

canadian partnership for



children's
health & environment

A Father's Day Report — Men, Boys and Environmental Health Threats

June 15, 2007



CPCHE gratefully acknowledges Danielle Schirmer, our health promotion intern, for her excellent work in preparing this report. We also thank those CPCHE partners and staff who helped with further research, writing and editorial review: Myriam Beaulne, Kathleen Cooper, Lynn Marshall, Barbara McElgunn, Caryn Thompson, Franca Ursitti and Loren Vanderlinden. As always, we thank Krista Friesen for her beautiful work on design and layout.

The Canadian Partnership for Children's Health and Environment (CPCHE) is a multi-sectoral collaboration of organizations (formed in 2001) that is working to protect children's health from environmental exposures and toxic chemicals by moving children's environmental health issues

into the minds of decision-makers, service provider organizations, individual practitioners, parents and the public. CPCHE members include:

Canadian Association of Physicians for the Environment
 Canadian Child Care Federation
 Canadian Environmental Law Association
 Environmental Health Clinic, Women's College Hospital
 Learning Disabilities Association of Canada
 Ontario College of Family Physicians
 Ontario Public Health Association
 Pollution Probe
 South Riverdale Community Health Centre
 Toronto Public Health



Table of Contents

Introduction	1
Boys at Risk	2
Cancer	2
Asthma	3
Learning and Developmental Disorders and Disabilities	4
Birth Defects	5
Testicular Dysgenesis Syndrome: Starts in the Womb	6
Fathers' Exposures and Their Children's Health	8
Fathers' Exposures and Potential Child Health Impacts	9
Playing It Safe: Childproofing Tips for Fathers	11
Endnotes	12

Front cover photos courtesy of Mark Surman

Introduction

The health of all children living in Canada is at risk from exposure to environmental hazards. Hundreds of toxic substances, such as air pollutants and pesticides are known, or are suspected of contributing to adverse child health outcomes. Much remains to be understood about environmental links to adverse health impacts. In the meantime, it is better to be safe than sorry. Much can be done to reduce or prevent exposures.

According to Health Canada:

- 12.2 per cent of children in Canada have asthma¹
- 26 per cent of children between the ages of six and eleven have one or more learning or behavioural problems²
- Birth defects are the leading cause of infant death³
- Several cancers are on the rise among young adults⁴

For a number of these health outcomes boys seem to be particularly at risk. The prevalence of cancer, asthma, learning and behavioural disorders, and some birth defects appears to be greater among boys than among girls. Health outcomes where girls appear to be at higher risk include breast cancer among young women, asthma after the age of 15, and two birth defects, *spina bifida* and congenital hip dislocation.

The reasons that boys appear to be at greater risk for the health outcomes discussed in this report are largely unknown. Several reasons have been suggested including genetic, hormonal, and physiological differences between the sexes.

Of concern is the fact that, according to US biomonitoring studies, men appear to have a greater body burden of certain chemicals than women including lead, polychlorinated dibenzofurans and some polycyclic aromatic

hydrocarbons, organophosphate pesticides, and polychlorinated biphenyls (PCBs).⁵ Large scale biomonitoring studies have not yet been conducted in Canada that would help determine whether a similar difference exists in the Canadian population. It is not known why this male-female difference is the case, although differences in metabolism have been suggested,^{6, 7} as well as potential differences in levels of exposure (e.g., through work).⁸

A father's exposure to toxic chemicals can affect the health of his children — male or female. Numerous studies have found a link between paternal occupational exposures and negative child health outcomes including birth defects, cancer and developmental delays.

This report summarizes the information currently available on the rates of diseases and disabilities of boys compared to girls and what is known about the environmental links to these health impacts. The issue of male vulnerability to environmental hazards is an emerging area of scientific research and public education.⁹ More is known about the hazards of maternal exposures to toxic chemicals. The reduction or prevention

of maternal exposures remains very important for the health of all children. This report focuses on environmentally-linked diseases and disabilities amongst boys. It also examines the role fathers play in ensuring children's environmental health.

The focus of this Father's Day report is also on exposure to toxic chemicals

commonly encountered in the indoor and outdoor environment rather than on well-known hazards such as smoking, alcohol and drugs. We need to know more about the reasons why boys appear to be more vulnerable. In the meantime, both parents, and all members of society, can take action to prevent exposure to toxic chemicals.



Photo credit: Loren Vanderlinden

Boys at Risk

Cancer

Although childhood cancer is very rare in Canada it is a serious condition that has great impacts on affected children and families, including the potential for long-term (late) health effects in survivors.¹⁰ Although mortality from childhood cancer has improved in recent decades, it is still the leading cause of illness-related death for children more than one year of age.¹¹ According to the Canadian Cancer Society and the National Cancer Institute of Canada, more boys between the ages of 0 and 19 are diagnosed with cancer every year in Canada than girls.¹² The reasons for this sex difference are unknown.

Among young adults (aged 20–44) cancer incidence shows a steady increase, although death from these cancers is dropping as well. Since the 1970s, there has been an overall upward trend in cancer incidence among young adults (aged 20–44) — at a rate of more than two per cent per year, or just under 20 per cent per decade.¹⁴ Although cancer incidence remains greater among young women than among young men overall, the following cancers in particular have increased among young men:

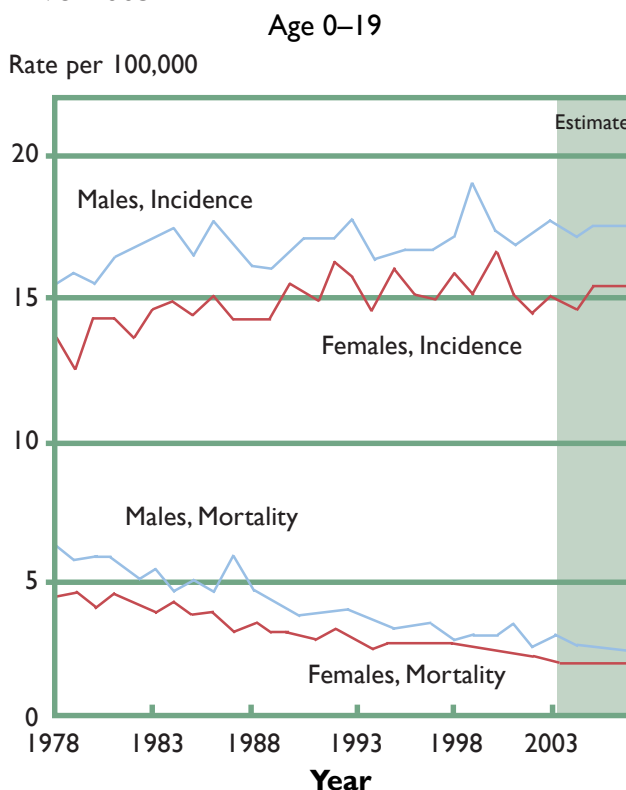
- Non-Hodgkin's lymphoma — 4.9 per cent per year increase between 1983 and 1994
- Testicular cancer — 2.2 per cent increase per year between 1983 and 1999

The causes of these increases are not known. Some of the increased incidence may be explained by improvements in diagnosis, but environmental health experts stress that it is not likely to be the whole explanation.¹⁵ Since cancers develop after a long latency period, early childhood, prenatal or exposures prior to conception may be a contributing factor. Exposures of both the mother and the father (prior to conception or during pregnancy) are of concern.^{16, 17, 18} Exposures that occur when cells are rapidly dividing (particularly during development in the womb) are thought to pose the greatest risk.¹⁹



Photo credit: Mark Surman

Incidence of All Childhood Cancers in Canada, 1978–2003¹³



Various cancers in children appear to be more likely with parental (often occupational) or childhood exposure to the following substances:²⁰

- Pesticides
- Solvents
- Petroleum products
- Motor vehicle exhaust
- Benzene
- Hydrocarbons

Childhood Cancer Statistics in Canada May Not Tell the Whole Story

The number of new cases of cancer per 100,000 children aged 0–19 per year increased in Canada between 1974 and 1984, but has not increased since that time. The lack of a clear upward trend in childhood cancer incidence could reflect Canada's relatively small population (especially since specific types of childhood cancer are relatively rare). In the US and the European Union — where the populations are larger — childhood cancer rates increased about one per cent per year over three decades. Steady increases occurred in the same cancers seen in children in other industrialized countries, including Canada. These cancers most often seen in children include leukemia, brain cancer and non-Hodgkin's lymphoma (NHL).²¹

Asthma

Asthma is an increasingly common childhood disease and it is more common in young boys than in young girls. In the 1996–1997 National Population Health Survey, asthma was reported in 12.2 per cent of children and youth in Canada under the age of 20 — up from just over two per cent in 1978.²² The prevalence rate of asthma was 13.3 per cent of boys aged 0–14 years, compared with only 9.5 per cent of girls.²³ Similarly, between 1980 and 1990, hospitalization of young children in Canada for asthma increased by 28 per cent among boys compared to 18 per cent among girls.²⁴

Why asthma is more common among boys is not clear but some researchers have suggested that it may be because they are born with smaller airways, relative to their lung size, than girls.^{25, 26} It may also be because they tend to have more allergies, which can predispose them to developing asthma.²⁷

While research continues on many fronts, scientists generally agree that asthma is the result of an interaction between genetic predisposition and environmental triggers.²⁸ Strong scientific evidence exists linking sources of indoor air pollution, such as dust mites and environmental tobacco smoke, to the development of asthma.²⁹ Similarly, there is considerable evidence linking exposure to outdoor air pollution (especially ozone) with the aggravation of asthma symptoms.³⁰ Outdoor air pollution may be related to the development of new-onset asthma, although this has yet to be conclusively demonstrated.³¹ Finally, there is suggestive evidence that pesticides and volatile organic compounds (VOCs) emitted from furniture, cleaning products and household furnishings may act as triggers.^{32, 33}

Learning and Developmental Disorders and Disabilities

The prevalence of neurobehavioural and neurodevelopmental effects among children in Canada is strikingly high:

- According to data collected for the 1997 National Longitudinal Survey on Children and Youth, 26 per cent of children in Canada aged 6 to 11 years had at least one identifiable emotional/behavioural, academic or social problem³⁴
- 16 per cent of children in Canada aged four to five years showed delayed vocabulary skills³⁵
- And, according to the Autism Society of Canada, autism rates in Canada have climbed from an estimated one in 10,000 children 20 years ago to an apparent rate of one in every 200 children (or 50 per 10,000).³⁶

For unknown reasons, boys are at an increased risk of having many of these disorders including autism, Attention Deficit Hyperactivity Disorder (ADHD), learning disabilities, Tourette's syndrome, cerebral palsy, and dyslexia.^{37, 38, 39, 40} Autism is approximately four times more common in boys than in girls,⁴¹ and according to



Photo credit: Canadian Association of Physicians for the Environment

the Learning Disabilities Association of Canada, among children with ADHD, boys outnumber girls by up to four to one.⁴²

The reasons why neurodevelopmental disabilities are more common in boys than girls are not well understood and have not been researched to any great extent.⁴³ Different explanations have been proposed including:⁴⁴

- Genetic differences
- A slower rate of biological maturation in males
- A greater vulnerability to accidental physical injuries in males
- A different pattern of prenatal hormone production with effects on the programming of brain development
- The fact that a greater number of stem cell divisions occur during male fetal development which gives rise to a greater risk of genetic errors
- Differences in brain structure, function and chemistry⁴⁵

Autism is a neurodevelopmental disorder characterized by difficulties with communication, impaired social interaction, and repetitive patterns of interests and behavior.⁴⁶

ADHD is a “neurobiological disability that interferes with a person’s ability to sustain attention or to focus on a task, and to control impulsive behaviour.”⁴⁷

Tourette’s syndrome is a “neurological or ‘neurochemical’ disorder characterized by tics — involuntary, rapid, sudden movements or vocalizations that occur repeatedly in the same way.”⁴⁸

Learning disabilities can be lifelong, range in severity, and result from impairments in processes related to learning. They may also impact organizational skills, social perception and social interaction.⁴⁹

"You get only one chance to develop your brain. Developmental neurotoxicity therefore has high priority in environmental health."

Source: Grandjean, P. 2004.

"Neurodevelopmental Disorders in Children: Some Old and Emerging Threats." *EEA/WHO/Collegium Ramazzini Workshop: Children in Their Environments*. Budapest, June 22.

A number of toxic substances in the environment are believed to contribute to the incidence of neurodevelopmental disabilities because they are neurotoxic, or in other words they have harmful effects on the developing brain and nervous system. Evidence of developmental neurotoxicity exists for a small number of substances. These include:^{50, 51, 52}

- Lead
- Methylmercury
- Arsenic
- Ionizing radiation
- Dioxins
- Some pesticides
- Solvents
- PCBs (industrial chemicals discontinued in the 1970s but still persistent in the environment)

However, scientists note that 201 chemicals have documented evidence of neurotoxicity in adults and over 1000 chemicals can cause neurotoxicity in laboratory studies. The ability of these or other chemicals to also cause neurotoxicity in the developing brain is poorly understood but of serious concern.⁵³

Evidence is increasing about substances that are similar in many important ways to dioxins and PCBs.⁵⁴ In particular, there is growing evidence from animal studies about the developmental neurotoxicity of the flame retardants known as PBDEs.^{55, 56, 57}

Birth Defects

Of the 350,000 babies born in Canada every year, two to three per cent are born with birth defects.⁵⁸ Overall, birth defects tend to be more common in boys than in girls.^{59, 60, 61} Note however that some specific defects are more common among girls (e.g., *spina bifida* and congenital hip dislocation).⁶² Canadian data do not indicate an overall upward trend in birth defects in recent decades.

Reproductive system defects, including cryptorchidism and hypospadias, account for about half of the increased risk of birth defects among boys.⁶³ In addition, some studies indicate that these two birth defects may be on the rise in industrialized countries across the last three to four decades.⁶⁴ Stillbirths and miscarriages — which are due in many cases to the presence of birth defects — also seem to be more common in boys.⁶⁵

Environmental factors have been identified as a cause with relative certainty in two to three per cent of all cases of birth defects.⁶⁶ Other known causes include genetic conditions and intra-uterine infections.⁶⁷ However, for the majority of defects — nearly 60 per cent — the cause is unknown.^{68, 69, 70} Experts agree that most birth defects likely result from multiple factors such as an interaction between one or more genes and the environment either prenatally or even prior to conception.⁷¹

Some environmental exposures are known to cause negative developmental outcomes, including birth defects. These include exposures of the mother or the father (before conception or during pregnancy) to high levels of:⁷²

- Lead
- Methylmercury
- Ionizing radiation
- PCBs contaminated by dioxins and furans

Other suspected chemicals include:⁷³

- Organic solvents
- Some pesticides
- Some air pollutants

Cryptorchidism is a condition where one or both testicles have not descended. It is a known risk factor for later development of testicular cancer.

Hypospadias refers to an abnormal opening of the urethra (or urinary tract) on the lower surface of the penis.

Environmental factors specifically associated with birth defects that are more common among boys have not been well studied. However, hypospadias in particular is associated with:⁷⁴

- Exposure to pesticides and dioxins
- Living near hazardous waste sites
- Maternal work in the leather industry
- Paternal work as vehicle mechanics

The apparent susceptibility of boys to birth defects is not well understood but several suggestions have been made:

- Fetal development of the male reproductive system occurs very rapidly making it more vulnerable to harm from exposures (rapidly growing cells have a greater potential to incorporate errors than those that grow more slowly).⁷⁵
- The development of the male reproductive system is more complex than the female reproductive system. The male system is dependent upon a progression from the female in the early embryo. With more developmental steps, greater opportunity arises for anomalies or birth defects to occur.⁷⁶
- In defects that originate in an X chromosome, females have a chance to “neutralize” this defect as they have two X chromosomes, whereas males only have one X chromosome.⁷⁷

Testicular Dysgenesis Syndrome: Starts in the Womb

Testicular Dysgenesis Syndrome (TDS) is a term used to describe a cluster of related reproductive effects that may have a common causal origin, including environmental exposures.⁷⁸ These effects include the birth defects cryptorchidism and hypospadias, poor semen quality (i.e., reduced sperm count, more abnormal sperm), lower fertility and perhaps also testicular cancer. Several researchers have noted that these male reproductive disorders appear to be increasing in most industrialized countries.^{79, 80, 81, 82, 83, 84} Between 1984 and 1996 there was a significant downward trend in sperm concentration in Canadian men,⁸⁵ and the Infertility Awareness Association of Canada estimates that infertility now affects over half a million Canadian men and women.⁸⁶



Photo credit: Mark Surman

Researchers hypothesize that TDS results from exposure to endocrine disrupting chemicals during pregnancy when male reproductive organs are developing.^{87, 88, 89} Interestingly, many endocrine disrupting chemicals began to be used in the 1970s when researchers started to observe TDS effects.^{90, 91, 92}

Endocrine disruptors are also suspected of being linked to the declining male to female sex ratio in many industrialized nations — that is, fewer male children are born every year.⁹³ Between 1970 and 1990, there was a decline of 2.2 males per 1000 live births in Canada.⁹⁴ Researchers suggest that endocrine disruptors may alter the normal path of male sexual development by altering the hormonal environment necessary to create the male reproductive structures. If certain male hormones are disrupted, the embryonic and fetal genital structures and gonads will develop differently and can appear feminized.⁹⁵ It is also thought that endocrine disruptors can trigger sex-specific mortality *in utero*.⁹⁶

What are Endocrine Disrupting Chemicals (EDCs)?

Often called “endocrine disruptors,” “hormone disruptors” or “endocrine toxicants,” EDCs are chemicals that can mimic or block hormones in our bodies. They can also be directly toxic to the endocrine system. The endocrine system coordinates and regulates communication between cells through hormones that act as chemical messengers, and it plays a crucial role in maintaining and coordinating normal growth, development and good health.⁹⁷

Substances known to be highly toxic and that are thought to be endocrine disruptors include:⁹⁸

- PCBs
- Dioxins and furans
- Organochlorine pesticides (e.g., DDT)⁹⁹

There is also limited but growing scientific evidence about the endocrine disrupting potential of the following substances:¹⁰⁰

- Phthalates — found in personal care products such as deodorant, cologne and aftershave, and in polyvinyl chloride plastic known as PVC or vinyl (identified by the #3 recycling symbol)
- Bisphenol A — found in soft drink cans, tin food cans, dental sealants, resins, dyes and polycarbonate plastic food and water containers (identified by the #7 recycling symbol)
- Brominated flame retardants such as PBDEs — found in many consumer products including foam, fabrics, casings for computers and electronic appliances
- Surfactants such as nonylphenol — used in detergents, cleaners, degreasers, paints, and some pesticides and cosmetics
- Lead, cadmium and mercury¹⁰¹ — toxic metals found in a variety of consumer products such as inexpensive jewellery (lead), thermometers or thermostats and paints (mercury), and some plastics (lead and cadmium).

People are exposed to these chemicals mainly through food and consumer products, although adult exposures in some work settings are likely higher than elsewhere (e.g., the application of pesticides by farmers, or the use of metals, vinyl chloride and other chemicals in many industrial processes).¹⁰² Experimental studies, mainly in animals, have shown that it takes only a relatively small amount of exposure to these chemicals to disrupt normal hormone functioning.¹⁰³

Fathers' Exposures and Their Children's Health

Numerous studies have found positive associations between fathers' exposures to environmental chemicals and fetal or child health problems (in both boys and girls) including low birth weight, spontaneous abortion, congenital anomalies, cancer and developmental delays.¹⁰⁴ Laboratory, epidemiological and animal studies all suggest that paternal exposures are important.¹⁰⁵

Some researchers think that fathers may be more vulnerable to harm from toxic chemicals than mothers in the sense that they are more likely to pass on damage from such exposures to the developing fetus.¹⁰⁶ This is because, once males reach maturity, sperm are constantly developing and are therefore continuously vulnerable to encountered harmful exposures.¹⁰⁷ Whether these exposures create greater vulnerability prior to conception or not, it is important to remember that maternal exposures to toxic chemicals are of equally serious concern.

Father's exposures have the potential to create risks across the entire course of fetal and child development.^{108, 109, 110, 111} The relative importance of different individual exposures and their origin and timing may vary. They may also occur simultaneously. For example, harmful exposures can occur prior to conception and lead to genetic mutations in sperm. Chemicals of concern can also be carried in seminal fluid and be transferred



Photo credit: Mark Surman

to the mother affecting conception or birth outcome. As well, fathers can bring home hazardous chemicals from the workplace, on their clothing, skin, hair, shoes or equipment, or they may create exposures at home through the use of pesticides or other toxic chemicals, especially during home renovations. These activities can in turn lead to maternal and then fetal exposure during pregnancy, or to contamination of the home environment during childhood.

The majority of studies on paternal exposures to toxic chemicals and their effects on child health have focused on exposures encountered in the workplace. These are highlighted in the table on the next page.

Fathers' Work Exposures and Potential Child Health Impacts

Chemicals of Concern	Work Settings	Potential Impacts
Some pesticides	Farms, pesticide production facilities, gardens and greenhouses, golf courses, etc.	<p>Fathers' work exposure to pesticides have been associated with a host of negative child health outcomes including:</p> <ul style="list-style-type: none"> • birth defects¹¹² • childhood cancers^{113, 114} • spontaneous abortion¹¹⁵ • developmental disorders^{116, 117} <p>In a study of male pesticide applicators working on farms, their children were found to have a higher rate of birth defects or developmental disorders in the first three years of life.¹¹⁸ This was especially the case in their male children.</p> <p>In a study of Ontario farmers "higher risks for miscarriage and prematurity...[were] found with direct paternal exposure to certain agricultural pesticides."¹¹⁹ Many studies have found that children of Vietnam veterans exposed to the banned herbicide Agent Orange, have an increased risk of <i>spina bifida</i>, cleft palate and tumors.¹²⁰</p>
Petroleum-derived hydrocarbons including motor vehicle exhaust, paints, solvents, etc.	Petroleum and chemical industries, rubber manufacturing, printing shops, motor vehicle related industries (e.g., body shops), painting and aircraft industries, hair salons, dry cleaners, electrical assembling, installing or repairing industries, etc.	<p>Fathers work exposures to solvents are associated with increased incidence of:¹²¹</p> <ul style="list-style-type: none"> • childhood cancer • spontaneous abortion • birth defects • low birth weight <p>More specifically, exposure to solvents and specifically chlorinated solvents, benzene, alcohols, methyl ethyl ketone, petroleum products and motor vehicle exhaust is associated with childhood leukemia.¹²²</p> <p>Exposure to benzene, alcohols, lacquer thinner, turpentine and hydrocarbons including diesel fuel is associated with neuroblastoma, a nerve cell cancer.¹²³</p> <p>Exposure to solvents and petroleum products is associated with acute non-lymphocytic leukemia.¹²⁴</p> <p>And exposure to vinyl chloride, organic solvents, toluene, xylene, gasoline, benzene, trichloroethane, and methylene chloride is associated with spontaneous abortions.^{125, 126, 127}</p> <p>There is some suggestive evidence of adverse effects on men's reproductive functioning from exposure to solvents. Glycol ethers are classified as toxic to reproduction in the European Union.¹²⁸</p>

Chemicals of Concern	Work Settings	Potential Impacts
Anesthetics	Operating rooms, dentists offices, veterinary clinics	Fathers' work exposure to inhalation anesthetics have been associated with: ^{129, 130} spontaneous abortions congenital malformations
Metals	Plate and steel industries, smelters, metal processing, welding industries, battery plants, car repair facilities	Paternal work exposures to various metals including mercury, lead, zinc and copper have been associated with an increased risk of: childhood cancers ^{131, 132} birth defects ¹³³ spontaneous abortions ^{134, 135, 136, 137} Some metals have also been linked to decreased sperm quality and fertility. ¹³⁸
Ionizing radiation	Nuclear power plants	Children of fathers exposed to low-level ionizing radiation at a nuclear plant in England had a six to eight fold increase risk of leukemia, although this finding remains controversial. ¹³⁹



Photo credit: Tatiana Morita

Playing It Safe: Childproofing Tips for Fathers

As a father, you can take steps to minimize the toxic substances that you, your partner and your children may be exposed to. You can do this at work, at home and in your community. Remember that these tips are just as important for mothers.

At work, become aware of possible environmental and occupational hazards. If you work with chemicals, or in construction or renovation, make sure you take all necessary precautions to protect yourself and your family:

- Wear protective clothing and equipment (e.g., masks, gloves, or other protections)
- Wash your hands when possible, especially before eating
- Change your clothes and shower when you get home if facilities are not available at your workplace
- Wash work clothes separately from other clothes
- Keep work equipment outside if possible (in the tool shed or garage for example)

For more information on the chemicals you may be exposed to on the job, and what you can do about them, contact the Canadian Centre for Occupational Health and Safety at 1-800-668-4284 or visit www.ccohs.ca.

At home, it is important to:

- Remove shoes at the door
- Wet dust, vacuum and ventilate your home regularly
- Minimize your use of toxic chemicals: buy personal care products and cleaning products that are less toxic. See www.lesstoxicguide.ca for a list of safe alternatives
- If your hobbies involve the use of hazardous substances make sure these are not practiced in the living areas of the house, that your workspace is kept well ventilated, and that you wear protective clothing



Photo credit: Mark Surman

For many more useful tips, see *Child Health and the Environment — A Primer* and the *Playing It Safe* brochure available at www.healthyenvironmentforkids.ca.

And in the community, as fathers you can:

- Become aware of the chemicals your children may be exposed to in childcare facilities, schools, playgrounds, parks, libraries, sports fields and arenas. Ask what products are being used — particularly cleaning products and pesticides — and whether they have been evaluated for health impacts. Find out if alternative products or approaches have been considered.
- Start or support campaigns to reduce pesticide use, promote energy efficiency, and reduce greenhouse gas emissions, etc.
- Voice your concerns to your elected officials — many issues require policy change at the municipal, provincial or federal level.

For more information on ways you can get involved and steps that you can take to ensure a healthy future for you and your children visit the Canadian Partnership for Children's Health and Environment at www.healthyenvironmentforkids.ca.

Endnotes

- ¹ Health Canada. 1999. *Measuring Up: A Health Surveillance Update on Canadian Children and Youth*. Rusen, ID and C McCourt, eds. Accessed at www.phac-aspc.gc.ca/publicat/meas-haut.
- ² Landy, S and KK Tam. 1998. "Understanding the Contribution of Multiple Risk Factors on Child Development at Various Stages. National Longitudinal Study in Children and Youth." Workshop paper given at *Investing in Children, A National Research Conference*.
- ³ Health Canada. 1999.
- ⁴ Cancer Care Ontario. 2006. *Cancer in Young Adults in Canada*. Accessed at www.cancercare.on.ca/pdf/CYAC2006E.pdf
- ⁵ Centers for Disease Control and Prevention. 2005. *Third National Report on Human Exposure to Environmental Chemicals*. Atlanta: National Center for Environmental Health.
- ⁶ Centers for Disease Control and Prevention. 2005.
- ⁷ Arbuckle, TE. 2006. "Are There Sex and Gender Differences in Acute Exposure to Chemicals in the Same Setting?" *Environmental Research*, 101: 195–204.
- ⁸ Arbuckle. 2006.
- ⁹ Daniels, C. 2006. *Exposing Men: The Science and Politics of Male Reproduction*. New York: Oxford University Press.
- ¹⁰ Savitz, DA. 2001. Environmental exposures and childhood cancers: Our best may not be good enough. *Am J. Pub Health*, 91(4): 562–563.
- ¹¹ Health Canada. 1999.
- ¹² Canadian Cancer Society and the National Cancer Institute of Canada. 2007. *Canadian Cancer Statistics 2007*. Accessed at www.ncic.cancer.ca/vgn/images/portal/cit_86751114/21/40/1835950430cw_2007_stats_en.pdf
- ¹³ The Canadian Cancer Society and the National Cancer Institute of Canada. 2007. *Canadian Cancer Statistics 2007*. Accessed at www.ncic.cancer.ca/vgn/images/portal/cit_86751114/21/40/1835950430cw_2007_stats_en.pdf
- ¹⁴ Cancer Care Ontario. 2006.
- ¹⁵ Kaiser, J. 1999. "Epidemiology: No Meeting of Minds on Childhood Cancer." *Science Magazine*, 286(5446): 1832–1834
- ¹⁶ Gouveia-Vigeant, T and J Tickner. 2003. *Toxic Chemicals and Childhood Cancer: A Review of the Evidence*. Lowell, MA: Lowell Center for Sustainable Production.
- ¹⁷ Olshan, AF, L Anderson, E Roman, N Fear, M Wolff, R Whyatt, V Vu, BA Diwan and N Potischman. 2000. "Workshop to Identify Critical Windows of Exposure for Children's Health: Cancer Work Group Summary." *Environmental Health Perspectives*, 108(S3): 595–597.
- ¹⁸ Anderson, LM, BA Diwan, NT Fear and E Roman. 2000. "Critical Windows of Exposure for Children's Health: Cancer in Human Epidemiological Studies and Neoplasms in Experimental Animal Models." *Environmental Health Perspectives*, 108(S3): 573–594.
- ¹⁹ Olshan et al. 2000.
- ²⁰ Gouveia-Vigeant and Tickner. 2003.
- ²¹ Canadian Partnership for Children's Health and Environment (CPCHE). 2005. *Child Health and the Environment — A Primer*. Accessed at www.healthyenvironmentforkids.ca/Primer.pdf
- ²² Health Canada. 1999.
- ²³ Health Canada. 1999.
- ²⁴ Dales, RE, M Raizenne, S El-Saadany, J Brook and R Burnett. 1994. "Prevalence of Childhood Asthma Across Canada." *International Journal of Epidemiology*, 23(4): 775–781.
- ²⁵ Venn, A, S Lewis, M Cooper, J Hill and J Britton. 1998. "Questionnaire Study of Effect of Sex and Age on the Prevalence of Wheeze and Asthma in Adolescence." *British Medical Journal*, 316: 1945–1946.
- ²⁶ Clough, JB. 1993. "The Effect of Gender on the Prevalence of Atopy and Asthma." *Clinical and Experimental Allergy*, 23: 883–885.
- ²⁷ Clough. 1993.
- ²⁸ Wigle, DT. 2003. *Child Health and the Environment*. New York: Oxford University Press.
- ²⁹ Toronto Public Health. 2005. *Environmental Threats to Children, Understanding the Risks, Enabling Prevention*. Accessed at www.toronto.ca/health/hp/he/pdf/boh_environmental_threats_summary_all.pdf
- ³⁰ As summarized in Toronto Public Health. 2005.
- ³¹ As summarized in Toronto Public Health. 2005.
- ³² As summarized in Toronto Public Health. 2005.
- ³³ Wigle. 2003.
- ³⁴ Landy and Tam. 1998.
- ³⁵ Landy and Tam. 1998.
- ³⁶ Autism Society of Canada. 2004. *Canadian Autism Research Agenda and Canadian Autism Strategy. A White Paper*. Accessed at www.autismsocietycanada.ca/pdf_word/finalwhite-eng.pdf.
- ³⁷ Autism Society of Canada. 2004.
- ³⁸ US Department of Health and Human Services (USDHHS). 2005. *Summary Health Statistics for US Children: National Health Interview Survey, 2003*. National Center for Health Statistics. Vital Health Statistics. 10(223). Accessed at www.cdc.gov/nchs/data/series/sr_10/sr10_223.pdf.
- ³⁹ Thompson, T, M. Caruso and K Ellerbeck. 2003. "Sex Matters in Autism and Other Developmental Disabilities." *Journal of Learning Disabilities*, 7(4): 345–362.
- ⁴⁰ Rutter, M, A Caspi and TE Moffitt. 2003. "Using Sex Differences in Psychopathology to Study Causal Mechanisms: Unifying Issues and Research Strategies." *Journal of Child Psychology and Psychiatry*, 44(8): 1092–1115.
- ⁴¹ Autism Society of Canada. 2004.
- ⁴² Learning Disabilities Association of Canada (LDAC). 2005. *Fact Sheet #1: An Overview of ADHD*. Accessed at www.ldac-taac.ca/InDepth/pdf/1.pdf.

- 43 Thompson. 2003.
- 44 Rutter et al. 2003.
- 45 Cosgrove, KP et al. 2007. Evolving Knowledge of Sex Differences in Brain Structure, Function and Chemistry. *Biol. Psychiatry*, in press.
- 46 Autism Society of Canada. 2007. What is *Autism Spectrum Disorder*? Accessed at www.autismsocietycanada.ca/pdf_word/info_ASC%27swhatisautisminfosheet_19_04_07_e.pdf.
- 47 LDAC. 2005.
- 48 Tourette Syndrome Foundation of Canada. 2007. *Questions and Answers: What is TS?* Accessed at www.tourette.ca/qa.html.
- 49 Learning Disabilities Association of Canada (LDAC), 2002. Official Definition of Learning Disabilities. Adopted by the Learning Disabilities Association of Canada, January 30, 2002. On-line at www.ldac-taac.ca/Defined/pdf/jan02eng.pdf.
- 50 Grandjean, P and PJ Landrigan. 2006. "Developmental Neurotoxicity of Industrial Chemicals." *The Lancet*, 368(9553): 2167–2179.
- 51 Schettler, T, G Solomon, M Valenti and A Huddle. 1999. *Generations at Risk*. Cambridge, MA: The MIT Press.
- 52 Wigle. 2003.
- 53 Grandjean and Landrigan. 2006.
- 54 Toronto Public Health. 2005.
- 55 Birnbaum, LF and DF Staskal. 2004. "Brominated Flame Retardants: Cause for Concern?" *Environmental Health Perspectives*, 112(1): 9–17.
- 56 Alm, H, B Scholz, C Fischer, K Kultima, H Viberg, P Eriksson, L Dencker and M Stigson. 2006. "Proteomic Evaluation of Neonatal Exposure to 2,2',4,4',5-Pentabromodiphenyl Ether." *Environmental Health Perspectives*, 114(2): 254–259.
- 57 Dingemans, MML, GMJ Ramakers, F Gardoni, RGDM van Kleef, A Bergman, M Di Luca, M van den Berg, RHS Westerink and HPM Vijverberg. 2007. "Neonatal Exposure to Brominated Flame Retardant BDE-47 Reduces Long-Term Potentiation and Postsynaptic Protein Levels in Mouse Hippocampus." *Environmental Health Perspectives*, 115(6): 865–870.
- 58 Health Canada. 2002. *Congenital Anomalies in Canada — A Perinatal Health Report*. Ottawa: Minister of Public Works and Government Services Canada.
- 59 Cui, W, C Ma, Y Tang, V Chang, PV Rao, M Ariet, MB Resnick and J Roth. 2005. "Sex-Differences in Birth Defects: A Study of Opposite Sex Twins." *Birth Defects Research (Part A): Clinical and Molecular Teratology*, 73: 876–880.
- 60 Lary, JM and LJ Paulozzi. 2001. "Sex Differences in the Prevalence of Human Birth Defects: A Population-Based Study." *Teratology*, 64: 237–251.
- 61 Shaw, GM, SL Carmichael, Z Kaidarova and JA Harris. 2003. "Differential Risks to Males and Females for Congenital Malformations Among 2.5 Million California Births, 1989–1997." *Birth Defects Research (Part A): Clinical and Molecular Teratology*, 67: 953–958.
- 62 Cui et al. 2005.
- 63 Lary and Paulozzi. 2001.
- 64 Wigle. 2003.
- 65 Kraemer, S. 2000. "The Fragile Male." *British Medical Journal*, 321: 1609–1612.
- 66 Davis, DL, G Friedler, D Mattison and R Morris. 1992. "Male-Mediated Teratogenesis and Other Reproductive Effects: Biologic and Epidemiologic Findings and a Plea for Clinical Research." *Reproductive Toxicology*, 6: 289–292.
- 67 Davis et al. 1992.
- 68 Davis et al. 1992.
- 69 Friedler, G. 1996. "Paternal Exposures: Impact on Reproductive and Developmental Outcome. An Overview." *Pharmacology, Biochemistry and Behavior*, 55(4): 691–700.
- 70 Health Canada. 2002.
- 71 Mekdeci, B and T Schettler. 2004. *Birth Defects and the Environment*. Accessed at www.protectingourhealth.org/news/science/birthdefects/2004-0501birthdefectspreview.htm.
- 72 Toronto Public Health. 2005.
- 73 Toronto Public Health. 2005; For a more detailed list of recognized and suspected developmental toxicants visit www.scorecard.org.
- 74 Mekdeci and Schettler. 2004.
- 75 Davis, DL, MB Gottlieb and JR Stampnitzky. 1998. "Reduced Ratio of Male to Female Births: A Sentinel Health Indicator?" *Journal of the American Medical Association*, 279(13): 1018–1023.
- 76 Lary and Paulozzi. 2001.
- 77 Lary and Paulozzi. 2001. The authors note however that it is possible that birth defects of the female reproductive system are under-reported because they are harder to diagnose.
- 78 Skakkebaek, NE, E Rajpert-De Meyts and KM Main. 2001. "Testicular Dysgenesis Syndrome: An Increasingly Common Developmental Disorder with Environmental Aspects." *Human Reproduction*, 16(5): 972–978.
- 79 Bhasin, S. 2007. "Secular Decline in Male Reproductive Function: Is Manliness Threatened?" *The Journal of Endocrinology & Metabolism*, 92(1): 44–45.
- 80 Davis, DL, P Webster, H Stainthorpe, J Chilton, L Jones and R Doi. 2007. "Declines in Sex Ratio at Birth and Fetal Deaths in Japan and U.S. Whites, but not in African Americans." *Environmental Health Perspectives*. Accessed at www.ehponline.org/members/2007/9540/9540.pdf.
- 81 Bay, K, C Asklund, NE Skakkebaek and A Anderson. 2006. "Testicular Dysgenesis Syndrome: Possible Role of Endocrine Disruptors." *Best Practice & Research Clinical Endocrinology & Metabolism*, 20(1): 77–90.
- 82 Aitken, RJ, NE Skakkebaek and SD Roman. 2006. "Male Reproductive Health and the Environment: Are Xenobiotics in the Environment Affecting Fertility in Australian Men?" *Medical Journal of Australia*, 185(8): 414–415.
- 83 Carbone, P, F Giordano, F Nori, A Mantovani, D Taruscio, L Lauria and I Figa-Talamanca. 2007. "The Possible Role of Endocrine Disrupting Chemicals in the Aetiology of Cryptorchidism and Hypospadias: A

- Population-Based Case-Control Study in Rural Sicily." *International Journal of Andrology*, 30: 3–13.
- 84 Skakkebaek et al. 2001.
- 85 Younglai, EV, JA Collins and WG Foster. 1998. "Canadian Semen Quality: An Analysis of Sperm Density Among Eleven Academic Fertility Centers." *Fertility and Sterility*, 70(1): 76–80.
- 86 Toronto Public Health. 2005.
- 87 Bay et al. 2006.
- 88 Skakkebaek et al. 2001.
- 89 Arbuckle TE, SM Schrader, D Cole, JC Hale, CM Bancej, LA Turner and P Claman. 1999. "2,4-Dichlorophenoxyacetic Acid Residues in Semen of Ontario Farmers." *Reproductive Toxicology*, 13(6): 421–429.
- 90 Daniels. 2006.
- 91 Aitken et al. 2006.
- 92 Colborn, T, D Dumanowski and JP Myers. 1996. *Our Stolen Future: Are We Threatening Our Fertility, Intelligence and Survival? A Scientific Detective Story*. New York: Dutton.
- 93 Davis et al. 2007.
- 94 Allan, BB, R Brant, JE Seidel and JF Jarrel. 1997. "Declining Sex Ratios in Canada." *Canadian Medical Association Journal*; 156: 37–41; Mackenzie, CA, A Lockridge and M Keith. 2005. "Declining Sex Ratio in a First Nation Community." *Environmental Health Perspectives*, 113(10): 1295–1298.
- 95 Davis et al. 1998.
- 96 Mackenzie et al. 2005.
- 97 CPCHE. 2005.
- 98 Wigle. 2003.
- 99 Although PCBs and most organochlorine pesticides have been banned in Canada, they continue to persist in the environment.
- 100 Wigle. 2003.
- 101 Labelle, C. 2000. *Endocrine Disruptors Update*. Government of Canada, Science and Technology Division. Accessed at <http://dsp-psd.pwgsc.gc.ca/Collection-R/LoPBdP/BP/prb0001-e.htm>.
- 102 Carbone et al. 2007.
- 103 Toronto Public Health. 2005.
- 104 Daniels. 2006.
- 105 Olshan, A and E Faustman. 1993. "Male-Mediated Developmental Toxicity." *Reproductive Toxicology*, 7: 191–202.
- 106 Davis et al. 1998.
- 107 Davis et al. 1998.
- 108 Olshan, A and E van Wijngaarden. 2001. "Paternal Occupation and Childhood Cancer" in Robaire, B and BF Hales, eds. *Advances in Male Mediated Developmental Toxicity*. New York: Kluwer Academic/Plenum Publishers.
- 109 Chia, SE and LM Shi. 2002. "Review of Recent Epidemiological Studies on Paternal Occupations and Birth Defects." *Occupational and Environmental Medicine*, 59: 149–155.
- 110 Cohen, FL. 1986. "Paternal Contributions to Birth Defects." *Nursing Clinics of North America*, 21(1): 49–64.
- 111 Hales, BF and B Robaire. 2001. "Paternal Exposure to Drugs and Environmental Chemicals: Effects on Progeny Outcome." *Journal of Andrology*, 22(6): 927–937.
- 112 Olshan and Faustman. 1993.
- 113 Friedler. 1996.
- 114 Gouveia-Vigeant and Tickner. 2003.
- 115 Olshan and Faustman. 1993.
- 116 Davis et al. 1998.
- 117 Daniels. 2006.
- 118 Garry, V, D Schreinemachers, ME Harkins and J Griffith. 1996. "Pesticide Appliers, Biocides, and Birth Defects in Rural Minnesota." *Environmental Health Perspectives*, 104: 394–399.
- 119 Savitz, DA, T Arbuckle, D Kaczor and KM Curtis. 1997. "Male Pesticide Exposure and Pregnancy Outcome." *American Journal of Epidemiology*, 146(12): 1025–1036.
- 120 Daniels. 2006.
- 121 Schettler et al. 1999.
- 122 Gouveia-Vigeant and Tickner. 2003.
- 123 Gouveia-Vigeant and Tickner. 2003.
- 124 Gouveia-Vigeant and Tickner. 2003.
- 125 Olshan and Faustman. 1993.
- 126 Davis et al. 1992.
- 127 Schettler et al. 1999.
- 128 Figa-Talamanca, I, ME Train and E Urbani. 2001. Occupational Exposures to Metals, Solvents, and Pesticides: Recent Evidence on Male Reproductive Effects and Biological Markers. *Occup Med*, 51(3): 174–188.
- 129 Friedler. 1996.
- 130 Olshan and Faustman. 1993.
- 131 Friedler. 1996.
- 132 Olshan and Faustman. 1993.
- 133 Olshan and Faustman. 1993.
- 134 Friedler. 1996.
- 135 Davis et al. 1992.
- 136 Olshan and Faustman. 1993.
- 137 Hales and Robaire. 2001.
- 138 Figa-Talamanca et al. 2001.
- 139 Friedler. 1996.