polluted children, toxic nation

A REPORT ON POLLUTION IN CANADIAN FAMILIES

ENVIRONMENTAL | DEFENCE

June 2006

Acknowledgements

Environmental Defence gratefully acknowledges the generous support of the Laidlaw Foundation for making possible the *Toxic Nation* project.

Laidlaw Foundation

Environmental Defence would also like to thank Dentsu Canada Inc. for its development of the *Toxic Nation* logo and for its strategic advice.

The report was prepared by Environmental Defence, and builds on the groundbreaking work of Environmental Working Group, Washington, D.C. and World Wildlife Fund (UK).

Much of the information provided on children's vulnerability to toxic chemicals has been adapted from the collaborative work of the Canadian Environmental Law Association (CELA) and the Ontario College of Family Physicians (OCFP), and the work of the Canadian Partnership for Children's Health and the Environment (CPCHE). These groups have been instrumental in researching and raising awareness around issues of toxins and children's health.

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Environmental Defence protects the environment and human health. We research. We educate. We go to court when we have to. All in order to ensure clean air, safe food and thriving ecosystems. Nationwide.

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Abbreviations

CDC	US Center for Disease Control and Prevention
CEPA	Canadian Environmental Protection Act
DDT	Dichlorodiphenyltrichloroethane
EPA	US Environmental Protection Agency
NPRI	National Pollution Release Inventory (Canadian)
PAHs	Polycyclic Aromatic Hydrocarbons
PBDEs	Polybrominated Diphenyl Ethers
PCBs	Polychlorinated Biphenyls
PFCs	Perfluorinated Chemicals
PFCAs	Perfluorinated Carboxylic Acids
PFOA	Perfluorooctanoic Acid
PFOS	Perfluorooctane Sulfonate
POPs	Persistent Organic Pollutants
VOCs	Volatile Organic Compounds

Executive Summary

Polluted Children, Toxic Nation: A Report on Pollution in Canadian Families

Polluted Children, Toxic Nation: A Report on Pollution in Canadian Families builds on the findings of Environmental Defence's study *Toxic Nation: A Report on Pollution in Canadians* (2005), and contributes important new information to the growing body of research on the chemical contamination of people. *Polluted Children, Toxic Nation* documents the chemical contamination of children, parents and grandparents from five Canadian families. It is the first study in Canada to assess pollution levels in youth.

Polluted Children, Toxic Nation identifies and quantifies chemicals of concern that are polluting the bodies of Canadians and examines a) what differences, if any, there are between the body burdens of adults and their children, and b) if there are any chemical concentrations detected that indicate an emerging concern for children's health.

For *Polluted Children, Toxic Nation*, children, parents and grandparents from five Canadian families provided blood and urine samples that were tested for 68 toxic chemicals—46 of which were detected. The presence of these chemicals in children as young as 10 raises concerns about what impact they may have on the health of Canadian families today and in the future.

Key Findings

- Laboratory tests detected 46 of the 68 chemicals tested for in 13 family members (six adults and seven children). These 46 chemicals include 5 PBDEs (polybrominated diphenyl ethers), 13 PCBs (polychlorinated biphenyls), 5 PFCs (perfluorinated chemicals), 9 organochlorine pesticides, 4 organophosphate insecticide metabolites, 5 PAHs (polycyclic aromatic hydrocarbons), and 5 heavy metals.

- On average, 32 chemicals were detected in each parent volunteer, and 23 chemicals were detected in each child volunteer.

- In total, 38 carcinogens, 23 hormone disruptors, 12 respiratory toxins, 38 reproductive/developmental toxins, and 19 neurotoxins were detected in the study volunteers. Three chemicals for which there is no data on health effects were detected in the volunteers; all three of these chemicals are PFCs.

- Although PCBs and many organochlorine pesticides were banned before the children in the study were born, these chemicals were detected in all of the children and all of the parents. However, in general, the child volunteers had lower numbers and concentrations of PCBs and organochlorine pesticides than their parents, which suggests that when governments take action to eliminate toxic chemicals, people's toxic load decreases, even if it takes several generations.

- There were several cases where the children were more contaminated than their parents by chemicals that are still in use. The median concentrations for three perfluorinated chemicals (PFOA, PFOS and PFHxS) were higher in children than in the adults. The children also had higher median concentrations of two PBDEs (47 and 153), and a higher median total concentration for the group of PBDEs. The children also had a higher median concentration of DMTP, an organophosphate insecticide metabolite, and they were more polluted by two PAHs (3-OH-chrysene and 3-OH-phenanthrene) than the adults.

Recommendations

Canadians expect their country to be a leader in the protection of human health and the environment. Despite the Canadian government's efforts to control toxic chemicals, the volume of harmful chemicals released into the environment and making their way into Canadians' bodies continues to increase.[1] And now the findings presented in this report reveal in some cases children are even more polluted than their parents.

Canada's pollution problems stem from the weak and ineffective regulation of toxic chemicals under the overarching national toxic chemicals law, the *Canadian Environmental Protection Act* (CEPA). The opportunity exists now to address the shortfalls of this Act during its mandatory five-year review, which began in the fall of 2005 and will continue through to 2007. Environmental Defence is calling upon the federal government to acknowledge the evidence of human contamination revealed in the Toxic Nation studies by taking action to strengthen the regulation of toxic chemicals in Canada.

Environmental Defence recommends that CEPA be amended to:

Establish timelines for the virtual elimination of toxic chemicals:

- Establish aggressive timelines to virtually eliminate carcinogens, respiratory toxins, endocrine disruptors, and reproductive and neurological toxins from use, release, manufacture, disposal and recycling. At a minimum, a 50 per cent reduction in these substances must be achieved by 2010, with virtual elimination being achieved by 2015.

- As a matter of priority, immediately ban PBDEs, PFCs and their precursors, and phthalates.

Make industry accountable for its chemicals:

- Shift the burden of proof onto industry to prove the safety of its chemicals before their introduction to or continued use in the market.

- Mandate industry to adopt a safe substitution policy to replace toxic substances with safer or non-toxic substances.

Regulate toxic chemicals in consumer products:

- Clarify CEPA to regulate toxic chemicals that may be released during the use or disposal of consumer products.

Reduce pollution in the Great Lakes Basin:

- Create a special section of CEPA to focus on Great Lakes protection.

- Provide new funding for a Canadian Great Lakes clean-up of toxic hot spots.

Environmental Defence also urges Canadians to make efforts to reduce their personal exposure to toxic chemicals wherever possible and to pay particular attention to the protection of children. Environmental Defence encourages people to visit the *Toxic Nation* web site, **www.ToxicNation.ca/pledge** and commit to at least five actions that will make a difference.

Living in a Chemical World

For the first time in the history of the world, every human being is now subjected to contact with dangerous chemicals, from the moment of conception until death. (Silent Spring, Carson, p.15)

The publication of Rachel Carson's groundbreaking work, *Silent Spring*, in 1962 brought the chemical contamination of the earth to the forefront of public awareness. Forty years later, our chemical dependence permeates every aspect of our lives and low levels of many toxic chemicals are detectable in Canadians no matter what their age. Most of us are unaware that we are surrounded by harmful chemicals in our homes, at work and at play, and that we carry the legacy of our chemical dependence in our bodies.

Polluted Children, Toxic Nation: A Report on Pollution in Canadian Families is the second study of chemical contamination in the Canadian population conducted by Environmental Defence and builds on the findings in *Toxic Nation: A Report on Pollution in Canadians* released in November 2005. For this study, children, parents and grandparents from five Canadian families provided blood and urine samples that were tested for 68 toxic chemicals– 46 of which were detected. The presence of these chemicals in children as young as 10 raises concerns about what impact they may have on the health of Canadian families today and in the future.

The range of chemicals detected in the family members also indicates that rather than being closer to winning the battle begun by Carson to reduce the production and use of harmful chemicals, the past half century has only created a more alarming situation. In the last 50 years, the global production and use of chemicals has escalated; more than 80,000 new chemicals have been created worldwide. In Canada, over 23,000 chemicals are registered for use in the market, many of which are particularly harmful to children's health, and each year approximately 300 new substances are added to this list.

The contamination of Canadians is the result of laws that permit industry to pollute our air, land and water with vast quantities of toxic chemicals and that fail to address the release of toxic chemicals during the use or disposal of a consumer product. The *Canadian Environmental Protection Act* (CEPA) regulates the manufacture, marketing, use, transport and disposal of toxic chemicals, and it has failed to protect the environment and human health. According to the most recently available data from PollutionWatch, Canadian industrial facilities that are required to report to Environment Canada's National Pollutant Release Inventory (NPRI) collectively released over 4.5 billion kilograms of pollutants in 2003.[2]

The contamination of Canadians is also the result of the failure of industry to re-orient its operations according to pollution prevention principles. A goal of CEPA is to ensure industry actively reduces and eliminates pollution, but since being passed in 1988, only five Pollution Prevention Plans have been developed and none have been implemented.[3] Clearly, when the public interest in a clean environment conflicts with the private interests of polluters, the public interest loses far too often.

Canadians also lose because industry is not required to prove a chemical is safe before it enters the market or ends up in consumer products. Historically, a ban or strict regulation of a toxic chemical has been achieved only after tragic consequences have established a direct link to the chemical's use. It has taken decades, but several pesticides that Carson focused on have been banned or subjected to regulation, at least in industrialized nations. *Silent Spring* documented the tragic effects of the pesticide dichlorodiphenylthrichloroethane (DDT) in 1962, but it took until 1990 for Canada to finally ban this destructive chemical, known to cause birth defects, cancer and a host of other ailments. However, the results of *Polluted Children, Toxic Nation* show that in 2006 the breakdown product of DDT can still be detected in the blood of Canadians as young as 10 and it will undoubtedly take generations to flush this persistent, harmful substance from our bodies.

While the Canadian government has acted to ban DDT and polychlorinated biphenyls (PCBs), and remove lead from gasoline and paint, these are rare examples of government action. Many chemicals that pose risks for human health are not yet regulated, but are in widespread use. Some of the newer groups of chemicals of concern include PBDEs, PFCs, and phthalates. Other chemicals that have been on the market for years, but have yet to be adequately regulated include volatile organic compounds (VOCs), heavy metals (such as lead, mercury, cadmium, etc.), polycyclic aromatic hydrocarbons (PAHs), and a wide range of pesticides, insecticides, fungicides and herbicides.

How We Are Polluted

Sources of Contaminants

Toxic chemicals are found at low levels in countless applications, in everything from personal care products, and cooking pots and pans, to electronics, furniture, clothing, food wrap and building materials. They make their way into our bodies through our food, air and water. Following the chemical trail through a typical day gives you a glimpse of the many sources of contaminants you may encounter.

A good night's sleep under wrinkle-resistant sheets, followed by a breakfast of eggs cooked up in a non-stick pan may expose you to some of the most harmful chemicals in use today:

- The mattress may contain PBDEs, chemicals used in brominated flame retardants which are known to cause cancer and suspected of disrupting hormones, and may also be protected against stains by a product containing PFCs, which are suspected of causing cancer.

- Wrinkle-resistant sheets are treated with formaldehyde, a chemical that is known to cause cancer, and is suspected of causing a host of other illnesses, particularly in the respiratory system.

- Non-stick pans may be another source of exposure to PFCs, in the form of perfluorooctanoic acid (PFOA), a substance used to manufacture non-stick and stain repellent coatings, such as those that can be found on numerous Teflon® products. PFOA has been identified as a likely carcinogen by an expert advisory panel to the US Environmental Protection Agency.[4]

In addition to mattresses, PBDEs are used as flame retardants in furniture, electronics, carpets and curtains. The volume of PBDEs used in the Americas far exceeds the amount used in Europe and to a lesser degree that of Asia.[5]
PFCs also have many uses beyond non-stick cookware. They are used for both their stain repellent and non-stick properties, for example, on clothing, upholstered furniture, bedding, carpets and fast food wrap, as well as in microwave popcorn bags, nail polish and windshield washer fluid.

Classes of Chemicals

Individual chemicals can be categorized according to the larger class of chemicals to which they belong. Chemicals that belong to the same class have similar chemical structures and/or environmental properties. Chemical classes of particular concern for human health include heavy metals, PCBs, PBDEs, organochlorine pesticides, organophosphate insecticides, PFCs, PAHs, dioxins and furans, and VOCs. For detailed information on many of these classes of chemicals, please see page18.

Table 1. Market Demand of PBDEs in 1999 (BSEF 2003)

Commercial product	Americas[a]		Europe[b]		Asia[c]	
	Market demand	Estimated consumption (tonnes)	Market demand	Estimated consumption (tonnes)	Market demand	Estimated consumption (tonnes)
DBDE	44%	24 500	13%	7 600	43%	24 050
OBDE	40%	1 500	16%	610	44%	1 680
PeBDE	95%	7 100	2%	150	3%	250

[a] All countries in North, South and Central America were included.
[b] All countries in Eastern and Western Europe were included.
[c] Australia, New Zealand and the Indian subcontinent were included.

Getting ready for school or work takes on a more sinister aspect when you consider that some of the chemicals used in your personal care products include:

- parabens (used as preservatives, known to disrupt hormones and suspected of causing cancer);
- phthalates (used to enhance fragrances, suspected of disrupting hormones);
- fragrances (a term which can indicate up to 4,000 separate petroleum based chemicals that can affect the central nervous system, trigger asthma and cause cancer);
- talc (used as a dusting or drying powder and known to cause cancer); and,
- diethanolamine (DEA) (used as a foaming and emulsifying ingredient, suspected of causing cancer and being toxic to the respiratory and nervous systems).

It has been reported, for example, that "by the time the average woman grabs her morning coffee, she has spritzed, sprayed and lathered with 126 different chemicals in nine different products".[6] Many of these chemicals are also common in household cleaning products, which can contain even harsher chemicals that carry warnings about their corrosive and poisonous nature.

Personal care products also emit VOCs, which are found in carpets, paints, adhesives, pressed-wood furniture and cleaning products as well. In fact, according to Environment Canada, VOC emissions from consumer and commercial products are the second largest contributors to overall emissions of VOCs and are expected to replace emissions from transportation as the largest source of VOCs released by people in Canada by 2010.[6] Most VOCs are recognized carcinogens and suspected hormone disruptors, and toxic to the respiratory and reproductive/developmental systems.

In the workplace you may be exposed to a similar variety of harmful chemicals that can be found in furnishings, computers, cleaning products, etc. Depending on your occupation, you may have a higher daily exposure, particularly if you work in chemicals manufacturing or directly with pesticides. Similarly, children at school or daycare are exposed to a host of chemicals, particularly VOCs from furnishings, carpeting, art supplies and cleaning products. Lead has also been found in children's PVC toys and jewellery. A more detailed discussion of the vulnerability of children follows on page 7.

When you sit down to a meal, you ingest low doses of heavy metals, pesticides and other persistent organic pollutants (POPs). In fact, heavy metals and POPs have become so ubiquitous in the environment that all food is contaminated to some degree, no matter where it is grown or harvested. Eating an organic diet reduces your exposure to pesticides.
Also, many food and beverage containers contain harmful chemicals that can migrate into food. Plastic containers with the recycling #3 are made of PVC (polyvinyl chloride), which contains phthalate additives. Polycarbonate plastic containers (recycling #7) contain bisphenol A; recent research has shown that this chemical is an estrogenic hormone disruptor that can cause reproductive damage and birth defects that may lead to prostate and breast cancer in adulthood.[9] Bisphenol A is also found in epoxy resins used in plastic food wrap and in the plastic lining in canned food.

> 94% to 99% of our exposure to persistent organic pollutants, such as PCBs, comes from diet, particularly from the consumption of breast milk, fish, fatty meats and dairy products.[8]

These examples of potential exposures to low levels of harmful chemicals offer a glimpse of what living in a chemical world means. They serve to emphasize the fact that toxic chemicals permeate every aspect of our lives and, as the Toxic Nation studies demonstrate, our bodies as well.

Environmental Media

Contaminants that may be found throughout the home, workplace or school are carried in environmental media such as air, water, soil, dust, and food. When mercury, for example, is emitted from a coal-fired power plant it attaches to particles and is carried in air until it falls to the ground in raindrops, dust, or through the force of gravity (known as 'air deposition'). It can then end up in water bodies and in soil, at which point it begins to circulate through the food chain. Household dust is a significant environmental media for the transportation of indoor pollutants. In a study conducted by Greenpeace in the UK, household dust samples tested positive for chemicals such as phthalates, brominated flame retardants

and organotin compounds.[10] In another study, researchers found that the inadvertent ingestion of house dust is the main exposure pathway for PBDEs.[11] Therefore, vacumning and dusting your home regularly is important in reducing your exposure to toxins.

Routes of Exposure

Chemicals are transferred to, or absorbed by, your body through your lungs, digestive system, and skin. Chemical uptake occurs through inhalation, ingestion and dermal (skin) contact. For instance, the main route of exposure for chemicals carried in food is your stomach, where chemicals are absorbed through digestion. Chemicals carried in air can enter your body through inhalation and skin contact. Contaminants in water and soil are absorbed through all three main routes of exposure.

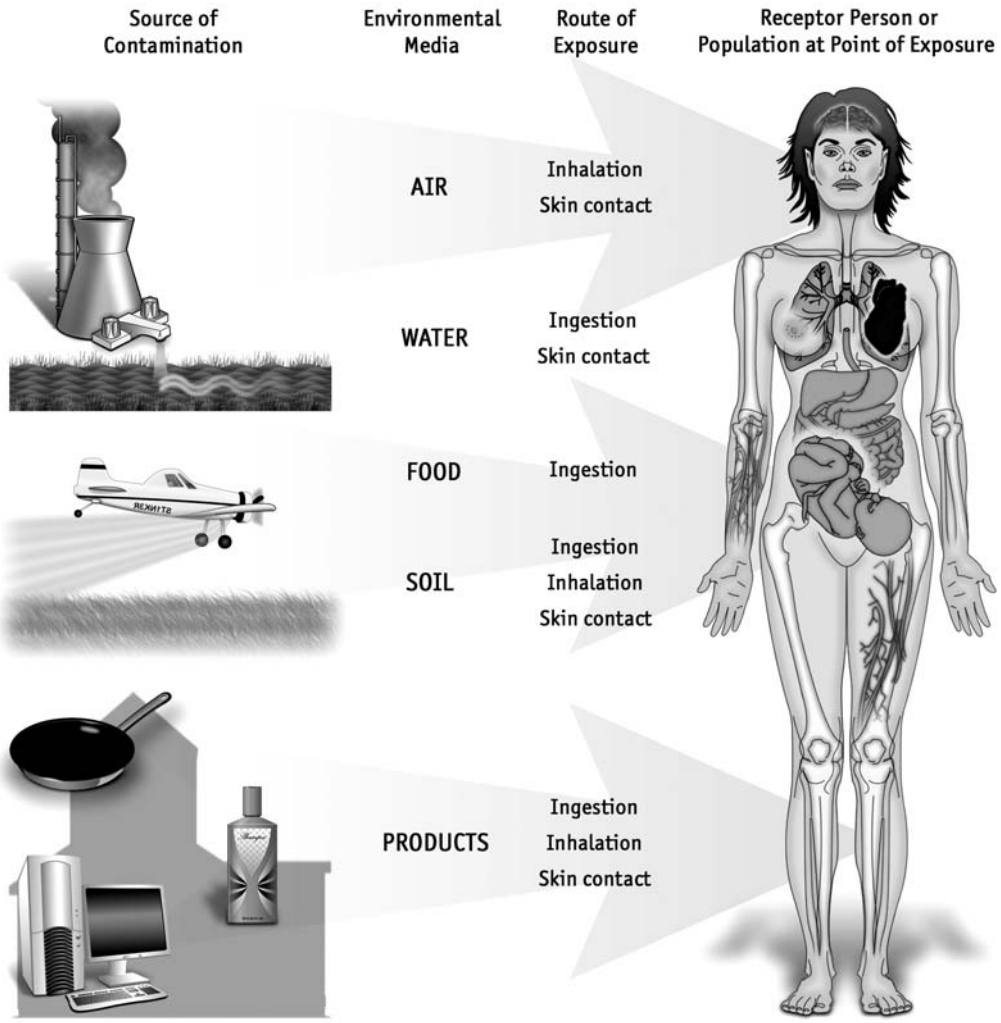


Figure 1. Human exposure to toxic chemicals

By the end of the day you have likely been exposed to low levels of hundreds of toxic chemicals. It is impossible to say how many or in what combination, but as the evidence in the Toxic Nation studies show, your body is living testimony to the chemical world we inhabit. It is time to take up the challenge that Rachel Carson issued in 1962, *"we should no longer accept the counsel of those who tell us that we must fill our world with poisonous chemicals; we should look about and see what other course is open to us."* [12]

Children's Vulnerability

A new course of action is urgently needed as we learn more about the evidence of multiple low level exposures in children. Children are more vulnerable than adults to negative health effects from environmental exposures due to their physiology and behaviour. Because children's bodies and physiological systems undergo substantial growth and development from conception through adolescence, they are particularly sensitive to chemical interference. As the most vulnerable members of society, children require a higher level of protection from their parents, caregivers, and policy makers.

Physiology, Development and Uptake

Certain physiological differences and vulnerabilities at different stages of development result in a greater uptake of chemicals by children. Per kilogram of body weight, children eat more food than adults, drink more water and breathe more air, all of which results in a proportionally greater uptake of pollutants.[13] In comparison, children aged one to 10 drink 35.5 mL of water/kg/day, while adults drink 19.9 mL water/kg/day; children aged three to five years eat 5.8 g of fruit/kg/day, while adults eat 1.3 g of fruit/kg/day.[14] If a child and an adult each eat an apple, they may ingest similar amouts of contaminants. However, the child receives a proportionally greater amount due to her lower body weight. Children also breathe more rapidly and exchange more air per kilogram of body weight, which results in a greater uptake of air pollutants. Children also tend to be more physically active than adults, and exercise enhances the uptake of air and air pollutants.[15]

Depending on age, the barriers that keep chemicals from entering the body, and the physiological mechanisms that usually protect the body from chemicals that do invade, may be undeveloped:

- *In utero*, the embryo and fetus are generally defenceless against chemicals that invade the mother's body and cross the placenta.

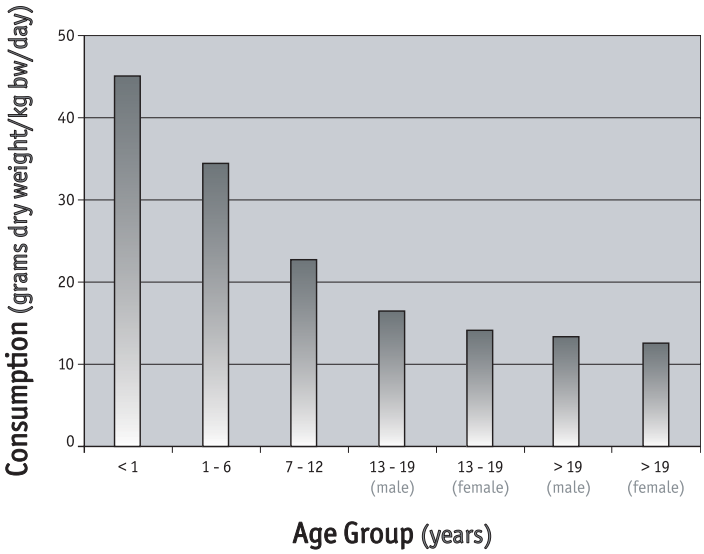


Figure 2. Average food consumption by age
Source: Canadian Partnership for Children's Health and the Environment. (2005) p.20.

- The blood-brain barrier, which partly protects the adult brain from toxic substances, is not fully developed until about six months after birth.[16]

- In the fetus and newborn, many metabolic systems that transform absorbed contaminants are not functioning at full capacity, and renal excretion takes the first six months of life to develop.[17]

- Up until about one year, the digestive tract, skin and lungs are extremely permeable and readily absorb substances.[18]

- Also, children absorb substances differently than adults; for example, it has been estimated that adults absorb 10 to 15 per cent of lead ingested with meals, but children and pregnant women can absorb up to 50 per cent.[19]

Developmental Vulnerability

At different stages of growth and development, particular immature organs and body systems are more susceptible to interference from environmental contaminants. By far the most vulnera-

ble time in a child's development is the first nine months from conception to birth, during which time environmental exposures may lead to anatomical abnormalities and physiological defects.[20]

- In utero, during both the embryonic and fetal stages, major organs, body structures and the nervous and reproductive systems are formed.[21]
- Up to the age of one, body structures experience rapid growth.[22]
- The body continues to grow and develop throughout the toddler years and childhood.[23]
- The brain and nervous system experience extensive growth after birth and are not fully developed until about the age of 10 or 12. The extended development phase of the brain and nervous system mean that they are especially vulnerable and have a broad window of susceptibility. Unlike other organs, the brain cannot readily repair cells after they have been damaged.[24]

The beginning of adolescence varies individually and represents the final stage of development between childhood and adulthood. Many important developments occur during this period:

- Reproductive tissues and structures (e.g. breasts, uterus, vagina, penis, scrotum, testicles) develop into their mature state, thereby making reproduction possible. Sperm production begins in males.
- Rapid skeletal and muscular growth occurs as young people reach their adult body size.[25]
- Higher brain functions, such as abstract thought, are achieved.[26]
- Myelination (the formation of the myelin sheath, which protects nerves and facilitates the transmission of nerve impulses) continues and ceases during adolescence.[27]

Tissues that have rapid turnover throughout life (i.e. blood, skin, sperm) are vulnerable targets for exposures at any stage in life.[28]

Exposure Through Behaviour

Regular childhood behaviour places children in closer contact with potential sources of contamination, mainly because of their exploratory nature, frequent hand-to-mouth activity, proximity to the ground, food preferences and inability to recognize hazards.

Children explore their surroundings by touching and tasting whatever they can get their hands on. Their frequent hand-to-mouth activity, particularly in the infant stage, can lead to the direct ingestion of contaminant residues in indoor dust, soil and on products such as toys.[29]

Children spend much more of their time at ground-level than adults. During the crawling stage, children are in frequent contact with soil, lawns, carpets and other floor surfaces, which have been shown to harbour chemical contaminants that can be transferred to the child through contact with skin, inhalation and digestion. Throughout childhood, a child's height also can make them vulnerable to inhaling air pollutants that concentrate at lower levels. The concentration of pesticides, for instance, has been shown to be much higher in the breathing zone of a child than of an adult (Figure 3).[30]

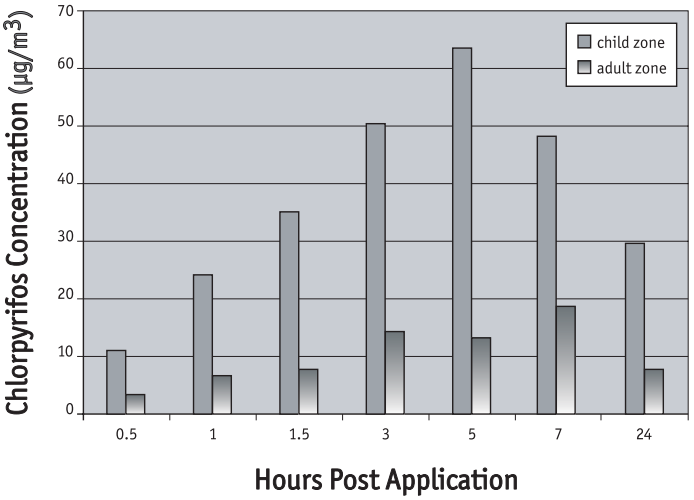


Figure 3.Pesticide air concentrations in child and adult breathing zones in a ventilated room
Source: Canadian Partnership for Children's Health and the Environment (2005) p.21

Children's exposure to pollutants is also affected by their diet. Breast milk, the main source of nourishment for many infants, is known to contain numerous persistent organic pollutants that bioaccumulate in fatty tissues, including PCBs, PBDEs, pesticides and heavy metals. However, while this source of contaminants during one of the most sensitive development phases is cause for concern, the other benefits of breast feeding (i.e. the provision of nutrients and antibodies for the immune system) still outweigh the risks, particularly since choosing an alternative food source does not ensure the child will not be exposed to contaminants.

Young children are also often unable to recognize hazards because they cannot read warning signs and labels, and do not understand the danger or need to avoid exposures. For these reasons, toxic products are kept out of the reach of children and when in use children rely on adult supervision to protect them.[31] Ironically, while parents are generally aware of the need to protect children from obvious hazards (such as bottles of Drano® or bleach), they unwittingly contribute to their child's exposure through the products they purchase and use in the home. PBDEs, for example, have been applied to mattresses, couches curtains and computers as flame retardants and without being aware of the dangers of PBDEs, millions of parents have introduced them into their home simply by purchasing new furnishings. Treating your kids to microwave popcorn, or fast food, means that they are likely ingesting PFCs as well since this non-stick chemical coats the inside of microwave popcorn bags and fast food wrap. In the absence of government regulations that restrict the use of hazardous substances, the responsibility of researching the full list of substances in a product and identifying potentially hazardous substances is unfairly left to parents or caregivers.

As a child grows and their behaviour changes, the main sources of potential exposure to toxic chemicals change as well. While the hand-to-mouth activity of toddlers may put them at risk of ingesting contaminants from objects or dust, adolescents are more vulnerable due to the products they may start using now that they are young adults. Teenagers begin to use more types of personal care products and use them more frequently, such as deodorant, shampoo, shaving creams, hair dyes and moisturizers. Most girls, for example, start experimenting with make-up and thereby open up a whole new range of exposures from these products. Older children and adolescents also spend much more time unsupervised, tend to be more adventurous and are prone to risky behaviour that can expose them to pollutants. When adolescents enter the workforce, they can also experience occupational exposures to contaminants.

Additional Factors that Affect Exposure and Vulnerability

Children's exposure and vulnerability to harmful chemicals are affected by additional factors, including genetic susceptibility, socioeconomic, nutritional and cultural factors.[32]

Genetic Susceptibility

Individual genetic and biological differences can affect the degree to which children are affected by exposures to harmful chemicals. Genes regulate growth, development, metabolism, and replication and repair at the organ, cellular and DNA levels, all of which can affect the impact of environmental exposures.[33] For example, genes that code for particular enzymes can affect the way toxins, such as lead and pesticides, act in the body, and make individuals with particular gene characteristics more susceptible to those exposures.[34] Certain conditions, such as asthma, can also make a individual more susceptible to adverse health effects from exposure to air pollutants.[35]

Socioeconomic and Nutritional Factors

Socioeconomic factors, particularly household income, have been associated with differences in the likelihood for exposure to toxins, as well as differences in susceptibility to harmful effects from those exposures. Children in low-income households are more likely to live in substandard housing that often contains more sources of environmental pollutants (Figure 4).[36] Contaminants such as lead and asbestos are more common in substandard housing. Lead, specifically, is often found in older homes that were once painted with lead-based paints. Parents in low-income households may not have the resources to contain or repair flaking lead paint on walls. Landlords that allow homes occupied by low-income tenants to fall into disrepair also contribute to higher lead exposures in poorer children.[37] Rental and low-income units with frequent turnover in occupancy are also more prone to cockroach infestations, and as a result are more frequently sprayed with pesticides.[38] Older furnishings, especially carpets, in homes of low-income families may contain higher concentrations of lead, pesticides and other contaminants.[39]

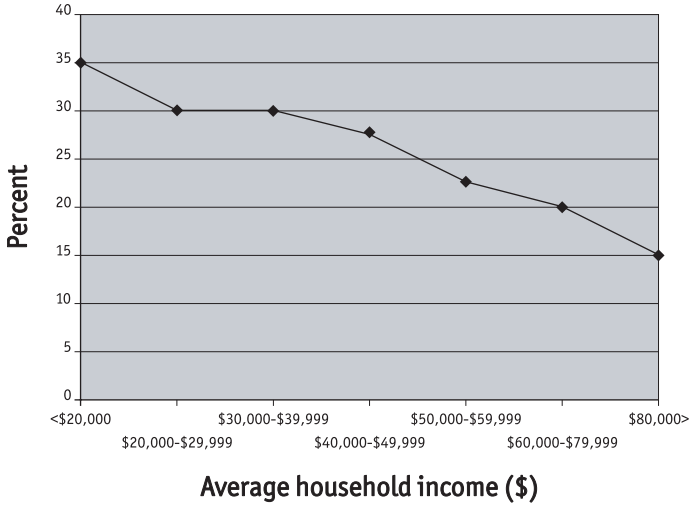


Figure 4. Children living in substandard housing and household income
Source: Ross, D. P. and P. Roberts (1999).
Note - Two-parent family aged 4-11 years

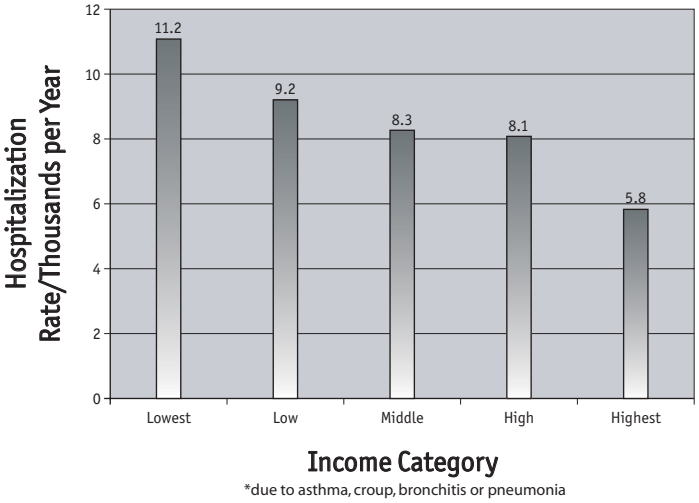


Figure 5. Respiratory hospitalization, children 0 to14, by income category, Toronto 1996-1999.
Source: Toronto Public Health (2005) p.47.

Low-income neighbourhoods are more often closer to sources of pollution, such as high traffic roadways, industrial facilities and hazardous waste disposal facilities.[40] Canadian data show that the health risks associated with close proximity to these sources of pollution are more likely to affect low-income areas, minority and Aboriginal communities (including reserves).[41] For instance, data from Toronto has shown that children from low-income areas are nearly twice as likely to be hospitalized for respiratory illnesses than children from higher income areas (Figure 5).

Inadequate nutrition is a key condition that predisposes children of lower income households to greater health risks resulting from exposure to environmental contaminants. Poor nutrition can include deficiencies in protein, calcium and iron, which can compound effects from exposures to toxic substances.[42] Poor nutrition can lead to a greater uptake of contaminants, and impair the body's capacity to deal with that exposure.[43] Even in cases where low-income households are able to provide good nutrition, they may not be able to afford organic food items. Compared to higher-income households who are able to afford organic food, members of low-income households may be more likely to experience exposures to pesticides through their diet.

Cultural Factors

Various groups within Canada's diverse population may be more vulnerable to environmental exposures and their effects on health. Two main factors related to diversity that affect children's environmental health are the influence of culture on dietary intake, and the socioeconomic challenges faced by visible minorities and new immigrants.

Particularly in the case of First Nation communities, cultural influence on diet can lead to an increase in exposure to harmful pollutants. The consumption of fish, marine mammals and wild game has cultural, spiritual and nutritional significance within the First Nation diet. Unfortunately, these food sources tend to have much higher concentrations of mercury and other persistent organic pollutants, such as PCBs, pesticides and PBDEs. Toxins build up in the fatty tissues of fish, marine mammals and other food sources through the process of bioaccumulation; in addition, many transboundary pollutants accumulate in the North (where many First Nation communities are located) due to air and water currents and climate conditions.

First Nations, other visible minorities and new immigrants also often face societal prejudice and discrimination, which affects

their access to education and employment, and as a result affects their socio-economic status.[44] Of the approximately one in six children in Canada who live in poverty, children of First Nation ancestry, those who represent a visible minority or are recent immigrants are disproportionately represented. For example, in Toronto, 47 per cent of First Nation children, 40 per cent of children who belong to a visible minority, and 54 per cent of children who are recent immigrants live in poverty, whereas 25 per cent of children born in Canada live in poverty.[45] As noted above, exposure to contaminants poses additional health risks for children from low-income households.

Types of Potential Health Effects

Only yesterday mankind lived in fear of the scourges of smallpox, cholera, and plague that once swept nations before them... Today we are concerned with a different kind of hazard that lurks in our environment.... (Silent Spring, Carson, p.187-188)

Chemicals can be grouped according to their effects on health. The categories of major chemical–health effects include respiratory toxins, neurotoxins, hormone disruptors, reproductive/developmental toxins, and carcinogens. Chemicals can also be toxic to the immune system, the kidneys, the gastrointestinal system and liver, skin and sense organs, the musculoskeletal system, and the cardiovascular system.

Respiratory Toxins

Respiratory toxins affect the breathing system. When these toxins are inhaled they affect the nasal passages, pharynx, trachea, bronchi, and lungs. These toxins cause both acute and chronic illnesses such as bronchitis, pulmonary fibrosis, emphysema, cancer, and general breathing problems. As irritants, respiratory toxins can also increase the severity and incidence of respiratory infections and can aggravate asthma.

Examples of known or suspected respiratory toxins: PAHs, components of smog (VOCs, nitrogen oxides, sulphur dioxide, particulate matter, carbon monoxide and ground-level ozone), pesticides and insecticides.

Neurotoxins

Neurotoxins cause damage to the brain and nervous system, and can lead to developmental and behavioural disabilities. Children are particularly vulnerable to neurotoxins because their developing brains are susceptible to chemical interference. Exposure to neurotoxins has been linked to several effects on brain development and functioning, including intellectual deficits, which reveal themselves in the form of lower school performance, IQ deficits, lower scores on aptitude tests and other cognitive and motor deficits; learning disabilities (such as dyslexia); autism spectrum disorders; Attention Deficit Hyperactivity Disorder (ADHD); visual or hearing deficits; behaviour problems (including the inclination towards violence); and altered thyroid function (which impacts brain development).[46] While neurotoxins particularly affect the child's developing brain and nervous system, the negative impacts of exposure to neurotoxins last through adulthood.

Examples of chemicals that are known or suspected neurotoxins: organophosphate insecticides, organochlorine pesticides, heavy metals (i.e. lead, mercury, arsenic, cadmium, manganese), VOCs, PCB and PBDEs.

Hormone Disruptors

Hormone disruptors (also known as 'endocrine disruptors') affect the endocrine system, which includes the body's hormone-producing glands–the pituitary gland and hypothalamus located in the brain, the adrenal glands on top of the kidneys, the female ovaries and male testicles, the pancreas in the abdomen, and the parathyroid and thyroid glands in the neck.[47] Hormones that are released from glands act as messengers that evoke a specific response in other cells throughout the body. These hormones regulate almost every cell, organ, and function of our bodies, including reproduction, metabolism, regulation of nutrients and minerals, body temperature, moods, the immune system, growth and development.

Hormone disrupting chemicals are structurally similar to natural hormones, and trick the body by either mimicking or blocking normal hormonal functions. Exposure to endocrine disrupting chemicals can cause a variety of health effects including adverse pregnancy outcomes (still births, changes in sex ratio, i.e. fewer male babies), male birth defects (undescended testes and hypospadius), decreased sperm count and quality, early onset of menstruation and puberty, neurobehavioural effects (resulting from altered thyroid hormone function *in utero*), endocrine-mediated immunotoxicity, and cancer promotion at endocrine sites (breast, endometrial, testes, prostate, and thyroid).[48]

Examples of chemicals suspected or known to disrupt hormones: heavy metals, PCBs, organochlorine pesticides, dioxins and furans, phthalates, bisphenol A, and PBDEs.

Reproductive and Developmental Toxins

Human reproduction and the development of the embryo, fetus and child are closely linked processes, and for this reason reproductive and developmental toxins are often considered together as a chemical-health effect group. Human reproduction involves the production, release and fertilization of gametes (sperm and ova). Child development encompasses the embryonic, fetal, infancy, toddler, childhood and adolescent phases (please see page 7 for more details on child development stages).

Reproductive toxins can affect reproductive ability and sexual function. Examples of reproductive disorders in women include endometriosis, failure to ovulate normally, tubal pregnancies, miscarriages, and still births. Male reproductive disorders include testicular cancer, low sperm count and motility, undescended testes and hypospadius-these four disorders have been identified as symptoms of one overarching disorder called testicular dysgenesis syndrome.[49] Reproductive disorders in both men and women can be caused by their parents' chemical exposures, and exposures in the womb and throughout childhood and adulthood.[50] If a parent is exposed to reproductive toxins, damage can occur to the cellular DNA of their gametes, which can cause genetic mutations inherited by the offspring. Males continually produce sperm from puberty throughout adulthood, and as such their sperm is susceptible to chemical interference throughout this timeframe. Females, on the other hand, produce their lifetime supply of eggs while they are a fetus, and therefore their gametes are only susceptible to chemical interference while they are in their mother's womb.[51]

Parental reproductive health and the developmental health of the embryo and fetus overlap to the extent that a reproductive toxin affects the outcome of a pregnancy (i.e causing a still birth or birth defects).

Developmental toxicity in a fetus can occur due to preconception and prenatal exposures to toxins of both the mother and father. Developmental toxicity after birth can occur from exposures that happen from infancy through adolescence. Developmental toxins can cause spontaneous abortion, stillbirth, low birth weight, birth defects, and behavioural and intellectual deficits that become apparent in later childhood. Developmental toxins have particular 'windows of opportunity' throughout a child's development, during which they can exert a negative effect. For instance, exposure to a developmental toxin during early pregnancy, when the child's limbs and organs are forming, can have particularly negative consequences. Developmental defects in the reproductive system of a child can affect their fertility in adulthood and the development of their own offspring as the cycle of reproduction continues.

Examples of chemicals known or suspected to be reproductive and/or developmental toxins: mercury, lead, PCBs, organic solvents, and pesticides.

Carcinogens

Carcinogenic chemicals can cause or aggravate cancer, which is the growth of abnormal cells that spread throughout the body, in some cases leading to death. In Canada, cancer is the leading cause of death, and it is now expected that 1 in 2.3 men and 1 in 2.6 women will have cancer in their lifetime. Between 1977 and 2006, the age-standardized incidence rate for cancer increased by 16.7 per cent in females, and 15 per cent in males.[52] During the same timeframe, the age-standardized incidence rates for breast cancer and prostate cancer, the most common forms of cancer in women and men respectively, increased by 25.9 per cent (breast) and 75 per cent (prostate).[53] Lung cancer remains the leading

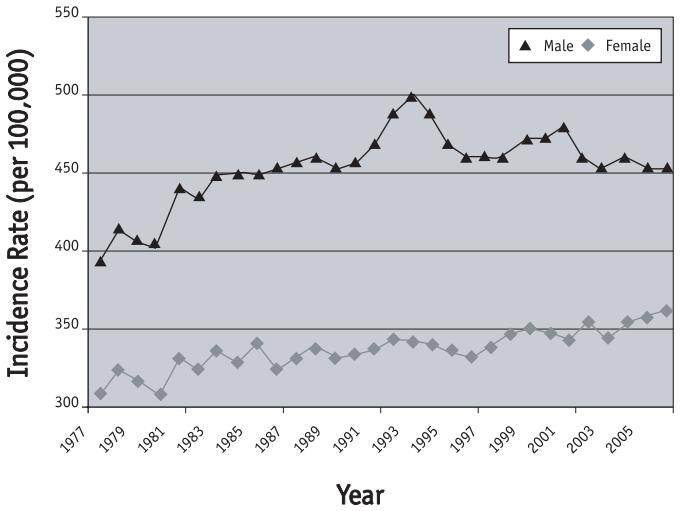


Figure 6. Trends in age-standardized incidence rates for cancer in Canadian adult males and females (1977-2006).
Source: Canadian Cancer Society/National Cancer Institute of Canada (2006)

cause of cancer death in both women and men.[54] In younger adults (aged 20 to 44 years), significant increases have been observed for non-Hodgkin's lymphoma and thyroid, lung and testicular cancer.[55]

Cancer is the most common cause of death by disease in Canadian children.[56] The most common form of cancer in children is leukemia, followed by cancers of the spinal cord and brain.[57] In children, exposure to carcinogens in the womb during rapid fetal cell division contributes the greatest risk to developing cancer.[58]

Some sources indicate that the incidence of childhood cancer has increased by 25 per cent since the 1970s.[59] The small population size in Canada, coupled with the rarity of childhood cancer, makes it difficult to use cancer statistics to establish trends in childhood cancer.[60] However, childhood cancer statistics on the much larg-

Table 2. Health statistics

Ailment	Statistics
Asthma	1.5 million Canadians[63]
	12% of Canadian Children[64]
	Four-fold increase in Canadian children since the 1980s[65]
Childhood Cancer	1,285 Canadian children diagnosed with cancer each year[66]
	227 Canadian children die of cancer each year[67]
	21% increase in childhood cancer in U.S. (1975-1998)[68,69]
	Approximately 20% increase in childhood cancer in Europe (1970s-1990s)[70]
Cancer in Canadian Adults (All cancer incidence rates are age-standardized.)	16.7 % increase in women(1977-2006)[71]
	15% increase in men (1977-2006)[72]
	25.9% increase in breast cancer (1977-2006)[73]
	75% increase in prostate cancer (1977-2006)[74]
Learning Disabilities	10% of Canadians[75]
Behaviour Problems	17-21% of Canadian children[76]
Birth Defects	Leading cause of infant mortality[77] 2%-3% of Canadian babies[78]
Infertility	10% of Canadian adults[79]
Miscarriages	15% of pregnancies in Canada[80]

er populations of both the US and Europe show a clear and similar upward trend of an approximately 20 per cent increase in the incidence of childhood cancer since the 1970s. Researchers in Europe found that between the 1970s and 1990s the incidence of cancer among children and adolescents increased by 1 per cent and 1.5 per cent per year, respectively.[61] In the US, the incidence of childhood cancer increased nearly 21 per cent between 1975 and 1998, or about one per cent per year over two decades.[62]

Examples of chemicals known and suspected of causing cancer: heavy metals, PCBs, organochlorine pesticides, organophosphate insecticides, PBDEs, and PFCs.

Risks of Low Dose Contamination

Like the constant dripping of water that in turn wears away the hardest stone, this birth-to-death contact with dangerous chemicals may in the end prove disastrous. Each of these recurrent exposures, no matter how slight, contributes to the progressive buildup of chemicals in our bodies and so to cumulative poisoning.
(Silent Spring, Carson, p.173)

Biomonitoring of the general population reveals that individuals carry a body burden of low concentrations of numerous toxic chemicals. For most chemicals much more is known about human health effects from acute, high dose poisonings[81], but that is not how most people are exposed to toxic chemicals. The prevalence of chemical use and the ubiquitous nature of many pollutants mean that people are exposed to low doses of multiple chemicals everyday of their life. Unfortunately, very few studies have examined the health effects of multiple low level exposures to toxic chemicals over a lifetime, and moreover, conducting studies that mimic real life exposures is extremely difficult. **As a result, chemicals on the market today have been identified as 'safe' based on *lack* of proof of harm, rather than on the basis of rigorous scientific proof of safety.**

Body burden refers to the amount of a chemical, or a number of chemicals (especially potential toxins), stored in the body at a given time. *Biomonitoring* is a scientific technique for assessing human exposures to chemicals by sampling and analyzing a person's tissues and fluids.

High Dose vs. Low Dose

We are accustomed to look for the gross and immediate effect and to ignore all else. Unless this appears promptly and in such obvious form that it cannot be ignored, we deny the existence of hazard...
(Silent Spring, Carson, p.190)

In *Body Burden: The Pollution in People*, a study conducted by Environmental Working Group which is based in Washington D.C., lead author Jane Houlihan provides a detailed discussion of the significance of low dose exposures for human health, a summary of which is provided here.

Historically, scientific studies on the health effects of chemicals involved feeding high doses of a single chemical to laboratory animals. Results from these studies have led to the false assumption that only a high dose of a chemical will negatively affect human health. There are several problems with this assumption, beginning with the fact that, by its very nature, a high dose test does not involve a test for health effects at low levels.

First and foremost, a high dose test will not necessarily reveal all the toxic properties of the chemical being studied. It is incorrect, therefore, to assume that a high dose study will accurately predict low dose toxicity. Chemicals produce a range of health effects that can vary with dose, and affect the target organ in different ways.[82] For example, some chemicals produce opposite effects at high and low levels, a phenomena that is referred to as biphasic dose response. Other chemicals produce different effects at high and low doses, and some produce effects at low doses but not at high ones.[83] Pyrethroid insecticides, for example, induce hyperactivity in rats at doses up to 0.7 mg/kg, but not at a dose 60 times higher (42 mg/kg). In prostate cancer cells, studies have shown that bisphenol A increases cell proliferation at concentrations 100 times lower than the levels that inhibit cell growth.[84] In addition, factors such as the age and genetic vulnerability of the exposed individual will also affect the impact of an exposure. When it comes to a chemical's effect on health, it turns out that 'the devil is in the details', which illustrates that the old adage 'the dose makes the poison' is an oversimplification of the way chemicals act in our bodies.

There are several differences between traditional high dose studies and more recent studies that have revealed adverse health effects at low doses. In general, traditional high dose studies have focused on obvious measures of toxicity, such as cancer and birth defects, and in general these studies have been based on adult subjects.[85] Recent studies on low dose health effects are often concerned with measuring more subtle, but critical, changes in physiological functions, such as immune function, enzyme activity, hormone levels, cellular changes in tissues, etc.[86] These studies often focus on the effects of low dose exposures

during critical periods of fetal development or infancy–effects, which often do not surface until later in life.[87] As children grow they are particularly vulnerable to chemicals that can interfere with developmental outcomes. As a result, exposures that cause no adverse health effects in adults can cause illness, disease and malformation in children. Given the particular vulnerabilities of children, it is especially important that toxicity studies test for chemicals' effects on the child at all stages of development, from conception to adolescence. For more information on the particular vulnerability of fetuses, infants and children please refer to pages 7 to 9.

Documenting Health Effects at Low Doses

Houlihan provides an overview of peer-reviewed scientific studies that have documented adverse health effects in people resulting from: a) low dose exposures, b) exposures at levels below those considered to be safe based on high dose testing, and c) exposure to multiple chemicals at low doses considered to be safe. Adverse health effects, such as neurological, developmental and behavioural problems, changes in sex ratio, low birth weight, and miscarriage have resulted from exposures to PCBs, dioxin, lead, methylmercury and pesticides at low doses that occur in the general population.[88] Scientific studies have documented adverse health effects resulting from exposure to bisphenol A at levels 2,500 times lower than the US Environmental Protection Agency's (EPA) 'lowest observed effect' dose.[89] In two rare studies on the effects of multiple low dose exposures, scientists dosed laboratory animals with a mixture of 16 organochlorine chemicals, lead and cadmium, each applied at its individual regulatory 'safe' dose, and found that the animals developed impaired immune response and altered thyroid function.[90]

The Evolving State of Scientific Knowledge and Safety Thresholds

Scientific 'facts' are not static; the more scientists study a subject, the more they learn. In the case of chemicals, there is much we do not yet know, but continued scientific study is revealing that even low level exposures that were previously considered safe can adversely affect human health. The observance of effects on health from low dose exposure is apparent both in scientific studies, and in the safety thresholds established by regulatory agencies. The examples of mercury and lead illustrate the uncertainty of safety levels; for both of these chemicals, the levels considered to be safe have been revised and lowered repeatedly throughout the years. Adding to the uncertainty, respected regulatory agencies, such as the World Health Organization (WHO), the US EPA and Health Canada, may concurrently establish different 'safe' levels for the same chemical.

Methylmercury is the form of mercury that most readily bioaccumulates in living organisms, including people, and it has been shown to cause several adverse health effects. In 2003, the World Health Organization revised the provisional tolerable weekly intake (PTWI) for methylmercury from 0.47 µg/kg body weight/day down to 0.23 µg/kg body weight/day. The US Environmental Protection Agency's current Reference Dose (RfD) for methylmercury is 0.1 µg/kg body weight/day, but in 1996 it was 0.3 µg/kg body weight/day. Health Canada's safe level for methylmercury is 0.3 µg/kg body weight/day. It is not unrealistic to suggest that these 'safe' levels may further be revised, and that, as in the case of lead, it may be found that there is actually no safe level.

The blood lead level considered to be safe by public health agencies around the world has been significantly lowered repeatedly since the 1960s when scientific studies began revealing adverse health effects, particularly in children, at lower and lower levels. Findings of adverse effects at low lead levels have resulted in the phasing out of many uses of lead, such as in gasoline and paint. In 1960, the US Centre for Disease Control (CDC) had established that lead poisoning in children occurred at a blood lead level of 60 ug/dL. In 1985, the lead poisoning level was lowered to 25 ug/dL, and in 1991 it was lowered again to 10 ug/dL.[91] Currently, even though a blood lead level of 10 ug/dL or greater is widely recognized as elevated, it has been demonstrated that adverse health effects can occur at concentrations even below 1 ug/dL. Thus, there is no safe concentration of lead in blood.[92] In Canada, and other jurisdictions, the blood lead level of 0.48 µmol/L is the intervention level adopted by government agencies.[93]

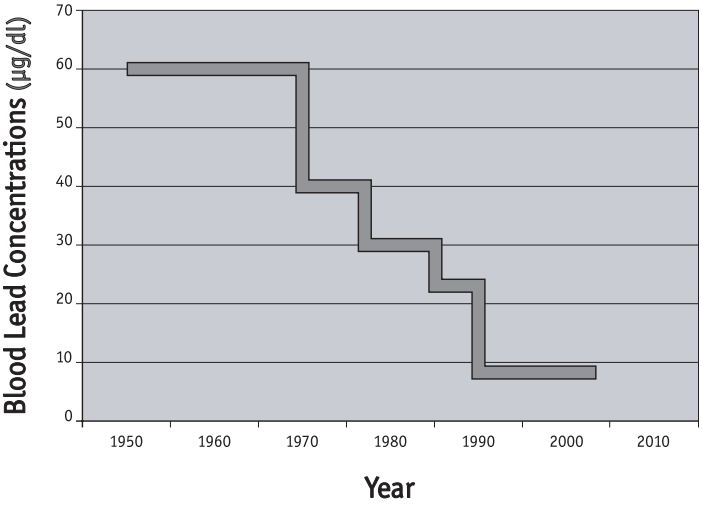


Figure 7. Blood lead concentrations considered to be elevated
Source: Canadian Partnership for Children's Health and the Environment (2005) p.89

About Biomonitoring

[Dangerous chemicals] have been found in fish in remote mountain lakes, in earthworms burrowing in soil, in the eggs of birds and in man himself. For these chemicals are now stored in the vast majority of human beings, regardless of age. They occur in mother's milk, and probably in the tissues of the unborn child.
(Silent Spring, Carson, p. 16.)

While all people are vulnerable to the health effects of harmful chemicals, children are particularly vulnerable because their developing bodies are susceptible to chemical interference. **This makes it particularly important to examine the types of chemical contaminants to which young Canadians are being exposed, as this information is vital to developing standards and regulations that effectively protect children's health.**

Although the presence of toxic chemicals in the body is unnatural, scientific knowledge about the health effects of a chemical body burden is limited, and biomonitoring results cannot be used to predict how exposure to a chemical will affect an individual's health. As stated in an authoritative document on children's environmental health prepared by the Canadian Environmental Law Association and the Ontario College of Family Physicians, "effects, or potential effects from contaminants vary according to the type and nature of the chemical, timing of exposure, frequency and duration of exposure and exposure dose. The effects also vary according to many factors inherent to the exposed individual."[94] While the presence of chemicals in a person's blood or other biological tissue or fluid does not necessarily mean that the exposure will cause illness in the individual, the detection of chemicals known to cause disease is reason for concern.

While biomonitoring information on the Canadian population is lacking, governments and researchers in other countries have conducted studies on their respective populations. Much of the research has focused on specific chemicals, such as lead and mercury, perfluorinated chemicals, PBDEs, PCBs and pesticides, rather than on capturing data on the cumulative body burden of people. In addition, the minimal amount of biomonitoring that has been conducted has mainly focused on measuring contaminant levels in adults. However, Rachel Carson's suspicion that chemicals are present in the tissues of unborn children has been proven in studies that detected toxic chemicals in umbilical cord blood.[95] An overview of international biomonitoring studies is provided in Appendix 3. The overview highlights studies conducted by non-profit organizations, including Environmental Defence, Washington-based Environmental Working Group and WWF in the United Kingdom, government organizations such as the United States Centre for Disease Control and Prevention, and numerous studies by scientific researchers that have appeared in peer-reviewed academic journals.

Benefits of Biomonitoring

Biomonitoring is a powerful tool for protecting public health from the potential adverse effects of chemicals and can be used for several purposes:

- gaining a better understanding of human exposures to a range of substances;
- establishing reference ranges that can be used to determine whether a person or a group has an unusually high level of a contaminant in their body;
- identifying potentially vulnerable groups that may experience higher levels of exposure;
- tracking, over time, trends in levels of exposure in a population;
- developing responsible and equitable chemical regulations and public health initiatives that reduce the population's exposure to chemicals of concern; and,
- evaluating the effectiveness of those regulations and initiatives.

Methodology

Volunteers

Environmental Defence selected families with children between the ages of 10 to 15 years from locations across the country, including Vancouver, Toronto, Sarnia, Montreal, and the town of Quispamsis in New Brunswick. Members of the five families in the study include six adults (two men and four women), and seven children (five girls and two boys), aged 10 to 66 years. Family members had no known medical condition that would preclude them from participating in the study. Each volunteer provided blood and urine samples which were tested for 68 individual chemicals.

Personal and Lifestyle Questionnaire

The volunteers were asked to complete a brief lifestyle questionnaire that was used to explore possible connections between a person's lifestyle and the chemicals found in his or her body. The questionnaire gathered responses about the following:

- age
- gender
- weight-gain, loss, stability
- place of residence
- occupation
- visit to malaria-infested area
- diet (vegan, dairy-and-egg-eating vegetarian, fish-eating vegetarian, omnivore)
- proportion of diet that is organic
- hours of computer use per day
- recent purchase of consumer products likely to contain brominated flame retardants, such as carpet, mattress, sofa or car
- use of products likely to contain perfluorinated chemicals, such as non-stick pans and stain-repellent furniture
- use of air fresheners
- pesticide use in and around the home, garden, and school yard
- consumption of cigarettes

Laboratories

To complete the analysis of the blood and urine samples, Environmental Defence contracted two laboratories–the Institut national de santé publique du Québec (INSPQ) in Sainte-Foy, Quebec, and AXYS Analytical Services (AAS) in Sidney, British Columbia. The analyses required to determine the presence and concentration of the chemicals in the samples are complex, and no single lab had the capacity to test for all the chemicals that Environmental Defence selected for inclusion in the study within the allocated budget.

The toxicology centre at INSPQ is a leader in the Canadian public health sector and has over 30 years experience in clinical, industrial and environmental toxicology. INSPQ was selected as the primary lab, and conducted the analyses for heavy metals, PBDEs, PCBs, organochlorine pesticides, organophosphate insecticide metabolites, and PAHs. AAS is an internationally recognized environmental laboratory specializing in custom and routine trace organic analyses for a broad spectrum of organic compounds in a range of mediums including human tissues, blood, and milk. AAS conducted the analysis for the group of perfluorinated chemicals.

Choice of Biological Materials

The selection of biological materials for analysis by the laboratories were consistent with established analytical protocols. Blood and urine are the preferred biological materials because, unlike breast milk, they can be taken from every volunteer. Drawing blood and urine samples is less invasive than sampling body fat and, in many cases, these materials also provide more accurate results than hair strand tests. Appendix 1 provides more detail about the analytical methods used in the study.

Selection of Chemicals for Testing

The chemicals were selected for testing according to the following criteria, in order of priority:

- generation of data that contributes to the international analysis of pollution in people,
- chemicals that are most harmful to human health, and particularly to children's development,
- chemicals that have the potential to be phased–out and added to the Virtual Elimination List under the *Canadian Environmental Protection Act*,
- reliability of testing procedure and cost of test.

Categorizing Chemicals According to Effects on Health

All chemicals included in the study were categorized according to five groups of health effects: carcinogens, hormone disruptors, respiratory toxins, neurotoxins and reproductive/developmental toxins. The chemicals were categorized according to information obtained from Scorecard Chemical Profiles in April 2006.[96] Scorecard differentiates between chemicals that are recognized and suspected of causing adverse health effects; in our study we included both in our total count of health effects. Our result, for example, of 38 carcinogens detected includes both recognized and suspected carcinogenic chemicals.

For many of the chemicals in the study, information was available on the health effects of individual compounds; however, this was not the case for organophosphate insecticide metabolites, PBDEs, PAHs or PFCs. Scorecard does provide a group assessment for organophosphate insecticide metabolites, PBDEs and PAHs, so all the compounds within each of these groups are coded for the same health effects. For PFCs, data on health effects is only available for two compounds, PFOA and PFOS (perfluorooctane sulfonate), the rest of the PFC compounds are coded as having 'no data on health effects'. The health effect data for PFOA and PFOS is based on the work of the Environmental Working Group, which for these two compounds, has more up-to-date data than Scorecard.

Chemical Groups Tested in Families

PFCs (perfluorinated chemicals)

PFCs and their precursors are a group of chemicals widely used in a range of consumer products for their resistance to environmental breakdown. PFCs are used to make non-stick coatings on items such as cooking pans, and stain repellent coatings on everything from carpets and furniture to microwave popcorn bags and fast-food packaging. Two of the most well-known PFCs are PFOS and PFOA. PFOA belongs to the subgroup of PFCs known as perfluorinated carboxylic acids (PFCAs), and PFOS to the subgroup known as perfluorinated alkyl sulfonates.

Existing studies show that perfluorinated chemicals are extremely persistent and bioaccumulative, as well as probably cancer-causing, hormone disrupting and toxic to reproduction and development. Recent research indicates that a major source of PFCs in the environment is the migration of PFC precursors from consumer products. In the first-ever cross-Canada measurement of PFOS levels, Environmental Defence detected PFOS in all volunteers tested for *Toxic Nation: A Report on Pollution in Canadians*.[97] Similar findings have been reported from numerous other countries[98], as well as in this report.

Many concerned people and organizations around the world have called for the phase-out of specific compounds within the group of PFCs. Sweden has proposed that PFOS be banned globally under the Stockholm Convention on Persistent Organic Pollutants, and along with Britain, has applied to the European Commission (EC) for a national ban on the substance. As of 2005, the EC had initiated a Directive requiring EU-wide restrictions on PFOS. In the US, the 3M company (the major manufacturer of PFOS) voluntarily agreed to stop using the chemical by 2003 after receiving pressure from the EPA. In February 2006, concerns about the possible health threats of PFOA led US regulators to reach a voluntary agreement with eight companies to phase-out the use of this controversial substance. Under the agreement, companies will reduce emissions of PFOA from their facilities and consumer products by 95 per cent by 2010, and work toward eliminating sources of PFOA by no later than 2015.

In July 2004, Canada was the first country to take precautionary regulatory action against PFCs by temporarily banning four fluorotelomers for two years. As of February 2006, Environment Canada and Health Canada had drafted a Risk Management Strategy for the four fluorotelomers, which recommends that a regulation be proposed to permanently prohibit the importation and manufacture of these substances. It is expected that regulations will be passed by June 2006.

Amidst consultations on the management of the four fluorotelomers, Health Canada and Environment Canada recognized the need to develop a plan to manage the group of PFCAs and their precursors as a whole, rather than make piece-meal regulations on individual chemicals. In February 2006, the Departments began consultations on their Proposed Action Plan for Assessment and Management of Perfluorinated Carboxylic Acids and Precursors, a subgroup of PFCs that includes PFOA but not PFOS.

While the proposed Action Plan on PFCAs is encouraging, the lack of action on PFOS is disconcerting. Both the Health and Environmental Screening Assessment Reports for PFOS were completed in February 2004. Although the Reports recommended that PFOS and its related substances be considered 'toxic' as defined by CEPA, and subject to virtual elimination under the Act, no regulatory actions have been pursued.

PBDEs (polybrominated diphenyl ethers)

PBDEs are used in flame retardants, which are applied to upholstered furniture, mattresses, curtains, carpets and electronics to slow the spread of fire. PBDEs can migrate from products, and have been detected in house dust, human blood and breast milk.

PBDEs are highly persistent and bioaccumulative in the environment and in people. As a group of chemicals, PBDEs are suspected of disrupting hormones, causing cancer and developmental disorders. These chemicals are suspected of having particularly damaging effects on the thyroid (which controls brain development), and as a result, PBDEs may cause learning disabilities and behaviour problems.

Research on human levels of PBDEs has found that the breast milk of Canadian women contains the second-highest levels of PBDEs in the world, second only to the US, and five to 10 times higher than that of breast milk from women from other industrialized countries, such as Japan and Germany.[99]

In February 2004, Health Canada and Environment Canada released their respective draft Screening Assessment Reports on PBDEs. Together, the departments recommended that PBDEs be considered 'toxic', as defined in section 64 of CEPA, 1999, and that certain PBDE compounds be considered for Virtual Elimination and other PBDEs for Track 1 substances under the Toxic Substances Management Policy.[100] The Canadian government, however, has not pursued regulatory action to phase-out the use of PBDEs. Instead, Environment Canada "is working with industry and other stakeholders to prepare a strategy to minimize the impact of PBDEs on the environment".[101] In light of the ubiquitous nature of PBDEs, and their potential health effects, other jurisdictions are taking precautionary action. By 2006, certain types of PBDEs will be banned in the European Union, Maine and California; in Hawaii, they will be banned by 2008.

PCBs (polychlorinated biphenyls)

PCBs have been banned in Canada since 1977, yet they continue to be released into the environment from sources in other countries and from PCB-containing industrial equipment that is still in use here in Canada.

PCBs are highly toxic and persistent chemicals that build up in wildlife and people through the process of bioaccumulation. PCBs cause many types of cancer, as well as reproductive and developmental disorders. These chemicals damage the nervous, immune and cardiovascular systems, leading to birth defects, brain damage and decreased immune function. PCBs are also suspected of being hormone disruptors.

Under the Stockholm Convention on Persistent Organic Pollutants, Canada is required to phase out the remaining uses of PCBs (in electrical transformers and other equipment) by 2025 and to dispose of these PCBS in an environmentally sound manner by 2028.

Organochlorine pesticides

Organochlorine pesticides are mainly used on agricultural crops–the fruits and vegetables we all eat. Canada still allows the use of many organochlorine pesticides, even though research has shown that these chemicals are persistent and bioaccumulative. As a group of chemicals, organochlorine pesticides are recognized carcinogens and reproductive/developmental toxins; they are also suspected hormone disruptors and respiratory toxins. The most notorious organochlorine pesticide, DDT, is banned in Canada, but continues to be used in other countries.

Organophosphate insecticide metabolites (a.k.a. dialkyl phosphate metabolites)

Dialkyl phosphate metabolites are breakdown products of organophosphate insecticides such as parathion, diazinon, malathion, and chloropyrifos, which have a variety of applications for lawns, agricultural crops, and mosquito and pest control. These chemicals are suspected of causing cancer and reproductive, developmental and neurological disorders.

In Canada, a variety of restrictions apply to the use of these chemicals, especially in residential settings. Many of these chemicals, however, are used extensively in agriculture in Canada and in other countries from which we import fresh produce.

Heavy metals (lead, mercury, arsenic, cadmium, and manganese)

Heavy metals in our environment include lead, mercury, arsenic, cadmium and manganese; some occur naturally, but most come from human-made sources. Overall, the most common source of exposure to metals is through food.

As a group, heavy metals are known to cause cancer and reproductive and developmental disorders. Many heavy metals are also suspected hormone disruptors and respiratory toxins. Canada has a variety of tools in place to regulate the production, use and disposal of these chemicals, but much stronger regulations are needed.

Releases of heavy metals by Canadian industry are reported annually to the NPRI. The most recent data available through PollutionWatch is for emissions in 2003 and shows that industry continues to contaminate the environment with dangerous heavy metals (Table 3).[102]

Exposure to arsenic can also come from wood that is pressure treated with chromated copper arsenate (CCA), which is found in playgrounds, fences, decks and other constructions. (Manufacturers of CCA-treated wood voluntarily agreed to stop producing it for consumer use by the end of 2003; however, CCA-treated wood will still be available in stores until existing stock is sold, and CCA-treated wood could remain on residential and public properties indefinitely.)

Exposure to lead comes from old lead paint and emissions from industrial facilities such as metal smelters. Mercury is emitted by coal-fired power plants, but it is also found in batteries, fluorescent light tubes,

Table 3. Total on and off-site releases for selected heavy metal compounds, as reported by Canadian industry to NPRI in 2003

Heavy Metal	Total On and Off-Site Releases
Arsenic (and its compounds)	580,838 kg
Cadmium (and its compounds)	183,024 kg
Lead (and its compounds)	3,535,270 kg
Manganese (and its compounds)	12,317,246 kg
Mercury (and its compounds)	112,287 kg

Source: Environmental Defence. (May 2006).

thermometers and related equipment. The main sources of cadmium are pigments and cigarette smoking; cadmium emissions also come from industrial sources such as lead and copper smelting and municipal waste incineration. While manganese is naturally occurring,human–made sources include the burning of fossil fuels, emissions from the steel industry, and the use of synthetic manganese compounds in pesticides. Although manganese is an essential element necessary for good health, at elevated levels it can become a neurotoxin.

PAHs (polycyclic aromatic hydrocarbons)

PAHs come from both natural and human-made sources, and are formed during the incomplete burning of coal, oil, gas, garbage, or other organic substances; some PAHs are manufactured. Forest fires are the largest natural source of PAHs in Canada. The greatest human-made sources of PAHs in air, water and soil are aluminum smelters, coking plants, creosote-treated products, spills of petroleum products, and transportation.

PAHs have been identified as 'probably carcinogenic to humans', and are suspected reproductive and respiratory toxins. In Canada, some PAHs have been assessed under the Priority Substances List of CEPA, and 17 of them are subject to reporting under the NPRI, Canada's national pollution reporting program.

Limitations of the Study

Polluted Children, Toxic Nation is a groundbreaking project that builds on the findings of *Toxic Nation: A Report on Pollution in Canadians*, Environmental Defence's first study of pollution in adults, by examining the chemical contamination of Canadian youth in comparison to their parents and grandparents. In other industrialized countries, considerable research has been conducted on the burden of chemicals found in the population; in Canada, however, research has been limited. Our studies begin to fill this knowledge gap.

Our sampling methodology was not randomized and our sample size of 13 is too small to produce statistically significant results, therefore the *Polluted Children, Toxic Nation* findings are largely demonstrative. The results, however, are consistent with body burden studies conducted in the US and Europe, and the detection of so many chemicals in every volunteer is cause for concern and further analysis.

It is also important to note that scientists have not yet developed reliable or affordable tests for detecting the vast majority of chemicals in human samples. For some chemicals reliable tests are not yet available. Testing for chemical concentrations in human samples is also expensive; testing for 68 chemicals included in this study cost over $2,000 CAD per volunteer. These technical limitations, combined with financial restrictions, mean that the group of chemicals included in our study is not a complete representation of all the chemicals people are exposed to daily. In reality most Canadians are exposed to many more each day.

While the results for a volunteer may show a level of a chemical higher or lower than the median level, the results cannot be used to predict how exposure to a chemical will affect the individual's health. Scientific knowledge about the human health effects of many individual chemicals is limited.[103] Even less information is available about the cumulative effects of long-term exposure to multiple chemicals at low levels.[104] As a result, Canadians are the test subjects in an uncontrolled experiment on the effects of daily exposure to a multitude of harmful toxic chemicals. For information on low dose and multiple exposures, please refer to pages 14 and 15.

Results and Discussion

Group Results

The findings presented in *Polluted Children, Toxic Nation*, demonstrate that Canadians young and old are polluted with toxic chemicals, no matter where they live, or what they do at work, school and play. The laboratory tests detected 46 of 68 chemicals in the 13 family members who volunteered to participate in the study. These 46 chemicals include 5 PBDEs, 13 PCBs, 5 PFCs, 9 organochlorine pesticides, 4 organophosphate insecticide metabolites, 5 PAHs, and 5 heavy metals (Table 4). On average, 32 chemicals were detected in each parent volunteer, and 23 chemicals were detected in each child volunteer (Table 4). Most of the chemicals detected in the family volunteers are either recognized for, or suspected of, causing adverse health effects. In total 38 carcinogens, 23 hormone disruptors, 12 respiratory toxins, 38 reproductive/developmental toxins, and 19 neurotoxins were detected in the study volunteers (Table 5). Three chemicals for which there is no data on health effects were detected in the volunteers (Table 5); all three of these chemicals are PFCs.

Abbreviations used in results charts:

PBDEs- polybrominated diphenyl ethers

PCBs- polychlorinated biphenyls

PFCs- perfluorinated chemicals

OCPs- organochlorine pesticides

OPIMs- organophosphate insecticide metabolites

PAHs- polycyclic aromatic hydrocarbons

Table 4. Number of chemicals detected in the study volunteers

Chemical Group	Total Number of Chemicals Tested For	Total Number of Chemicals Detected			Average Number of Chemicals Detected		
		In Adults (n=6)	In Children (n=7)	In All Volunteers (n=13)	In Adults (n=6)	In Children (n=7)	In All Volunteers (n=13)
PBDEs	5	5	5	5	2	2	2
PCBs	16	13	10	13	11	7	9
PFCs	13	5	4	5	3	3	3
OCPs	13	9	7	9	8	4	5
OPIMs	6	4	3	4	2	1	2
PAHs	10	4	5	5	1	1	1
Heavy Metals	5	5	5	5	5	5	5
Total	68	45	39	46	32	23	27

* The 'average number of chemicals detected' has been rounded to the nearest whole number.

Table 5. Number of chemicals detected in the study volunteers that are linked to a listed health effect

Chemicals' Effect on Health	Number of Chemicals Detected in Study Volunteers that are Linked to a Listed Health Effect*								
	In All Volunteers (n=13)			In Adults (n=6)			In Children (n=7)		
	Total	Average	Range	Total	Average	Range	Total	Average	Range
Carcinogen	38	22	14-30	37	26	20-30	33	19	14-26
Hormone Disruptor	23	16	10-21	23	18	14-21	20	14	10-19
Respiratory Toxin	12	7	5-10	11	8	5-10	11	6	5-8
Reproductive/ Developmental Toxin	38	23	14-31	37	26	21-31	33	20	14-27
Neurotoxin	19	12	7-17	19	14	11-17	17	11	7-15
No Data On Health Effects	3	1	0-2	3	1	0-2	2	1	0-2

* The 'average number of chemicals detected' has been rounded to the nearest whole number.

Among all the volunteers in the study, children generally had a lower total number of chemicals detected in their samples than the adults. Most children had fewer PCBs and organochlorine pesticides detected compared to the adults, likely because many of these compounds were banned throughout the 1970s, 80s and 90s, before the children in the study were born. Adults in the study, therefore, most likely had higher and more frequent exposures to these chemicals than the children. Certain organochlorine pesticides and PCBs that have been banned can still be detected in children because the chemicals are persistent and bioaccumulative, and therefore they continue to pollute the land, air, water and food supply. It is also possible for children to become polluted by chemicals in their mother's body while they are in the womb and through breast-feeding. On average, seven PCBs and four organochlorine pesticides were detected in the children, compared to 11 PCBs and eight organochlorine pesticides detected in adults (Table 4). The median total concentration of PCBs in children was 0.574 µg/L in plasma, compared to 1.934 µg/L in parents; and the median total concentration of organochlorine pesticides in children was 0.286 µg/L in plasma, compared to 0.787 µg/L in parents (Table 6). **A decreased presence of PCBs and organochlorine pesticides in the child volunteers suggests that when governments take action to eliminate toxic chemicals, people's toxic load decreases, even if it takes several generations.**

It is common to expect adults to be more contaminated by harmful chemicals than children because they have had a longer time to accumulate chemicals in their bodies. The results of this study, however, show that this is not always the case. While the children in the study were generally less contaminated than their parents by 'older' chemicals, such as PCBs and organochlorine pesticides, there were cases where the children were more contaminated than their parents by chemicals that are still in use, including PFCs, PBDEs, heavy metals, organophosphate insecticide metabolites and PAHs. Individual cases of children being more contaminated than their parents are highlighted in the results for each family. Across all volunteers, children in the study were more polluted than the adults by specific chemicals:

- PBDE 153 was detected in five of seven children at a median concentration of 0.029 µg/L in plasma. Three of six adults had PBDE 153 detected in their samples, at a lower median concentration of <0.010 µg/L (or not detected).

Table 6. Chemical concentrations detected in the study volunteers

Chemical Group		Chemical Concentrations					
		In All Volunteers (n=13)		In Adults (n=6)		In Children (n=7)	
		Median Total Concentration	Range of Concentrations Detected	Median Total Concentration	Range of Concentrations Detected	Median Total Concentration	Range of Concentrations Detected
PBDEs (µg/L in plasma)		0.118	<0.010 - 0.71	0.042	<0.010 - 0.71	0.118	<0.010 - 0.13
PCBs (µg/L in plasma)		1.041	<0.010 - 2.6	1.934	<0.010 - 2.6	0.574	<0.010 - 0.76
PFCs (ng/mL in serum)		17.345	<0.46 - 76.4	17.345	<0.46 - 76.4	17.329	<0.46 - 19.1
OCPs (µg/L in plasma)		0.602	<0.005 - 1.5	0.787	<0.005 - 1.5	0.286	<0.005 - 0.48
OPIMs (µg/L in urine)		7.9	<1 - 55	7.9	<1 - 45	7.7	<1 - 55
PAHs (µg/L in urine)		0.273	<0.013 - 1.2	<LLD	<0.057 - 0.36	0.273	<0.013 - 1.2
		Median Concentration	Range	Median Concentration	Range	Median Concentration	Range
Heavy Metals (in whole blood)	Mercury (nmol/L)	3.5	0.51 - 16	3.7	1.6 - 16	1.4	0.51 - 4.5
	Lead (µmol/L)	0.046	0.023 - 0.16	0.052	0.033 - 0.16	0.033	0.023 - 0.082
	Arsenic (nmol/L)	12	8.1 - 56	12	8.1 - 56	12	11 - 19
	Cadmium (nmol/L)	3.2	2.2 - 36	5.4	4.1 - 36	2.5	2.2 - 3.2
	Manganese (nmol/L)	180	110 - 360	170	120 - 260	180	110 - 360

*Values of less than (<) = not detected at lowest level of detection (LLD).

- PBDE 47 was detected in five children and four adults, at a median concentration of 0.078 µg/L in children, and 0.042 µg/L in adults.

- PFOA was detected in all volunteers, but the children had a higher median concentration of 2.38 ng/mL in serum, compared to 1.71 ng/mL in the adults.

- PFOS was detected in all volunteers as well, but once again, at a slightly higher median concentration in children than adults (13.8 ng/mL in children versus 13.5 ng/mL in adults).

- PFHxS was detected in five children and two adults. The children's median concentration for PFHxS was 1.19 ng/mL, compared to the adults' median concentration of <1.01 ng/mL (or not detected).

- DMTP, an organophosphate insecticide, was detected in six children and four adults. The median concentration for DMTP in children was 7.7 µg/L, compared to a median of 3.3 µg/L in adults.

- Two PAHs (3-OH-chrysene and 3-OH-phenanthrene) were more common in children than in adults. 3-OH-chrysene was detected in one child and no adults, and 3-OH-phenanthrene was detected in three children and only one adult.

The children in the study also had higher median total concentrations for the groups of PBDEs and PAHs. The children's median total concentration for PBDEs was 0.118 µg/L in plasma, compared to the adults' median total concentration of 0.042 µg/L (Table 6). For PAHs, the median total concentration among children was 0.273 µg/L in urine, compared to the adults median total concentration of <LLD (or not detected) (Table 6). Although we cannot be sure why children in the study had higher levels of certain chemicals than their parents, we suspect that physiological differences, such as differences in metabolism, as well as different patterns of exposure, can cause children to absorb some chemicals more readily than adults. **The fact that children had higher median concentrations for two of the five PBDEs and three of the five PFCs that were detected may indicate that these chemicals are an emerging concern for children's environmental health.**

PCBs

13 of 16 PCBs tested for were detected; 10 in children and 13 in adults (Table 4). Four of the 16 (PCB Aroclor 1260, 138, 153, and 180) were detected in all volunteers. The median concentration for these four PCBs are as follows: PCB Aroclor 1260 was 0.73 µg/L in adults and 0.43µg/L in children; PCB 138 was 0.095 µg/L in adults and 0.028 µg/L in children; PCB 153 was 0.099 µg/L in adults and 0.054 µg/L in children; and PCB 180 was 0.11 µg/L in adults and 0.028 µg/L in children. PCB 28, 52 and 128 were not detected, and PCB 105 was detected in only one volunteer, an adult.

Organochlorine Pesticides

9 of 13 organochlorine pesticides tested for were detected; 7 in children and 9 in adults (Table 4). Oxychlordane (a breakdown product of the pesticide chlordane) and p,p'-DDE (a breakdown product of the pesticide DDT) were detected in all volunteers. The median concentration of oxychlordane was 0.04 µg/L in adults and 0.019 µg/L in children. The median concentration of p,p'-DDE was 0.52 µg/L in adults and 0.2 µg/L in children. Four organochlorine pesticides, aldrin, α-chlordane, γ-chlordane, and p,p'-DDT, were not detected in any volunteers.

PBDEs

5 of 5 PBDEs tested for were detected in both children and adults (Table 4). The most common PBDE detected was PBDE 47, which was detected in five children and four adults. As mentioned above, the children in the study had higher median concentrations of PBDE 47 and 153 than the adults, as well as a higher median total concentration for PBDEs.

PFCs

5 of 13 PFCs tested for were detected; four in children and five in adults (Table 4). Two PFCs were detected in all volunteers, PFOS and PFOA. The median concentration of PFOS was 13.5 ng/mL in adults and 13.8 ng/mL in children. The median concentration of PFOA was 1.71 ng/mL in adults and 2.38 ng/mL in children. PFUnA was detected in only one volunteer, an adult. As mentioned above, the children in the study had higher median concentrations of three PFCs: PFOA, PFOS and PFHxS. Eight PFCs were not detected in any of the volunteers: PFBA, PFPeA, PFHxA, PFHpA, PFDA, PFDoA, PFBS, and PFOSA.

Organophosphate Insecticide Metabolites

4 of 6 organophosphate insecticide metabolites tested for were detected; three in children and four in adults (Table 4). The most common organophosphate insecticide metabolite detected was DMTP (dimethyl thiophosphate), which was detected in six children and four adults. DMTP is a metabolite of several organophosphate insecticides, including: azinphos methyl, chlorpyrifos methyl, dimethoate, fenitrothion, fenthion, isazaphos-methyl, malathion, methidathion, methyl parathion, oxydemeton-methyl, phosmet, pirimiphos-methyl, and temephos. As mentioned above, the children in the study had a higher median concentration of DMTP than the adults. DEDTP (diethyl dithiophosphate) and DETP (diethyl thiophosphate) were not detected in any of the volunteers.

Heavy Metals

5 of 5 heavy metals tested for were detected in all volunteers (Table 4). Four adults in the study had above normal levels of cadmium, and one adult had an above normal level of mercury. Two children in the study had above normal levels of manganese. The normal levels for these heavy metals as established by the laboratory that conducted the analysis are as follows: 0-5 nmol/L for cadmium, 0-15 nmol/L for mercury, and 0-300 nmol/L for manganese.

PAHs

5 of 10 PAHs tested for were detected; five in children and four in adults (Table 4). The most common PAH detected was 1-OH-phenanthrene, which was detected in three children and three adults. As mentioned above, two PAHs (3-OH-chrysene and 3-OH-phenanthrene) were more common in children than in adults. The five PAHs that were not detected in the volunteers are 1-OH-benz(a)-anthracene, 3-OH-benz(a)-anthracene, 3-OH-fluoranthene, 4-OH-phenanthrene and 6-OH-chrysene.

Family Results

From left to right:
Wilson Sr. (grandfather, age 66),
Jessie (daughter, age 14), and
Wilson Jr. (father, age 44)

The Sarnia Family (Ontario)

The Plain family is from the Aamjiwnaang First Nation Community in Sarnia, Ontario. Wilson Sr., now retired, spent his working life in various industrial factories in the Sarnia area. Of all the volunteers in the study, Wilson Sr. had the highest concentration of PFOS, at 35.8 ng/mL, as well as the highest total concentrations for PCBs (at 3.77 µg/L) and organochlorine pesticides (at 1.9053 µg/L) (Table 8).

Wilson Jr. has also worked in various industrial facilities in the Sarnia area and is now a truck driver. Along with one other volunteer, Wilson Jr. had the highest total number of chemicals detected in a volunteer (36 of 68), as well as the highest total concentrations for PBDEs (at 0.941 µg/L) and PFCs (86.93 µg/L)(Table 8). For individual chemicals, Wilson Jr. had by far the highest level of PFOS detected at a concentration of 76.4 ng/mL, and the highest concentration of cadmium at 36 nmol/L. According to his Lifestyle Questionnaire, Wilson Jr. is a smoker, which is the likely cause of the elevated level of cadmium detected in his sample.

Jessie, the daughter, had a lower than average number of chemicals detected in her samples, as well as the least number of PCBs and organochlorine pesticides of any volunteer in the study. A relatively high level of manganese was detected in Jessie's sample, at a concentration of 300 nmol/L.

Table 7. Number of chemicals detected in the Sarnia family that are linked to a listed chemical health effect, and the study averages

Chemicals' Effect on Health	Number of Chemicals Detected in Study Volunteers that are Linked to a Listed Health Effect*				
	Grandfather	Father	Daughter	Average in Adults	Average in Children
Carcinogen	25	29	16	26	19
Hormone Disruptor	16	21	12	18	14
Respiratory Toxin	6	9	7	8	6
Reproductive/ Developmental Toxin	25	29	17	26	20
Neurotoxin	13	17	10	14	11
No Data On Health Effects	2	2	1	1	1

* The average number of chemicals has been rounded to the nearest whole number.

Family Results

Table 8. Number and concentration of chemicals detected in the Sarnia family, and the median total concentrations in all volunteers

Chemical Group		Grandfather			Father			Daughter			In All Volunteers
		Number of Compounds Detected	Total Concentration	Range	Number of Compounds Detected	Total Concentration	Range	Number of Compounds Detected	Total Concentration	Range	Median Total Concentration
PBDEs (µg/L in plasma)		0 of 5	na	<0.01-<0.03	4 of 5	0.941	<0.01-0.71	2 of 5	0.116	<0.01-0.084	0.118
PCBs (µg/L in plasma)		12 of 16	3.775	<0.01-2.6	9 of 16	1.214	<0.01-0.81	4 of 16	0.239	<0.01-0.19	1.041
PFCs (ng/mL in serum)		4 of 13	45.03	<0.046-35.8	4 of 13	86.93	<0.46-76.4	3 of 13	23.49	<0.46-18	17.345
OCPs (µg/L in plasma)		8 of 13	1.9053	<0.005-1.5	9 of 13	0.7869	<0.005-0.42	2 of 13	0.151	<0.005-0.14	0.602
OPIMs (µg/L in urine)		3 of 6	47	<1-20	3 of 6	25.5	<1-9.6	2 of 6	4.3	<1-2.2	7.9
PAHs (µg/L in urine)		0 of 10	na	<0.057-<0.13	2 of 10	0.5	<0.057-0.25	2 of 10	0.273	<0.059-0.19	0.273
		Detected	Concentration		Detected	Concentration		Detected	Concentration		Median
Heavy Metals (in whole blood)	Mercury (nmol/L)	1 of 1	1.6		1 of 1	7.1		1 of 1	0.86		3.5
	Lead (µmol/L)	1 of 1	0.052		1 of 1	0.033		1 of 1	0.026		0.046
	Arsenic (nmol/L)	1 of 1	12		1 of 1	8.1		1 of 1	12		12
	Cadmium (nmol/L)	1 of 1	4.6		1 of 1	36		1 of 1	2.2		3.2
	Manganese (nmol/L)	1 of 1	230		1 of 1	120		1 of 1	300		180
Total Number of Chemicals Detected		32 of 68			36 of 68			20 of 68			

*Values of less than (<) = not detected at lowest level of detection (LLD).

Family Results

From left to right:
Johanna (daughter, age 15),
Amy (mother, age 42), and
Satchel (son, age 13)

The Vancouver Family (British Columbia)

The Robertson's now live in Vancouver, but have spent several years living on Cortez Island and in the Fraser Valley in British Columbia. Amy works at home and grows much of the family's food in their organic garden. The children in this family have higher concentrations of several chemicals than their mom, and they are the only children in the study who have a higher total concentration of PCBs than their parent. Of all the children in the study, Johanna, the daughter, had the highest total number of chemicals (32 of 68)(Table 10). Johanna is the only child in the study to have a greater number of chemicals than her parent. Satchel, the son, had the second highest number of chemicals detected in a child in the study (29 of 68) (Table 10).

Among the children in the study, Johanna and Satchel both had a higher than average number of PCBs and organochlorine pesticides detected in their samples. Both children have higher levels than their mom for four PCBs (Aroclor 1260, 153, 163 and 180); Johanna also had a higher concentration than her mom for PCB 180, and she was the only child in the study with PCB 101 detected in her sample (which was not detected in her mom). Both children also had PCB 156, which was not detected in their mom.

For organochlorine pesticides, Johanna and Satchel both had a higher concentration than their mom for oxychlordane, and they were the only children with β-HCH detected in their samples. Johanna was the only child in the study with toxaphene parlar 50. Johanna was the only person in the study to have all five PBDEs, and both she and Satchel had higher levels of PBDE 153 than their mom; Satchel also had a higher level of PBB 153 than his mom. Both children had PBDE 47, which was not detected in their mom. Johanna also had PBDE 99, which was not detected in her mom.

Both Johanna and Satchel also had a higher level of 1-OH-phenanthrene (a PAH) than their mom. In addition, Satchel had a higher level of 2-OH-phenanthrene than his mom, and 3-OH-phenanthrene, which was not detected in his mom. Satchel also had the highest concentrations in the family for three PFCs: PFOA, PFOS, and PFHxS.

Amy had the least number of PCBs of any of the adults in the study. However, Amy did have the highest concentration of PFNA in the study, at a concentration of 1.73 ng/mL, and she was the only volunteer with PFUnA, at a concentration of 1.19 ng/mL. Amy also had an above normal level of cadmium (although she is not a smoker), and the highest concentration of arsenic in the study, at 56 nmol/L.

Table 9. Number of chemicals detected in the Vancouver family that are linked to a listed chemical health effect, and the study averages

Chemicals' Effect on Health	Number of Chemicals Detected in Study Volunteers that are Linked to a Listed Health Effect*				
	Mother	Daughter	Son	Average in Adults	Average in Children
Carcinogen	24	26	24	26	19
Hormone Disruptor	18	19	16	18	14
Respiratory Toxin	10	7	8	8	6
Reproductive/ Developmental Toxin	25	27	25	26	20
Neurotoxin	13	15	13	14	11
No Data On Health Effects	2	2	2	1	1

* The average number of chemicals has been rounded to the nearest whole number.

Family Results

Table 10. Number and concentration of chemicals detected in the Vancouver family, and the median total concentration in all volunteers

Chemical Group		Mother			Daughter			Son			In All Volunteers
		Number of Compounds Detected	Total Concentration	Range	Number of Compounds Detected	Total Concentration	Range	Number of Compounds Detected	Total Concentration	Range	Median Total Concentration
PBDEs (µg/L in plasma)		2 of 5	0.028	<0.02-0.017	5 of 5	0.23	0.02-0.078	3 of 5	0.136	<0.02-0.086	0.118
PCBs (µg/L in plasma)		9 of 16	0.888	<0.1-0.62	10 of 16	1.104	<0.01-0.76	9 of 16	1.041	<0.01-0.73	1.041
PFCs (ng/mL in serum)		4 of 13	23	<0.46-18.6	4 of 13	14.871	<0.46-11.5	3 of 13	23.47	<0.46-19.1	17.345
OCPs (µg/L in plasma)		8 of 13	0.8237	<0.005-0.54	6 of 13	0.509	<0.005-0.31	5 of 13	0.661	<0.005-0.48	0.602
OPIMs (µg/L in urine)		0 of 6	na	<1-<2	1 of 6	7.7	<1-7.7	1 of 6	9.6	<1-9.6	7.9
PAHs (µg/L in urine)		3 of 10	0.539	<0.057-0.24	1 of 10	0.41	<0.057-0.41	3 of 10	1.28	<0.059-0.69	0.273
		Detected	Concentration		Detected	Concentration		Detected	Concentration		Median
Heavy Metals (in whole blood)	Mercury (nmol/L)	1 of 1	13		1 of 1	1.4		1 of 1	3.9		3.5
	Lead (µmol/L)	1 of 1	0.046		1 of 1	0.031		1 of 1	0.04		0.046
	Arsenic (nmol/L)	1 of 1	56		1 of 1	15		1 of 1	19		12
	Cadmium (nmol/L)	1 of 1	10		1 of 1	2.5		1 of 1	2.7		3.2
	Manganese (nmol/L)	1 of 1	170		1 of 1	190		1 of 1	110		180
Total Number of Chemicals Detected		31 of 68			32 of 68			29 of 68			

*Values of less than (<) = not detected at lowest level of detection (LLD).

Family Results

From left to right:
Hanna (daughter, age 14),
Patty (mother, age 45), and
Mary (daughter, age 14)

The Quispamsis Family (New Brunswick)

The Donovans live in the town of Quispamsis in New Brunswick. Patty, a Program Facilitator at a women's centre, had the lowest total number of chemicals of all the adults in the study (24 of 68). Her twin daughters, Mary and Hanna, had the same number of chemicals detected in their samples (17 of 68)(Table 12), which was the lowest total number of chemicals of all the volunteers in the study. All three family members also had the least number of PFCs detected in their samples; while the other volunteers in the study each had three or four PFCs, the Donovans each had only two (PFOS and PFOA).

Patty had a slightly above normal level of cadmium, at a concentration of 5.4 nmol/L (Table 12). Hanna and Mary, along with one other volunteer (Wilson Sr.) were the only volunteers in the study with no PBDEs detected in their samples (Table 12). Both daughters had a higher level of PFOA than their mom, and Mary also had a higher level of PFOS than her mom. Hanna had a significantly higher level of DMP (an organophosphate insecticide metabolite) than the median in the study. The level of DMP detected in Hanna was 29 µg/L, while the study median was 4.6 µg/L.

Table 11. Number of chemicals detected in the Quispamsis family that are linked to a listed chemical health effect, and the study averages

Chemicals' Effect on Health	Number of Chemicals Detected in Study Volunteers that are Linked to a Listed Health Effect*				
	Mother	Daughter (M)	Daughter (H)	Average in Adults	Average in Children
Carcinogen	20	14	14	26	19
Hormone Disruptor	14	10	11	18	14
Respiratory Toxin	5	5	6	8	6
Reproductive/ Developmental Toxin	21	15	14	26	20
Neurotoxin	11	7	8	14	11
No Data On Health Effects	0	0	0	1	1

* The average number of chemicals have been rounded to the nearest whole number.

Family Results

Table 12. Number and concentration of chemicals detected in the Quispamsis family, and the median total concentration in all volunteers

Chemical Group		Mother			Daughter (M)			Daughter (H)			In All Volunteers
		Number of Compounds Detected	Total Concentration	Range	Number of Compounds Detected	Total Concentration	Range	Number of Compounds Detected	Total Concentration	Range	Median Total Concentration
PBDEs (µg/L in plasma)		1 of 5	0.042	<0.01-0.042	0 of 5	na	<0.01-<0.03	0 of 5	na	<0.01-<0.03	0.118
PCBs (µg/L in plasma)		10 of 16	1.934	<0.01-1.4	7 of 16	0.574	<0.01-.043	4 of 16	0.313	<0.01-0.25	1.041
PFCs (ng/mL in serum)		2 of 13	14.3	<0.46-13.1	2 of 13	15.66	<0.46-13.8	2 of 13	12.13	<0.46-10.5	17.345
OCPs (µg/L in plasma)		5 of 13	0.724	<0.005-0.52	2of 13	0.206	<0.005-0.2	3 of 13	0.286	<0.005-0.14	0.602
OPIMs (µg/L in urine)		1 of 6	5	<1-5	1 of 6	5.7	<1-5.7	2 of 6	4	<1-29	7.9
PAHs (µg/L in urine)		0 of 10	na	<0.057-<0.13	0 of 10	na	<0.057-0.13	1 of 10	0.11	<0.057-0.11	0.273
		Detected	Concentration		Detected	Concentration		Detected	Concentration		Median
Heavy Metals (in whole blood)	Mercury (nmol/L)	1 of 1	3.7		1 of 1	0.88		1 of 1	0.51		3.5
	Lead (µmol/L)	1 of 1	0.071		1 of 1	0.023		1 of 1	0.033		0.046
	Arsenic (nmol/L)	1 of 1	12		1 of 1	14		1 of 1	12		12
	Cadmium (nmol/L)	1 of 1	5.4		1 of 1	3.2		1 of 1	2.4		3.2
	Manganese (nmol/L)	1 of 1	180		1 of 1	160		1 of 1	180		180
Total Number of Chemicals Detected		24 of 68			17 of 68			17 of 68			

*Values of less than (<) = not detected at lowest level of detection (LLD).

Family Results

From left to right:
Aladin (son, age 10) and
Viviane (mother, age 33)

The Montreal Family (Quebec)

Viviane and Aladin live in Montreal, Quebec, where Viviane is a Coordinator of Project Integration at a wind energy consulting firm. Along with one other volunteer in the study, Viviane had the highest total number of chemicals (36 of 68)(Table 14). Viviane was the only volunteer with PCB 105, at a concentration of 0.011 µg/L.

Both Viviane and Aladin had high levels of organophosphate insecticides. Viviane had the highest level of DMTP, at 45 µg/L, while Aladin had the highest level of DMP, at a concentration of 55 µg/L. Aladin was also one of only two volunteers with DEP, and he had the highest total concentration of organophosphate insecticide metabolites in the study, at 80.1 µg/L. Viviane had the second highest total concentration of organophosphate insecticide metabolites in the study, at 66 µg/L(Table 14).

Of all the children, Aladin had the highest total concentration for PFCs, at 28.823 ng/mL; his levels of PFOS and PFOA were higher than his mom's, and he had the highest level of PFHxS in the study, at a concentration of 4.75 ng/mL (PFHxS was not detected in Viviane). In addition, the level of lead in Aladin's blood was higher than his mom's. Aladin's lead level was the second highest lead level in the study.

Table 13. Number of chemicals detected in the Montreal family that are linked to a listed health effect, and the study averages

Chemicals' Effect on Health	Number of Chemicals Detected in Study Volunteers that are Linked to a Listed Health Effect*			
	Mother	Son	Average in Adults	Average in Children
Carcinogen	30	19	26	19
Hormone Disruptor	20	14	18	14
Respiratory Toxin	9	7	8	6
Reproductive/ Developmental Toxin	31	20	26	20
Neurotoxin	15	13	14	11
No Data On Health Effects	1	2	1	1

* The average number of chemicals has been rounded to the nearest whole number.

Family Results

Table 14. Number and concentration of chemicals detected in the Montreal family, and the median total concentrations in all volunteers

Chemical Group		Mother			Son			In All Volunteers
		Number of Compounds Detected	Total Concentration	Range	Number of Compounds Detected	Total Concentration	Range	Median Total Concentration
PBDEs (µg/L in plasma)		4 of 5	0.576	<0.01-0.3	2 of 5	0.118	<0.01-0.077	0.118
PCBs (µg/L in plasma)		12 of 16	2.119	<0.01-1.5	5 of 16	0.358	<0.01-0.28	1.041
PFCs (ng/mL in serum)		3 of 13	14.04	<0.46-11.5	4 of 13	28.823	<0.46-18.7	17.345
OCPs (µg/L in plasma)		7 of 13	1.0644	<0.005-0.83	4 of 13	0.3	<0.005-0.23	0.602
OPIMs (µg/L in urine)		2 of 6	66	<1-45	3 of 6	80.1	<1-55	7.9
PAHs (µg/L in urine)		3 of 10	0.89	<0.059-0.36	2 of 10	1.5	<0.13-1.2	0.273
		Detected	Concentration		Detected	Concentration		Median
Heavy Metals (in whole blood)	Mercury (nmol/L)	1 of 1	3.5		1 of 1	4.5		3.5
	Lead (µmol/L)	1 of 1	0.076		1 of 1	0.082		0.046
	Arsenic (nmol/L)	1 of 1	17		1 of 1	11		12
	Cadmium (nmol/L)	1 of 1	4.1		1 of 1	2.3		3.2
	Manganese (nmol/L)	1 of 1	150		1 of 1	120		180
Total Number of Chemicals Detected		36 of 68			25 of 68			

*Values of less than (<) = not detected at lowest level of detection (LLD).

Family Results

From left to right:
Ada (daughter, age 10) and
Barri (mother, age 40s)

The Toronto Family (Ontario)

Barri and Ada live in Toronto, Ontario, where Barri is a filmmaker and writer. Compared to the other volunteers in the study, Barri's test results were fairly average, except that she had the highest levels of both mercury and lead in the study; she also had an above normal level of cadmium. According to her Lifestyle Questionnaire, Barri occasionally smokes cigarettes, which may be a cause of her elevated level of cadmium.

Ada had a higher number of PBDEs and PFCs than her mom, as well as a higher total concentration of PBDEs (Table 16). Ada's level of PBDE 99 was 0.032 µg/L, which was higher than her mom's level of 0.024 µg/L; Ada also had one PBDE that her mom did not, PBDE 153. For PFCs, Ada had a higher concentration of PFOA than her mom, at 3.29 ng/mL, compared to 2.91 ng/mL; she also had PFHxS, which was not present in her mom. Of all the volunteers in the study, Ada had the highest concentration of manganese at 360 nmol/L, which is above the normal range.

Table 15. Number of chemicals detected in the Toronto family that are linked to a listed chemical health effect, and the study averages

Chemicals' Effect on Health	Number of Chemicals Detected in Study Volunteers that are Linked to a Listed Health Effect*			
	Mother	Daughter	Average in Adults	Average in Children
Carcinogen	25	18	26	19
Hormone Disruptor	18	14	18	14
Respiratory Toxin	6	5	8	6
Reproductive/ Developmental Toxin	25	19	26	20
Neurotoxin	13	9	14	11
No Data On Health Effects	1	2	1	1

* The average number of chemicals has been rounded to the nearest whole number.

Family Results

Table 16. Number and concentration of chemicals detected in the Toronto family, and the median total concentration in all volunteers

Chemical Group		Mother			Daughter			In All Volunteers
		Number of Compounds Detected	Total Concentration	Range	Number of Compounds Detected	Total Concentration	Range	Median Total Concentration
PBDEs (µg/L in plasma)		2 of 5	0.154	<0.01-0.13	3 of 5	0.174	<0.01-0.13	0.118
PCBs (µg/L in plasma)		11 of 16	2.159	<0.01-1.5	9 of 16	0.709	<0.01-0.5	1.041
PFCs (ng/mL in serum)		3 of 13	17.345	<0.46-13.5	4 of 13	17.329	<0.46-12.1	17.345
OCPs (µg/L in plasma)		8 of 13	0.6018	<0.005-0.36	3 of 13	0.218	<0.005-0.18	0.602
OPIMs (µg/L in urine)		2 of 6	7.9	<1-4.6	0 of 6	na	<1-<2	7.9
PAHs (µg/L in urine)		0 of 10	na	<0.057-0.13	0 of 10	na	<0.057-<0.13	0.273
		Detected	Concentration		Detected	Concentration		Median
Heavy Metals (in whole blood)	Mercury (nmol/L)	1 of 1	16		1 of 1	2.2		3.5
	Lead (µmol/L)	1 of 1	0.16		1 of 1	0.058		0.046
	Arsenic (nmol/L)	1 of 1	31		1 of 1	11		12
	Cadmium (nmol/L)	1 of 1	15		1 of 1	2.8		3.2
	Manganese (nmol/L)	1 of 1	260		1 of 1	360		180
Total Number of Chemicals Detected		31 of 68			24 of 68			

*Values of less than (<) = not detected at lowest level of detection (LLD).

Conclusion and Recommendations

The results of *Polluted Children, Toxic Nation: A Report on Pollution in Canadian Families* show that Canadians as young as 10 are contaminated with a host of harmful chemicals that can exert particularly damaging effects in the growing body of a child. Of the 68 chemicals the families were tested for, 46 were detected. On average, 32 chemicals were detected in each parent volunteer, and 23 chemicals were detected in each child volunteer. Good and bad news can be found in the results. Overall, the children in the study were less contaminated than the adults by 'older' chemicals, such as organochlorine pesticides and PCBs. The decreased presence of PCBs and organochlorine pesticides in the child volunteers suggests that when governments take action to eliminate toxic chemicals, people's toxic load decreases, even if it takes several generations. It is alarming, however, that in some cases the children in the study were more contaminated than the adults by chemicals that are still in use. The detection of higher concentrations of certain chemicals, including PBDEs and PFCs, in the children suggests that these chemicals are an emerging concern for children's environmental health, and require immediate attention by industry and government.

Recommendations for Parents and Childcare Providers

Parents, childcare professionals, school teachers and others who work with children have an important role to play in protecting children from exposure to harmful chemicals. The four most important things parents and other childcare providers can do to protect children from exposure to chemicals are:

- Learn more about chemicals of concern
- Reduce the use of products that contain toxic chemicals
- Control dust in childrens' indoor environments
- Get involved in achieving a toxic-free future

Learn more about chemicals of concern

The first step in childproofing for pollutants is to learn about chemicals of concern–what they are, how they affect health, where they may be found in the home or school, and how children can become exposed. If you know the hazards you will be better equipped to evaluate risks and identify measures you can take to avoid exposures.

Chemicals of concern for children's health include:

- Metals (i.e. lead, mercury)
- Pesticides
- VOCs
- PBDEs
- PCBs
- PFCs
- Phthalates
- Bisphenol A
- Dioxins and Furans
- PAHs
- Components of smog (sulphur dioxide, nitrogen oxides, particulate matter, carbon monoxide, ground-level ozone)

Online resources for parents and childcare providers:

- Canadian Partnership for Children's Health and the Environment (CPCHE), *Child Health and the Environment- A Primer*. Available at http://www.healthyenvironmentforkids.ca/img_upload/13297cd6a147585a24c1c6233d8d96d8/Primer.pdf

- Canadian Environmental Law Association (CELA) and Ontario College of Family Physicians (OCFP) Environmental Health Committee, Ch.2: *Relationship Between Children's Health and Environmental Contaminants* in Environmental Standard Setting and Children's Health. Available at http://cela.ca/uploads/f8e04c51a8e04041f6f7faa046b03a7c/Ch2.pdf

- CELA and Pollution Probe, *Toxic Substances- Focus on Children: Developing a Canadian List of Substances of Concern to Children's Health*. Available at: http://cela.ca/uploads/f8e04c51a8e04041f6f7faa046b03a7c/List_Project_Full_Report.pdf

- *Toxic Nation* - http://www.ToxicNation.ca

- US Agency for Toxic Substances and Disease Registry (ATSDR): ToxFAQs: Hazardous Substances Fact Sheets-- http://www.atsdr.cdc.gov/toxfaq.html

- Scorecard Chemical Profiles-- http://www.scorecard.org/chemical-profiles/

Reduce the use of products that contain toxic chemicals

These web sites provide information on reduction strategies.

- Toxic Nation's Toxic Free Home Tour and the Chemical Reduction Pledge-- www.toxicnation.ca/pledge

- Guide to Less Toxic Products-- http://www.lesstoxicguide.ca/

- Health Canada, Indoor Air Quality: Tools for Schools-Action Kit for Canadian Schools-- http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/air/tools_school-outils_ecoles/tools_school-outils_ecoles_e.pdf

Get Involved in Achieving A Toxic-Free Future

- Share information with family, friends and colleagues about the importance of reducing our daily exposure to toxic chemicals.

- Visit **www.ToxicNation.ca** and join our campaign to strengthen the *Canadian Environmental Protection Act* and make industry accountable for proving that chemicals are safe before they go on the market.

Recommendations for the Federal Government and Industry

Canadians expect their country to be a leader in the protection of human health and the environment. Despite the Canadian government's efforts to control toxic chemicals, the volume of harmful chemicals released into the environment and making their way into Canadians' bodies continues to increase. And now the findings presented in this report reveal in some cases children are even more polluted than their parents.

Canada's pollution problems stem from the weak and ineffective regulation of toxic chemicals under the overarching national toxic chemicals law, CEPA. The opportunity exists now to address the shortfalls of this Act during its mandatory five-year review, which began in the fall of 2005 and will continue through to 2007. Environmental Defence is calling upon the federal government to acknowledge the evidence of human contamination revealed in the Toxic Nation studies by taking action to strengthen the regulation of toxic chemicals in Canada.

Environmental Defence recommends that CEPA be amended to:

- establish timelines for the virtual elimination of toxic chemicals,

- make industry accountable for its chemicals,

- regulate toxic chemicals in consumer products, and

- reduce pollution in the Great Lakes basin.

Establish timelines for the virtual elimination of toxic chemicals:

- Establish aggressive timelines to virtually eliminate carcinogens, respiratory toxins, endocrine disruptors, and reproductive and neurological toxins from use, release, manufacture, disposal and recycling. At a minimum, a 50 per cent reduction in these substances must be achieved by 2010, with virtual elimination being achieved by 2015.

- As a matter of priority, immediately ban PBDEs, PFCs and their precursors, and phthalates.

At present, Canada has no goals or timelines for the elimination of toxic chemicals, in fact under current regulations our federal government will allow chemicals that harm health to remain on the market and be released into the environment indefinitely. In comparison, other jurisdictions are well on their way to reducing the amount of toxic chemicals released into the environment, with set goals for the elimination of chemicals that harm human health. Canadians may be particularly surprised by the fact that the US is

doing a much better job of protecting people from harmful chemicals than our own government.

In 2003-2004 major reductions in the release of some of the most toxic chemicals were achieved in the United States, including a 58 per cent decrease in dioxin and dioxin compounds, a 16 per cent decrease in mercury and mercury compounds and a 92 per cent decrease in PCBs.[105] Internationally, the most aggressive toxics reduction goals have been set by Sweden, which aims to eliminate carcinogenic, mutagenic and reproductive toxins from products by 2007, and ban all persistent and bioaccumulative substances by 2015.

In Canada we have the information needed to start targeting the most harmful chemicals to achieve elimination by 2015. Health Canada and Environment Canada are currently categorizing over 23,000 existing substances that make up the Domestic Substances List (DSL) based on persistence, bioaccumulation and inherent toxicity. The categorization is to be complete by September 2006. Environmental Defence urges the federal government to announce an immediate action plan to reduce and eliminate the top 200 toxic substances, as determined by the DSL categorization.

Compared to Canada, the US also leads the way when it comes to regulating emerging chemicals of concern, including PBDEs and PFCs. In the US, under pressure from regulators, manufacturers of PBDEs and PFCs have chosen to voluntarily phase-out the use of these chemicals. These voluntary phase-outs have been solidified by subsequent regulations that ensure that any future uses will be evaluated and that the government will have the power to prohibit or limit activities before they occur. In contrast, the Canadian government has not acted on recommendations from both Health Canada and Environment Canada to virtually eliminate PBDEs and PFCs, and no regulations are in place to prohibit the manufacture and import of these substances.

Environmental Defence urges the federal government to finalize the draft assessments for PFOS and its precursors, PFOA, and PBDEs, and to enact immediate bans on perfluorinated chemicals and their precursors, as well as all three commercial mixtures of PBDEs (OctaBDE, PentaBDE and DecaBDE). Environmental Defence also urges the federal government to acknowledge the international body of research indicating the hazardous characteristics of phthalates, and to follow the precedent set by the European Union by banning phthalates in children's toys and products and in cosmetics.

Make industry accountable for its chemicals:

- Shift the burden of proof onto industry to prove the safety of its chemicals before their introduction to or continued use in the market.

- Mandate industry to adopt a safe substitution policy to replace toxic substances with safer or non-toxic substances.

There is one basic, overarching problem with the approach to the safety assessment of chemicals under CEPA: companies have not been required to conduct adequate safety testing before their chemicals enter the market; rather, the government has been responsible for proving a chemical is hazardous after it is already in use. For existing substances, which make up the majority of chemicals on the market, companies are not required to conduct retroactive safety testing. For new substances, the requirements for safety assessment are inadequate and do not ensure that all new toxic substances will be identified and kept off the market. This substance-by substance approach to regulating toxic chemicals is ineffective for several reasons. First, the approach is extremely time and labour intensive and places an unfair burden on the public purse. Second, because of a lack of government resources, the majority of chemicals on the Canadian market will never be adequately assessed for their safety, and many chemicals that are extremely harmful to human health will continue to be used and released. Third, when a chemical is identified as potentially hazardous, the safety assessment process is dangerously slow–it typically takes 10 to 15 years for the safety assessment to be completed and for any restrictions to take effect.

The burden of proof must be shifted onto industry to prove that a chemical is safe before it is permitted to enter, or continue to be used, on the market. To prove the safety of all existing and new chemicals industry should be required to submit safety data, including the latest information on health effects for assessment by Health Canada and Environment Canada, as well as for peer-review by a scientific panel.

To support the phase-out of existing chemicals that are identified as toxic, industry must adopt a safe substitution policy and develop safer or non-toxic substances. Substitution involves replacing a toxic chemical with a safer or non-toxic substance, or redesigning the product or system to eliminate the need for the toxic chemical. Substitution must be mandatory for all chemicals that are, or are suspected to be, persistent, bioaccumulative or inherently

toxic, and substitution should involve strict timelines.
Regulate toxic chemicals in consumer products:

- Clarify CEPA to regulate toxic chemicals that may be released during the use or disposal of consumer products.

CEPA focuses on toxic chemicals that industry uses, manufactures and releases but does not address the release of toxic chemicals during the use of a product or its disposal. This major gap in the scope of CEPA leaves consumers vulnerable to exposure to toxic chemicals through the use of everyday products. Toxic chemicals in products are mainly regulated under the *Hazardous Products Act*, which has been ineffective in protecting people, and particularly children, from exposure to toxic chemicals in products. The *Hazardous Products Act* includes no requirements for a pre-market assessment of risks associated with a product, and only after Health Canada receives complaints or recognizes a potential risk is a post-market assessment conducted. Even after Health Canada determines that a product poses risks, it has no authority to mandate product recalls; it can seize only products in storage, meaning that products already on store shelves remain there unless companies take voluntary action to impose their own recall. The primary tools Health Canada relies on to protect consumers are public advisories and warnings.

Clarifying the scope of CEPA to cover toxic chemicals in consumer products can ensure that all toxic chemicals in products are regulated and, in the case of new products, that at least some pre-market assessment will occur. Inclusion of toxic chemicals used in consumer products under CEPA will also give Health Canada the authority to regulate chemicals in products in a precautionary manner and to develop programs to eliminate their use through phase-outs. As the overarching law on toxic substances, CEPA should cover all chemicals in Canada, whether they are released through industrial activities or from products.

Reduce pollution in the Great Lakes Basin:

- Create a special section of CEPA to focus on Great Lakes protection.
- Provide new funding for a Canadian Great Lakes clean-up of toxic hot spots.

The Great Lakes - Superior, Michigan, Huron, Erie, and Ontario - form the largest surface freshwater system on the Earth. The Great Lakes basin is home to 30 million people, including about one third of the Canadian population. The basin is also home to industrial facilities that produce 45 per cent of Canada's total toxic air emissions. Given that nearly half of all of Canada's toxic air emissions originate in the Great Lakes basin, a special section of CEPA is needed to focus on this pollution hot spot.

Protecting the Great Lakes from pollution, and remediation the effects of past (and present) contamination, requires cooperative efforts on both sides of the border, and so far, the US has stepped up with a strategy and funding for Great Lakes restoration and protection, while Canada has stalled.

There are several concrete examples of the ways in which the US treats the Great Lakes as a priority. For example, in 2002, the US *Great Lakes Legacy Act* authorized $270 million over five years (beginning in 2004) to help with the remediation of contaminated sediment in Areas of Concern in the Great Lakes basin. In May 2004, President Bush issued an Executive Order recognizing the Great Lakes as a "national treasure"; the Order also directed the EPA to convene a "regional collaboration of national significance for the Great Lakes". The Collaboration was launched in December 2004, and one year later they released a *Strategy to Restore and Protect the Great Lakes*. The strategy included recommendations on a number of issues, including: accelerating the clean-up of the 31 Areas of Concern; actions to address the non-point sources of pollution; address the problems of toxic pollutants; and, ensuring long-term sustainability of the Great Lakes resource.

In contrast to US efforts in the Great Lakes, Canada has seemingly abandoned previous efforts to remediate the effects of contamination in the Great Lakes and prevent future pollution. In the 2001 *Report of the Commissioner of the Environment and Sustainable Development*[108] , concerns were expressed "about the loss of momentum in recent years and the implications this has for the future" in reference to protecting the state of the Great Lakes and St. Lawrence River basin. The audit revealed that "many of the federal government's priorities and commitments for the basin are general and vague. The results it hopes to achieve are difficult to measure" and, "many key commitments have not been met; many key initiatives have not been completed; and departments are spreading their efforts thin". Furthermore, the audit found that funding to deal with many issues in the basin is "unstable, declining, and insufficient to meet the government's objectives".

The Commissioner's report highlighted several recommendations for areas where the federal government can do better. The recommendations included: "adequately fund its commitments; reassess

whether the legislative and other tools it uses are sufficient to manage threats to the basin; set-up consistent data gathering to understand the nature and trends in key threats to the basin; and analyze and demonstrate how federal activities have improved the basin's sustainability".

To date, no new funding has been allocated to protecting the Great Lakes and St.Lawrence River basin, nor have any new action plans been released. Of the 17 Areas of Concern identified in Canada in 1985, 16 are still on the list. Without funding or action plans, it is not clear how or when the federal government plans to restore and protect the Great Lakes and St. Lawrence River basin. Environmental Defence recommends creating a section in CEPA that specifically addresses pollution in the Great Lakes basin as a tool for generating concrete actions for reinvesting in the remediation and protection of the basin.

References

[1] Environmental Defence and Canadian Environmental Law Associaiton. (2004, December). Shattering the Myth of Pollution Progress: A National Report. p.1.

[2] Environmental Defence. (2005, October 15). PollutionWatch Compilation: Create a Pollution Report. Retrived online October 2005 from http://www.pollutionwatch.org

[3] Canadian Environmental Network (CEN) Toxics Caucus. (2005, March). The ENGO Agenda for the Review of the Canadian Environmental Protection Act (1999). p. 14. Canadian Environmental Law Association (CELA) publication # 504, ISBN #1-897043-33-3.

[4] Mittelstaedt, M. (2006, February 4). US acts against chemical in non-stick pans: Washington wants cuts to controversial emissions while Health Canada holds off. Globe and Mail. p.A6.

[5] BSEF (Bromine Science and Environmental Forum). (2003). Major brominated flame retardants volume estimates. BSEF, Brussels. Retrieved from: Environment Canada. (2004, February). Environmental Screening and Assessment Report on Polybrominated Diphenyl Ethers (PBDEs). p.4.

[6] Page, S. and S. Allan. (2005, April 16). Not so pretty: most beauty routines include the use of carcinogens, allergens and other harmful substances. Ottawa Citizen.

[7] Environment Canada. (2004). Clean Air Online: Pollution Sources: Consumer and Commercial Products. Retrieved online April 24, 2006 from http://www.ec.gc.ca/cleanair-airpur/Commercial_and_Consumer_Products-WS2B169239-1_En.htm

[8] Cooper, K. et al. (2000, May 25). Environmental Standard Setting and Children's Health. Canadian Environmental Law Association and Ontario College of Family Physicians. Toronto. p.51.

[9] Muñoz-de-Toro, M. et al. (2005). Perinatal exposure to bisphenol A alters peripubertal mammary gland development in mice. Endocrinology. v.146, n.9, pp. 4138-4147.

Timms, B. G. et al. (2005). Estrogenic chemicals in plastic and oral contraceptives disrupt development of fetal mouse prostate and urethra. Proceedings of the National Academy of Sciences of the United States of America. v.102, n.19, pp.7014-7019.

Cone, M. (2005, May 3). Estrogen imitator in womb may lead to cancer in men, study finds. LA Times.

Meikle, J. (2005, May 30). Scientists link plastic food containers with breast cancer. Guardian.

[10] Greenpeace Research Laboratories. (2003, May). Consuming Chemicals: Hazardous Chemicals in House Dust as an Indicator of Chemical Exposure in the Home. Retrieved online May 9, 2006 fromhttp://eu.greenpeace.org/downloads/chem/Consuming%20Chemicals.pdf

[11] Jones-Otazo, H.A. et al. (2005). Is house dust the missing exposure pathway for PBDEs? An analysis of urban fate and human exposure to PBDEs. Environmental Science and Technology. v.39. pp. 5121-5130.

[12] Carson, R. (2002). Silent Spring: 40th Anniversary Edition. Mariner Books/ Houghton Mifflin Company: New York. p.277.

[13] Cooper, K. et al. (2000, May 25). p.30.

[14] Toronto Public Health. (2005, September). Environmental Threats to Children: Understanding the Risks, Enabling Prevention. Technical Report. p.31.Retrieved online April 26, 2006 from http://www.toronto.ca/health/hphe/pdf/tr_environmental_threats_all.pdf

[15] Cooper, K. et al. (2000, May 25). p.30.

[16] Cooper, K. et al. (2000, May 25). p.35.

[17] Cooper, K. et al. (2000, May 25). p.35.

[18] Cooper, K. et al. (2000, May 25). p.34.

[19] Government of Canada. (2006). Children's Health and the Environment in North America: A First Report on Available Indicators and Measures-Country Report: Canada. p.20. Retrieved online February 16, 2006 from http://www.cec.org/files/PDF/POLLUTANTS/CountryReport-Canada-CHE_en.pdf

[20] Cooper, K. et al. (2000, May 25). p.34.

[21] Cooper, K. et al. (2000, May 25). p.34.

[22] Cooper, K. et al. (2000, May 25). p.34.

[23] Cooper, K. et al. (2000, May 25). p.35.

[24] Cooper, K. et al. (2000, May 25). p.35.

[25] Cooper, K. et al. (2000, May 25). p.36.

[26] Cooper, K. et al. (2000, May 25). p.36.

[27] Cooper, K. et al. (2000, May 25). p.36.

[28] Cooper, K. et al. (2000, May 25). p.36.

[29] Cooper, K. et al. (2000, May 25). p. 30.

[30] Canadian Partnership for Children's Health and the Environment (CPCHE). (2005, August). Child Health and the Environment: A Primer. p.21.

[31] Cooper, K. et al. (2000, May 25).p.30.

[32] Cooper, K. et al. (2000, May 25). p.48

[33] Cooper, K. et al. (2000, May 25). p.48

[34] Cooper, K. et al. (2000, May 25). p.48

[35] Cooper, K. et al. (2000, May 25). p.48

[36] Ross, D.P. and P. Roberts. (1999). Income and Child Well-Being: A New Perspective on the Poverty Debate.: Part One: Family Income and Children's Development. Canadian Council on Social Development. Retrieved online April 26, 2006 from http://www.ccsd.ca/pubs/inckids/1.htm

[37] Toronto Public Health. (2005, September). p.37

[38] Toronto Public Health. (2005, September). p.37

[39] Ross, D.P. and P. Roberts. (1999).
[40] Toronto Public Health. (2005, September). p.37.
[41] Toronto Public Health. (2005, September). p.37
[42] Toronto Public Health. (2005, September). p.38.
[43] Toronto Public Health. (2005, September). p.38.
[44] Toronto Public Health. (2005, September). p.18.
[45] Toronto Public Health. (2005, September). p.19.
[46] CPCHE. (2005, August). p.36.
[47] CPCHE. (2005, August). p. 51.
[48] CPCHE. (2005, August). p. 53
[49] Skakkebaek, N.E. et al. (2001, May). Testicular dysgenesis syndrome : an increasingly common developmental disorder with environmental aspects. Human Reproduction. v.16 n.5 pp.972-978.
[50] CPCHE. (2005, August). p. 46
[51] Cooper, K. et al. (2000, May 25). p.33.
[52] Canadian Cancer Society/National Cancer Institute of Canada. (2006, April). Canadian Cancer Statistics 2006. p.16. Retrieved online April 12, 2006 from http://129.33.170.32/vgn/images/portal/cit_86751114/31/21/935505792cw_2006stats_en.pdf.pdf
Based on Table 7.1 Age-Standardized Incidence Rates for Selected Cancer Sites, Males, Canada, 1977-2006, p.40; and Table 8.1 Age-Standardized Incidence Rates for Selected Cancer Sites, Females, Canada, 1977-2006. p.42.
[53] Canadian Cancer Society/National Cancer Institute of Canada. (2006, April).
Based on Table 7.1 Age-Standardized Incidence Rates for Selected Cancer Sties, Males, Canada, 1977-2006, p.40; and Table 8.1 Age-Standardized Incidence Rates for Selected Cancer Sites, Females, Canada, 1977-2006. p.42.
[54] Canadian Cancer Society/National Cancer Institute of Canada. (2006, April). p.16.
[55] Canadian Cancer Society. (2002, April 18). More young adults being diagnosed with cancer: first Canadian research in this area: Canadian Cancer Statistics 2002. Retrieved online August 19, 2005 from http://www.cancer.ca/ccs/internet/mediarelease list/0,3208,3172_210504898_333099_langId-en, 00.html
[56] Public Health Agency of Canada. (1999). Measuring Up: A Health Surveillance Update on Canadian Children and Youth. Retrieved online June 10, 2005 from http://www.phac-aspc.gc.ca/publicat/meas-haut/mu_h_e.html
[57] Canadian Cancer Society/National Cancer Institute of Canada. (2006, April). p.68.
[58] CPCHE. (2005, August). p.42.
[59] Cooper, K. et al. (2000, May). p. 71.
[60] CPCHE. (2005, August). p.42. and Canadian Association of Physicians for the Environment (CAPE). (2000). Children's Environmental Health Project: Childhood Cancer. Retrieved online April 19, 2006 from http://www.cape.ca/children/cancer2.html
[61] Steliarova-Foucher, E. et al. (2004, December). Geographical patterns and time trends of cancer incidence and survival among children and adolescents in Europe since the 1970s (the ACCIS project) : an epidemiological study. The Lancet. v.364, i.9451, pp. 2097-2105.
[62] CPCHE. (2005, August). p. 42.
[63] Government of Canada. (2006). p.17.
[64] Government of Canada. (2006). p.17.
[65] Government of Canada. (2006). p.iv.
[66] Canadian Cancer Society/National Cancer Institute of Canada. (2006, April). p.68.
[67] Canadian Cancer Society/National Cancer Institute of Canada. (2006, April). p.68.
[68] CPCHE. (2005, August). p.42. and CAPE. (2000).
[69] CPCHE. (2005, August). p. 42.
[70] Steliarova-Foucher, E. et al. (2004, December).
[71] Canadian Cancer Society/National Cancer Institute of Canada. (2006, April).
Based on Table 7.1 Age-Standardized Incidence Rates for Selected Cancer Sties, Males, Canada, 1977-2006, p.40; and Table 8.1 Age-Standardized Incidence Rates for Selected Cancer Sites, Females, Canada, 1977-2006. p.42.
[72] Canadian Cancer Society/National Cancer Institute of Canada. (2006, April).
Based on Table 7.1 Age-Standardized Incidence Rates for Selected Cancer Sties, Males, Canada, 1977-2006, p.40; and Table 8.1 Age-Standardized Incidence Rates for Selected Cancer Sites, Females, Canada, 1977-2006. p.42.
[73] Canadian Cancer Society/National Cancer Institute of Canada. (2006, April).
Based on Table 7.1 Age-Standardized Incidence Rates for Selected Cancer Sties, Males, Canada, 1977-2006, p.40; and Table 8.1 Age-Standardized Incidence Rates for Selected Cancer Sites, Females, Canada, 1977-2006. p.42.
[74] Canadian Cancer Society/National Cancer Institute of Canada. (2006, April).
Based on Table 7.1 Age-Standardized Incidence Rates for Selected Cancer Sites, Males, Canada, 1977-2006, p.40; and Table 8.1 Age-Standardized Incidence Rates for Selected Cancer Sites, Females, Canada, 1977-2006. p.42.
[75] Learning Disabilities Association of Saskatchewan. Statistics. (Based on the 1970 Commission on Emotional and Learning Disorders in Children.) Retrieved online June 9, 2005 at

http://www.ldas.org/statistics.htm
[76] McElgunn, B, Phillips, A., Kasperski, J., Ogilivie, K., and Cooper, K. (2003, June 10). Letter to Anne McLellan, Minister of Health, regarding action needed on PBDEs. Learning Disabilities Association of Canada, South Riverdale Community Health Centre, Ontario College of Family Physicians, Pollution Probe, Canadian Environmental Law Association. (Statistic is based on 2002 National Longitudinal Survey of Children and Youth). Retrieved online March 16, 2005 at http://www.healthyenvironment-forkids.ca/english/resources/fulltext.shtml?x=1857
[77] Pastuszak. A.L. (2000). Improving our knowledge of birth defects. The Hospital for Sick Children Journal. v.2 i.4. Retrieved April 21, 2006 from http://www.sickkids.on.ca/journal/vol2issue4/birthdefects.asp
[78] Public Health Agency of Canada. (2002). Congenital Anomalies in Canada. p. xi. Retrieved April 20, 2006 from http://www.phac-aspc.gc.ca/publicat/cac-acc02/pdf/cac2002_e.pdf
[79] Hughes, E.G. and A. Moore. (2004). Clinical Focus: Current Issues in Diagnosis and Treatment of Infertility: Riding the Infertility Rollercoaster. Canadian Fertility and Andrology Society. Retrieved online April 20, 2006 from http://www.cfas.ca/english/content/pdf/ClinicalFocus2004-Infertility.pdf
[80] Laskin, C. Recurrent Miscarriage: A Common Problem with an Uncommon Solution. Infertility Awareness Association of Canada. Retrieved online April 20, 2006 from http://www.iaac.ca/english/articles/recurrent.asp
[81] Cooper, K. et al. (2000, May 25) p.50.
[82] Houlihan, J. (2003, January). Body Burden: The Pollution in People. Environmental Working Group. p.9.
[83] Houlihan, J. (2003, January). p.9.
[84] Houlihan, J. (2003, January). p. 51.
[85] Houlihan, J. (2003, January). p.41.
[86] Houlihan, J. (2003, January). p.7.
[87] Houlihan, J. (2003, January). p. 41.
[88] Houlihan, J. (2003, January). pp.44-45.
[89] Houlihan, J. (2003, January). p.8.
[90] Houlihan, J. (2003, January). p.10.
[91] Akron Health Department. (2002). CDC (Centers for Disease Control) may lower the acceptable blood lead level in children. Lead Issues. v.5 i.2. Retrieved online April 28, 2006 from http://www.ci.akron.oh.us/Health/Promotions/health_issues_v5_i2.htm
[92] US Environmental Protection Agency (EPA). (2003, February). America's Children and the Environment: Measures of Contaminants, Body Burdens and Illnesses. 2nd ed. Retrieved online April 13, 2006 from www.epa.gov/envirohealth/children
[93] Government of Canada. (2006). p.20.
[94] Cooper, K. et al. (2000, May 25). p. 50.
[95] Houlihan, J. et al. (2005, July 14). Body Burden: The Pollution in Newborns. Environmental Working Group. And WWF UK. (2005, September). A Present for Life: Hazardous Chemicals in Umbilical Cord Blood.
[96] Green Media Toolshed. (2005). Scorecard: Chemical Profiles. Retrieved online April 16, 2006 from http://www.scorecard.org/chemical-profiles/
[97] Environmental Defence. (2005, November).
[98] Kannan, K. et al. (2004). Perfluorooctanesulfonate and related fluorochemicals in human blood from several countries. Environmental Science and Technology. v. 38, n.17, pp. 4489-4495.
[99] Ryan, Jake. (2004). Polybrominated Diphenyl Ethers (PBDEs) in Human Milk: Occurrence Worldwide. Health Canada, Health Products and Food Branch, Ottawa, Ontario, Canada. Mittlestaedt, Martin. (2004, June 7). Flame retardant in breast milk raises concern: Canadian women have second-highest level in world, Health Canada finds. Globe and Mail. Toronto.
[100] Environment Canada. (2004, February). Environmental Screening Assessment Report on Polybrominated Diphenyl Ethers (PBDEs). p.14.
[101] Environment Canada. (2004). Frequently Asked Questions: Polybrominated Diphenyl Ethers (PBDEs). Retrieved online March 13, 2006 from http://www.ec.gc.ca/CEPARegistry/documents/subs_list/PBDE_draft/PBDEfaq.cfm
[102] Environmental Defence. (2006, May 23). Pollution Summary for Canada, 2003. Available online at http://www.pollution-watch.org/country.do;jsessionid=44185392FEE45F8649B5CDA887EC6D4A
[103] Houlihan, J. et al. (2003, January). p.6-10.
[104] Houlihan, J. et al. (2003, January). p. 10.
[105] US Environmental Protection Agency (EPA). (2006, April 12). News release: EPA report shows decrease in toxic chemicals released. Washington, D.C.
[106] Cooper, K. et al. (2000, May 25). Chapter 7: Consumer Products, in Environmental Standard Setting and Children's Health. Canadian Environmental Law Association and Ontario College of Family Physicians. pp.215-216.
[107] Great Lakes Regional Collaboration of National Significance. (2005, December). Great Lakes Regional Collaboration Strategy to Restore and Protect the Great Lakes. http://www.glrc.us/documents/GLRC_Strategy.pdf
[108] For the full report, visit http://www.oag-bvg.gc.ca/domino/reports.nsf/html/c101list_e.html

Glossary

Arsenic
Most exposures to the heavy metal arsenic come from wood that is pressure-treated with Chromated Copper Arsenate (CCA), which is found in playgrounds, fences, decks and other constructions. (Manufacturers of CCA wood stopped producing in at the end of 2003, although stores can still sell the wood until the stockpiles are gone). Arsenic is a carcinogen and has been shown to cause lung, skin, bladder, liver, kidney and prostate cancer. Arsenic can also cause blood disorders, cardiovascular diseases, and is a known hormone disruptor that affects metabolism and immune function.

Bioaccumulation
Bioaccumulation is the increase in concentration of a substance in the tissues of a living organism throughout its lifetime. Everyday we are exposed to a mixture of substances through contaminated air, water, food and products. As exposure occurs, certain chemicals that are very slowly metabolized or excreted build up in the tissues of living organisms.

Bisphenol A
Bisphenol A is primarily used to make polycarbonate plastic (recycling # 7) food and beverage containers and epoxy resins that are used to line metal cans for foods, such as cans of soup. Bisphenol A can leach from these products as they age, to be subsequently ingested by people. Recent research has shown that this chemical is an estrogenic hormone disruptor that can cause reproductive damage and birth defects that may lead to prostate and breast cancer in adulthood.

Body burden
Body burden refers to the amount of a chemical, or a number of chemicals, stored in the body at a given time, especially a potential toxin in the body as the result of exposure.

Brominated Flame Retardants (BFRs)
Brominated flame retardants (BFRs) are used to slow the spread of fire in upholstered furniture, mattresses, curtains, carpets and electronics. BFRs contain PBDEs (polybrominated diphenyl ethers), a group of chemicals that are highly persistent and bioaccumulative; they are suspected hormone disruptors and can cause cancer, reproductive and developmental disorders. PBDEs are suspected of having particularly damaging effects on the thyroid (which controls brain development), and as a result, PBDEs may cause neurodevelopmental disorders such as learning disabilities and behaviour problems. PBDEs leach from products, and have been detected in house dust, human blood and breast milk.

Cadmium
Cadmium is a heavy metal that comes from both natural and man-made sources. Most exposures to cadmium come from pigments and bakeware, as well as electronic equipment, car parts, batteries, phosphate fertilizer, sludge applications in agriculture and contaminated food. This heavy metal is known to cause lung and prostate cancer, and is toxic to the gastrointestinal tract, the kidneys, and the respiratory, cardiovascular and hormonal systems.

Carcinogen
Any substance that can cause or aggravate cancer.

Domestic Substances List
The Domestic Substances List has been compiled under the Canadian Environmental Protection Act (CEPA).The list includes more than 23,000 substances that were manufactured in, imported into or used in Canada on a commercial scale from 1984 to 1986. Health Canada and Environment Canada are aiming to classify and assess all substances on the Domestic Substances List by September 2006. All substances not on the list are considered new and must be reported prior to importation or manufacture so that they can be assessed to determine if they are toxic.

Hormone disruptors (a.k.a. Endocrine disruptors)
Hormone or endocrine disruptors are substances that can interfere with the normal functioning of the hormone system of both people and wildlife in a number of ways to produce a wide range of adverse effects including reproductive, developmental and behavioural problems.

Hypospadias
Hypospadias is a condition that affects approximately one in 500 newborn males. This congenital defect results in the urethral opening being somewhere other than the tip of the penis. In severe cases, the penis is also deformed. In these instances, the condition is usually corrected through surgery. Less serious occurrences are often left alone but this can add to fertility problems when the man is older.

Lead
Lead is a heavy metal that occurs naturally in the environment and is produced from man-made sources. Most exposures to lead come from lead paint and emissions from industrial facilities like metal smelters. Other sources of exposure include crystal tableware, porcelain enamel and contaminated food. Lead is a suspected carcinogen, a known hormone disruptor, and can damage almost every organ and system in the human body, particularly the nervous system. Lead has been indicated as a cause of decreased men-

tal ability, developmental delays, behavioural disorders and reproductive defects.

National Pollutant Release Inventory (NPRI)

The National Pollutant Release Inventory is a program managed by Environment Canada. It is a database of information on annual releases to air, water, land and disposal or recycling from all sectors - industrial, government, commercial and others. The National Pollutant Release Inventory is the only legislated, nation-wide, publicly-accessible inventory of its type in Canada.

Neurodevelopmental Disorders

Neurodevelopmental disorders are disabilities in the functioning of the brain that affect a child's behaviour, memory, or ability to learn. These effects may result from exposure of the fetus or young child to certain environmental contaminants, though current data do not indicate the extent to which environmental contaminants contribute to overall rates of neurodevelopmental disorders in children. A child's brain and nervous system are vulnerable to adverse impacts from pollutants because they go through a long developmental process beginning shortly after conception and continuing through adolescence.

Neurotoxins

Exposure to chemical substances can cause adverse effects on the nervous system (neurotoxicity). Chemicals toxic to the central nervous system can induce confusion, fatigue, irritability, and other behavioural changes. Exposure to methyl mercury and lead cause central nervous system toxicity, and can also cause degenerative diseases of the brain (encephalopathy). Chemicals toxic to the peripheral nervous system affect how nerves carry sensory information and motor impulses from the brain to the rest of the body.

Organochlorine Pesticides

Organochlorine pesticides (OPs), such as DDT, were introduced in the 1940s. Many of their uses have been restricted because they persist in the environment. These chemicals are highly toxic and as a group of chemicals are recognized carcinogens and reproductive/developmental toxins, they are also suspected hormone disruptors and respiratory toxins. Organochlorine pesticides can enter the environment from direct application and runoff, emissions from waste incinerators, releases from manufacturing plants and disposal of contaminated waste in landfill.

Organophosphate Insesticides

A broad group of pesticides still in use that represent the most commonly used insecticides in agriculture and home uses. Organophosphate insecticides are suspected of causing cancer, reproductive, developmental and neurological disorders.

Organophosphate Insecticide Metabolites (Dialkyl phosphate metabolites)

Most organophosphate pesticides are metabolized in the body to measurable breakdown products known as dialkyl phosphate metabolites. Dialkyl phosphates themselves are not considered toxic, but they are markers of exposure to organophosphate insecticides.

Persistent

Compounds that are not easily broken down in the environment and therefore stay in the environment for a very long time are known as "persistent".

Perfluorooctane Sulfonate (PFOS)

A key ingredient in stain-repellants, PFOS is widely used in a variety of consumer products - from wrapping for microwave popcorn to fire extinguishing foam. PFOS is a perfluorinated chemical, and although much more research is needed on these chemicals, existing studies have shown that perfluorinated chemicals are extremely persistent. Studies also suggest that these chemicals can cause cancer and disrupt hormones.

Perfluorooctanoic Acid (PFOA)

PFOA belongs to a group of perfluorinated chemicals (PFCs) that are widely used in consumer products for their resistance to environmental breakdown. PFOA and its precursors (substances that under the right conditions form into PFOA) are most commonly used to make non-stick cookware, and stain and water repellents on clothes, upholstery and carpeting. As these types of products are used, harmful chemicals actually break away from the product and enter our household air and food-and our bodies. Although much more research is needed on the health impacts of perfluorinated chemicals, existing studies have shown that PFCs are extremely persistent and can cause numerous types of cancer, as well as neurological and reproductive defects.

Phthalates

Phthalates are a group of man-made chemicals that are widely used as plasticizing additives in a broad range of consumer products, including cosmetic and personal care products, PVC consumer products and construction materials. These chemicals are also used in synthetic fragrances to extend the scents' staying power. Phthalates are relatively persistent in the environment and have been found in drinking water, soil, household dust, wildlife, fatty foods (meat and dairy products) and in the blood and breast

milk of people. Scientific research has shown that phthalates disrupt hormones, and can cause birth defects of male reproductive organs.

Pollution Prevention Plans

The *Canadian Environmental Protection Act*, 1999 (CEPA 1999) gives the Minister of the Environment the authority to require the preparation and implementation of pollution prevention plans for CEPA 1999 toxic substances (substances that have been added to Schedule 1 of CEPA 1999).

Pollution prevention is defined in the *Canadian Environmental Protection Act* as "the use of processes, practices, materials, products, substances or energy that avoid or minimize the creation of pollutants and waste and reduce the overall risk to the environment or human health."

Polybrominated diphenyl ethers (PBDEs)

See brominated flame retardants.

PCBs (polychlorinated biphenyls)

PCBs have been banned in Canada since 1977, yet they continue to be released into the environment from sources in other countries, and from PCB-containing industrial equipment that is still in use here at home. PCBs are highly toxic and persistent chemicals that have been building up in wildlife and people through the process of bioaccumulation. PCBs cause many types of cancer and damage the nervous, immune and cardiovascular systems, leading to birth defects, brain damage and decreased immune function.

Reproductive/Developmental Toxins

Reproductive toxins can affect sexual behaviour, onset of puberty, sperm count, fertility, gestation time, pregnancy outcome, lactation and premature menopause. Developmental toxins, a subgroup of reproductive toxins, can cause adverse effects for the developing child, such as birth defects.

Respiratory Toxins

Respiratory toxins cause adverse effects to the structure or functioning of the respiratory system (nasal passages, pharynx, trachea, bronchi, and lungs), and produce a variety of acute and chronic pulmonary conditions, including local irritation, bronchitis, pulmonary edema, emphysema, and cancer.

Respiratory toxins include categories of substances like toxic gases, vapors from solvents, aerosols, and particulate matter. Ozone and fine particles are known to pose a significant threat to respiratory health. Ground-level ozone, the main component in smog, causes breathing problems, aggravates asthma, and increases the severity and incidence of respiratory infections

Testicular Dysgenesis Syndrome

Testicular dysgenesis syndrome (TDS) is a term that includes a number of male reproductive health disorders, including poor sperm quality, undescended testes, hypospadias and testicular cancer. Scientific research suggests these symptoms of testicular dysgenesis syndrome all originate during the development of the fetal testes.

Toxaphene

Toxaphene was one of the most widely used insecticides, but is now banned in many countries. People are most often exposed to toxaphene through their diet, especially if it includes fish from contaminated sources. Toxaphene has been measured in oils and fats, root vegetables, meats and grains.

Toxic

Materials that cause death, disease, or birth defects in organisms that ingest or absorb them. The quantities and exposures necessary to cause these effects can vary widely.

Virtual Elimination

Under the *Canadian Environmental Protection Act*, virtual elimination is the reduction of releases to the environment of the most dangerous toxic substances to a level below which these releases cannot be accurately measured.

VOCs (Volatile and Semi-volatile organic compounds)

VOCs, such as the chemicals xylene, benzene, and toluene, are found in many household products, including paints, varnishes, paint stripping products, and adhesives. VOCs are air borne particles that contribute to poor air quality indoors and out. VOCs are one of the building blocks of smog, and are toxic to the nervous system. Some VOCs are cancer-causing. The health effects of different VOCs range from damage to the reproductive, neurological and respiratory systems, birth defects, and impaired kidney and liver function.

Measurements

µg/L (microgram per litre), equivalent to parts per billion (ppb)
ng/mL (nanogram per millilitre), equivalant to parts per billion (ppb)
µmol/L (micromoles per litre)
nmol/L (nanomoles per litre)

Appendix 1. Sampling and Analytical Methodology

Laboratories:

- Centre de Toxicologie, Institut National de Santé Publique du Québec (INSPQ) in Ste-Foy, Quebec, conducted the analysis for:
  - heavy metals, polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs), organochlorine pesticides, organophosphate insecticide metabolites, and polycyclic aromatic hydrocarbons (PAHs)
- AXYS Analytical Services in Sidney, British Columbia, conducted the analysis for:
  - perfluorinated chemicals (PFCs)

Polychlorinated Biphenyls (PCBs), Organochlorine Pesticides, Polybrominated Diphenyl Ethers (PBDEs)

Compounds	**PCBs:** PCB Aroclor 1260, PCB-28, PCB-52, PCB-99, PCB 101, PCB-105, PCB-118, PCB 128, PCB 138, PCB-153, PCB-156, PCB 163, PCB-170, PCB-180, PCB 183, PCB 187 **Organochlorine pesticides:** Aldrin, -chlordane, -chlordane, -HCH, Cis-nonachlore, p,p'-DDT, p,p'-DDE, Hexachlorobenzene, Oxychlordane, Mirex, Trans-nonachlore, toxaphene 26, Toxaphene 50 **PBDEs:** PBDE 47, PBDE 99, PBDE 100, PBDE 153, PBB 153
Specimen	Plasma
Specimen collection container	10 mL glass Lavender top (EDTA) Becton Dickinson Vacutainers
Specimen collection	Immediately invert tube 8 to 10 times. Cool slowly to 4°C. Centrifuge for 10 minutes. Transfer plasma using a plastic transfer pipette into shipping container.
Shipping container	Pre-cleaned 7 mL screw-cap glass tube with Teflon disc
Storage	4°C (samples to reach laboratory within 3 days)
Shipping	Shock-resistant cooler. Include ice pack.
Methodology	Gas Chromatography Mass Spectrometry (GC-MS)
Detection limit	0.01 µg/L (ppb)

Organophosphate Insecticide Metabolites

Compounds	Diethyl phosphate, Dimethyl phosphate, Diethyl thiophosphate, Dimethyl thiophosphate, Diethyl dithiophosphate, Dimethyl dithiophosphate
Specimen	Urine
Specimen container	125 mL polyethylene bottle
Storage	4°C (-20oC)
Shipping	Shock-resistant cooler. Include ice pack.
Methodology	Gas Chromatography Mass Spectrometry (GC-MS)
Detection limit	1 µg/L

Heavy Metals

Compounds	Arsenic, Cadmium, Manganese, Lead, Mercury
Specimen	Whole blood
Specimen container	6 mL Lavender top (EDTA) Becton Dickinson Vacutainers (plastic)
Specimen collection	Immediately invert tube eight to 10 times.
Storage temperature	4°C (maximum 1 month)
Shipping	Shock-resistant cooler. Include ice pack.
Methodology	Inductively Coupled Plasma Mass Spectrometry (ICP-MS) Graphite furnace atomic absorption spectrometry (Manganese)
Detection limits	Arsenic 3 nmol/L Cadmium 0.4 nmol/L Lead 0.001 µmol/L Manganese 7 nmol/L Mercury 0.5 nmol/L

Polycyclic Aromatic Hydrocarbons (PAHs)

Compounds	3-OH-benz(a)-anthracene, 3-OH-chrysene, 6-OH-chrysene, 3-OH-fluoranthene, 1-OH-phenanthrene, 2-OH-phenanthrene, 3-OH-phenanthrene, 4-OH-phenanthrene, 1-OH-pyrene
Specimen	Urine
Specimen container	125 mL Nalgene bottle
Specimen collection	Collect a spot sample preferably the first morning sample.
Storage temperature	Maintain frozen at -20oC
Shipping	Shock-resistant cooler. Include ice pack.
Methodology	Gas Chromatography Mass Spectrometry (GC-MS)
Detection limit	~0.1 µg/L

Perfluorinated Chemicals (PFCs)

Compounds	PFBA, PFPeA, PFHxA, PFHpA, PFOA, PFNA, PFDA, PFUnA, PFDoA, PFBS, PFHxS, PFOS, PFOSA
Specimen	Serum
Specimen collection container	7 mL Red top (EDTA) Becton Dickinson Vacutainers (plastic)
Specimen collection	Avoid all contact of samples with Teflon, glass surfaces, sticky labels and adhesive tape. For each lot of number of vacutainers retain a single vacutainer unused and sealed to send to laboratory as a blank. Clot at room temperature for 60 minutes. Centrifuge vacutainer as soon as possible (same day). Decant/pour the serum into lavender top plastic vial. If using glass pipettes do not pipette the serum out.
Storage temperature	Refrigerate if sending within 24-48 hours, otherwise freeze.
Shipping	Place cool or frozen samples upright in shock-resistant cooler with ice packs.
Methodology	Liquid chromatography Mass spectrometry (LC-MS/MS)
Detection limit	0.5 - 0.8 ng/mL for a 0.5 mL serum sample

Appendix 2. List of Chemicals Tested and their Health Effects

Heavy Metals (5)

Cadmium	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor, respiratory toxin, neurotoxin
Lead	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor, respiratory toxin, neurotoxin
Manganese	*Suspected:* Respiratory toxin, reproductive/developmental toxin, neurotoxin
Arsenic	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor, respiratory toxin, neurotoxin
Mercury	*Recognized:* Reproductive/developmental toxin *Suspected:* Hormone disruptor, respiratory toxin, neurotoxin

Polybrominated Diphenyl Ethers (PBDEs) (5)

PBB 153	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor, neurotoxin
PBDE 100	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor, neurotoxin
PBDE 153	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor, neurotoxin
PBDE 47	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor, neurotoxin
PBDE 99	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor, neurotoxin

Polychlorinated Biphenyls (PCBs) (16)

PCB Aroclor 1260	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor
PCB-101	*Recognized:* Carcinogen, reproductive/developmental toxin
PCB-105	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor
PCB-118	*Recognized:* Carcinogen, reproductive/developmental toxin
PCB-128	*Recognized:* Carcinogen, reproductive/developmental toxin
PCB-138	*Recognized:* Carcinogen, reproductive/developmental toxin
PCB-153	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor
PCB-156	*Recognized:* Carcinogen, reproductive/developmental toxin
PCB-163	*Recognized:* Carcinogen, reproductive/developmental toxin

PCB 170	*Recognized:* Carcinogen, reproductive/developmental toxin
PCB-180	*Recognized:* Carcinogen, reproductive/developmental toxin
PCB-183	*Recognized:* Carcinogen, reproductive/developmental toxin
PCB-187	*Recognized:* Carcinogen, reproductive/developmental toxin
PCB-28	*Recognized:* Carcinogen, reproductive/developmental toxin
PCB-52	*Recognized:* Carcinogen, reproductive/developmental toxin
PCB-99	*Recognized:* Carcinogen, reproductive/developmental toxin

Organochlorine Pesticides (13)

Aldrin	*Recognized:* Carcinogen *Suspected:* Hormone disruptor, respiratory toxin, reproductive/developmental toxin, neurotoxin
a-chlordane	*Suspected:* Hormone disruptor
Cis-nonachlor	*Suspected:* Hormone disruptor
g-chlordane	*Recognized:* Carcinogen *Suspected:* Hormone disruptor, respiratory toxin, reproductive/developmental toxin, neurotoxin
Hexachlorobenzene	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor, neurotoxin
Mirex	*Recognized:* Carcinogen *Suspected:* Hormone disruptor
Oxychlordane	*Suspected:* Hormone disruptor
toxaphene parlar 26	*Recognized:* Carcinogen *Suspected:* Hormone disruptor, respiratory toxin, reproductive/developmental toxin, neurotoxin
toxaphene parlar 50	*Recognized:* Carcinogen *Suspected:* Hormone disruptor, respiratory toxin, reproductive/developmental toxin, neurotoxin
p,p'-DDE	*Recognized:* Carcinogen *Suspected:* Hormone disruptor, neurotoxin
p,p'-DDT	*Recognized:* Carcinogen, reproductive/developmental toxin *Suspected:* Hormone disruptor, respiratory toxin, neurotoxin
b-HCH	*Recognized:* Carcinogen *Suspected:* Hormone disruptor, reproductive/developmental toxin, neurotoxin
Trans-nonachlor	*Suspected:* Hormone disruptor, neurotoxin

Perfluorinated Chemicals (PFCs) (13)

PFBA	No data on health effects
PFPeA	No data on health effects
PFHxA	No data on health effects
PFHpA	No data on health effects
PFOA	*Suspected:* Carcinogen, hormone disruptor, reproductive/developmental toxin

PFNA	No data on health effects
PFDA	No data on health effects
PFUnA	No data on health effects
PFDoA	No data on health effects
PFBS	No data on health effects
PFHsX	No data on health effects
PFOS	*Suspected:* Carcinogen, hormone disruptor, reproductive/developmental toxin
PFOSA	No data on health effects

Polycyclic Aromatic Hydrocarbons (PAHs) (10)

1-OH-benz(a)-anthracene	*Suspected:* Carcinogen, respiratory toxin, reproductive/developmental toxin
3-OH-benz(a)-anthracene	*Suspected:* Carcinogen, respiratory toxin, reproductive/developmental toxin
3-OH-chrysene	*Suspected:* Carcinogen, respiratory toxin, reproductive/developmental toxin
6-OH-chrysene	*Suspected:* Carcinogen, respiratory toxin, reproductive/developmental toxin
3-OH-fluoranthene	*Suspected:* Carcinogen, respiratory toxin, reproductive/developmental toxin
1-OH-phenanthrene	*Suspected:* Carcinogen, respiratory toxin, reproductive/developmental toxin
2-OH-phenanthrene	*Suspected:* Carcinogen, respiratory toxin, reproductive/developmental toxin
3-OH-phenanthrene	*Suspected:* Carcinogen, respiratory toxin, reproductive/developmental toxin
4-OH-phenanthrene	*Suspected:* Carcinogen, respiratory toxin, reproductive/developmental toxin
1-OH-pyrene	*Suspected:* Carcinogen, respiratory toxin, reproductive/developmental toxin

Organophosphate insecticide metabolites (6)

Diethyl phosphate	*Suspected:* Carcinogen, reproductive/developmental toxin, neurotoxin
Dimethyl phosphate	*Suspected:* Carcinogen, reproductive/developmental toxin, neurotoxin
Diethyl thiophosphate	*Suspected:* Carcinogen, reproductive/developmental toxin, neurotoxin
Dimethyl thiophosphate	*Suspected:* Carcinogen, reproductive/developmental toxin, neurotoxin
Diethyl dithiophosphate	*Suspected:* Carcinogen, reproductive/developmental toxin, neurotoxin
Dimethyl dithiophosphate	*Suspected:* Carcinogen, reproductive/developmental toxin, neurotoxin

Determining the Health Effects of a Chemical

- The determination of potential health effects of each chemical is based on Chemical Profiles provided in the Scorecard database (available at www.scorecard.org).
- Information is provided for five health effect categories: carcinogens, reproductive/developmental toxins, hormone disruptors, respiratory toxins and neurotoxins.
- Organophosphate insecticide metabolites. Health data for individual compounds was not available; however, Scorecard does provide an assessment for the group of chemicals, so all organophosphate insecticide metabolites were coded for the same health effects.
- Polybrominated diphenyl ethers (PBDEs). Health data for individual compounds was not available; however, Scorecard does provide an assessment for the group of chemicals, so all PBDEs were coded for the same health effects.
- Polycyclic aromatic hydrocarbons (PAHs). Health data for individual compounds was not available; however, Scorecard does provide an assessment for the group of chemicals, so all PAHs were coded for the same health effects.
- Perfluorinated chemicals (PFCs). No health data is available for the group of PFCs, and there is no health data available for individual chemicals, with the exception of PFOA and PFOS.

Appendix 3. An Overview of International Biomonitoring

While biomonitoring information on the Canadian population is lacking, governments and researchers in other countries have conducted studies on their respective populations. Much of the research has focused on specific chemicals, such as lead and mercury, perfluorinated chemicals (PFCs), polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs) and pesticides, rather than on capturing data on the cumulative body burden of people. In addition, the minimal amount of biomonitoring that has been conducted has mainly focused on measuring contaminant levels in adults.

Biomonitoring Campaigns–the Environmental Working Group (EWG), WWF UK, and Environmental Defence

Non-governmental organizations have carried out biomonitoring projects. In the US the Environmental Working Group (EWG) has conducted body burden tests on adults and umbilical cord blood. In *Body Burden: The Pollution in People*, EWG's first study, 167 of the 210 chemicals tested for were detected in the nine adults included in the study.[1] These chemicals included PCBs, organochlorine pesticides, dioxins and furans, metals, organophosphate insecticides, phthalates and volatile organic compounds (VOCs). In EWG's groundbreaking study, *Body Burden: The Pollution in Newborns*, 287 of the 413 chemicals tested for were detected in umbilical cord blood samples.[2] Once again, these chemicals included PCBs, organochlorine pesticides, PBDEs, mercury, perfluorinated chemicals, polycyclic aromatic hydrocarbons (PAHs), dioxins and furans and polychlorinated naphthalenes.

The United Kingdom branch of the World Wildlife Fund (WWF) has taken the lead across Europe in testing umbilical cord blood, adults, families, celebrities and politicians for their chemical content. In its first study, *ContamiNation* (2003), WWF UK tested 155 adult volunteers for 78 synthetic chemicals, including pesticides, PCBs and PBDEs. The results showed that every person in the survey was contaminated by chemicals from each of the chemical groups tested.[3] WWF UK also tested 47 adult volunteers from 17 countries across Europe and found similar results. In *Contamination: The Next Generation*, a WWF UK study on the body burdens of 33 individuals (aged nine to 88 years) from seven families in Britain, they once again found that all volunteers were contaminated by a host of chemicals, and that in some cases the children were more contaminated by higher numbers and levels of 'newer' chemicals (such as PBDEs and perfluorinated chemicals) than their parents or grandparents.[4] In the WWF UK's latest biomonitoring study, 42 maternal blood samples and 27 umbilical cord blood samples tested positive for a range of artificial musks, alkylphenols, PBDEs, perfluorinated chemicals, phthalates, organochlorine pesticides, triclosan and bisphenol A.[5]

Polluted Children, Toxic Nation: A Report on Pollution in Canadian Families, builds on Environmental Defence's study, *Toxic Nation: A Report on Pollution in Canadians* (2005). In the first Toxic Nation study, Environmental Defence tested blood and urine samples from 11 adults from across Canada for the presence of 88 chemicals, including heavy metals, PBDEs, PCBs, PFOS, organochlorine pesticides, organophosphate insecticide metabolites and VOCs. Laboratory tests detected 60 of the 88 chemicals in the 11 adults, and on average 44 chemicals were detected in each volunteer.

The National Biomonitoring Program in the United States

Since 1999, the US Center for Disease Control and Prevention (CDC) has conducted bi-annual biomonitoring studies in which they have tested representative samples of the US population (ages one year and older) for over 100 chemicals (including metals, PAHs, PCBs, dioxins and furans, phthalates, phytoestrogens, and various types of pesticides, insecticides, and herbicides). The CDC's reports on these biomonitoring studies do not provide an overview of the average number of chemicals detected in people, but a review of the findings on individual chemicals shows that many chemicals known to harm human health were detected in the US population.[6]

Child Biomonitoring

Between 2000 and 2002, environmental health researchers, Sexton et al., conducted a body burden study of children (age 3-6 years) living in a poor inner-city neighbourhood in Minneapolis.[7] The children's blood samples were tested for 54 chemicals, including 11 VOCs, 11 organochlorine pesticides, 30 PCBs, lead and mercury. VOCs were detected in 42% of samples; the highest concentrations detected were for m-p-xylene (median 0.24 ng/mL), 1,4-dichlorobenzene (median 0.10 ng/mL), and toluene (median 0.10 ng/mL). Lead was detected in 98.3% of samples, at a median of 2.9 ug/dL, and mercury was detected in 51.5% of samples at a median of 0.20 µg/L. The most common pesticide detected was p,p'-DDE (a metabolite of DDT) at a median of 0.30 ng/g serum. Of the 30 PCBs, 16 were detected, with a total PCB median concentration of 0.08 ng/g serum.

Biomonitoring Studies on Individual Chemicals

Other governments and researchers across the globe have conducted biomonitoring tests in their respective countries for specific groups of contaminants, such as PBDEs, PFCs, metals and pesticides.

Polybrominated diphenyl ethers (PBDEs)

Many studies on PBDE concentrations in people have been based on breast milk samples, including a Canadian study that found a median concentration of 22 ppb.[8] International median PBDE levels in breast milk include: 2.9 ppb in Sweden; 2.8 ppb in Norway; 3.3 ppb in Holland; 2.9 ppb in Belgium; 6.6 ppb in Germany and the UK; 1.6 ppb in Japan; and 55 ppb in the US (Figure 1).[9] Previous median PBDE concentrations found in blood include 5.6 ppb in the UK[10] and 61 ppb in the US.[11] Measures of median PBDE concentrations in human adipose tissue include 77 ng/g lipid wt in New York[12] and 1.3 ng/g lipid wt in Japan. PBDEs have also been detected in indoor and outdoor air[13], house dust[14], soil, sediments of the Great Lakes[15], the food supply[16], and wildlife .[17]

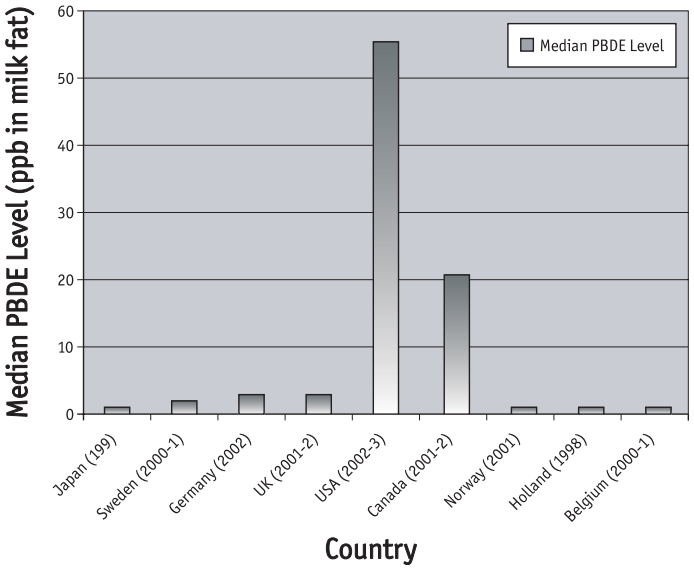
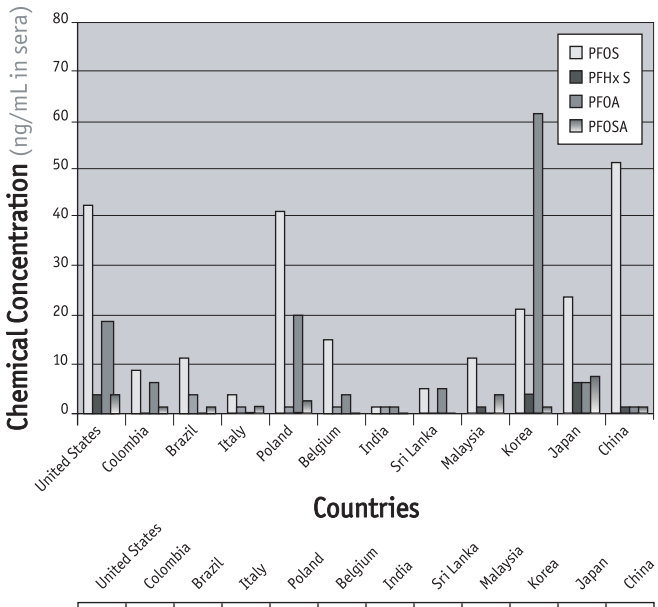


Figure 1. International comparison of PBDE levels in breast milk
Source: Ryan, Health Canada (2004).

Perfluorinated chemicals (PFCs)

Several international studies have measured levels of perfluorinated chemicals in people, young and old. Environmental Defence's Toxic Nation study on adults was the first to measure levels of perfluorinated chemicals in Canadians from across the country, and the results showed a median PFOS concentration of 10 µg/L in plasma.[18] A study on individuals in Nunavut and the Northwest Territories found a mean PFOS concentration of 36.9 ng/ml in plasma.[19] International measures of four perfluorinated chemicals in the blood of adults are summarized in Figure 2. In the US, perfluorinated chemicals have been measured in the serum of children aged 2-12 years at median concentrations of 36.7 ng/mL for PFOS, 5.1 ng/mL for PFOA, 3.8 ng/mL for PFHS, and 3.7 ng/mL for PFOSAA.[20] Allsopp et al. (2005) also provides an overview of PFC levels detected in the environment and in wildlife, as well as in people.[21]



	United States	Colombia	Brazil	Italy	Poland	Belgium	India	Sri Lanka	Malaysia	Korea	Japan	China
PFOS	43.2	8.28	11.7	4.32	42.1	15.7	1.85	5.03	12.7	21.1	24.6	52.7
PFHx S	4	0.2	3.77	1.64	1.3	1.04	1.6	.57	1.98	3.95	5.92	1.88
PFOA	19.1	6.16	0	0	21.3	4.82	2.64	6.38	0	61.8	6.4	1.59
PFOSA	4.14	1.57	1	1.78	2.06	0	0	0	4.57	1.3	7.92	1.82

Figure 2. International comparison of four fluorotelomer chemicals in human blood sera (ng/mL)*
*Median concentrations are provided.
Source: Yeung, L.W.Y. et al. (2006).

Lead

In a recent review of North American children's environmental health, the Commission for Environmental Cooperation (CEC) reported blood lead levels for children in Canada, the US and Mexico.[22] Although a blood lead level of 10 ug/dL is considered elevated, there is no demonstrated safe concentration of lead in blood, and adverse health effects can occur at lower levels.[23] The levels found in Canadian children were taken from Wang et al. (1997), which reported blood lead concentrations of 0.17 µmol/L in Toronto children, 0.15 µmol/L in children from Moosonee for the year 1992.[24] Mean blood lead levels in Mexican children were reported at 14.80 ug/dL for rural children aged three to six years[25], 12.60 ug/dL for urban children in Mexico City aged six to eight years[26] , and 9.70 ug/dL for semi-urban children aged five to 13 years.[27] The median concentration of blood lead in American children aged five years and under was 2.2 ug/dL in 1999-2000.[28]

Measures of blood lead levels in adults have also been documented. A global study conducted under the United Nations Environmental Program looked at lead levels in eight countries - Belgium, India, Israel, Japan, People's Republic of China, Sweden, USA, Yugoslavia. The study found that the geometric means for lead in blood ranged from 0 mug Pb/l in Beijing and Tokyo to 225 Pb/l in Mexico City. Other levels were < 100 mug Pb/l in Baltimore, Jerusalem, Lima, Stockholm and Zagreb, and 100-200 mug Pb/l in Brussels and India.[29] The geometric mean level reported by the CDC for the US population is 1.45 ug/dL.[30] In Canada, the median concentration of lead in the whole blood of the Toxic Nation study adult volunteers was 0.11345 µmol/L.[31]

Pesticides

Measurable levels of pesticides in people have been documented worldwide. In the CDC's most recent biomonitoring study of the US population p,p'-DDE (a metabolite of DDT) was detected at a geometric mean of 1.81 ng/g in serum, and TCPy (a metabolite of chlorpyrifos) was detected at a geometric mean of 1.76 µg/L (or 1.73 ug/g cre).[32] In Germany, urinary metabolites of pyrethoid insecticides were detected in an urban population of 1,177 people, including 331 children under 6 years and 247 children aged 6 to 12 years. The 95th percentile levels in urine were 0.30 µg/L for Br2CA; 0.51 µg/L for cis-Cl2CA; 1.43 µg/L for trans-Cl2CA; and 0.27 µg/L for F-PBA.[33] In a study on pregnant women from Southwest Quebec (aged 15 to 39 years), median plasma concentrations of organochlorine pesticides at delivery were recorded at 0.05 µg/L for trans-nonachlor, 0.06 µg/L for HCB (hexachlorobenzene), 0.04 µg/L for DDT, 0.47 µg/L for p,p'-DDE, and 0.05 µg/L for ß-BHC (beta hexachlorocyclohexane).[34] Children (aged 3 to 11 years) in a suburb of Seattle were found to have organophosphate pesticide metabolite levels in urine at median levels of 1.5 µg/L for MDA (a metabolite of malathion), and 6.0 µg/L for TCPy (a metabolite of chloropyrifos). After switching to an organic diet, the children's levels of these pesticide metabolites dropped immediately and significantly to 0 µg/L for MDA and 0.9 µg/L for TCPy.[35]

The overview of body burden studies provided here is by no means exhaustive. It indicates, however, a significant body of scientific research documenting the chemical contamination of people at all ages around the world. How we interpret these scientific findings of human contamination, and what we do with that information, is a matter of interest for concerned citizens, environmental health researchers, public health advocates, environmental groups, policy makers and industry. Regardless of who the stakeholder is, biomonitoring data should be used primarily to protect human health.

References

[1] Houlihan, J. et al. (2003, January). Body Burden: The Pollution in People. Environmental Working Group.

[2] Houlihan, J. et al. (2005, July 14).Body Burden: The Pollution in Newborns. Environmental Working Group.

[3] World Wildlife Fund, United Kingdom (WWF UK). (2004, October). Contamination: The Next Generation. p. 8. Retrieved online May 14, 2005 from http://www.wwf.org.uk/filelibrary/pdf/family_biomonitoring.pdf

[4] WWF UK. (2004, October). p.4.

[5] WWF UK. (2005, September). p.4.

[6] Centre for Disease Control and Prevention (CDC). (2005, July). Third National Report on Human Exposure to Environmental Chemicals.

[7] Sexton, K. et al. (2006). Using biological markers in blood to assess exposure to multiple environmental chemicals for inner-city children 3-6 years old. Environmental Health Perspectives. v.114, n.3, pp. 453-9.

[8] Ryan, John Jake. Health Canada, Health Products and Food Branch. (2004). Polybrominated Diphenyl Ethers (PBDEs) in Human Milk: Occurrence World wide.

[9] Ryan. (2004).

[10] WWF-UK. (2003). National Biomonitoring Survey. Retrieved

online April 7, 2005 at http://www.wwf.org.uk/filelibrary/pdf/biomonitoringresults.pdf

[11] Andreas Sjödin, Richard S. Jones, Jean-François Focant, Chester Lapeza, Richard Y. Wang, Ernest E. McGahee III, Yalin Zhang, Wayman E. Turner, Bill Slazyk, Larry L. Needham, and Donald G. Patterson Jr. (2004, May). Retrospective time-trend study of polybrominated diphenyl ether and polybrominated and polychlorinated biphenyl levels in human serum from the United States. Environmental Health Perspectives, v 112, n. 6. Table 1.

[12] Johnson-Restrepo, B. et al. (2005). Polybrominated diphenyl ethers and polychlorinated biphenyls in human adipose tissue from New York. Environmental Science and Technology. v.39, n14, 5177-5182.

[13] Wilford, B.H., et al..(2004). Passive sampling survey of polybrominated diphenyl ether flame retardants in indoor and outdoor air in Ottawa, Canada: implications for sources and exposure. Environment Canada. Environmental Science and Technology. v.38 n20, 5312-5318. and
Butt, C.M. et al. (2004). Spatial distribution of polybrominated diphenyl ethers in Southern Ontario as measured in indoor and outdoor window organic films. Environmental Science and Technology. v.38 n3, 724-731.

[14] Jones-Otazo, H.A. et al. (2005). Is house dust the missing exposure pathway for PBDEs? An analysis of the urban fate and human exposure of PBDEs. Environmental Science and Technology. v.39 n14, 5121-5130.

[15] Song, W. et al. (2005). Polybrominated diphenyl ethers in the sediments of the Great Lakes. 3. Lakes Ontario and Erie. Environmental and Science Technology. v.39 n15, 5600-5605.

[16] Schecter, A. et al. (2004). Polybrominated diphenyl ethers contamination of United States food. Environmental and Science Technology. v38 n20, 5306-5311.
Picard, A. and A. Favaro. (2005, February 14). Common foods laced with chemical: levels of PBDEs rise, new research shows. Globe and Mail. (Toronto). p.A1.

[17] Elliott, J.E., Wilson, L.K. and Wakeford, B. (2005). Polybrominated diphenyl ether trends in eggs of marine and freshwater birds from British Columbia, Canada, 1979-2002. Environmental Science and Technology. v.39 n15, 5584-5591.
Lindstrom, G. Wingfors, H., Dam, M. and Bavel B.V. (1999). Identification of 19 Polybrominated diphenyl ethers (PBDEs) in long-finned pilot whale (Globicephala melas) from the Atlantic. Archives of Environmental Contamination and Toxicology. v.36, 355-363.

[18] Environmental Defence. (2005, November). Toxic Nation: A Report on Pollution in Canadians. Available online: http://www.environmentaldefence.ca/toxicnation/report/Rev_English%20Web.pdf

[19] Tittlemier S., Ryan J.J. and Van Oostdam J. (2004). Presence of anionic perfluorinated organic compounds in serum collected from northern Canadian populations. Organohalogen Compounds. v.66, pp.4009-4014.

[20] Olsen, G.W. et al. (2004). Quantitative evaluation of perfluooctanesulfonate (PFOS) and other fluorochemicals in the serum of children. Journal of Children's Health. v.2, n.1, pp. 53-76.

[21] Allsopp, M. et al. (2005, April). Perfluorinated Chemicals: An Emerging Concern. Greenpeace Research Laboratories, University of Exeter, UK.

[22] Commission for Environmental Cooperation (CEC). (2006, January). Children's Health and the Environment in North America. pp.39-51. Retrieved online February 6, 2006 from http://www.cec.org/files/PDF/POLLUTANTS/CEH-Indicators-fin_en.pdf

[23] US Environmental Protection Agency (EPA). (2003, February). America's Children and the Environment: Measures of Contaminants, Body Burdens and Illnesses. 2nd ed. p.52. Retrieved online March 30, 2006 from http://www.epa.gov/envirohealth/children/ace_2003.pdf

[24] Wang, S.T. et al. (1997). Decline in blood lead in Ontario children correlated to decreasing consumption of leaded gasoline, 1983-1992. Clinical Chemistry. v.43, pp.1251-1252.

[25]Carrizales, L. et al. (2005). Exposure to arsenic and lead of children living near a copper-smelter in San Luis Potosí, Mexico: Importance of soil contamination for exposure of children. Environmental Research. In press.

[26] Romieu, I. et al. (1992). Vehicular traffic as a determinant of blood-lead levels in children: A pilot study in Mexico City. Archives in Environmental Health. v.47, n.4, pp. 246-49.

[27] Díaz-Barriga, F. et al. (1997). The El Paso smelter 20 years later: Residual impact on Mexican children. Environmental Research. v.74, pp. 11-16.

[28] US Environmental Protection Agency (EPA). p.53.

[29] Toxnet, Toxicology Data Network. (1983). Assessment of exposure to lead and cadmium through biological monitoring: Results of a United Nations Environmental Program-WHO global study. Environmental Resources. v. 20, n.1, pp. 95-128.

[30] Centers for Disease Control and Prevention. (2005). Third National Report on Human Exposures to Environmental Chemicals. Department of Health and Human Services. p.38.

[31] Environmental Defence. (2005, November). Toxic Nation: a report on pollution in Canadians. Toronto. Available online at http://www.environmentaldefence.ca/toxicnation/report/Rev_English%20Web.pdf

[32] Centre for Disease Control and Prevention (CDC). (2005, July).

[33] Heudorf, U. and J. Angerer. (2001, March). Metabolites of

pyrethroid insecticides in urine specimens: current exposure in an urban population in Germany. Environmental Health Perspectives. v.109, n.3, pp.213-217.
Full names of abbreviated compounds: cis-3-(2,2-dibromo-vinyl)-2,2-dimethylcyclo-propanecarboxylic acid (Br2CA), cis- and trans-3-(2,2-dichlorovinyl)-2,2-dimethylcyclopropane-carboxylic acid (cis-Cl2CA and trans-Cl2CA), and 4-fluoro-3-phenoxybenzoic acid (F-PBA).

[34] Takser, L. et al. (2005, August). Thyroid hormones in pregnancy in relation to environmental exposures to organochlorine compounds and mercury. Environmental Health Perspectives. v.113, n.8, pp.1039-1045.

[35] Chensheng, L. et al. (2006, February). Organic diets significantly lower children's dietary exposure to organophosphate pesticides. Environmental Health Perspectives. v.114, n.2, pp. 260-263.

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