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The Reality of Children's Risk in Canada

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Contents

<i>Executive Summary</i>	3
<i>The Debate over Children's Risk</i>	5
<i>The Reality of Children's Risk in Canada</i>	10
<i>Is More Regulation the Answer?</i>	12
<i>Conclusions & Recommendations</i>	16
<i>Appendix—Current Approaches to Protecting Children</i>	18
<i>References</i>	20
<i>About the Author & Acknowledgments</i>	24

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Executive summary

According to Environment Canada, 89% of Canadians believe that their children's health is being affected by environmental threats. For example, many people are concerned about children's pesticide exposures. The Canadian Environmental Law Association and the Ontario College of Family Physicians have stated: "The cumulative effects of being exposed to many different pesticides over a lifetime represent an unquantified and unacceptable risk to all Canadian children." Activist organizations such as Greenpeace and the Natural Resources Defense Council continue to call for additional regulatory stringency, based on the claim that children are especially sensitive to environmental chemical exposures. While people agree that protecting children's health and achieving a clean and safe environment are important, many disagree about how best to attain those goals. The debate begins with the very definition of the "environment," and proceeds through the question of what mix of regulatory, research, and educational efforts is the best for protecting children's health. Activist groups are adamant that children are at special risk from exposure to chemicals both indoors and out, and conduct continual campaigns calling for additional regulating—and in some cases, banning—of chemicals despite a lack of evidence demonstrating any harm. But the Canadian Institute of Child Health acknowledges that the impact of life-long exposure to environmental contaminants on life expectancy or on disease is yet to be determined.

Conclusions of this study

After examining the challenges to understanding and assessing children's environmental risk, this study concludes that:

- Canada's children are healthier than ever before, and while risks remain, most are not environmental.
- Great progress has been made in protecting and promoting the health of children in Canada. Infant

mortality rates have decreased by 81% from 1960 to 2000. Life expectancy has increased from 61 years for women and 59 years for men born in 1920 to 82 years for women and 77 years for men born in 2000. Looking at the environment in terms of chemical contaminants, the news is also good.

- Air quality continues to improve, with a 49% decrease in average levels of the six primary air pollutants in Canada since 1980. In 1992, ambient lead concentrations in Canada had been reduced by 97% compared to 1974; Canadian children's average blood lead levels were reduced by 70% between 1984 and 1992. The levels of benzene in air and emissions of mercury to air both decreased by 35% from 1995 to 2000.
- The levels of contaminants in breast milk also show downward trends, with the levels of PCBs decreasing 73% between 1982 and 1992 and the levels of DDT decreasing by 94% between 1967 and 1992. Studies of children's metabolizing of pharmaceuticals, and experiments using juvenile experimental animals suggest that children's susceptibility to environmental chemicals is not particularly more pronounced than that of adults, varying from equally susceptible, to greater susceptibility, to lesser susceptible depending on the chemical involved.
- Canadian children are well protected by existing programs—education and research are needed more than additional regulation. Numerous programs in Canada focus on the protection of children's health. Canada has at least seven programs intended to protect children at the federal level alone. A review of 17 studies that performed quantitative analyses of the extent of variability in sensitivity due to age or other factors in both humans and animals found that a high percentage of the population, including children, is protected by the margin of safety now used

by North American regulatory agencies to account for sensitivity differences. Studies using larger populations that include sensitive individuals suggest that the value is close to 100%. Thus it appears that the current approach used by regulatory agencies to limit chemical exposures protects most of the people—including children—most of the time

The reality of risk and the precautionary principle

Despite such programs and research findings, there is an increasing tendency to base decisions about children's environmental health protection on the precautionary principle, which stipulates that action should be taken to reduce a suspected risk even though there may not be scientific evidence supporting the extent or even existence of the proposed risk. In other words, if the available science is inadequate to guide a decision about how best to treat a potential risk to children, that inadequacy is not a basis for inaction. Thus, chemicals can be banned simply because, in high-dosage animal testing, they are found to pose a risk to animal health. But beyond a few examples, there are virtually no well-substantiated correlations between exposure to chemicals at the levels normally found in the environment and public health problems, in children or in adults.

The measures by which we usually characterize the environment—water quality, air quality, reportable emissions—show that substantial improvements have been made. What we lack is an ability to measure those accomplishments in terms of an impact on public health, if there is one. We do not have the disease surveillance data or the environmental exposure data needed to determine how, and to what extent, the two may be re-

lated. In such situations of limited knowledge, regulatory efforts to manage risk have not shown a good history of prioritization or return on investment. In the United States, where the benefits of regulating risk have been studied closely, the attempts to eliminate ever smaller, less-defined risks has resulted in a growing irrationality in risk-reduction investment choices. Public-policy priorities often bear little relationship to any rational ranking of risks and potential risk-reduction investments of tax dollars. As risk analyst Aaron Wildavsky has demonstrated, in conditions of high uncertainty about risk, an agenda of research and education is superior to one of regulation based on precaution.

Poorly established risks from environmental exposures to chemicals often receive more attention from policy-makers than do the well-established leading causes of childhood morbidity and mortality. Prenatal factors are the leading cause of death among children. Maternal smoking and alcohol consumption during pregnancy impair infant development and are regrettably common. Unintentional injuries are the second leading cause of death among children and, while cancer is the third leading cause of death, most evidence points at non-environmental factors for cancer deaths among children. Children spend more than 80% of their time indoors but little responsibility has been taken to ensure a healthy indoor environment. Limiting children's chemical exposures remains an important goal. But in the absence of strong evidence implicating childhood exposure to individual chemicals at low, environmental levels as causes of childhood or adult disease, actions taken to protect children from the many well-characterized and established threats to their health are likely to have greater and more demonstrable impacts.

The debate over children's risk

According to Environment Canada, 89% of Canadians believe that their children's health is being affected by environmental threats (Environment Canada, 2002b). For example, many people are concerned about children's pesticide exposures. The Canadian Environmental Law Association and the Ontario College of Family Physicians have stated:

The cumulative effects of being exposed to many different pesticides over a lifetime represent an unquantified and unacceptable risk to all Canadian children. (Children's Health Project, 2000)

Advocacy groups active in both the United States and Canada lead the public to presuppose that children face special environmental and chemical risks. In the Sierra Club's so-called "fact sheet" against pesticides, for example, children are portrayed as especially sensitive to environmental exposures. The Sierra Club claims,

Pound for pound of body weight, children consume considerably more pesticides than adults. Kids are especially vulnerable to the toxic effects because their metabolic systems don't process or excrete toxins the way adults' systems do. (Sierra Club 2003).

The advocacy group, Environment and Human Health Inc., in a report on the safety of pesticides, claims that

Children are often more susceptible to the toxic effects of pesticides than adults; they take in more pesticides relative to body weight than adults, and have developing organ systems that are less able to detoxify toxic chemicals.

They go on to claim that this special vulnerability persists to 5 years of age:

Children are especially vulnerable to carcinogens before the age of five, when their cells are reproducing most

rapidly, may be more susceptible to loss of brain function if exposed to neurotoxins, and may be more susceptible to damage to their reproductive systems. (EHHI, 2003)

Another group, the Grassroots Environmental Education, in its so-called "fact sheet," "Why Kids Are at Risk," claim that

we do know that all children are uniquely vulnerable to a wide range of health effects from environmental exposures due to physiological and behavioral differences. Their small and developing bodies cannot detoxify or eliminate poisons the way an adult can and they play close to the ground on lawns and floors where residues of chemicals, especially pesticides, are found. Play habits and typical hand-to-mouth behaviour increases their risk for accidental ingestion of toxins. (Grassroots Environmental Education, 2003)

The Children's Health Environmental Coalition, which boasts Erin Brokovich as a board member, claim that children need special protection from environmental chemicals:

Children are not little adults. They are vulnerable to chemicals that adults can tolerate. Their bodies are simply not ready to process and remove toxins. And kids receive proportionately larger doses of environmental toxins than adults. (CHEC, 2003)

The Natural Resources Defense Council, in *Our Children at Risk*, asserts:

That children are uniquely vulnerable to environmental hazards is well established in the scientific literature. In fact, the World Health Organization recommended more than a decade ago that, "when health risks from chemicals are evaluated, the special characteristics of infants and children must be recognized. (NRDC, 1997)

Finally, in a synopsis of a study entitled *Overexposed: Organophosphate Insecticides in Children's Food*, the Environmental Working Group (EWG) lauds the US Food and Drug Administration for using the precautionary principle to enshrine special protections for children's health (an additional 10-fold safety factor for setting allowable concentrations of pesticide residuals in food) into law:

The Food Quality Protection Act (FQPA) requires EPA to act to protect infant and child health, even in the absence of total scientific certainty regarding the toxicity or exposure of pesticides to the fetus, infant or young child. This is a dramatic reversal of previous statutory requirements where EPA had no mandate, and arguably could not act to protect the public health, even child health, in the absence of complete data on the risk from a pesticide. Now the law is clear. In the absence of complete and reliable data on pre- and postnatal toxicity and exposure to a pesticide, the EPA must err on the side of child safety and apply an additional ten-fold margin of safety to food tolerances for the pesticide. (EWG 1998)

The EWG goes on to condemn the US Environmental Protection Agency, however, for sticking to an individualized, evidence-based risk assessment methodology, saying,

Contrary to the clear requirements of the law, the EPA has devised and implemented an official policy in response to FQPA that disregards the requirement for a ten-fold safety factor. This policy plainly undermines protection of the nation's children from pesticides. (EWG, 1998; references internal to the quotations have been omitted)

Are Children at Special Risk from Environmental Chemicals?

A number of scientists have attempted to investigate quantitatively if the margin of safety used in safety assessment adequately accounts for the variability in sensitivity to chemical toxicity between the overall human population and its potentially more sensitive groups, including children. A review of 17 studies that performed

quantitative analyses of the extent of variability in sensitivity due to age or other factors in both humans and animals found that a high percentage of the population, including children, is protected by the margin of safety now used by regulatory agencies to account for sensitivity differences. Based on specific comparisons for newborns, infants, children, and adults, the percentage of the population protected is between 67 and 100. Studies using larger populations that included sensitive individuals suggest that the value is closer to 100%. Thus it appears that the current approach used by regulatory agencies to limit chemical exposures protects most of the people—including children—most of the time (Dourson et al., 2002).

But establishing reliable relationships between chemical contaminants in the environment and children's health outcomes is difficult. Environmental monitoring data are few, so understanding children's (or adults') exposures to chemicals is limited. The indicators of children's health that are available generally involve trends in hospitalization, specific diseases, and causes of death, but there is as yet no way to link those outcomes with contaminant exposures. Furthermore, the "environment" includes many more complexities than just chemical contaminants, such as physical safety, nutrition, and socioeconomic factors.

Complexity of assessing risks to children

The reality of risks to children's health from the environment is complex and difficult to characterize. Children do eat and drink more on a body-weight basis than do adults, so may have higher exposures to chemical and microbial contaminants in food. Infants would have lower exposures to food-borne contaminants than adults but might be exposed to chemicals via breast milk. Crawling babies are exposed to higher levels of contaminants in the home and from soil. As a result, infants' and children's exposures are different from those of adults, although those differences have not always been accounted for explicitly in the past by environmental regulations limiting chemical exposures. As more data are collected on how children's exposures are different, they are being incorporated into regulatory practices.

There are many physiological and pharmacological reasons why susceptibility to the impacts of chemical

exposures may differ between children and adults. A developing fetus undergoes many complex, integrated processes that involve cell growth, differentiation, and morphogenesis. If mutation or altered cell division, enzyme function, or energy sources interfere with these processes, they can have significant adverse impacts on development (Wilson, 1997; Faustman et al., 2000).

A number of environmental factors (construed broadly) are known to have an impact on normal fetal development—including maternal nutrition, folic acid in the diet, prescription drugs, maternal smoking, and alcohol consumption during pregnancy. Similarly, environmental factors can have an influence on normal childhood development, including ingestion of chemical contaminants such as lead (in paint), arsenic (in drinking water), and organic mercury (in fish).

Children also may be more sensitive to some kinds of toxicity than adults because their bodies are growing and could be harmed if something interfered with normal growth and development. Furthermore, different organs grow at different rates and that difference has toxicological implications, notably with respect to “windows of vulnerability,” which are the critical time periods during which chemical exposures may produce particular effects. One reason growing bodies can be more sensitive is that, if changes in DNA such as mutations occur in cells

that are multiplying in order to grow, those changes can be copied into future cells, potentially resulting in cancer or other disorders.

Young children are more sensitive than adults to the toxic effects of some chemicals, such as lead and organic mercury. At the same time, children are less sensitive than adults to other chemicals. Studies examining children’s sensitivity to pharmaceuticals shed some light on how children’s sensitivity differs from that of adults. For example, unlike the situation in adults, liver toxicity and death from acetaminophen poisoning is extremely rare in children (Penna and Buchanan, 1991). Reduced chemical toxicity in children is generally due to their more rapid rates of metabolism and elimination, resulting in lower body burdens of drugs or chemicals than adults would have for the same exposures. As Table 1 shows, morphine is cleared about 50% faster by younger infants than by newborns, while older infants eliminate morphine from the bloodstream three times faster than newborns. Morphine clearance is slower in adults than in older infants and children, but approximately the same as in newborns and younger infants. The chemotherapy drug methotrexate is cleared six times faster by children less than 10 years of age than by adults. The anti-psychotic drug, Thorazine® (*chlorpromazine*), is cleared five times faster by children than by adults.

Table 1: Drug Clearance Rates as a Function of Age

Compound	Age	Clearance
Morphine		(ml/minute/kg)
	< 7 days	8.7 ± 5.8
	7 days–2 months	11.9 ± 5.1
	2–6 months	28.0 ± 8.9
	Children	20.5–25.7
	Adults	6.2–15.6
Methotrexate		(l/kg/hour)
	< 10 years	0.6
	10–15 years	0.2
	Adults (> 15 years)	0.1
Thorazine® (<i>chlorpromazine</i>)		(l/kg/hour)
	Children (0.3–17 years)	3.1 ± 0.6
	Adults (> 17 years)	0.6 ± 1.2

Source: adapted from Renwick 1998.

Note: Clearance rates are expressed as units metabolized (cleared) per unit of body weight, per unit of time.

Children's susceptibility to carcinogens

Much attention has been focused on the susceptibility of children to chemical carcinogens in the environment. The US EPA's cancer-risk assessment guidelines conclude that "[m]ost often differences between carcinogenic effects in the young vs. adults can be traced to differences in the handling of chemical agents" (US EPA, 1999). With notable exceptions, very few human cancers occur in children; cancer is a set of diseases that occur with advancing age. The incidence of cancer in children, like that for all ages, has shown a net increase of about 20% since 1975—although children's mortality from cancer has declined by almost 50% while mortality from cancer for all ages has remained unchanged (NCI, 2001). A number of environmental exposures, including pesticides, parental occupational exposures, ionizing radiation (gamma rays, radon), non-ionizing radiation (power lines, electrical appliances), and infectious organisms, have been suggested as possible precursors to cancer in children; however, the considerable research conducted to date has yielded inconsistent or limited evidence linking those factors to cancer in children (Public Health Policy Advisory Board, 1999).

Since there are so few cancers among children, researchers are forced to rely on laboratory experiments using animals to explore whether or not young animals are generally more sensitive to chemical carcinogens than older animals. But the evidence does not suggest such heightened sensitivity in young animals. The EPA's 1996 report, *Comparison of the Effects of Chemicals with Combined Perinatal and Adult Exposure vs. Adult-Only Exposure in Carcinogenesis Bioassays*, concluded that lowering the age of first chemical exposure in rodents to include the perinatal stage (from the twentieth week of gestation to the twenty-eighth day of life) neither increased the sensitivity of the bioassays nor produced tumors of different types than did the standard bioassays (US EPA, 1996). That report also showed that increasing total doses by adding a perinatal stage can slightly increase tumor incidence and sometimes decrease tumor latency. Studies of the effects of anticancer drugs, viral infections, and ionizing radiation demonstrate that both the young and old develop a similar spectrum of tumors (US EPA, 1996).

Taken together, those observations do not provide strong support for the idea that children are generally more sensitive to carcinogens than adults. Rodent bioassays show that younger animals are less susceptible to chemical carcinogens in some cases and more susceptible in others, depending on the chemical. *Pesticides in the Diets of Infants and Children*, published by US National Academy of Sciences/National Research Council, included a table summarizing the results of studies (performed through 1983) in which the effects of age on chemically-induced carcinogenesis in rodents had been evaluated (NAS/NRC, 1993). Charnley and Putzrath updated those results to include studies performed since 1983 (Charnley and Putzrath, 2001). The data indicate that there are a similar number of studies showing that younger animals are less susceptible than adults (47%) to chemically-induced carcinogenesis as there are showing that they are more susceptible (40%) under the conditions of the bioassays. A number of studies showed that age played no role at all in susceptibility (13%). The report from the NAS/NRC concluded that the rodent bioassays reviewed clearly demonstrate that age may be an important factor in susceptibility to chemically induced carcinogenesis, but they do not support the conclusion that younger animals are always more susceptible than older animals (NAS/NRC, 1993).

The data also illustrate the difficulty associated with assessing quantitatively the extent of the differences in susceptibility due to age. Virtually all of the studies evaluated used only one dose level, so the underlying dose-response relationships are unknown and comparison of sensitivities is possible only at the relatively high, single-dose levels. Generalizations about the effect of age on susceptibility to chemical carcinogens are thus difficult to make. Experiments using laboratory animals suggest that young animals are not generally more sensitive to chemical carcinogens than older animals.

Children's susceptibility to toxic chemicals

Data on acute chemical toxicity show similar results. A review of the data available on the lethal doses of a variety of chemicals for 50% of exposed laboratory rodents (LD₅₀ milligram-per-kilogram body weight) showed only small differences due to age (Calabrese, 1986). In some

cases, infants were more susceptible and, in some cases, adult animals were more susceptible. In only a few cases did the differences exceed an order of magnitude. In many cases, there were no differences. Data on the maximum tolerated doses (MTDs) of chemotherapeutic agents in humans show that MTDs were frequently higher for children than adults, indicating greater susceptibility of adults, although the differences between age groups were usually less than or equal to two (Bruckner, 2000).

Sensitivity to organophosphate pesticides

Studies of acute toxicity from organophosphate pesticides also show variability. For 36 pesticides given orally to weanling and young adult rats, no more than two-fold to three-fold differences in sensitivity were observed, with the younger animals more sensitive to toxicity than older animals in only four cases (Gaines and Linder, 1986). In contrast, 14 of 15 organophosphate pesticides showed greater acute toxicity to weanling rats than to adult rats. (Brodeur and DuBois, 1963). Newborn rats were more sensitive than adult rats to malathion poisoning but less sensitive than adult rats to dieldrin toxicity (Lu et al., 1965).

One of the few chemicals for which low-dose data do exist is the insecticide *chlorpyrifos*. Those data show that young animals are more sensitive than adults to the nervous-system toxicity of *chlorpyrifos* at high doses but are less or similarly sensitive than adults at low doses (Mattson et al., 2000). The reason young animals are less sensitive to *chlorpyrifos* toxicity at low doses is that they can compensate for it faster than adult animals can. In this case, extra regulatory protection of the young based on the high-dose observations would have no advantage because the low-dose data show that the young are not more sensitive than adults at the low exposure levels expected to occur in the environment.

Since environmental exposure levels are negligible and risks from negligible levels of pesticides are far less than those from naturally occurring toxic chemicals and microbial contamination in food, others defend pesticides as improving children's health due to their contribution to abundant and affordable produce (Winter, 2001). The

Canadian Institute of Child Health acknowledges that the impact of life-long exposure to environmental contaminants on life expectancy or on disease is yet to be determined (CICH, 2000).

Conclusions

The available evidence on age-related susceptibility of laboratory animals to the effects of chemical contaminants thus suggests that children may be more than, less than, or just as sensitive as adults, depending on the chemical and the exposure situation. Most of the available information on age-related differences in sensitivity, however, comes from experiments using single, high doses of chemicals that produced short-term, acute toxicity. Those observations may be poor predictors of what occurs when low doses of chemicals are received over long periods of time or at key times during development. Long-term exposure to low doses of chemicals can produce different types of toxicity than short-term exposure to high doses—or no discernible toxicity. On the other hand, low environmental exposures to chemicals are less likely to overwhelm developing detoxification mechanisms, so age-related differences at low doses may be quantitatively less pronounced than at high doses.

In sum, scientists' current understanding of the rates of maturation of metabolic capability indicate that human infants up to approximately 6 months of age typically—but not always—have lower rates of metabolic transformation and elimination than adults. For most chemicals, the immaturity of infant biotransformation, elimination, and other physiologic systems produces higher concentrations in the blood for longer periods, although for some chemicals, unique metabolic pathways not available in the adult human can be used by the newborn. The newborn's metabolic capacity rapidly matures and by about 6 months of age most metabolic systems are reasonably mature, becoming almost completely adult-like by one year of age. Children over 6 months of age can be more sensitive metabolically to chemical toxicity than adults but they usually are not; in many cases they are less sensitive (Scheuplein et al., 2002).

The reality of children's risk in Canada

From a historical perspective, great progress has been made in protecting and promoting the health of children in Canada (Myres, 2001). Infant mortality rates were cut by 81% between 1960 and 2000 (OECD, 2003). Life expectancy has increased from 61 years for women and 59 years for men born in 1920 to 82 years for women and 77 years for men born in 2000. Gains in life expectancy are due both to improved treatments for adults with diseases and other health problems and to better nutrition, immunization, and safer environments that contribute to the health of infants and children (CICH, 2000).

Chemical contaminants

Looking at the environment in terms of chemical contaminants, the news is good. Air quality, for example, continues to improve, with a 49% decrease in average levels of the six primary air pollutants in Canada since 1980 (Environment Canada, 2003). During that same period there was a 28% increase in kilometers traveled by automobile, a 26% increase in energy consumption, and an 83% increase in the Canadian GDP. In 1992, ambient lead concentrations in Canada had been reduced by 97% compared to 1974; Canadian children's average blood lead levels were reduced from 11.9 µg/dL (micrograms per deciliter of blood) in 1984 to 3.5 µg/dL in 1992 (CICH, 2000). The levels of benzene in air and emissions of mercury to air both decreased by 35% from 1995 to 2000 (Environment Canada, 2002a). The levels of contaminants in breast milk also show downward trends, with the levels of PCBs decreasing from 26 ng/g fat (nanograms of PCBs per gram of fat—a nanogram is a millionth of a gram) in 1982 to 7 ng/g fat in 1992 and the levels of DDT decreasing from 140 ng/g fat in 1967 to 8 ng/g fat in 1992 (Environment Canada, 1996).

Despite such improvements, cultural, economic, and geographic disparities exist. For example, infants from

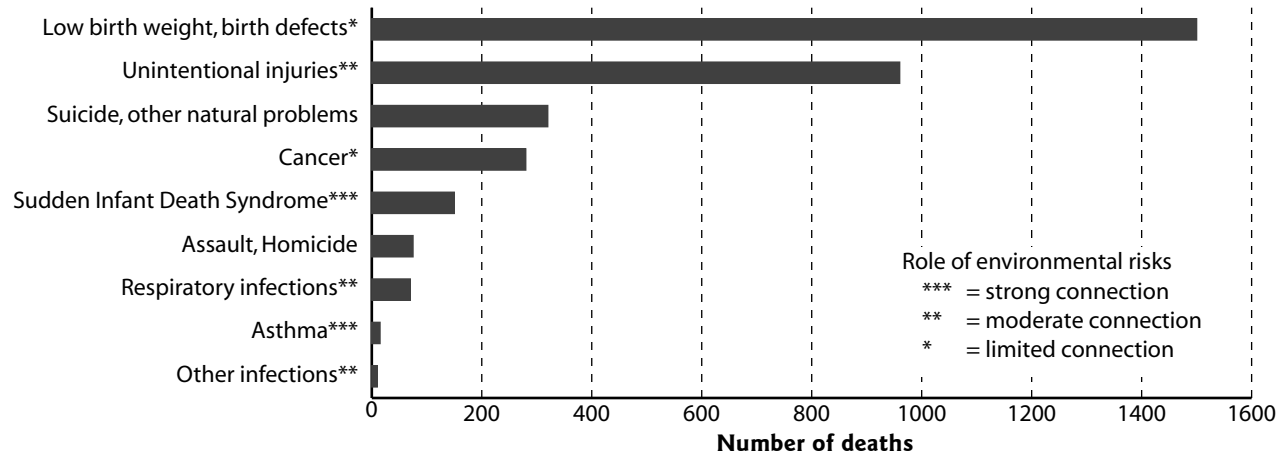
Nunavik have the highest known exposure to PCBs via breast milk in the world. The concentration of PCBs in breast milk is four times higher among Nunavik women than among women in southern Quebec (CICH, 2000). Studies of the effects of PCBs in infants are inconsistent, showing some temporary developmental problems and “modest” effects upon the immune system. Cancer incidence rates among Canadian children have remained stable over the past 15 years while the cancer mortality rate has decreased at a rate of 4% per year since 1970. Although rare, cancer is second only to injuries as the leading cause of death among Canadian children from one to 14 years of age (Health Canada, 1999b).

Other risks

While the evidence suggests children are not at particular risk from environmental factors, children face many other risks. Figure 1 shows the leading causes of death among children from zero to 19 years of age in Canada in 1997, with asterisks denoting the extent to which environmental hazards play a role (CICH, 2001, as reported in Myres 2001). But even such straightforward statistics can be deceptive: Sudden infant death syndrome is denoted as having a strong environmental influence, but “the environment” in this case is the physical nature of a child's bed and his or her position in it. That “environment” is not affected by chemical pollutants, which people often assume to be important environmental hazards.

Poverty

Poverty is also a known risk factor for children, with children of lower economic status tending to live in environments that are associated with higher exposures to lead, vehicle exhaust, pesticides, and industrial contaminants than other children. Children of lower economic status also have poorer nutrition, which can make them more vulnerable to disease (Newacheck, 2003).

Figure 1: Leading causes of death among children from zero to 19 years of age in Canada, 1997

Note: asterisks denote the extent to which environmental hazards play a role.

Source: CICH, 2001, as reported in Myres 2001.

Smoking and alcohol

Maternal smoking and alcohol consumption remain important prenatal risk factors. About one quarter of pregnant women in Canada reported that they drank alcoholic beverages during pregnancy, with the prevalence increasing with both age and income (CICH, 2000). Alcohol consumption during pregnancy can lead to serious developmental defects, both physical and mental, in children. Among smokers, about 90% reported that they continued to smoke during pregnancy (CICH, 2000). The infants of mothers who smoke during pregnancy tend to be smaller than those whose mothers do not smoke; birth weight is one of the most important determinants of health throughout life.

Asthma

Other important problems remain unsolved. The prevalence of asthma in Canadian children increased five-fold between 1978 and 1999 (Health Canada, 2001). Some of that increase may be attributable to better diagnosis and reporting but allergens in indoor air, genetic predisposition, lung development, and exposure to immune suppressants and infectious agents may also contribute (CICH, 2000). Outdoor air pollution is often blamed in the increase in the prevalence of children's asthma but most types of air pollutant have decreased significantly while the rates of asthma in children have increased, suggesting that there is no causal mechanism linking outdoor air quality to asthma in children (Koenig et al., 2001; NAS, 2003).

Is more regulation the answer?

Numerous programs in Canada focus on the protection of children's health. At the federal level alone, Canada has at least seven programs intended to protect children:

- *Canadian Environmental Protection Act* (CEPA), 1999
- *The Pest Control Products Act* (PCPA), 2002
- *The Canadian Institute of Child Health* (CIH)
- Health Canada's Division of Childhood and Adolescence's Centres of Excellence for Children's Well-Being
- Health Canada's Office of Children's Environmental Health
- Commission for Environmental Cooperation (CEC)
- The Domestic Substances List

(See Appendix, page 18 for a more complete description of these programs.)

The Precautionary Principle

Despite the extensive coverage of children's risk by regulatory agencies, however, there is continuous pressure by activist groups to base children's environmental health protection on the precautionary principle, the idea that it is better to be "safe than sorry." In other words, if the available science is inadequate to guide a decision about how best to treat a potential risk to children, that inadequacy is not a basis for inaction. The precautionary principle was statutorily enshrined in 1999 with the *Canadian Environmental Protection Act*, which states that the Government of Canada is "committed to implementing the precautionary principle that, where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation" (see preamble and §§ 1(1)(a)). The precautionary principle is also the basis of the requirement in the 2002 *Pest Control Products Act* for regulators to assume that, in the absence of information to indicate otherwise, chil-

dren should be considered more susceptible to risks from pesticides than adults, with additional stringency taken to avoid children's potential risks.

An example of the precautionary principle in action is the ban on cosmetic (aesthetic) uses of pesticides in Hudson, Quebec. Although there is no clear scientific evidence demonstrating that such pesticides pose a risk to children or the environment when used according to their labels, the citizens of Hudson decided that the potential benefits of the pesticides were not enough to justify their potential risks and took the precautionary measure of banning them.

Is current regulation adequate?

The question is, does banning chemicals or regulating them more stringently protect or improve children's health? Certainly, banning lead in gasoline contributed to the substantial reduction in the environmental burden of lead and in children's blood lead levels. High blood lead levels are associated with toxicity to the nervous system and intelligence deficits in children. Environmental mercury emissions have also decreased significantly. Exposure of pregnant women to fish or sea mammals containing high levels of methylmercury, which is produced naturally by bacteria and fungi but can also result in part from the burning of fossil fuels, has been associated with toxicity to the developing fetus. Beyond those two examples, there are virtually no well-substantiated correlations between exposure to chemicals at the levels normally found in the environment and public-health problems, in children or in adults.

Evaluating the effectiveness of regulation

One key difficulty with evaluating whether these current environmental regulatory approaches are adequate to protect public health is that virtually everything we know about differences in the susceptibilities of infants,

children, and adults comes from observing the effects of substances following exposure levels—such as those encountered in clinical studies with drugs, intentional or accidental exposures, accumulation after repeated exposures, and improper use or abuse—that are higher than the exposure levels typically encountered in the environment. There seems to be little in the literature suggesting that low doses of chemicals that are modestly well excreted are generally more hazardous in infants and children than in adults. Low environmental exposures to chemicals are less likely to overwhelm developing detoxification mechanisms so, where they occur, age-related differences at low doses may be quantitatively less pronounced than at high doses.

Further proliferation of safety measures aimed at small risks and considered in isolation will only make it more difficult to compare the benefit of existing or suggested regulatory efforts. And such comparisons are further complicated when complex mitigation measures are required and when risk-reduction measures only benefit a small percentage of the population. Such complications make it difficult to ensure that resources used to address risks produce the desired outcome without introducing new risks, or by shifting risk from one person or avenue of life to another.

Risk-trading and risk-shifting

Like most of the actions taken to improve safety, actions intended to reduce environmental health risks are rarely pure in their effects. Choices have consequences, and it is a true Pollyanna who thinks that any significant action, risk-reducing or otherwise, can have purely positive consequences. While some safety improvement may be gained through the impact of a given risk-reduction measure, in many cases, the unintended consequences of the measure can produce countervailing impacts that erase some or all of the perceived benefit.¹

Part of the reason that risk-reducing measures are ambiguous is that risks themselves are often ambiguous. Even for a simple risk scenario, people do not face risks in the same way. Rather, each person has a highly individualized portfolio of risks of various types: environmental, nutritional, hereditary, social, and so on. Generally, we address risk issues by sectioning them off into

risk areas: environmental risk, transportation risk, food safety, and so on.

The tendency to section off risks into easily manageable categories increases the likelihood of unintended consequences such as those following from airbag regulations. In the most extreme cases, such tunnel vision can lead to actions that might ultimately cause more harm than good. For example, regulators afflicted with tunnel vision frequently overlook the phenomenon that some call “death by regulation.” Although regulatory costs and job losses are not often considered risk-relevant in themselves, they should be. The linkage between income and risk is subtle but intuitive. We know, for example, that people’s safety is related to their income. Those with lower incomes are proportionately less able to take the safety measures that higher-income earners can. Families with higher incomes can better withstand short-term health problems than those with lower incomes. Families with higher incomes eat higher-quality foods, drive safer cars, live in safer neighborhoods, train their children for safer jobs, and so on (Keeney, 1997; Keeney and Green, 1997; Wildavsky, 1980; Viscusi, 1994).

Peer-reviewed studies over two decades have examined the relationship between income and risk. The general conclusion of such studies is this: people use their disposable income to weave a personal safety net around themselves and their loved ones. The more disposable income they have, the tighter the weave of their personal safety net. The less disposable income they have, the looser the weave. As systems engineer Ralph L. Keeney points out in *Estimating Fatalities Induced by the Economic Costs of Regulations*:

Regulatory costs are paid by individuals, which leaves them with less disposable income. Since individuals on average use additional income to make their lives safer and healthier, the regulatory costs lead to higher mortality risks and fatalities. Based on data from the *National Longitudinal Mortality Study* relating income to the risk of dying, approximately each \$5 million of regulatory cost induces a fatality if costs are borne equally among the public. If costs are borne proportional to income, approximately \$11.5 million in regulatory costs induces a fatality. (Keeney, 1997)

Cost-effectiveness of risk-reducing measures

In the United States, where the benefits of regulating risk have been studied closely, the complicating factors above result in a growing irrationality in risk-reduction investment choices. As risk analysts Tammy Tengs and John Graham have shown, public-policy priorities often bear little relationship to any rational ranking of risks and potential risk-reduction investments of tax dollars:

- To regulate the flammability of children's clothing, the United States spent \$1.5 million per year of life saved, while some 30% of those children live in homes without smoke alarms, an investment that costs about \$200,000 per year of life saved (Tengs and Graham, 1996).
- To regulate potentially carcinogenic benzene emissions during waste operations, the United States spent \$19 million per year of life saved, while 70% of women over age 50 do not receive regular mammograms, an intervention that would achieve benefits that cost roughly \$17,000 per year of life saved (Tengs and Graham, 1996).
- While the United States spent approximately \$21.4 billion in 1994 on 185 life-saving interventions that averted approximately 56,700 premature deaths, spending that same amount of money based on priorities to maximize effectiveness could have saved an additional 60,200 people (Tengs and Graham, 1996).

Tengs and colleagues demonstrated in their study of the cost-effectiveness of risk-reduction measures undertaken by various governmental agencies that all investments in risk reduction do not yield equal results (Tengs et al., 1995). Table 2 shows the median cost of intervention for five regulatory agencies, each charged with reducing risk within its sphere of authority. To date, saving lives through environmental regulations has been, in the aggregate, more expensive than saving lives through other types of safety regulation, though some individual environmental measures may be highly cost-effective ways to reduce risk.

As Graham, Tengs, and others have argued, rational risk management requires a net-benefit framework for portraying both the nature of environmentally conveyed health risks and the consequences of taking action to reduce them (Green, 1997). It also requires an easily understood framework for choosing a risk-reduction strategy. Such a rational risk-management framework can help decision-makers make sensible choices regarding risk policy. It can also help individuals evaluate whether they are getting a net benefit from the taxpayer resources invested in environmental risk reduction.

In a landmark work on risk management, policy analyst Aaron Wildavsky pointed out that the success of policy responses to a risk depends acutely on the state of knowledge about the risk and the efficacy of one's proposed response. Employing both theory and empirical observation, Wildavsky observed that a strategy of risk-interception (i.e., by passing a specific regulation, or setting a specific exposure limit) is likely to be successful only in situations of excellent information (Wildavsky, 1991). So, for example, for a power plant owner who

Table 2: Median value of cost/life-year saved for five regulatory agencies

Regulatory Agency	Median cost/ life-year saved
Federal Aviation Administration	\$23,000
Consumer Product Safety Commission	\$68,000
National Highway Traffic Safety Administration	\$78,000
Occupational Safety and Health Administration	\$88,000
Environmental Protection Agency	\$7,600,000

Source: Tengs et al., 1995.

knows that a particular part is going to burn out every 150 days an interception strategy of replacing the part every 149 days to prevent the risk may be cost-effective. But where less information exists, strategies that focus on resilience (such as education, adaptation, and case-by-case responsiveness) are likely to succeed where interception is either infeasible or expensive. For example, if a power plant had 8,000 critical pieces of equipment that, upon failure, would create a fire but the plant owner did not know the failure rate of each piece, trying to intercept the risk by replacing pieces before they failed would be enormously costly. Further, trying to have backup systems on all 8,000 pieces would be technologically difficult and financially infeasible. Instead, a strategy of resilience, such as educating employees about how to minimize the chance of fire, and having a sophisticated fire-response system, is more likely to be a feasible and efficient way of dealing with this risk (Green, 1999).

Figure 2 shows how uncertainties about the nature and scope of a risk and uncertainties about intervention measures and their effects constrain strategy selection, favoring certain approaches over others.

Conclusion

Children's health protection is already extensively regulated in Canada. The efficacy of additional, "precautionary," regulation is dubious. Experience in the United States suggests that risk-reduction investments based

on diffuse principles and without regard for prioritization or risk shifting are unlikely to produce significant social benefits. Indeed, such haphazard risk-reduction investment is likely to lead to poor investment strategies that fail to save as many lives as could be saved either through more science-based investment or through leaving resources in the hands of individuals to purchase additional safety for themselves and their loved ones with their daily spending decisions.

Note

- 1 Of course, there may be ancillary effects (both positive and negative) of a given action that do not relate to risk. For example, air pollution reduction has the ancillary benefit of providing greater visibility, which some people would value highly. But ozone-reduction regulations would also likely have negative impacts on the costs and availability of recreational activities such as motorhome camping, motorcycle touring, or powerboating, which other people would consider a negative impact on lifestyle. Such considerations would be important in a holistic regulatory analytical framework that looked beyond risk to total regulatory impact. However, classification of a regulatory effort as "risk-reducing" generally excludes considerations that do not relate directly (or indirectly) to health risk, either by legislative decree or by environmental agency interpretation of existing law.

Figure 2: Appropriate strategies for different conditions

		Amount of knowledge about intervention measures	
		<i>Small</i>	<i>Large</i>
Knowledge of the nature and scope of risks and future conditions	<i>High</i>	More resilience, less interception	Interception
	<i>Low</i>	Resilience	More resilience, less interception

Source: adapted from Wildavsky, 1991: 122.

Conclusions & recommendations

Canada's children are healthier than ever before and are not at special risk from environmental chemicals

Great progress has been made in protecting and promoting the health of children in Canada. Infant mortality rates have decreased by 81% from 1960 to 2000. Life expectancy has increased from 61 years for women and 59 years for men born in 1920 to 82 years for women and 77 years for men born in 2000. Looking at the environment in terms of chemical contaminants, the news is also good. Air quality continues to improve, with a 49% decrease in average levels of the six primary air pollutants in Canada since 1980. In 1992, ambient lead concentrations in Canada had been reduced by 97% compared to 1974; Canadian children's average blood lead levels were reduced by 70% between 1984 and 1992. The levels of benzene in air and emissions of mercury to air both decreased by 35% from 1995 to 2000. The levels of contaminants in breast milk also show downward trends, with the levels of PCBs decreasing 73% between 1982 and 1992 and the levels of DDT decreasing by 94% between 1967 and 1992. Studies of children's metabolism of pharmaceuticals and experiments using juvenile experimental animals suggest that children's susceptibility to environmental chemicals is not particularly more pronounced than that of adults, varying from equally susceptible, to greater susceptibility, to lesser susceptibility depending on the chemical involved.

Canadian children are well protected by existing programs—education and research are needed more than additional regulation

Numerous programs in Canada focus on the protection of children's health. Canada has at least seven programs intended to protect children at the federal level alone.

Current margin of safety is protective

A review of 17 studies that performed quantitative analyses of the extent of variability in sensitivity due to age or other factors in both humans and animals found that a high percentage of the population, including children, is protected by the margin of safety now used by regulatory agencies to account for sensitivity differences. Studies using larger populations that include sensitive individuals suggest that the value is close to 100%. Thus, it appears that the current approach used by regulatory agencies to limit chemical exposures protects most of the people—including children—most of the time.

Low-level risks over emphasized

Poorly-established risks from environmental exposures to chemicals often receive more attention from policy-makers than do the well-established leading causes of childhood morbidity and mortality. Unintentional injuries remain the leading cause of death among children. Maternal smoking and consuming alcoholic beverages during pregnancy impair infant development and are regrettably common. Children spend more than 80% of their time indoors but little responsibility has been taken to ensure a healthy indoor environment (Pollution Probe, 1998). Although limiting children's chemical exposures remains an important goal, in the absence of strong evidence implicating childhood exposure to individual chemicals at low, environmental levels in childhood or adult disease, actions taken to protect children from the many, well-defined and established threats to their health are likely to have greater and more demonstrable impacts.

More data needed for case-by-case risk evaluation

The measures by which we usually characterize the environment—water quality, air quality, reportable emissions—show that substantial improvements have been made. What we lack, however, is an ability to measure

those accomplishments in terms of an impact on public health, if there is one. We do not have the disease surveillance data or the environmental exposure data needed to determine how, and to what extent, the two may be related. For example, information correlating trends in air quality and morbidity from respiratory disease could help Health Canada evaluate not only whether air quality has improved but also whether rates of respiratory problems associated with air pollution have fallen and, if not, why. There is little evidence that environmental exposures play a leading role in childhood disease but, even if they play a minor role, they could constitute a public-health problem by virtue of the numbers of people affected. Without the right data, the role of the environment—and the most effective actions needed to have an impact on that role—are likely to remain speculative and controversial, and could produce less return on investment than alternative risk-reduction efforts available in other public-policy areas.

Unfocused risk regulations offer little benefit

Regulatory efforts to manage risk have not shown a good history of prioritization or return on investment. In the United States, where the benefits of regulating risk have been studied closely, the pursuit of ever smaller, less-defined risk management has resulted in a growing irrationality in risk-reduction investment choices. As risk analysts at Harvard University have shown, public-policy priorities in the United States often bear little re-

lationship to any rational ranking of risks and potential risk-reduction investments of tax dollars. As risk analyst Aaron Wildavsky has demonstrated, in conditions of high uncertainty about risk, an agenda of research and education is superior to one of regulation based on precaution.

Studies like the proposed *National Children's Study* will gather much of the types of data that are needed to help scientists infer the roles of environmental, genetic, and social factors in disease, where “environment” is defined broadly to include natural and man-made environmental factors, biological and chemical factors, physical surroundings, social factors, behavioural influences and outcomes, genetics, cultural and family influences and differences, and geographic locations (*National Children's Study*, 2003). What scientists are likely to find, however, is that both good and bad health result from complex interactions among the factors. Whether, and to what extent, regulating individual chemicals that are present at low levels in the environment has an impact on those interactions and, ultimately, on public health in general or children's health in particular may never be clear. When “environment” is defined as broadly as it is by the *National Children's Study*, there is no question that environment plays an important role in both health and disease. When “environment” is defined much more narrowly as relating to low, regulated chemical exposures such as those from pollution, consumer products, energy production, or food, its role in health and disease is less clear.

Appendix—current approaches to protecting children

Children’s health does not suffer from a lack of regulatory or agency attention. Many laws are already in place and efforts are underway in Canada to protect children’s health. Several of these are described below.

Canadian Environmental Protection Act (CEPA), 1999

Health Canada has the responsibility for administering the provisions of the *CEPA* that are intended to protect human health (at all ages) from toxic substances. While this law does not specifically address children, the standards used to limit exposures to toxic substances generally protect both children and adults. Where information about children’s unique exposures or sensitivities is available, it can be incorporated into the regulatory structure used to limit exposures.

Pest Control Products Act (PCPA), 2002

The *PCPA* is used to regulate pesticides and to minimize their potential risks to human health while acknowledging health, economic, and other benefits that pesticides bring. The law requires Health Canada to “apply appropriate margins of safety to take into account . . . different sensitivities to pest control products of major identifiable subgroups, including pregnant women, infants, children” and to use an extra 10-fold margin of safety to protect infants and children against pesticides intended for use in the home or schools. All regulatory decisions about pesticides specifically consider children’s unique exposures and the potential for developmental toxicity.

Canadian Institute of Child Health (CICH)

The CICH is a national, charitable, institute dedicated to protecting and improving children’s health through advocacy and building community resources. The CICH’s primary activities include monitoring children’s health, educating parents and policy-makers, and advocating legislation and policies to improve children’s health. The CICH’s environmentally focused activities address safety issues and injury prevention (car seat restraints, bicycle helmets) as well as chemical exposures (poison control, removal of lead from gasoline).

Health Canada, Division of Childhood and Adolescence, Centres of Excellence for Children’s Well-Being

The vision of the Centres of Excellence for Children’s Well-Being is to improve the understanding of, and responsiveness to, the physical and mental-health needs of children and the critical factors needed for healthy child development. The Centres are mandated to ensure that important knowledge about children and their healthy

development is broadly distributed among families, community-based organizations, educators, health professionals, non-government organizations and governments.

Health Canada, Office of Children's Environmental Health

This office is the focal point within Health Canada both for understanding the special sensitivity of children to environmental threats and for promoting action to reduce the risk of these threats to children's health. The office monitors and analyzes scientific evidence, identifies knowledge gaps and advocates research to fill the gaps, coordinates the development of policies and strategies to reduce children's environmental health threats, and develops public education materials. The office is considering a study that would complement the *US National Children's Study*, which hopes to follow a cohort of 100,000 children from the prenatal period to adulthood to study environmental influences on health and development (USDHHS 2003). A life-stage approach will be used to investigate the interactions between chemical, genetic, behavioural, physical, and social factors that affect children's health and development.

Commission for Environmental Cooperation (CEC)

Established by Canada, Mexico, and the United States as a result of the North America Free Trade Agreement, the CEC is developing a cooperative agenda to protect children's health from environmental threats, with a focus on asthma and toxic substances, particularly lead. The overall objective is reducing human-made pressures on children's health.

Regulatory activities—chemicals

The *Domestic Substances List* is an inventory of all substances in commerce in Canada. Exposure to those substances is limited through the use of regulatory safety assessment. The goal of regulatory safety assessment is to restrict chemical exposures in order to ensure that children and adults are protected from chemical risks. Safety assessments are based on information about potential hazard (the intrinsic nature of a substance's toxicity), about the relationship between dose and response (the level of exposure that is needed to produce toxicity), and about anticipated or, in some cases, conservative (i.e., higher) estimates of exposure patterns and levels (via air, water, food, soil). Exposures are limited by regulatory tolerances such as acceptable daily intakes. Regulatory tolerances generally are based on animal toxicity tests and use margins of uncertainty to limit allowable exposures to well below those doses shown to have no effect in test animals. When chemicals are found to produce toxicity specific to developmental processes, they are regulated more stringently than if they produce other types of toxicity. If tolerances are determined accurately and, in particular, by tests in which laboratory animals are exposed prenatally and throughout their complete lifetimes, humans of whatever age, exposed at or below the tolerances, are unlikely to be at significant risk (Dourson et al. 2002).

References

- Alhakami, A.S., and P. Slovic (1994). "A Psychological Study of the Inverse Relationship between Perceived Risk and Perceived Benefit." *Risk Analysis* 14: 1085–96.
- Brodeur, J., and K.P. DuBois (1963). "Comparison of Acute Toxicity of Anticholinesterase Insecticides to Weanling and Adult Male Rats." *Proceedings of the Society for Experimental Biology and Medicine* 114: 509–11.
- Bruckner, J.V. (2000). "Differences in Sensitivity of Children and Adults to Chemical Toxicity: The NAS Panel Report." *Regulatory Toxicology and Pharmacology* 31: 280–85.
- Calabrese, E.J. (1986). *Age and Susceptibility to Toxic Substances*. New York City: John Wiley & Sons.
- Canadian Institute of Child Health [CICH] (2000). *The Health of Canada's Children*. Third Edition. Ottawa: Canadian Institute of Child Health. <<http://www.cich.ca>>.
- Charnley, G., and R.M. Putzrath (2001). "Children's Health, Susceptibility, and Regulatory Approaches to Reducing Risks from Chemical Carcinogens." *Environmental Health Perspectives* 109: 187–92.
- Children's Health Environmental Coalition [CHEC] (2003). *Protect Your Kids*. <http://www.checnet.org/improve_main.asp>.
- Children's Health Project (2000). *Environmental Standard Setting and Children's Health*. Canadian Environmental Law Association and Ontario College of Family Physicians Environmental Health Committee. <http://www.cela.ca/ch_health/titlepg.htm>.
- Choonara, I.A., A. Lawrence, et al. (1992). "Morphine Metabolism in Neonates and Infants." *British Journal of Clinical Pharmacology* 34: 434–37.
- Choonara, I.A., P. McKay et al. (1989). "Morphine Metabolism in Children." *British Journal of Clinical Pharmacology* 28: 599–604.
- Commission for Environmental Cooperation (2003). <<http://www.cec.org>>.
- Commission of the European Communities (2003). *A European Environmental Health Strategy*. Communication from the Commission to the Council, the European Parliament, and the European Economic and Social Committee. Brussels, Belgium.
- Danish Environmental Protection Agency (2001). *Environmental Project, 589 Children, and the Unborn Child: Exposure and Susceptibility to Chemical Substances—an Evaluation*. <http://www.mst.dk/udgiv/Publications/2001/87-7909-574-7/html/default_eng.htm>.
- Donelli, M.G., M. Zucchetti, et al. (1995). "Pharmacokinetics of HD-MTX in Infants, Children and Adolescents with Nonlymphoblastic Leukemia." *Medical and Pediatric Oncology* 24: 154–59.
- Dourson, M., G. Charnley, and R. Scheuplein (2002). "Differential Sensitivity of Children and Adults to Chemical Toxicity II. Risk and Regulation." *Regulatory Toxicology and Pharmacology* 35: 448–67.
- Environment Canada (1996). *Conserving Canada's National Legacy*. CD-ROM. <http://www.msc-smc.ec.gc.ca/library/cdrom_e.html#C>.
- Environment Canada (2002a). *Comprehensive Mercury Inventory and National Air Pollution Surveillance Network*. <<http://www.ec.gc.ca/soer-ree/English/headlines/ind8.cfm>>.
- Environment Canada (2002b). *Envirozine*, 21 May. <http://www.ec.gc.ca/envirozine/english/home_e.cfm>.
- Environment Canada (2003). *Environmental Signals: Canada's National Environmental Indicator Series 2003*. Ottawa. <<http://www.ec.gc.ca/soer-ree/English/default.cfm>>.

- Environment Canada National Air Pollution Surveillance Network (2003). Personal communication between Steven Hansen and John Shelton, May 7.
- Environment and Human Health, Inc. [EHHI] (2003). *Risks from Lawn-Care Pesticides*. <http://www.ehhi.org/pubs/pesticides/ehhi_pesticides_full.pdf>.
- Environmental Working Group [EWG] (1998). *Overexposed: Organophosphate Insecticides in Children's Food*. <<http://www.ewg.org/pub/home/reports/ops/fqpa.html>>.
- European Commission [EC] (2003). *A European Environment and Public Health Strategy*. Press Release. <<http://europa.eu.int/>>.
- Faustman, E.M., S.M. Silbernagel, et al. (2000). "Mechanisms Underlying Children's Susceptibility to Environmental Toxicants." *Environmental Health Perspectives* 108: 13–21.
- Federal, Provincial and Territorial Advisory Committee on Population Health [ACPH] (1999). *Toward a Healthy Future: Second Report on the Health of Canadians*. Prepared for the meeting of the Ministers of Health, Charlottetown, P.E.I., September 1999. Ottawa: Minister of Public Works and Government Services.
- Furlanut, M., P. Benetello, M. Baraldo, G. Zara, G. Montanari, and F. Donzelli (1990). "Chlorpromazine Disposition in Relation to Age in Children." *Clinical Pharmacokinetics* 18: 329–31.
- Gaines, T.B., and R.E. Linder (1986). "Acute Toxicity of Pesticides in Adult and Weanling Rats." *Fundamental and Applied Toxicology* 7: 299–308.
- Grassroots Environmental Education (2003). *Kids and Toxins*. <<http://www.grassrootsinfo.org/>>.
- Health Canada (1999a). *Measuring Up: A Health Surveillance Update on Canadian Children and Youth*. <<http://www.hc-sc.gc.ca/pphb-dgspsp/publicat/meas-haut/index.html>>.
- Health Canada (1999b). *A National Children's Agenda: Developing a Shared Vision—Background*. <<http://www.hc-sc.gc.ca/english/media/releases/1999/nca99ebk1.htm> and http://unionsociale.gc.ca/nca_e.html>.
- Health Canada (2001) *Respiratory Disease in Canada*. Ottawa. <http://www.hc-sc.gc.ca/pphb-dgspsp/ccdpc-cpcmc/crd-mrc/pubs_e.html>.
- Health Canada (2002). *Centres of Excellence for Children's Well-Being*. <http://www.hc-sc.gc.ca/dca-dea/allchildren_touslesenfants/centres_main_e.html>.
- Health Canada (2003a). *Children's Environmental Health*. <<http://www.hc-sc.gc.ca/hecs-sesc/oceh/index.htm>>.
- Health Canada (2003b). *Safe, Healthy Environments*. <http://www.hc-sc.gc.ca/dca-dea/allchildren_touslesenfants/she_main_e.html>.
- Keeney, Ralph L. (1997). "Estimating Fatalities Induced by the Economic Costs of Regulations." *Journal of Risk and Uncertainty* 14: 5–23.
- Keeney, Ralph L., and Kenneth Green (1997). *Estimating Fatalities Induced by the Economic Impacts of EPA's Proposed Ozone and Particulate Standards*. Policy Study No. 225. Los Angeles, CA: Reason Public Policy Institute.
- Koenig, Harold, et al. (2001) *Asthma: Separating Fact from Fiction*. Annapolis, MD: The Annapolis Center for Science-Based Public Policy.
- Lynn, A.M., and J.T. Slattery (1987). "Morphine Pharmacokinetics in Early Infancy." *Anesthesiology* 66: 136–39.
- Mattson, J.L., J.P.J. Maurissen, R.J. Nolan, and K.A. Brzak (2000). "Lack of Differential Sensitivity to Cholinesterase Inhibition in Fetuses and Neonates Compared to Dams Treated Perinatally with Chlorpyrifos." *Toxicological Sciences* 53: 438–46.
- Moore, R.A., D. Baldwin, et al. (1984). "Sensitive and Specific Morphine Radioimmunoassay with Iodine Label: Pharmacokinetics of Morphine in Man after Intravenous Administration." *Annals of Clinical Biochemistry* 21: 318–25.
- Myres, A.W. (2001). "Child Health: The Past Is Prologue." In *Precautionary Principle and Children's Health*, Workshop Report, Toronto, Ontario (February 19–20, 2001). Network for Environmental Risk Assessment and Management, Institute for Risk Research, University of Waterloo.

- Nahata, M.C., A.W. Miser, and R.H. Reuning (1985). "Variation in Morphine Pharmacokinetics in Children with Cancer." *Developmental Pharmacology and Therapeutics* 8: 182–88.
- National Research Council [NRC] (1993). *Pesticides in the Diets of Infants and Children* Washington, DC: National Academy Press.
- Natural Resources Defense Council [NRDC] (1997). *Our Children at Risk*. <<http://www.nrdc.org/health/kids/ocar/ocarinx.asp>>.
- Newacheck, Paul W., et al. (2003). "Disparities in the Prevalence of Disability between Black and White Children." *Archives of Pediatric Adolescent Medicine* 157: 244–48.
- Organisation for Economic Co-Operation and Development [OECD] (2003). *OECD Health Data 2003*. 2nd ed. Electronic Version.
- Penna, A., and N. Buchanan (1991). "Paracetamol Poisoning in Children and Hepatotoxicity." *British Journal of Clinical Pharmacology* 32: 143–49.
- Pokela, M.L., K.T. Olkkola, et al. (1992). "Pharmacokinetics of Single-dose Oral Ciprofloxacin in Infants and Small children." *Antimicrobial Agents and Chemotherapy* 36: 1086–90.
- Pollution Probe (1998). *The Air Children Breathe: The Effects on Their Health*. Downsvew. <<http://www.pollution-probe.org/Publications/Childrens.htm>>.
- Public Health Policy Advisory Board (1999). "Health and the American Child. Part 1: A Focus on Mortality among Children." *Risks, Trends, and Priorities for the 21st Century*. Washington, DC: Public Health Policy Advisory Board.
- Renwick, A.G. (1998). "Toxicokinetics in Infants and Children in Relation to the ADI and TDI." *Food Additives and Contaminants* 15S: 17–35.
- Ries, L.A.G., M.P. Eisner, et al. (eds) (2001). *SEER Cancer Statistics Review, 1973–1998*. Bethesda, MD: National Cancer Institute.
- Scheuplein R., G. Charnley, and M. Dourson (2002). "Differential Sensitivity of Children and Adults to Chemical Toxicity. I. Biological Basis." *Regulatory Toxicology and Pharmacology* 35: 429–47.
- Sierra Club (2003). *Fact Sheet: The Truth about Pesticides*. <<http://www.sierraclub.ca/national/pest/pesticide-truth.html>>.
- Stanski, D.R., L. Paalzow, and P.O. Edlund (1982). "Morphine Pharmacokinetics: GLC Assay versus Radioimmunoassay." *Journal of Pharmaceutical Science* 71: 314–17.
- Statistics Canada (1997). *Life Expectancy at Birth*. <<http://www.statcan.ca/english/Pgdb/health26.htm>>.
- Tengs, Tammy O., et al. (1995). "Five-Hundred Life-Saving Interventions and Their Cost-Effectiveness." *Risk Analysis* 15, 3: 369–89.
- Tengs, Tammy O., and John D. Graham (1996). "The Opportunity Costs of Haphazard Social Investments in Life-Saving." In R. Hann (ed.), *Risks, Costs, and Lives Saved: Getting Better Results from Regulation* (New York: Oxford University Press).
- Thompson, K.M. (2001). "Navigating the Maze: Federal Activities to Address Children's Environmental Health Risks." In *Children's Environmental Health: What Role for the Federal Government?*: 151–69. Printed for the Senate Committee on Environment and Public Works, 107th Congress, 2nd Session. Senate Report 107-62. Washington, DC: US Government Printing Office.
- United States Department of Health and Human Services [USDHHS] (2003). *The National Children's Study*. <<http://www.nationalchildrensstudy.gov/about/overview.cfm>>.
- United States Environmental Protection Agency [EPA] (1996). *Comparison of the Effects of Chemicals with Combined Perinatal and Adult Exposure vs. Adult Only Exposure in Carcinogenesis Bioassays*. Washington, DC: Office of Research and Development).

- United States Environmental Protection Agency [EPA] (1999). *Guidelines for Carcinogen Risk Assessment*. July 1999 draft. Washington, DC: Risk Assessment Forum.
- United States Environmental Protection Agency [EPA] (2003). *Assessing Cumulative Risk: Revised Organophosphate Cumulative Risk Assessment*. See: <http://www.epa.gov/pesticides/cumulative/>.
- United States National Academy of Sciences/National Research Council [NAS/NRC] (1993). *Pesticides in the Diets of Infants and Children*. Washington, DC: National Academy Press.
- United States Presidential/Congressional Commission on Risk Assessment and Risk Management [CRARM] (1997). *Final Report, Volume 1. Framework for Environmental Health Risk Management*. GPO #055-000-00567-2. Washington, DC: Government Printing Office
- Vandenbergh, H., S. MacLeod, et al. (1983). "Pharmacokinetics of Intravenous Morphine in Balanced Anaesthesia: Studies in Children." *Drug Metabolism Reviews* 14: 887–903.
- Viscusi, William Kip (1994). "Mortality Effects of Regulatory Costs and Policy Evaluation Criteria." *Rand Journal of Economics* 25: 94–109.
- Wildavsky, Aaron (1980). "Richer Is Safer." *The Public Interest* 60: 23–29.
- Wildavsky, Aaron (1991). *Searching for Safety*. New Brunswick, NJ: Transaction.
- Wilson, J. (1977). "Current Status of Teratology; General Principles and Mechanisms Derived from Animal Studies." In J. Wilson and F. Fraser, eds., *Handbook of Teratology; General Principles and Etiology* (New York: Plenum).
- Winter, C.K. (2001) "Pesticide Residues in the Food Supply." In W. Helferich and C.K. Winter, eds., *Food Toxicology* (Boca Raton, FL: CRC Press): 163–85.

Statutes cited

- Canadian Environmental Protection Act* (CEPA), 1999, c. 33.
- Pest Control Products Act* (PCPA), 2002, c. C-8.

About the author & Acknowledgments

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