberta College of Physicians and Surgeons. The charge was met with political outrage and eventually dismissed. In the last few years, one can witness a proliferation of nongovernmental organizations (NGOs) now investigating the Tar Sands and rallying behind Fort Chipewyan.[1]

While Americans often have made a stereotype of their neighbor to the north as a land of socialist compassion, Canada is also the United States's largest supplier of oil and a participant in a larger transnational political economy of accumulation and dispossession. And not only the Canadian state, but the people

Michelle Murphy, "Chemical Regimes of Living," *Environmental History* 13 (October 2008): 695-703.

of Fort Chipewyan, who live downstream from the Tar Sands—whose chemical byproducts most likely caused cancer in their community, as well as all the molecular relations externalized and imperceptible that we do not yet know about, are caught in this larger political economy. Cars, militarization, water, laws, the direction of a river, the price of oil, the properties of sand, the rise of neoliberalism, histories of colonial dispossession—are all part of a complex of *molecular relations* that extend outward in place, and into the past, as well as forward to uncertain futures.

It has become a truism that synthetic chemicals have traveled to distant crevices and niches of the globe. Largely produced by over a century of petroleum-dependent industrialized capitalism, these varied molecular modifications range in duration, mobility, and effect, offering us a world changed in ways both subtle and overwhelming. The intensification of production and consumption in recent decades has yielded a chemically recomposed planetary atmosphere to alarming future effect, while it has penetrated the air, waters, and soils to accumulate into the very flesh of organisms, from plankton to humans. Not only are we experiencing new forms of chemical embodiment that molecularly tie us to local and transnational economies, but so too processed food, hormonally altered meat, and pesticide-dependent crops become the material sustenance of humanity's molecular recomposition. We are further altered by the pharmaceuticals imbibed at record-profit rates, which are then excreted half metabolized back into the sewer to flow back to local bodies of water, and then again redispersed to the populace en masse through the tap. In the twenty-first century, humans are chemically transformed beings.

Historians are able to offer this grand claim—that humans are chemically transformed—not only because the material world has indeed changed, but also because the last fifty years have seen the rise of technoscientific practices and modes of governmentality that together make the molecular realm newly legible and politicizable. The British sociologist Nikolas Rose, in his work elaborating on Michel Foucault's notion of "biopolitics," has argued that we are witnessing a new politics of life within contemporary biomedicine, a central feature of which is the *molecularization of life,* defined as the emergence of technoscientific practices—such as within genomics, biotechnology, and neurochemistry— that refocus health and life at a molecular register, thereby populating life with new molecular-scale entities, processes, and relationships.[2] The molecularization of life, moreover, has been accompanied by a new "bioeconomy" encompassing everything from from commodified organisms, to biotechnology, to biobanking, to pharmaceutical development.[3] For Rose, the molecularization of life in biomedicine also is characterized by a new "style of thought" modeled on genomics that emphasizes information, individualized risks and individualized variations.[4]

The geographer Bruce Braun helpfully builds on Rose's thinking to draw attention less to an epochal shift toward molecularization, and more to its layering onto other already existing forms of biopolitics that we inherited from the nineteenth and twentieth centuries—from sewers to eugenics.[5] Here we can also

include the history of infectious disease, as Nash argues in this forum (pp. 651-658). Importantly, for Braun, our understanding of molecularization also needs to widen to include the production of new ways of naming and managing *precarious lives* unable to achieve the individualized monitoring of health and hypervaluation of life that Rose discusses. Braun argues that molecularization offers—through such disciplines as virology and immunology—a vision of a world chaotically and dangerously interconnected by unpredictable viral exchanges. While Braun and Rose both theorize the molecularization of life through biomedicine and microorganic domains, I want to suggest that some of these insights might fruitfully extend to questions about the nonorganic molecular realm of pollution and toxicity.

That it is now possible to detect multiple, individualized, and low-level accretions of synthetic chemicals in organisms can be understood as a symptom of this molecularization of life. But when it comes to questions of pollution, perhaps it is more appropriate to discuss the historical emergence of a *chemical regime of living*, in which molecular relations extend outside of the organic realm and create interconnections with landscapes, production, and consumption, requiring us to tie the history of technoscience with political economy.[6] Through such practices as toxicology, gas spectrometry, and body burden testing, it is now possible to render legible (and contestable) the molecular relations that characterize the conditions of a factory floor, a body of water, food, or breast milk. Even without directly using these techniques, it is commonplace to postulate the existence of unwanted and unseeable molecular exposures in everyday life linked to both processes of production and habits of consumption. We are in a new chemical regime of living in which not just genomes but the atmosphere, water, soil, nourishment, commodities and our very bodies are apprehendable as caught in possibly toxic molecular relations.

Inquiring about the history of the molecular relations of life as understood through synthetic chemicals involves excavating a more fraught and complicated relation to capitalism than Rose's account provides.[7] First, our current chemical regime of living is not simply the result of new epistemological or technical innovations, but rather the accumulated result of some two hundred years of industrialized production, such as coal-based energy of the nineteenth century, or petroleum and plastic processing of the twentieth century. While recent decades have certainly seen new forms of production—such as those associated with electronic and digital devices—the most important recent shifts in this chemical regime of living have been the intensification of consumption combined with the geographical extension of industrialized consumption to more and more of the world's people, thereby accelerating the rates and variety of toxic pollutants released. Second, while synthetic molecular relations are clearly the result of activities which generate capital, they also tend to be "externalized" material effects of production and consumption practices—that is, effects that are purposively posited as existing outside the accountability of corporations, and in the context of neoliberal governments, outside the scope of regulation. Our chemical regime of living is characterized by the way it allows the fumes of

petrochemicals or the off-gassing of plastic commodities to be detectable but nonetheless irrelevant to corporate accountability. The costs—in lives and dollars—of externalized molecular relations are distributed into proximate, peripheral, or even distant landscapes. The anthropologist Sarah Lochlann Jain uses the term *commodity violence* to describe the kinds of injurious relations built into commodities for which producers are unaccountable.[8] Such commodity violence is typically statistical, rather than specific, in kind: that is, it is externalized when only predictable as a statistical probability in aggregate, and not in specific individual—such as in the way breast cancer caused by pollution can happen to anyone, but not necessarily anyone in particular.[9]

Of course, the criterion of harmful molecular relations is not always externalized to production. When acknowledged, however, it tends to be posited as the acceptable contractual risks of laborers, or as the legitimate cost-beneficial risks to consumers. Despite the ubiquity of risk calculi, it is fair to say, (and many scholars have documented, including Allen and Nash here) that much effort has gone into obscuring, rather than revealing, synthetic molecular relations, fostering a chemical regime of living in which it is commonplace and legally acceptable for such molecular relations to escape state regulation or the spotlight of research.

Since there is a regime of imperceptibility that has been purposively assembled around synthetic molecular relations, efforts to render visible such relations—by scientists, by bureaucrats, by community groups, or by NGOs—are political acts.[10] This chemical regime of living, then, is less about harnessing life to profit as in the bioeconomy, than it is about contestations over making legible the distributions of molecular harm and precarious life as effects of a complex political economy. For example, chemical harm concentrates in zones of dispossession, that is, zones in which life is rendered not just precarious to chemical effects, but also more disenfranchised and devalued in the larger political economy.[11] In the case of the Tar Sands and Fort Chipewyan, chemical exposures are built on histories of colonial dispossession. In other words, I want to attend to the history of a chemical regime of living in which the molecularization of life as an epistemologically contestable fact is interlinked with contestations over the physical production and distribution of chemical harm and dispossession.

The economic and epistemological aspects of the chemical regime I have so far sketched are joined by modes of governance that help to establish their condition of possibility. In the case of the Tar Sands, we might note that while chemical exposures are studied and contested more then ever before, Canada nonetheless overwhelmingly encourages the intensification of Tar Sands production in a neoliberal era in which the health of the economy tends to trump the health of ecosystems or human populations as a goal of national governance. Yet, at the same time, there has been a blossoming of nonstate grassroots efforts to render chemical exposures legible, regulatable, or preventable as an aspect of citizenship. The anthropologist Adriana Petryna has coined the term *biocitizenship* to name the ways people in the Ukraine took the condition of their

bodies relative to radiation as a point of entry into demanding entitlements from the state in the wake of the Chernobyl disaster.[12] Biocitizenship, then, is a useful term naming efforts that take life—from human bodies to ecosystems—as points of entry into making demands to the state, and thereby articulating the terms of citizenship via health and living-being. We can trace a century or more of biocitizenship projects responding to the effects of pollution on human well being. Since the 1970s in particular, scholars have tracked a history of such efforts, often by citizen-scientist alliances mobilizing mapping and survey techniques to render legible and politicize the effects of specific chemicals on communities. In the United States, the scholarship on environmental racism, on popular epidemiology in locations such as Love Canal, New York, Warren County, North Carolina, Woburn, Massachusetts, or in the chemical corridor of Louisiana, has charted the variety of places and projects that might fruitfully be grouped together as biocitizenship projects that took chemical exposure as an entry into renegotiating the terms of citizenship.[13]

Biocitizenship projects, by their very focus, tend to conjure a hopeful relation to the state—an optimism about the possibilities of pollution regulation, or about the state's commitment to health, product testing, safe food, and so on. Unfortunately, the story of the state's accountability to the new molecular relations of life has recently been largely a tale of deregulation and even subsidization, in which corporations, from tobacco to oil, have developed sophisticated tactics to obscure the chemical molecular relations of life, or to promote the kinds of risk calculi that legitimate the violent effects of production and consumption. All these features are at work in the case of the Tar Sands. Thus, increasingly multisited political economies of research and contestation have emerged.

Historically, the NGO has become the form that most engages and fosters biocitizenship projects. Yet, as the chemical violence of production is unevenly intensified in zones already shaped by other forms of dispossession (such as incinerators in minority neighborhoods, or workplace exposures for undocumented workers, or waste sent off-shore), biocitizenship projects that turn to the state are not available for noncitizens; for disenfranchised, illegal residents; for informal or illegal economic sectors; for communities across national borders; and so on. The question here is not whether biocitizenship projects, and NGOs more broadly, do a better job than the state does at monitoring chemical exposures (a low bar indeed), but instead in which ways these nonstate, nonprofit forms of governmentality are themselves constrained and productive in historically specific ways. Thus, nation-focused biocitizenship projects, as one dominant form of political grassroots tactic, have fostered new tactics of governing, researching, and contesting chemical exposure, while simultaneously reiterating a focus on nation that can sometimes obscure the transnational scale of political economy and leave unexamined contemporary forms of disenfranchisement.

As Daemmrich notes elsewhere in this forum (pp. 684-694), there has been a historical shift from what I am calling biocitizenship projects directed at the state to NGOs more directly confronting, or even collaborating with, the private sector,

calling attention to the plurality of kinds of NGOs, both over time and in place. Along with biocitizenship projects, then, we might also attend to what Partha Chatterjee has called the "politics of the governed," a term he uses to describe the political, technical, and social strategies of dispossessed peoples, without access to formal mechanisms of citizenship, who seek to organize themselves into legible, governable, ethically charged communities amenable to the attentions of NGOs, development programs, or even the state.[14] An example might be the spread of bucket sampling—a technique that uses plastic buckets to capture the evidence of transient, night-time, or other hard to prove pollution episodes developed by environmental justice activists in the Louisiana petroleum corridor—to communities in South Africa, India, Mexico, Canada, and other sites joined, not by a common nation-state, but rather by common conditions of dispossession created by mutual proximity to petrochemical processing.[15] Ironically, bucket sampling turns the very products of the chemical industry—cheap plastic commodities—against them. Bucket sampling has traveled across national lines, facilitated by collaborations between the San Francisco-based NGO Global Community Monitoring and local "industrial communities" in diverse "chemical corridors" tied together, not by shared identity, but by shared proximity to multinational corporations Tactically, the evidence collected from bucket sampling and the sharing of tactics across sites, has not led to national regulation, but instead to successful negotiations of local out-of-court settlements. Bucket sampling even links back to the Tar Sands, the oil of which is subsequently refined in Sarnia, Ontario. In both Sarnia and Alberta, it has been First Nation communities, already politicized in relation to colonial dispossession, that have initiated the resistance around which NGOs, including Global Community Monitoring, have rallied.

The particular technical practice of biomonitoring that allows us to list the diversity of synthetic chemicals accruing in ordinary humans has emerged, I would argue, within this larger chemical regime of life composed by molecularization, economic externalization, neoliberalism, diverse NGOs, and the tactics of ethically charged communities. Within this regime, biomonitoring can work on many levels. On the one hand, biomonitoring promises an individualized enumeration of chemical injury and risk resonant with visions of individualized genetics, holding open a promise of boutique medicine for the bourgeois risk-calculating subject exposed to chemical injury through consumption and the insidious spread of molecular relations even into the domains of relative privilege. On the other hand, biomonitoring can render legible the sheer abundance of synthetic molecular relations that make up human life and that exceed geographically bounded, racialized, or classed zones of dispossession.

Unfortunately, awareness of multiplicity is not inherently contestatory (as my own work on the history of Sick Building Syndrome has shown) because chemical, pesticide, and tobacco industries already have strategically encouraged awareness of the multiplicity of exposures, which subverts the ability to isolate the harmful effects of any specific exposure in an era when chemical exposures

are only regulatable and litigable as specific entities.[16] All of this is further evidence of the claim that making synthetic molecular relations visible is a complexly political act.

Often when historians of science and the environment have studied toxic exposures, we tended to follow the chain from production to consumption in our attempts to show the violence wrought by industrial processes. The emergence of such techniques as biomonitoring and bucket sampling underline how, in this chemical regime of living, effects and injuries are not chained in this way. Molecular relations, as the Tar Sands show, not only move spatially across the earth on currents and winds, and not only spread transnationally through the proliferation and redistribution of industrial processes in global capital, but also are part of transnational conjunctures of militarism, activism, research, citizenship, and dispossession that exceed the chain metaphor.

In the Alberta Tar Sands, while the government has out-sourced monitoring of environmental exposures to the very industry that creates those exposures, environmental NGOs are working to create counterdocumentation of the process.[17] Environmental Defence, for example, one of many Canadian NGOs concerned with the Tar Sands, has compiled a report of this "giant slow-motion oil spill" that extends the effects of the Tar Sands not only to Fort Chipewyan, but through pipelines to refineries in Louisiana and Sarnia, Ontario, Canada's chemical corridor. Sarnia is home of the Aamjiwnaang First Nation, members of which have in turn used bucket sampling to document their exposure to endocrine-disrupting chemicals that may be linked to a decade-long halving of the number of boys born.[18] Ironically, Environmental Defence also performed a national biomonitoring study called "Toxic Nation," including not only the family of a Aamjiwnaang environmental activist, but also a handful of high-ranking politicians, who proved to have overall higher concentrations of tested chemicals than the citizen volunteers.[19] Add to this conjuncture of NGOs, communities, and biomonitoring the activities of British Petroleum, one of the companies exploiting the Tar Sands, which is in turn protested both in its home country of Britain, but also in Whiting, Indiana, where it wants to expand refinery processing of tar sand oil. Air pollution from the Tar Sands is transforming into acid rain and blowing into neighboring Saskatchewan, while CO_2 emission, for extraction alone, makes the Tar Sands the largest source in Canada, contributing to global warming. We historians too are now part of this conjuncture, tracking molecular pathways that overflow the norms of social status and national borders, or even the time span of a human life. We too are struggling to find conceptual tools through which to capture this complex and uncertain set of phenomena. Instead of a chain, or a focus on bodies, the notion of a chemical regime of living might better provoke questions more adequate to the history of this entangled and enfolded political economy of molecular relations.

Michelle Murphy *is a professor of women and gender studies at the University of Toronto. She is the author of* Sick Building Syndrome and the Problem of Uncertainty: Environmental Politics, Technoscience, and Women Workers *(Duke, 2006).*

NOTES

1. See, for example, To the Tar Sands(Sierra Youth Coalition Campaign); Oil Sands Truth; Oil Sands Watch (Pembina Institute); Tar Sands Watch (Polaris Institute); Indigenous Tar Sands Campaign
2. Nikolas Rose, "Molecular Biopolitics, Somatic Ethics and the Spirit of Biocapital," *Social Theory and Health* 5 (2007).
3. Sarah Franklin, *Dolly Mixtures* (Durham, NC: Duke University Press, 2006).
4. See also Carlos Novas and Nikolas Rose, "Genetic Risk and the Birth of the Somatic Individual," *Economy and Society* 29 (2000).
5. Bruce Braun, "Biopolitics and the Molecularization of Life," *Cultural Geographies* 14(2007).
6. Collier and Lakoff develop a notion of "regime of living" as a "tentative and situated configuration of normative, technical, and political elements" in which "how to live is at stake," emphasizing in their definition the questions of ethics and kinds of reasoning. In this essay, my use of the term diverges from this definition in its emphasis on an assemblage of political economy, governmentality, and epistemology. Stephen Collier and Andrew Lakoff, "On Regimes of Living," in *Global Assemblages*, ed. Aihwa Ong and Stephen Collier (Oxford: Blackwell, 2004).
7. "Bioeconomy" is a term developed within the biotechnology industry sector, not by academicians. See Kean Birch, "The Neoliberal Underpinnings of the Bioeconomy: The Ideological Discourses and Practices of Economic Competitiveness," *Genetics, Society, and Policy* 2 (2006).
8. Sarah Lochlann Jain, *Commodity Violence* (Durham: Duke University Press, forthcoming).
9. Sarah Lochlann Jain, "Living in Prognosis: Toward an Elegiac Politics," *Representations* 98 (2007).
10. On regimes of imperceptibility, see Michelle Murphy, *Sick Building Syndrome and the Problem of Uncertainty: Environmental Politics, Technoscience, and Women Workers* (Durham, NC: Duke University Press, 2006).
11. On the interrelationship between laborers considered disposable and distributions of legitimated violence, see Melissa Wright, *Disposable Women and Other Myths of Global Capitalism* (New York: Routledge, 2006).
12. Adriana Petryna, *Life Exposed: Biological Citizens after Chernobyl* (Princeton: Princeton University Press, 2002). The concept is also developed in Vinh-Kim Nguyen, "Antiretroviral Globalism, Biopolitics, and Therapeutic Citizenship," in *Global Assemblages*, ed. Aihwa Ong and Stephen Collier (Oxford: Blackwell, 2004); Nikolas Rose and Carlos Novas, "Biological Citizenship," in *Global Assemblages*, ed. Aihwa Ong and Stephen Collier (Oxford: Blackwell, 2004).
13. The literature here is large. See, for example, Barbara Allen, *Uneasy Alchemy: Citizens and Experts in Louisiana's Chemical Corridor Disputes* (Cambridge: MIT Press, 2003); Phil Brown and Edwin Mikkelsen, *No Safe Place: Toxic Waste Leukemia, and Community Action* (Berkeley and Los Angeles: University of California Press, 1997); Robert Bullard, "Anatomy of Environmental Racism and the Environmental Justice Movement," in *Confronting Environmental Racism: Voices from the Grassroots*, ed. Robert Bullard (Boston: South End Press, 1993), 15-39; Giovanna Di Chiro, "Living Is for Everyone: Border Crossings for Community Environment and Health," in *Landscapes of Exposure: Knowledge and Illness in Modern Environments*, ed. Gregg Mitman, Michelle Murphy, and Christopher Sellers, *Osiris* (Chicago: University of Chicago Press, 2004); and Eileen McGurty, *Transforming Environmentalism: Warren County, Pcbs and the Origins of Environmental Justice* (Rutgers University Press, 2007).

14. Partha Chatterjee, *The Politics of the Governed: Reflections on Popular Politics in Most of the World* (New York: Columbia University Press, 2006).
15. Importantly, activists have been able to negotiate acceptance of bucket air sample results by the U.S. EPA. See http://www.labucketbrigade.org/index.shtml.
16. Murphy, *Sick Building Syndrome*.
17. Christopher Hatch and Matt Price, "Canada's Toxic Tar Sands: The Most Destructive Project on Earth" (Toronto: Environmental Defence, 2008).
18. Constanze Mackenzie, Ada Lockridge, and Margaret Keith, "Declining Sex Ratio in a First Nation Community," *Environmental Health Perspectives* 113 (2005).
19. Environmental Defence, "Toxic Nation on Parliament Hill: A Report in Four Canadian Politicians" (Toronto: 2007).