NATURAL RESOURCES JOURNAL

Volume 50
Issue 2 *"As if Equity Mattered" in Natural Resources*

Spring 2010

Toxic Inequities: Chemical Exposures and Indigenous Communities in Canada and the United States

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ABSTRACT

Endocrine-disrupting chemicals have permeated global ecosystems, crossing international boundaries to contaminate people far from initial sources of production and consumption. Not only do toxic residues complicate political boundaries, they also confuse temporal distinctions, for their legacies persist long after they have been banned. Moreover, the risks of exposure to these chemicals are rarely equitably distributed in a human population. This article examines chemical exposures in four indigenous communities in the United States and Canada, arguing that quantitative risk assessment protocols have failed to protect these communities from harm.

I. INTRODUCTION

New technologies and methods for the detection of toxic chemicals, particularly endocrine disruptors, have drawn increasing attention toward the pervasive presence of industrial chemicals in our bodies.[1] Many of these chemicals can interfere with the hormonal signaling systems that control reproduction, and many are persistent, resisting the metabolic processes that bind and break down the body's own hormones.

Since World War II, the production and use of synthetic chemicals has increased by more than 30-fold. The modern chemical industry, now a $2 trillion/year global enterprise, is central to the world economy, generating millions of jobs and consuming vast quantities of energy and raw materials. Each year, over 70,000 different industrial chemicals are synthesized and sold, which means that billions of pounds a year of endocrine-disrupting chemicals make their way into watersheds and aquatic organisms.

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1. Jody A. Roberts & Nancy Langston, *Toxic Bodies/Toxic Environments: An Interdisciplinary Forum*, 13 ENVTL. HIST. 629, 629 (2008), *available at* http://www.historycooperative.org/ (search for "Toxic Bodies"; then follow the hyperlink to the article).

Because consumption of fish is an important vector of human exposure to endocrine-disrupting chemicals, exposure is particularly problematic in indigenous communities in the United States and Canada where levels of fish consumption are high, or where chemical industries have been sited close to indigenous reserves and food sources. This article examines chemical exposures in the Shoalwater Tribe in the United States, the Cree in Canada, and Great Lakes Ojibwe bands in both Canada and the United States. Quantitative risk assessment protocols have failed to protect these communities from reproductive health risks associated with endocrine-disrupting chemicals.

II. THE SHOALWATER TRIBE: UNITED STATES

During the 1990s, pregnant women on the Shoalwater Bay Reservation in Washington State began experiencing a mysterious run of miscarriages. In 1997 and 1998, of 13 pregnancies, only one healthy baby was born.[2]

> "You look around the village and you start asking yourself, 'Where are the children?'" said Lisa Shipman, who had a stillborn son in 1990 and whose sister-in-law, Vernita Norman, has miscarried three times and recently left the reservation, in fear, when she became pregnant. . . . "[W]here are all the others? Where is the next generation coming from?"[3]

The U.S. Centers for Disease Control did not find a definitive cause for the miscarriages; it could simply be random events or possibly the result of genetic flaws. Or they could stem from diet, poverty, alcohol, or drug abuse, all of which can contribute to miscarriages. Yet the federal study found no evidence that either alcohol or drug abuse were contributing to the problem.[4] When several tribal women left the reservation during their pregnancies, they had successful pregnancies, and when non-tribal women came onto the reservation, they experienced miscarriages. Gale Taylor, the tribal health director, is one of the nonnative women who had a miscarriage while living on the reservation. Taylor told Sam Howe Verhovek, a reporter from the *New York Times*, "When you have to tell people that maybe the water they drink is the

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2. Kim Barker, *CDC Takes Closer Look at Tribe's Pregnancy Woes—Health Officials Following Miscarriages*, SEATTLE TIMES, Feb. 23, 1999, http://community.seattletimes.nwsource.com/archive/?date=19990223&slug=2945775.

3. Sam Howe Verhovek, *Mysterious Force Attacks Small Western Tribe's Young in the Womb*, N.Y. TIMES, Mar. 26, 2000, http://www.nytimes.com/library/national/science/health/032600hth-tribe-miscarriages.html.

4. *Id.*

cause, or just walking on the beach is a cause, or that maybe there's no cause at all, that's frightening. . . . A lot of women feel, if you want to stay pregnant, get the hell out of the reservation. It's hard."[5]

Many people in the tribe fear that the problem is environmental. Farmers spray pesticides on cranberry bogs north of the reservation; foresters spray herbicides on surrounding forest. "State and federal environmental reports found high levels of three pesticides in the drainage areas of the cranberry bogs" even though the drinking water was not found to contain levels of pesticides that exceeded federal standards.[6] The tribe is immersed in chemical exposure, and all these chemicals have the potential to disrupt the actions of hormones that shape fetal development. The people of the Shoalwater Tribe are exposed to something, but no one is certain what. Yet because fetal development is so complex and because synthetic chemicals are so difficult to monitor, no one can determine exactly what is harming the developing children. Quantitative risk assessments cannot prove that any single exposure has caused reproductive harm, so no legal action has been taken to restrict activities off the reservation that might be increasing chemical exposure on the reservation.[7]

While the Shoalwater Tribe's experience is extreme, it is not unique. Similar issues with reproductive health and toxic exposures, as well as profound uncertainty, confront native communities across the United States and Canada.

III. THE CREE IN CANADA

In 1962, Rachel Carson argued in *Silent Spring* that widespread spraying of the pesticide DDT was harming reproduction in many species of wildlife, and she warned that people might be subject to the same reproductive harm. Carson was particularly worried about DDT use in the boreal forests of Canada, homeland of the Cree Tribe of Canada. The Cree had suffered high rates of premature births linked to high rates of gestational diabetes among pregnant mothers.[8] Few public health officials, however, recognized that pesticide spraying might also be implicated in premature births, for reasons Rachel Carson had recognized:

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5. *Id.*

6. *Id.*

7. *See* NANCY LANGSTON, TOXIC BODIES: HORMONE DISRUPTORS AND THE LEGACY OF DES (2010). *See also* Linda L. Layne, *In Search of Community: Tales of Pregnancy Loss in Three Toxically Assaulted U.S. Communities*, 29 WOMEN'S STUD. Q. 25 (2001).

8. Shaila Rodrigues, Elizabeth Robinson & Katherine Gray-Donald, *Prevalence of Gestational Diabetes Mellitus Among James Bay Cree Women in Northern Quebec*, 160 CMAJ 1293 (1999).

people are intimately connected to the rest of nature, and wildlife can serve as sentinels of human exposure to toxic chemicals sprayed in the boreal forests.

A. Rachel Carson's *Silent Spring*

In *Silent Spring*, Rachel Carson argued that eagles, peregrine falcons, and other predatory birds were experiencing increasing rates of reproductive failure. Carson singled out the persistent organic pollutant dichlorodiphenyltrichloroethane, commonly known as DDT, as the likely culprit in eagle eradication, and she noted that "the insecticidal poison affects a generation once removed from initial contact with it."[9]

In the chapter "Rivers of Death," Carson described the ecological devastation caused by the widespread DDT spraying of Canadian boreal forests after World War II. Large-scale spruce budworm infestations have always been part of boreal forest disturbance cycles, occurring in cycles of roughly 35 years. Before World War II, spruce budworm infestations posed little commercial threat, but soon after the war, insect outbreaks began to complicate efforts to develop pulp and paper production in the boreal region. When spruce budworm populations exploded in the late 1940s, Canadian foresters were armed with new technologies made possible by the war: DDT and planes released from military service that could spray the chemical over millions of acres. Aerial spraying of DDT did indeed suppress budworm populations temporarily, but inadvertently prolonged the budworm cycles by keeping some food source available for the insects, leading to ever more defoliation, and ever more spraying of DDT in an attempt to control the outbreaks. As the botanist George Woodwell noted:

> Spraying half a pound of DDT in oil per acre reduced the budworm population by 95 to 98 percent, but the remaining population would explode the next year and create the problem all over again. Year after year DDT was sprayed, and the population crashed and then exploded again. The net effect was to preserve the insects' food supply and thereby perpetuate the outbreak. After watching the cycle repeat itself, I could see that while spraying was protecting the trees, it was prolonging the outbreak and the need for spraying.[10]

This is illustrated in what happened in the decades that followed.

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9. RACHEL CARSON, SILENT SPRING 121 (1962).

10. George Woodwell, *Toxic Food Web*, *in* LIFE STORIES 74, 74–75 (Heather Newbold ed., 2000). For the original research, see G.M. Woodwell & F.T. Martin, *Persistence of DDT in Soils of Heavily Sprayed Forest Stands*, 145 SCIENCE 481 (1964).

For example, the infestation of 1910–20 defoliated 10 million hectares. The infestation of 1945–55, when DDT was first used heavily, defoliated more than twice the earlier infestation: 25 million hectares. And the infestation of 1968–85 defoliated even more: 55 million hectares. As a comparison, the combined area of New York, Pennsylvania, Maryland, West Virginia, Virginia, and North Carolina is about 57 million hectares.[11]

DDT spraying did not stop the budworm, but it did ignite concerns about the environmental effects of massive spray campaigns in the boreal forests. Carson described how the Miramichi River, once the most abundant Atlantic salmon run in the world, became "a picture of death and destruction."[12] She described the spraying:

> So in 1954, in the month of June, the planes visited the forests of the Northwest Miramichi and white clouds of settling mist marked the crisscross pattern of their flight. The spray—one half pound of DDT to the acre in a solution of oil—filtered down through the balsam forests and some of it finally reached the ground and the flowing streams. . . .
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> Soon after the spraying had ended there were unmistakable signs that all was not well. Within two days dead and dying fish, including many young salmon, were found along the banks of the stream. . . . All the life of the stream was stilled. Before the spraying there had been a rich assortment of the water life that forms the food of salmon and trout. . . . But now the stream insects were dead, killed by the DDT, and there was nothing for a young salmon to eat.[13]

Although *Silent Spring* was published in 1962, it was not until 1985 that the Canadian government completely banned the use of DDT in forestry. Even then, the use of existing stocks was permitted until 1990.[14]

B. Pesticides and the Cree

In "Rivers of Death," Carson focused on DDT's acute poisoning of fish and insects. But she also noted that DDT and other pesticides had

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11. Nicholas C. Bolgiano, *Cause and Effect: Changes in Boreal Bird Irruptions in Eastern North America Relative to the 1970s Spruce Budworm Infestation*, 58 AMERICAN BIRDS 26 (2005), *available at* http://birds.audubon.org/documents/ (search "Audubon" for "Bolgiano"; then follow hyperlink to the article).

12. CARSON, *supra* note 9, 131.

13. *Id.*

14. Environment Canada, Dichlorodiphenyltrichloroethane (DDT), http://www.ec.gc.ca/toxiques-toxics/default.asp?lang=en&n=13272755-1 (last visited Aug. 27, 2010).

the potential to alter sexual development and reproduction. We now know that DDT is an endocrine-disrupting chemical that can cause reproductive health problems in people as well as wildlife. Did women in Canada exposed to DDT spraying during the spruce budworm campaigns experience any reproductive harm? Answering this question definitively is impossible, but several lines of evidence support a possible association.

Like other aboriginal women in Canada, Cree women who reside in the boreal forests, where high levels of pesticides, including DDT, have been sprayed, experience high rates of premature births, as well as prenatal, stillbirth, and newborn deaths—2 to 2.5 times the national average.[15] Most efforts to understand environmental health issues among the Cree have focused on mercury contamination, which is indeed a major concern.[16] But persistent organic pollutants such as DDT were used widely in boreal forests, and growing evidence shows that boreal forest ecosystems may intercept and retain these compounds. Predatory fish that reside in boreal aquatic ecosystems then bioaccumulate the chemical, and, for people who eat those fish, human exposures may be significant.[17]

In 2001, Matthew Longnecker and his colleagues measured levels of dichlorodiphenyldichloroethylene, or DDE—a metabolite of DDT—in the stored blood sera of 2,380 mothers who gave birth between 1959 and 1966, when DDT was being heavily used in agriculture and forestry. Of these women, 361 delivered prematurely.[18] The higher the level of the endocrine disruptor DDE in the mother's blood, the greater the odds of preterm birth.[19] These associations are not proof that boreal forest spray

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15. Beverley Chalmers & Shi Wu Wen, *Perinatal Care in Canada*, 4 BMC WOMEN'S HEALTH S28, S28 (2004), http://www.biomedcentral.com/content/pdf/1472-6874-4-S1-S28.pdf; PUB. HEALTH AGENCY OF CANADA, CANADIAN PERINATAL HEALTH REPORT xxii (2000), http://www.phac-aspc.gc.ca/publicat/cphr-rspc00/pdf/cphr00e.pdf.

16. JILL TORRIE, ELLEN BOBET, NATALIE KISHCHUK & ANDREW WEBSTER, The Evolution of Health Status and Health Determinants in the Cree Region (Eeyou Istchee): Eastmain-1-A Powerhouse and Rupert Diversion Sectoral Report: Volume 2: Detailed Analysis 18–20 (2005), http://www.gcc.ca/pdf/QUE000000012.pdf.

17. *See id.* at 25–27. *See also* Derek C. G. Muir & Neil L. Rose, *Lake Sediments as Records of Arctic and Antarctic Pollution*, *in* LONG-TERM ENVIRONMENTAL CHANGE IN ARCTIC AND ANTARCTIC LAKES 209, 209–39 (Reinhard Pienitz et al., eds., 2004); Derek C.G. Muir et al., *Bioaccumulation of Toxaphene Congeners in the Lake Superior Food Web*, 30 J. OF GREAT LAKES RES. 316, 316–40 (2004); Dorothea F.K. Rawn et al., *Historical Contamination of Yukon Lake Sediments by PCBs and Organochlorine Pesticides: Influence of Local Sources and Watershed Characteristics*, 280 THE SCI. OF THE TOTAL ENV'T 17, 17–37 (2001).

18. Matthew P. Longnecker et al., *Association Between Maternal Serum Concentration of the DDT Metabolite DDE and Preterm and Small-for-Gestational-Age Babies at Birth*, 358 LANCET 110, 110 (2001).

19. *Id.* at 113.

campaigns caused the elevated rates of reproductive problems experienced by Cree women. They do suggest, however, a plausible link between exposure to persistent organic pollutants and fetal harm.

IV. THE GREAT LAKES OJIBWE: AAMJIWNAANG RESERVE IN CANADA

Sarnia, a town that lies 100 kilometers, approximately 62 miles, north of Detroit on Lake Huron, is at the epicenter of Canadian petrochemical development. During World War II, the Canadian government constructed a synthetic rubber plant in Sarnia when allied sources of Asian rubber were cut off by military actions. "After the war, industries flocked to Sarnia to take advantage of the new 'petrochemical revolution.' With access to raw materials, a network of pipelines, a prime location, and exploding consumer demand, Sarnia became an economic powerhouse. . . . [Just] south of Sarnia's 'Chemical Valley' is the Aamjiwnaang First Nations reserve."[20] As the filmmaker Pamela Calbert reports on the community:

> 40% of Canada's petrochemical industry is located in the immediate vicinity of Sarnia, ON. There are currently forty[-]two chemical companies listed on the National Pollution Release Inventory within 10 km (6.2 miles) of Aamjiwnaang First Nation. . . . Beginning in 1993-94, girl babies at the Aamjiwnaang First Nation (AFN) began to outnumber boys, at a cumulative rate of 2:1 over the following decade. . . . The normal human birth ratio is for 105 boys to be born for every 100 girls . . . 39% of the women at Aamjiwnaang over the age of 18 have had at least one miscarriage or stillbirth. This compares to 25% in the general population.[21]

In 2006 families across Canada were tested for the presence of 68 toxic chemicals in their blood and urine.[22] A resident of Aamjiwnaang

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20. Pamela Calvert, Discussion Guide, *The Beloved Community*, http://newsreel.org/guides/belovedcommunity/bcdiscuss.htm (last visited Nov. 16, 2009).

21. *Id.* For a map showing some of these chemical plants in relation to the Aamjiwnaang Reserve and Sarnia, Ontario, see http://www.cec.org/images/trio/20/aam_en.jpg (last visited July 4, 2010). *See generally* Constanze A. Mackenzie, Ada Lockridge & Margaret Keith, *Declining Sex Ratio in a First Nation Community*, 113 ENVTL. HEALTH PERSP. 1295 (2005), http://www.ehponline.org/docs/2005/8479/abstract.html (describing a community research project); Paolo Mocarelli et al., *Paternal Concentrations of Dioxin and Sex Ratio of Offspring*, 355 LANCET 1858, 1858–63 (2000) (describing a research project); Jack Poirier, *Band to Monitor Industry*, THE SARNIA OBSERVER, May 10, 2007, http://www.gcmonitor.org/article.php?id=582; Toxic Nation, http://www.toxicnation.ca/ (last visited Nov. 16, 2009).

22. Calvert, *supra* note 20.

reserve "had the highest body burden of toxics, including the presence of 30 carcinogens and 31 reproductive/developmental toxins," of any person tested in Canada. Another local resident, age 14, carried "12 hormone disruptors and 17 reproductive/developmental toxins in her body."[23]

The women of Sarnia fear that "because of their own exposure to a cluster of hormone-mimicking chemicals . . . the next generation may be at risk."[24] But while the associations between chemical exposure and reproductive harm concern the tribe, the associations do not offer enough proof of a link between exposure and harm for the chemicals to be banned under Canadian law. Risk assessment protocols demand a standard of proof that epidemiological studies can rarely provide, yet this is small comfort to families experiencing miscarriages and premature births.

V. THE GREAT LAKES OJIBWE: TOXAPHENE EXPOSURE

In the late 1990s, Canadians noticed that levels of a persistent organic pollutant (POPS) named toxaphene were rising in the fish of Lake Superior, even though the chemical had not been used anywhere near Lake Superior for decades. Large predatory fish such as lake trout can accumulate many toxic chemicals, making fish consumption a significant source of human exposure. Even after these chemicals are banned, levels may continue to rise in lakes, fish, and people.[25]

High toxaphene levels were startling because, along with other POPS, toxaphene had been banned in the 1980s. But levels of toxaphene were for some reason extraordinarily high. When researchers looked, it was clear that people who ate fish from the Great Lakes were accumulating significant levels of banned toxic chemicals. Additional studies found that during pregnancy and breastfeeding, women passed those chemicals to their developing fetuses at their most vulnerable stages of development.[26] The legacies of the past were becoming the body burdens of future generations.

Lake Superior, on the border between Canada and the United States, is cold, vast, and distant from most industrial development. Of all the Great Lakes, it is easily the cleanest, and in many ways it seems almost pristine. So why would toxaphene be highest in this particular lake, in a region where the chemical had never been produced and had hardly

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23. *Id.*
24. *Id.*
25. *See* MELVIN J. VISSER, COLD, CLEAR, AND DEADLY: UNRAVELING A TOXIC LEGACY (2007).
26. *Id.*

even been used? What might that contamination mean for one of the great recovery stories of modern conservation: the restoration of Lake Superior fisheries? Even more pressing, what might that contamination mean for Ojibwe bands attempting to restore culturally significant foods, such as lake fish, to their diets?

The name toxaphene refers to a group of turpentine-smelling chemicals made from pine oil and chlorine—two natural chemicals combined into a synthetic substance. First introduced in 1945 by Hercules Company of Wilmington, Delaware, toxaphene was immediately found to be toxic to fish, birds, and mammals. In the late 1940s, research was published showing its toxicity, and over the next two decades, studies showed it to be mutagenic and carcinogenic in mammals.[27]

Even though much of the research on the toxicity of toxaphene was published well before the EPA cancelled federally registered uses of DDT in the early 1970s, soon after the DDT was removed from widespread use, toxaphene manufacturers began promoting toxaphene as a safe alternative to DDT—safe because it was made from nature's own building blocks.[28] It was quickly mass produced and widely used as an insecticide, particularly in the cotton-growing industry in the American South.[29]

The writer Vickie Monks notes that "[t]oxaphene became one of the most heavily used pesticides ever—as much as 46 million pounds a year during the height of its use in the 1970s, according to the U.S. Agency for Toxic Substances and Disease Registry."[30] Production peaked in 1975, at 59.4 million pounds.[31] "In the United States, most of that was sprayed on cotton and soybeans in the South, but it was registered for use in more than 800 products—for everything from tick control on livestock to the killing of unwanted fish species in lakes and ponds."[32]

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27. Henk-Jan de Geus et al., *Environmental Occurrence, Analysis, and Toxicology of Toxaphene Compounds*, 107 Supp. 1 ENVTL. HEALTH PERSP. 115, 115–44 (1999).

28. *See* U.S. EPA, DDT REGULATORY HISTORY: A BRIEF SURVEY (1975), http://www.epa.gov/history/topics/ddt/02.htm (excerpted from report EPA-540/1-75-022).

29. *Id. See generally* Scott Fields, *Great Lakes: Resource at Risk*, 113 ENVTL. HEALTH PERSP. A164–73 (2005).

30. Vickie Monks, *How Did the Poison Get into the Trout?*, NATIONAL WILDLIFE para. 10 (1998), http://www.nwf.org/nationalwildlife/article.cfm?issueID=19&articleID=157. Approximately one billion pounds of year of pesticides were used annually on row crops in the 1970s. *See generally* David Pimentel et al., *Benefits and Costs of Pesticide Use in U.S. Food Production*, 28 BIOSCIENCE 772 (1978).

31. *See* Agency for Toxic Substances & Disease Registry, Dep't of Health & Human Serv., *Production, Import/Export, Use, and Disposal in* TOXICOLOGICAL PROFILE FOR TOXAPHENE, 99, 99 (NTIS Accession No. PB97-121057, 1996), http://www.atsdr.cdc.gov/toxprofiles/tp94-c4.pdf [hereinafter ATSDR].

32. Monks, *supra* note 30, at para. 10.

After further research showed it to be carcinogenic, mutagenic, and teratogenic, its use was banned in the United States and Canada in the 1980s.[33] But instead of destroying existing stocks of the chemical, manufacturers shipped them overseas and soon began marketing the chemical worldwide. Throughout Russia, China, and Africa, the chemical continued to be used widely.[34]

In the 1950s, lake trout in the Great Lakes had been driven to the verge of extinction. With intensive fishing harvests, invasion by sea lampreys and alewives, and accumulation of persistent organic pollutants that affected reproductive health, the cumulative effects had led to a collapse of lake trout populations, with economic, cultural, and health consequences for native peoples and for non-natives alike. Commercial fishing restrictions and the banning of many persistent organic pollutants led to a substantial recovery for these trout, which became one of the great recovery stories of conservation.[35]

Breeding populations appeared to have recovered in Lake Superior by the late 1980s. So the finding, a decade later, that lake trout were newly contaminated with toxaphene unsettled fisheries biologists deeply. Where was that toxaphene coming from? Researchers initially suspected the culprit was pulp mills lining the Canadian shores of Lake Superior near Thunder Bay, where deforestation of regional boreal forests had begun in the 1980s. The harvests supplied a growing paper industry, which dumped pulp mill wastes directly into Lake Superior. Those wastes contained chlorine and pine oils, which could combine under certain natural conditions to form toxaphene.[36] But even in inland lakes such as Lake Siskiwit on Isle Royale—unaffected by toxins directly carried over from Thunder Bay—contamination was high.[37]

Evidence suggests that the chemical continues to be volatized from old cotton fields in the American South, and that global wind currents may also be transporting toxaphene still used in Africa into Lake Superior and other boreal lakes—where it finds its way into fish, and eventually into the people eating that fish. Once it falls into Lake Supe-

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33. VISSER, *supra* note 25.

34. *Id*. The Agency for Toxic Substances and Disease Registry notes that "[w]hile it is impossible to quantify production figures or usage rates, India and many countries in Latin America, Eastern Europe, the former Soviet Union, and Africa still use various toxaphene products as pesticides." ATSDR, *supra* note 31 (citation omitted).

35. Michael J. Hansen, *Lake Trout in the Great Lakes; Basinwide Stock Collapse and Binational Restoration*, *in* GREAT LAKES FISHERIES POLICY AND MANAGEMENT: A BINATIONAL PERSPECTIVE 417, 417–53 (C. Paola Ferreri & William W. Taylor eds., 1999).

36. Monks, *supra* note 30.

37. Derek C.G. Muir et al., *Bioaccumulation of Toxaphene Congeners in the Lake Superior Food Web*, 30 J. GREAT LAKES RES. 316, 316–40 (2004).

rior, it tends to concentrate, as the lake never warms enough to allow much toxaphene to go airborne again. Canadian researcher Terry Bidleman of the Atmospheric Environment Service has documented toxaphene as far north as the polar ice cap, stored in the fat of marine mammals, fish, and northern peoples.[38]

The Canadian government had advised citizens to avoid eating most Lake Superior trout because of toxaphene contamination. Across the border in Wisconsin and Minnesota, levels are equally high, but no advisories warn against consumption of fish contaminated with toxaphene.[39] Nevertheless, because the present concentrations in fish are high enough to be causing reproductive problems for the fish themselves, researchers are concerned that human health may also be at risk.

Among those most concerned are indigenous communities who live along the shores of Lake Superior. Fish is particularly important for the health of fetuses and young children,[40] and eating fish is of great cultural significance. But its potential contamination forces communities to make trade-offs between their beliefs and possible harm to themselves and their children.

Many tribal health departments are urging members to eat much more traditional or country food, i.e., wild food, in order to reduce the risks of diabetes, heart disease, and obesity from non-traditional sources such as high-fructose corn syrup, white flour, and trans fats that make up the average American diet. But much traditional food, such as lake trout, is at the top of long food chains, and therefore quite high in contaminants that have bioaccumulated in the fat of prey species.[41]

As the writer Paul Rauber notes, "Like dead fish rising to the surface, poisons dumped down drains or pumped into the wind return again—often on the plates of the poor."[42] Rauber reports on a visit to the Red Cliff Band of Lake Superior, where he talked with biologist and tribal member Judy Pratt-Shelley.

> "We don't need people to tell us, 'Don't eat the fish,'" says Pratt-Shelley. "This is a guaranteed, federally reaffirmed right

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38. Monks, *supra* note 30.

39. MARK ELSTER, U.S. EPA, EPA-160-R-97-005, UNITED STATES GREAT LAKES PROGRAM REPORT ON THE GREAT LAKES WATER QUALITY AGREEMENT (1997), *available at* http://www.epa.gov/grtlakes/glwqa/usreport.pdf.

40. Dariush Mozaffarian & Eric B. Rimm, *Fish Intake, Contaminants, and Human Health: Evaluating the Risks and Benefits*, 296 JAMA 1885, 1885–89 (2006).

41. *See* Susan T. Glassmeyer et al., *Toxaphene in Great Lakes Fish: A Temporal, Spatial, and Trophic Study*, 31 Envtl. Sci. & Tech. 84, 84–88 (1997).

42. Paul Rauber, *Fishing for Life: Thousands of Americans Face the Choice of Eating Contaminated Fish or Not Eating at All*, SIERRA MAGAZINE, Nov.–Dec. 2001, at para. 7, *available at* http://www.sierraclub.org/sierra/200111/fishing.asp.

> that we retained in our treaties when we ceded this territory: the right to hunt, fish, and gather. . . . What we need is for the laws to change so that those [chemicals] aren't allowed in the ecosystem to begin with. That's the only way they're not going to end up in our breast milk." Giving up local fish would mean giving up her culture, and while the effects of eating tainted fish are chronic, subtle, and often hard to separate from the manifold ailments of poverty, the effects of losing one's culture are there for anyone to see: alcoholism, broken families, drifting children.
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> . . .
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> In the end, the only problem fish advisories solve is that of informed consent. People have a right to know the risks involved in what they eat, but they have an even more fundamental right not to face those risks in the first place.[43]

VI. QUANTITATIVE RISK ASSESSMENT

In all four cases discussed above, environmental regulations have failed or neglected to protect wildlife or human health from risks posed by exposure to endocrine-disrupting chemicals. The reason for this regulatory failure lies in the risk assessment techniques that were intended to protect public health. Since the 1980s, a technique called "quantitative risk assessment" has dominated the oversight of most occupational and environmental chemicals in both the United States and Canada. Quantitative risk assessment assumes that risk is an unavoidable fact of modern life, something to be managed rather than eliminated. Risk assessors estimate the size of a given risk from a given chemical, and risk managers decide whether that risk is acceptable. This process relies on estimates of how an average person can be exposed to particular toxic chemicals without suffering significant harm. Harm is typically defined as getting cancer, although other endpoints such as reproductive failure are possible to assess. Risk assessors then manage pollution by permitting chemicals to be used and released, just so discharges do not exceed a standard of contamination deemed to be an acceptable trade-off for economic gains.[44]

Risk assessment assumes human bodies can accommodate some degree of chemical exposure, so long as the exposure is below a thresh-

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43. *Id.* at para. 3, 4, 15.

44. LANGSTON, *supra* note 7; RISK ASSESSMENT OF CHEMICALS: AN INTRODUCTION (C.J. van Leeuwen & T.G. Vermeire eds., 2d ed. 2007).

old of toxicity.[45] Although this "dose makes the poison" concept may be true for certain chemical exposures, it is rarely true for endocrine disruptors. At low concentrations, hormones normally stimulate receptors, but at high concentrations, hormones can saturate receptors, thus inhibiting their pathways. This means that low doses of endocrine disruptors might produce adverse impacts, even though higher doses might not. The idea that a substance can have more powerful effects at low doses than high doses fundamentally challenges traditional toxicological approaches, and with them quantitative risk assessment models.[46]

Endocrine disruptors have their most profound effects on sexual development and reproductive health, but because these are complex and ignored by quantitative risk assessments, the chemicals have largely escaped regulation. Quantitative risk assessments have focused on the potential of chemicals to cause cancer by directly damaging DNA, leading to genetic mutations. Endocrine disruptors, however, often do not alter genes, but instead change the way they are expressed, which is an outcome not captured by risk assessments. Risk assessment typically measures effects on adults, but for endocrine disruptors, fetal effects are most problematic. A tiny exposure to the fetus may have effects that are not obvious at birth, but may lead to reproductive failure decades later.[47]

If drinking a poison kills you five minutes later, that is an acute effect, and it is fairly easy to understand because the compound quickly produces a dramatic change in the state of the organism. But chronic, low-level effects are much more difficult to identity or measure, much less regulate. As the biologist Joe Thornton writes: "[T]here is no single 'normal' state for any of these functions, all of which vary naturally within some range in the population. This natural variability greatly increases the noisiness of the results and reduces a study's power to establish a statistically significant association of exposure with effect."[48]

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45. JOE THORNTON, PANDORA'S POISON: CHLORINE, HEALTH, AND A NEW ENVIRONMENTAL STRATEGY 7 (2000).

46. Frederick S. vom Saal & Daniel M. Sheehan, *Challenging Risk Assessment: Traditional Toxicological Testing Cannot Detect the Adverse Effects of Very Low Doses of Environmental Chemicals*, 13 F. FOR APPLIED RES. & PUB. POL'Y, 11, 11–18 (1998); Frederick S. vom Saal & Claude Hughes, *An Extensive New Literature Concerning Low-Dose Effects of Bisphenol A Shows the Need for a New Risk Assessment*, 113 ENVTL. HEALTH PERSP. 926, 926–33 (2005); Frederick S. vom Saal et al., Letter to the Editor, *Implications for Human Health of the Extensive Bisphenol A Literature Showing Adverse Effects at Low Doses: A Response to Attempts to Mislead the Public*, 212 TOXICOLOGY 244, 244–52 (2005).

47. Retha R. Newbold et al., *Developmental Exposure to Diethylstilbestrol Alters Uterine Gene Expression That May Be Associated with Uterine Neoplasia Later in Life*, 46 MOLECULAR CARCINOGENESIS 783, 783–96 (2007).

48. THORNTON, *supra* note 45, at 111.

VII. CONCLUSION

Risk assessment has meant that society hands over "regulatory decision making to a technological elite, whose members alone can understand the increasingly arcane basis and data analysis of quantitative risk assessment."[49] The quantifications appear precise, because they are so complex and difficult to understand—yet "few groups (industry, regulators, scientists, or the public) have faith in their precision."[50] Communities suspicious of risk assessments have usually been dismissed as ignorant and largely excluded from the decision-making process.[51]

Failures of risk assessment are expressed in the body of a child diagnosed with learning disabilities, a child whose mother ate Great Lakes fish contaminated with toxic chemicals throughout her pregnancy, a child whose father worked in the chemical plants of Sarnia. Quantitative risk assessment assumes that a certain amount of risk is acceptable. Yet with chemical releases, the risks are rarely distributed equally or fairly among the population.[52] Indigenous communities with less economic and political power have often borne a substantial share of the risks from toxic releases as we have seen illustrated in the case studies of chemical exposure in the Shoalwater Tribe, the Cree, and the Great Lakes Ojibwe bands in Canada and the United States.

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49. Ellen K. Silbergeld, *Risk Assessment: The Perspective and Experience of U.S. Environmentalists*, 101 ENVTL. HEALTH PERSP. 100, 103 (1993).

50. *Id.* at 102.

51. *See id.* at 100–04.

52. Raul P. Legano & Climis A. Davos, *Fair Share: Siting Noxious Facilities as a Risk Distribution Game under Nontransferable Utility*, 43 J. ENVTL. ECON. & MGMT 251, 251–66 (2002); Beverly Wright, *Living and Dying in Louisiana's "Cancer Alley*," *in* THE QUEST FOR ENVIRONMENTAL JUSTICE: HUMAN RIGHTS AND THE POLITICS OF POLLUTION 87, 87–107 (Robert Bullard ed., 2005); Robert Bullard, UNEQUAL PROTECTION: ENVIRONMENTAL JUSTICE AND COMMUNITIES OF COLOR (1997).