

Environmental Threats to Children

Understanding the Risks, Enabling Prevention



Technical Report

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ABOUT THIS PROJECT

Toronto Public Health (TPH) has a longstanding interest in how the City's biophysical and built environment affects the health of its children. Our vision is that all of Toronto's children, as well as future generations of children, should enjoy the highest achievable level of health and well-being. This vision recognizes that the determinants of health are complex, and include potent influences such as safe and nutritious food, adequate shelter, education, financial resources, peace, equity and social justice. However, this vision also recognizes the importance of a healthy sustainable ecosystem in which children are protected from environmental hazards. As a result, Toronto Public Health embarked on studies to understand what needed to be done to better protect the health of children (TPH, 1999a).

In 2001, TPH conducted a Needs Assessment Framework Study which provided a broad outline and strategy for assessing information needs and mapping out future planning and evaluation of public health strategies to address children's environmental health issues (TPH, 2002b). In 2002, TPH implemented a survey to assess the knowledge, attitudes and practices of Toronto parents regarding various environmental hazards (TPH, 2002c). Through these early studies and the involvement of representatives from leading health and environmental organizations, the Environmental Protection Office of TPH became a founding member of the *Canadian Partnership for Children's Health and the Environment* (CPCHE). CPCHE's overall goals include moving children's environmental health (CEH) issues into mainstream decision-making in Canada, promoting CEH issues among caregivers, health professionals and the public, increasing awareness about how to prevent harm by adapting the "childproofing" theme for a major health promotion campaign, and coordinating activities and creating efficiencies among the partners. From seven founding organizations, CPCHE has grown to a mature organization, active in healthy public policy and educational outreach to parents and caregivers.

The current report expands on the results and recommendations of TPH's earlier studies. It discusses the wider topic and context of environment and child health and while it focuses on key issues for Toronto the information summarized is relevant to urban children elsewhere in Canada. Adding to the results of the Needs Assessment Framework Study, this report completes a baseline assessment of the state of knowledge of child health and environment issues in Toronto. From this assessment, the report culminates in key strategic directions for future action.

The research was conducted in a collaborative manner by a team of external consultants and public health staff. An advisory committee of experts and

practitioners, including several from among the CPCHE partners, guided the project and preparation of this technical report. This report has benefited additionally from an external peer review.

This TPH report is the technical companion piece for CPCHE's flagship educational resource, **Child Health and the Environment – A Primer**. The CPCHE Primer and associated fact sheets for the public serve as a shorter, plain language version of the present document.

EXECUTIVE SUMMARY

Children are at risk

Children in Canada, including those within the City of Toronto, are at risk from environmental contaminants. There is scientific consensus that the developing fetus, infants and children up to age three years can experience greater exposure than adults to substances in the environment. The degree of risk arising from environmental exposures is often poorly understood. Risks vary across different contaminants, age groups and individual circumstances. Scientific evidence exists of associations between environmental hazards and asthma, cancer, learning, behavioural and developmental effects, low birth weight and birth defects. Emerging evidence exists for additional, equally serious, health effects such as impaired functioning of the immune system and interference with the hormones of the endocrine system.

There are many thousands of contaminants in the environment. Hundreds of them are suspected of contributing to these health outcomes although only a small number of them have been fully evaluated for their effects on prenatal and child development. Nevertheless, multiple exposures continue during pregnancy and throughout the early years of child development. Full scientific certainty about the effect of these exposures is in fact impossible. Such scientific proof would require carefully controlled experiments on children, an ethically abhorrent proposition.

Such “experiments” on children do not occur in the controlled environment of a scientific laboratory, but, in the environments where children live, learn and play. Uncontrolled “experiments” have been conducted for many years and they continue. For example, knowledge about the way lead can harm children’s brains comes from many years of exposing children to lead from its use in gasoline and paint and documenting negative effects on brain development.

Likewise, strong evidence exists that air pollution triggers asthma attacks in people with asthma and there is now suggestive evidence that some outdoor air pollutants can cause the onset of asthma as well. Increasing evidence exists of many other serious effects from air pollution in the developing fetus and child. This evidence comes from measuring these effects in children exposed to air pollution.

Health trends

Patterns of disease among children have changed dramatically in the last 100 to 200 years. Infant mortality is substantially lower and the historically common illnesses of early childhood are very rare in the developed world. Life expectancy has nearly doubled. However, chronic diseases and other debilitating conditions, including several with suspected or strong associations with environmental exposures, are on the rise among children.

Two health outcomes that are observable in large numbers of children include respiratory conditions, particularly asthma, and a range of conditions related to cognitive and neurobehavioural functioning. Substantial evidence demonstrates associations between respiratory effects and indoor and outdoor air pollutants. This evidence includes numerous studies, including in Canada, showing associations between increased air pollution and increased respiratory illness among vulnerable populations including children.

For effects and related outcomes in the developing nervous system, the situation is poorly understood for most contaminants although effects on the developing nervous system are well documented for some of the more extensively studied substances including lead, mercury, dioxins, PCBs and some solvents. Increasing concern exists about nervous system effects of the organophosphate insecticides and polyhalogenated compounds such as the flame retardants known as polybrominated diphenyl ethers (PBDEs). As well, mounting evidence of effects from early life exposure to environmental tobacco smoke on neurological development is of concern.

For each of the major health concerns with known or suspected links to environmental exposures it is generally possible to assemble some data on trends of these health outcomes in the Toronto population. Or, where Toronto-specific data are lacking, trend information from the provincial or national or even the international level can be noted. However, it is only rarely possible to draw a direct relationship between these health outcomes and exposures to environmental contaminants or to establish the relative impact of various factors such as socioeconomic status versus environment.

In Toronto, asthma, learning disabilities, cancer, low birth weight and birth defects occur in the child population at rates that are similar to, or in some cases higher than, rates that occur among children in the rest of Canada and in other industrialized countries. Children in Toronto are disproportionately affected by poverty, compared to children living in adjoining regions of the Greater Toronto

Area (GTA). Poverty is a known risk factor for both poor health and greater exposure to environmental contaminants.

Cancer rates have been rising among children in the US and countries in Europe for many years but such increases are not apparent in children in Canada. Although still very rare, cancer remains the leading cause of illness-related death for children in Canada older than one year of age. Moreover, cancer rates among young adults (aged 20 - 44 years) in Canada have increased gradually since the 1970s. For certain cancers, such as thyroid and testicular cancer in men, brain cancer in women and non-Hodgkin's lymphoma in both men and women, incidence rates increased by more than two percent per year or just under 20 percent per decade. Causes for these increases are unknown but given the long latency period for most carcinogens, that is, the time period between an exposure and the onset of disease, early childhood, prenatal or parental preconceptional exposures, especially during windows of vulnerability, could be contributing factors.

Effects on the Developing Respiratory and Nervous Systems

Current estimates place the prevalence of childhood asthma at 12% and Toronto physicians report treating children for acute and chronic respiratory symptoms more than any other health complaint. Data about these conditions among Toronto's children indicate that poorer children may be particularly vulnerable to general respiratory health effects from air pollution.

The prevalence of learning disabilities, Attention Deficit Hyperactivity Disorder (AD/HD), autism and other neurobehavioural deficits appear comparable among data from the US, Canada and Ontario. Although data are limited, Toronto appears to be on the higher end of the scale with about 13% of enrolled students with at least one or more learning or behavioural exceptionalities of concern.

Although not universally accepted, there is limited evidence that prevalence rates for most of these neurocognitive and neurobehavioural conditions have increased in recent years with some US-based physicians referring to the problem as having reached epidemic proportions given the numbers of children affected. This apparent trend of increasing prevalence over time may be influenced by more aggressive diagnostic practices, nonetheless the burden of disabling conditions is high.

The social costs are considerable. Research into the economic burden of the diseases and disorders of concern with respect to toxic exposures suggests that

exposure prevention could result in substantial savings in health care, human productivity and myriad social costs.

Understanding Exposure

Like the wide range of health effects of concern that may arise from environmental contaminants, describing the range of substances to which children are exposed is challenging. There is both a vast range of substances and, in many cases, multiple sources or settings where exposure can occur. The gaps in information are even more profound for exposure data than for the scientific investigation of health effects. This gap also explains the inability to accurately describe the exposures of children in Toronto in this report. Moreover, many of the substances of greatest concern are often known or suspected of being associated with multiple effects and exposure occurs in many ways. Timing is also critically important given the reality of windows of vulnerability during prenatal development and the many stages of childhood.

Biomonitoring is one, quite direct means of measuring exposure. Although such data collection has never been done systematically in Canada, recent data looking at the US population has found that people of all ages have measurable evidence of exposure and, for persistent substances, body burdens of many contaminants, or their metabolites, at levels that, in most cases, are of uncertain health significance. These data provide a baseline of information to better understand the nature of exposure. Although useful to medical and scientific experts, biomonitoring results have also provided an unexpected and often unwelcome indication to many people of the pervasiveness of environmental contamination.

For pesticide residues in particular, biomonitoring data seem to be essential to improving understanding of exposure and to evaluating both the need for and the efficacy of regulatory measures to minimize exposure. Canadian Food Inspection Agency data appear to reveal a multi-year trend of lowering levels of exposure to pesticide residues on food. The proportion of samples with non-detectable residues is quite high and the proportion of samples showing exceedences of regulatory limits, that is, of the maximum residue limits (MRLs), is consistently low. These data paint a picture of fairly strong regulatory compliance and could lead to a conclusion of very low exposure. However, the US biomonitoring data reveal that pesticides and their metabolites are extremely common in people's bodies.

It is difficult to know either the extent, or the implications to children's health, of combined exposures to multiple pesticide residues on food, either at detection levels or for those found (fairly rarely) in excess of MRLs. It seems essential to

combine such data with actual biomonitoring results to know whether the record of minimal exceedences of MRLs in food is reflective of “safe” exposure levels. This work is of particular importance to the work required to re-evaluate the majority of pesticides in use, in most cases, to determine their potential for exposure and health effects in children.

Compared to the lower levels of pesticide residues on food, pesticide exposure can be much higher and unsafe when it occurs more directly from use outdoors, but particularly in the indoor environment. It is because of this potential for overexposing children that limits have increasingly been placed on the use of pesticides in areas frequented by children.

Less uncertainty exists with respect to the known hazards of exposure to outdoor and indoor air pollution. Air remains one of the most significant media for environmental exposures. All too well understood in Toronto, urban outdoor air is a complex mix of chemicals, including numerous substances that are harmful to children’s health. The burden of illness attributed to air pollution is also very high and estimated to be considerable for people, including children, in Toronto.

Indoor air quality is a largely unregulated source of exposure to a variety of contaminants. Globally, scientific experts acknowledge that poor indoor air quality is a significant environmental health issue requiring further study and monitoring. Indoor exposures are as relevant in a child’s home as they are in schools and child care facilities. Contaminant levels in indoor dust, arising from the tracking in of contaminants from outdoors, the deterioration of old paint or the use and break-down of consumer products, are also of concern.

Exposures will often occur through many media although certain media will be of greater significance than others depending on the particular contaminant or substance. For example, lead exposure can occur via water, food, soil or air. However, exposure to lead in indoor dust appears to be the single greatest exposure pathway for children due to its greater presence in dust than in other media as well as children’s exploratory and hand-to-mouth behaviour. It is a pathway for which greater awareness and precautionary responses are needed. Certain exposures are probably unsafe at any level, including lead, ozone, particulate matter but also the persistent organic pollutants (POPs).

For many of the substances of emerging concern, those that are also persistent and bioaccumulative need immediate attention. Full understanding of their toxic effects in humans will likely only be possible once environmental contamination is pervasive enough or has occurred for long enough that toxic effects can be discerned at which point preventive responses may be too late.

For example, unexpectedly high levels of polybrominated diphenyl ethers (PBDEs) and other substances originating from consumer products are turning up in indoor dust, air and a variety of foods. In Sweden, where longitudinal monitoring revealed a dramatic rise of PBDEs in breast milk over time, swift regulatory action to phase out these substances resulted in reduced exposure as measured via breast milk. The presence of PBDEs in the breast milk of North American women (in amounts that are the highest in the developed world) reflects this multi-media exposure and the fact that production and use of these substances is higher here than elsewhere in the world.

What Toronto Parents Know About Environmental Risks to Children

Results of a telephone survey show that Toronto parents have high awareness of the harmfulness of exposures to children's health for exposures that are high profile or are already well covered in TPH health promotion work (e.g. air pollution, water quality, pesticides). Parents also feel these same areas are worthy of action on the part of the City. While most parents feel they can do a fair amount to protect their children themselves, there is a need to enhance that sense among some parents by providing written information at appropriate literacy levels and by considering development of educational materials other than in written format.

Most parents or caregivers are already practising or ensuring they take some simple measures that may reduce their child's exposures in and around the home such as shoe removal, frequent floor cleaning and child's hand-washing, attention to sources of drinking water and use of sunscreen. They also widely report precautionary household practices that mean children's exposures to potentially harmful substances are minimized. For example, a substantial proportion of Toronto parents report avoiding the use of pesticides and providing a smoke-free home environment.

The survey also identified public education needs such as support for further reducing pesticide use, additional sun protection measures, avoiding smoking indoors, avoiding pets sleeping in the child's room and informing Toronto parents about the benefits of extending protective, cautionary behaviours to all children, regardless of age. The results should allow for risk communication and children's environmental health resources and programs that are relevant, appropriate and tailored to the needs of Toronto parents.

The Policy Response

For most environmental exposures control measures are often delayed or opposed until solid proof of harm is obtained. This delay in applying control measures or finding alternatives is also because the activities in question, such as automobile dependence, are part of entrenched patterns in society that are difficult to change.

Some progress has occurred in terms of revising regulatory approaches to take children's health into account. But the fact remains that widespread exposure to thousands of potentially hazardous substances continues. Equally daunting, there is an enormous number of substances in commercial use, or that result from industrial emissions, that have never been fully evaluated for toxicity during prenatal and childhood life stages. The ability, and political willingness, to control even those situations where strong evidence of harm exists has not been impressive. Part of the problem is the reality that it can be far more difficult to address environmental problems after the fact compared to preventing their occurrence in the first place.

There is much to learn from past experience. Waiting until there is proof of harm from environmental exposures can result in undue exposure and other well-studied toxic substances is the need to act sooner when impacts on children, while the science is debated. The regulatory lesson from the case of lead early evidence suggests a problem. Taking action earlier, despite scientific uncertainty, is an approach that seeks to prevent harm in the first place rather than scientifically documenting it in one generation of children and then, if possible, belatedly preventing harm in the next.

There will never be full scientific certainty in environmental debates. An approach of waiting for proof of harm before controlling or eliminating harmful exposures will continue to place the developing fetus and child at unnecessary and avoidable risk. Advocates for changing traditional approaches to environmental hazards call for a precautionary approach. This approach speaks directly to the reality of forever having incomplete information. It denotes a duty, on all members of society, to prevent harm, when it is within our power to do so, even when the evidence is uncertain or unattainable.

Numerous international agreements have been signed by Canada recognizing the vulnerability of children and committing to policy and related efforts to address these risks. Federal and provincial law and policy has been revised to better account for child health and exposure risks but much remains to be done to either re-evaluate pesticides or assess and manage the tens of thousands of substances in commerce or resulting from transportation emissions, industrial and other human

activities. The incomplete and obsolete nature of Canada's regulation of potentially toxic substances in consumer products is an area of particular concern.

There is an urgent need for strong political leadership and clear accountability and resources for children's environmental health at both the federal and provincial level. There must be greater integration across departments where policies and programs can minimize exposure to environmental hazards. At the provincial level, particular attention needs to be directed at coordinating the activities of the Ministries of the Environment, Health and Long-Term Care, and Children and Youth services into a comprehensive cross-cutting provincial program.

Many Canadian municipalities have exercised leadership in applying precautionary action to recognized risks through the passage of progressive bylaws and other actions. Toronto has been in the forefront of this precautionary activity as have Toronto school boards. However, both the municipal ability and that of school boards to apply progressive or proactive environmental controls is limited by funding constraints, their respective arenas of policy and regulatory authority and influence, and the magnitude of issues needing to be confronted.

Based on a review of key policies and initiatives in Canada, the report concludes that some progress has occurred in terms of revised federal and provincial regulatory approaches to take children's health into account. However, much remains to be done at the federal, provincial and local levels. The recommendations in the report encompass measures that will assist the City of Toronto in reducing and preventing children's exposure to harmful substances in the environment.

The strategic directions recommended address the gaps that are identified from the review of literature and policy responses. Recommendations identify needs in: (1) research; (2) policy; and (3) education. Priorities for action are guided by the need to address exposure risks that are: a) preventable; b) have the potential to affect large numbers of children, including children whose health status is compromised by other circumstances such as poverty; and c) associated with serious or irreversible health effects or with long-term consequences. Given that the authority, responsibility and mandate for the key issues of relevance to children's environmental health in Toronto do not fall solely under one government jurisdiction, recommendations are directed at federal, provincial or local governments as indicated. Recommendations for school boards and independent/private schools in the City are included as well, in light of the importance of the school environment for children's health.

The public health mandate of health protection and health promotion is fundamentally one of applying a precautionary approach. This report makes

recommendations that are intended to assist the City of Toronto in choosing a course of action in the face of uncertainty and to continue to apply a precautionary approach to reducing and preventing children's exposure to harmful substances in the environment.

CHAPTER ONE – INTRODUCTION

1.1 Key Issues in Children’s Environmental Health

There is a large and rapidly increasing literature on environmental exposures and health outcomes in children. There is scientific consensus that the developing fetus, infants and children up to age three years can experience greater exposure than adults to substances in the environment. They can also be more vulnerable to the effects of these substances than adults or even older children.

Beyond the general agreement on these statements about fetal, infant and early childhood exposure and vulnerability, this field includes a vast amount of uncertainty, controversy, and fundamental gaps in knowledge. This lack of information stems from four overarching realities:

- The breadth and complexity of the topic (encompassing all stages of human development from parental germ cells to the end of adolescence);
- Incomplete and imprecise exposure assessment and the reality of a vast number and range of circumstances of possible exposures (over 23,000 substances, including pesticides, in commercial use in Canada and many additional pollutant emissions resulting from the activities of modern life);
- The fact that health or ill-health often results from multiple, interacting factors including parental behaviours and genetic factors; and
- The challenges associated with assessing prenatal and childhood exposures to factors other than environmental contaminants including prenatal maternal tobacco, alcohol and drug use, infectious agents and adverse social environments.

As a first step in formulating an agenda for action by the City of Toronto, this report summarizes the large and growing field of child health and the environment. It focuses on three major topic areas: the vulnerability of children; health outcomes of concern; and exposure sources and settings. For each of these three areas, the information summarized in this report makes note of the degree of scientific consensus. Less emphasis is placed on those areas where the experts do not agree or where information is still emerging or controversial. Emerging issues are important to include, but they are not explored in detail here.

A key observation made by Toronto Public Health in earlier work on this topic is that data for exposure and health outcome indicators among Toronto’s children

are needed to help characterize the nature of children's environmental health and to strengthen future program planning activities. These Toronto-specific data remain elusive.

For each of the major health concerns that have suspected or strong associations with environmental exposures it is generally possible to assemble some data on trends of these health outcomes in the Toronto population. Or, where Toronto-specific data are lacking, trend information from the provincial or national or even the international level can be noted. Such trend information is provided in this report. However, it is only rarely possible to draw a direct relationship between these health outcomes and exposures to environmental contaminants, for several reasons.

First, causal relationships are often not simple or direct, with health outcomes resulting from several interacting factors. Second, experts do not know enough about the interacting causes of these health outcomes, or the relative importance of individual factors, including exposure to substances in the environment. Third, there is a lack of knowledge about the nature of exposure to these substances. Although there have been many research studies in this field, many have been limited by small sample size and crude exposure indices. Indeed, only in recent years have a few large studies been initiated that include new methods to directly measure exposure.

In the area of respiratory effects from air pollution, however, there is strong scientific evidence for causal relationships between indoor and outdoor air and asthma episodes (i.e., aggravation of symptoms among known asthmatics). There is now also suggestive evidence of a causal relationship between incident asthma (i.e., newly developed cases) and outdoor air pollutants. Experts hasten to add that asthma is a complex condition and air pollution is one of several contributing factors including genetic traits and infections. For a handful of other well-studied contaminants there are clear lines of evidence linking exposure with specific health outcomes. However, even for these well-known hazards, such as lead, mercury, PCBs and ionizing radiation, exposures are often inadequately measured.

1.2 Definitions

Some key definitions are provided here to augment the glossary of terms and abbreviations.

It is necessary to consider multiple stages of “**childhood**” within the field of environmental health. Since health concerns can relate to exposures affecting

development, the life stages of interest extend from three months pre-conception, (accounting therefore for parental occupational or environmental exposures and, to a lesser extent, accumulated body burden of persistent toxicants), through conception, *in utero*, birth, infancy, childhood and adolescence. The age-range for adolescence varies among different information sources. Some place childhood and adolescence extending up to age 20. The World Health Organization defines children as persons age 0-17 years. Because some developmental processes continue through adolescence, such as those associated with reproductive maturity and nervous system development, there is good reason to recognize the developmental vulnerability of children as extending to the end of adolescence. Also, prenatal and childhood exposures can have delayed effects including some cancers. This report is concerned with “childhood” across these diverse life stages. However, since health outcomes and environmental exposures of concern can vary considerably at different stages, and since data collection does not always correspond to the life stages noted here, age ranges are noted, where appropriate.

Environment is a similarly broad term. Put simply, a child’s environment includes everything around the child, in both indoor and outdoor settings, and including all that is taken into the body. It includes consumer products where these contribute to toxic exposures. Since babies develop for nine months *in utero*, the mother’s body is a child’s first environment and her breast milk the first food. The physical environment includes natural and built environments. Potential threats to child health in these environments include chemical (e.g., pesticides), biological (e.g., infectious agents) and physical agents (e.g., ionizing radiation) from natural and human activity related sources. While biological hazards in the environment are important threats to child health, they are beyond the scope of what is covered here.

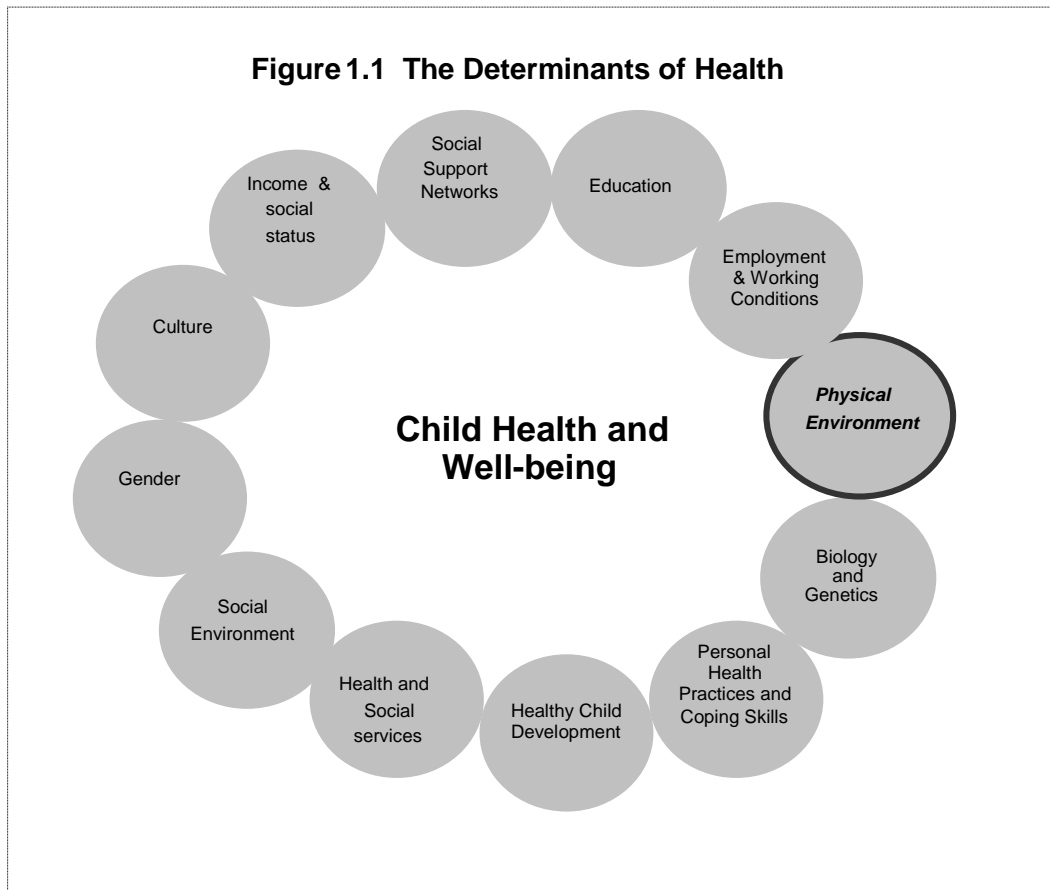
This broad definition of environment is necessary since exposure to substances of concern can occur from multiple sources. These sources can be captured within several major categories of human activity including fossil fuel combustion and other industrial activities, the use of consumer products, waste disposal and accidents. Substances of concern can include those that are released to the air, water or soil and are considered environmental contaminants. These can range from industrial emissions, to smog-forming particles in tailpipe emissions to second-hand tobacco smoke. Substances of concern can also include those that are not “contaminants” but are intentionally used (such as pesticides) or incorporated into consumer products (such as plasticizers or flame retardants), or that are by-products of intentional uses of substances for health reasons (such as disinfection by-products that result from the use of chlorine to disinfect drinking water).

Exposure to substances varies widely since, once in the environment (indoors or outdoors), substances can travel, change and combine. Air, water, soil, and food are generally considered the media through which substances travel or accumulate. A fifth exposure medium of particular relevance to children is house dust, a medium that also illustrates the importance of both indoor and outdoor sources. A large fraction of house dust comes from soil tracked into homes; other sources include clothing, carpets, rugs, furniture, bedding and human/animal dander. Depending on the physical and chemical nature of different substances, exposure can occur via inhalation, skin contact or ingestion, and *in utero* via the placenta. Understanding the many pathways from source to receptor (that is, the child) and the relative contribution of exposures from different media can be very complex.

The definition of **health** is also very broad. Like most health agencies, Toronto Public Health applies a positive definition of health that includes more than just the absence of injury, disease, or disability. Drawing upon widely accepted definitions developed within the World Health Organization, Toronto Public Health defines health as including the notion of individuals possessing and applying skills, abilities and other resources in everyday activity, in order to pursue needs and desires, and to cope with and adapt to their environment. Health is therefore a continuum where many factors contribute to health by degrees from poor to optimal health.

In further describing **child health** TPH divides this positive concept of health into two sub-components: healthy development and healthy functioning of the child. **Healthy development** includes the ability to develop skills and abilities fully as brain and body mature. **Healthy functioning** includes a child's use of these developmental skills and abilities as well as having and using all other resources necessary for good health. TPH's current, positive understanding of the health of young children in Toronto will be covered in greater detail in an upcoming series of Child Health Status reports.

The determinants of health include twelve individual and collective factors that work interactively and in complex ways to influence health (shown schematically in Figure 1.1) (PHAC, no date).



Clearly the topic of how to define and characterize the entire range of influences on child health is much broader than can be adequately addressed in the current, issue-focussed report. How the determinants of health operate within the families, neighbourhoods and communities of Toronto's children will be explored in the upcoming Child Health Status report series.

Finally, **environmental health**, as defined by the World Health Organization, includes aspects of human health and disease that are determined by environmental factors. It can also include the theory and practice of assessing and controlling factors in the environment that can potentially affect health. It includes both the direct pathological effects of chemicals, radiation and some biological agents, and the effects (often indirect) on health and well-being of the broad physical, psychological, social and aesthetic environment which includes housing, urban development, land use and transport (WHO Regional Office for Europe, 1989).

Experts in the field of **children’s environmental health** sometimes refer to the “new pediatric morbidity” when discussing child health trends for which environmental factors may be contributing causes (Landrigan et al, 1998). Alongside the traditional measures or indicators of child health, such as infant mortality or infectious diseases, they look at trends in respiratory health (especially asthma), cancer, neurodevelopmental and neurobehavioural outcomes and various reproductive and developmental indicators such as fertility rates or birth defects. This new perspective embraces the broad definition of child health described above. It looks at both developmental and functional health endpoints in investigations that try to unravel the complexity of the environment-human health interface.

1.3 Toronto’s Children – part of a large and diverse population

With a population of over 2.4 million (2001 Census), Toronto is one of the most populous cities in North America. It has a larger population than most Canadian provinces. Note that a distinction is made here between the City of Toronto and the Toronto Census Metropolitan Area (CMA) as used by Statistics Canada. The Toronto CMA includes the surrounding municipalities within the Greater Toronto Area. Total population of the Toronto CMA was 4.7 million in 2001. This report focuses on data for the City of Toronto.

There are over 434,000 children (0-14 years) living in Toronto (2001 census data), accounting for 17.5% of Toronto’s population. Overall, the population of children in Toronto grew by about 2% between 1996 and 2001 (Statistics Canada, 2001 Census data on-line). In some census tracts, however, the population of children grew by over 35% (Toronto, 2002).

Toronto is one of the most culturally diverse cities in the world. The 2001 Census data indicate that 49% of Toronto’s population was born outside the country and 43% were members of a visible minority. In 2001, 40% of Toronto children and one out of every five children in first grade were born outside Canada (Toronto, 2002). Over 160 languages and dialects are spoken in Toronto and nearly 30% of residents speak a language other than English or French at home (Statistics Canada 2001 on-line data; Toronto, 2002).

1.3.1 The Socioeconomic, Sociocultural Picture

As determinants of health, socio-economic and cultural factors can impact upon a child’s exposure to environmental contaminants. Among the many ways that

Health Canada recognizes culture or ethnicity as influencing health, most relevant to this report is that culture and ethnicity have important links to practices (PHAC, no date), that is, cultural customs that can modulate or heighten exposure. For example, the use of metallic mercury in certain religious rites contributes to significant exposure to mercury vapour in the home (Riley et al, 2001). Some Chinese herbal medicines are known to contain forms of mercury as additives or as contaminants (Wong, 2004). Of importance in Canada is the influence of culture on dietary exposure to contaminants for people in Aboriginal communities. The Aboriginal diet places great cultural, spiritual as well as nutritional health significance on “country foods” (that is, fish, marine mammal and terrestrial wildlife species), which has resulted in greater contaminant exposure in these populations because of transboundary pollution and bioaccumulation of persistent toxic substances (INAC, 2003).

Secondly, culture and ethnicity can interact with social and economic well-being to compound negative influences on health (or exposure to environmental contaminants) where, because of societal prejudice or discrimination, people of a particular culture do not have the same access to education, employment or housing (PHAC, no date).

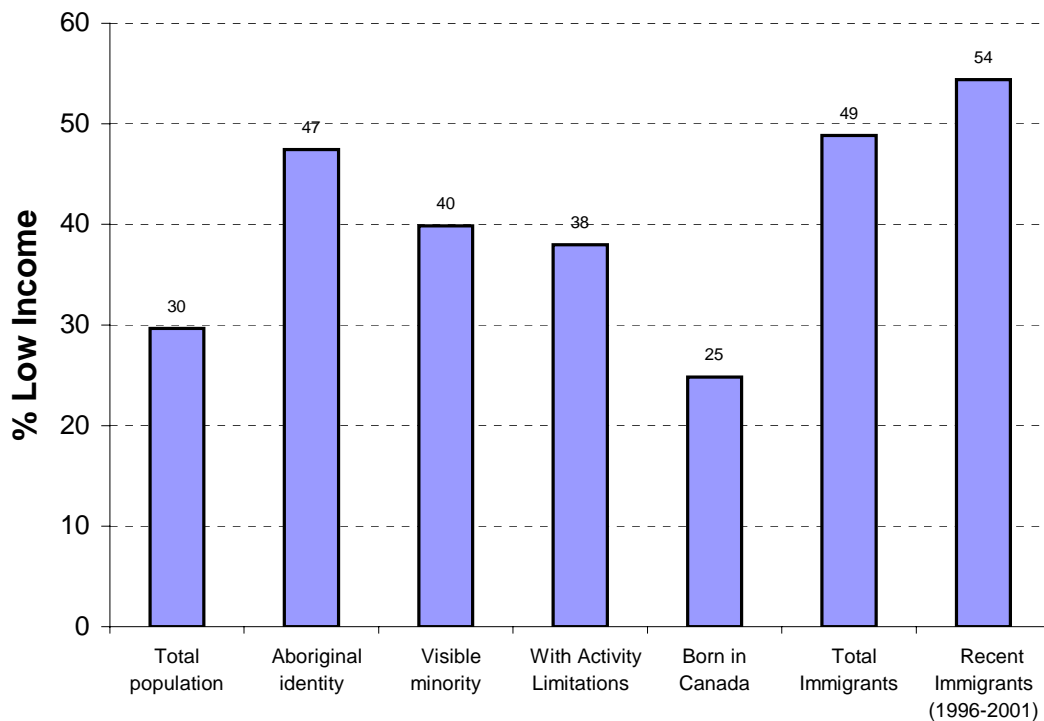
Socio-economic factors in particular can influence the physical condition of a child’s home, the characteristics of a child’s neighbourhood and the occupational exposure of a child’s parents. Poverty as a risk factor that influences environmental exposures is discussed in greater detail in Section 2.4.

In 2001, about 1 in 6 children in Canada lived in poverty. Children of Aboriginal ancestry, those who represent a visible minority or are recent immigrants are disproportionately represented among children living in poverty (Campaign 2000, 2004; Chaudhuri, 1998; and WHO-EEA, 2002). Compared to figures for all of Canada, children in Toronto are disproportionately living in poverty. These are figures for children aged 0 to 14 and the poverty measure is Statistics Canada’s low-income cut off (LICO) which is an income level at which families or unattached individuals spend a significantly higher proportion of their income on food, shelter and clothing than the average Canadian family or individual living in a comparable setting. The Toronto LICO for a family of two in 2000 was \$22,964 and for a family of four was \$34,572 (Toronto, 2002).

Even compared to neighbouring communities in southern Ontario, poverty levels are absolutely and relatively higher for Toronto’s children. Census statistics from 2001 undoubtedly underestimate the degree of poverty in Toronto in 2005 as current economic indicators suggest more families are likely affected (Toronto, 2002, 2003).

Toronto's children are more likely to live in poverty if they: live in lone-parent families dependent on a single income, are of Aboriginal descent, are in recent immigrant families often in low paying jobs and, by definition, are in families living below the low-income cut off (LICO) (Toronto, 2003). See Figure 1.2 below.

Figure 1.2 – Toronto Child Poverty by Diversity Attributes (Percent Low Income by Category)



Source: Statistics Canada, Census of Canada, 2001

Toronto has only 40% of the child population (ages 0 to 12) of the GTA, but it has 66% of the GTA's poor children, 69% of the GTA's low income families, 62% of the lone-parent families, and 80% of the GTA's recent immigrants (Toronto SDSSC, 2001, as reported in Toronto, 2002). The United Way confirms this disproportionate level of poverty in Toronto in a report noting overall high rates of poverty in Toronto and the increase in number and changing distributions of higher poverty neighbourhoods. Noting trends since 1981, the report finds that the traditional inner city location of higher poverty neighbourhoods has expanded to

include large amounts of the inner suburbs of the five municipalities within the former Metropolitan Toronto particularly in southeastern Scarborough and North York (UWGT and CCSD, 2004).

The adequacy and affordability of housing for low-income families is a serious problem in Canada. Much research confirms that stable, safe and secure housing is vital to healthy growth and development in children. Research also confirms that inadequate housing (defined as houses that are in need of major repairs) and poor quality housing is linked to poor health outcomes in children. While the detrimental factors at work are broader than those under consideration in this report, several are related to environmental health. For example, research has shown that children in poor and inadequate housing are at greater risk for asthma and other respiratory problems and for exposure to diverse respiratory allergens and toxicants including lead, asbestos and pesticides (as reviewed in Cooper, 2001; CHEO, 2003).

Homelessness is a reality for many children in Toronto. In 1999, Toronto's total shelter/hostel admissions for the year stood at 30,000, an increase of 40% from about ten years earlier. Throughout the 1990s various public policy decisions dramatically affected the availability of affordable housing. While improving, this situation will remain a problem for several more years (Toronto, 2002). Also in 1999, it was estimated that 6,200 children were using emergency shelters, an increase of 120% from ten years earlier (TPH, 2001a). Shortage of food is also an issue for many families in Toronto. In 2000, it was estimated that one-third of the children whose families rely on food banks (i.e. over 13,000 families) miss at least one meal a week (TPH, 2001a) and data collected in 2003 show increasing use of food banks by families with children (Toronto, 2003).

The influence of poverty, quality of housing and nutrition on children's environmental health are discussed further in Section 2.5.

1.4 Health Trends

Fortunately, overall rates of serious illness and death among children in Canada, including in Toronto, are generally quite low. Traditional measures or indicators of child health look at rates of infant mortality, infectious disease, and injuries. Across the 20th century, rates of infant mortality and infectious diseases dropped dramatically thanks in large part to advances in public health, such as improvements in water and sewage sanitation, housing and nutrition, alongside overall advancements in medicine, especially immunization and antibiotics. It is important to remember this overall context when looking at trends in health outcomes that may be influenced by environmental exposures.

As noted in section 1.2, health status results from many interacting factors in society, the environment and the individual. Factors influencing a child's health begin in the womb and are directly linked to the mother's overall physical and mental health, prenatal care, nutrition and other health-influencing habits such as maternal smoking, alcohol or drug use. Optimum child development and functioning occurs with good nutrition and a suite of emotional, social, psychological and physical supports and stimulations throughout childhood and adolescence.

Similarly, most health outcomes, including those for which environmental contaminants are suspected, result from a complicated mixture of influences – biological, social and environmental. For example, asthma is related to indoor and outdoor air pollution but there are other factors at work including an individual's genetic makeup or inherited susceptibility. The point to underline is that the focus on environmental influences, described in this report, should not suggest that the influence of environmental factors is of primary concern or that other influences are less important.

Health effect trends are often reported in terms of leading causes of death, illness or disability. In children these leading adverse health outcomes include birth defects, low birth weight, neurodevelopmental outcomes, unintentional injuries, cancer and asthma (Wigle, 2003).

Among the most common causes of disability in children are several important conditions for which environmental exposures are implicated. These include conditions such as asthma and other respiratory illnesses as well as various neurodevelopmental outcomes. Environmental contaminants are suspected of contributing to these outcomes, but the extent of any contributions is difficult to determine. The evidence concerning such links is discussed in this report.

1.5 Evaluating the Scientific Evidence

This report discusses the evidence, primarily about chemical substances, that are associated with, or suspected of contributing to, a wide range of health outcomes. Without exception, these are complex conditions that can result from multiple contributing factors, not all of which are fully understood.

Only for respiratory effects from air pollution and other effects from a small number of well-studied pollutants such as lead, PCBs, environmental tobacco smoke (ETS) and ionizing radiation, has a direct causal relationship been drawn between environmental exposures as a contributing cause (among many) to increased levels of disease in the child population.

For the rest of the health outcomes discussed in this report, it is possible to describe the scientific evidence about suspected associations between these outcomes and certain substances. It is also possible to describe health trends and that information is provided in Chapter Three at varying levels of detail, including, where available, information about children in Toronto. However, only rarely is there sufficient scientific evidence to trace a direct or causal relationship between the prevalence or trends of most of these health outcomes in the child population and specific environmental exposures.

This difficulty arises because of the nature of scientific inquiry. Demonstrating causal relationships involves detailed and methodical scientific investigation. Testing for drug safety illustrates some key issues and important differences. Drugs are typically used to provide therapeutic benefits and before they can be sold they are tested in many ways including clinical trials on human subjects. Requirements for the ethical conduct of such experiments on humans have evolved over time and key among these are requirements of informed consent of (usually adult) test subjects as well as careful monitoring for adverse effects which, if found, require termination of the experiment. Where such drug testing is conducted on children, consent must be obtained from their parents. While drug testing is not without problems, this kind of controlled testing carefully refines the knowledge base about a specific drug in the process of evaluating whether it is safe for use.

In contrast, it is not possible nor would it ever be ethically acceptable to run similarly controlled experiments on human subjects to determine the safety of exposure to environmental contaminants. Instead, various lines of evidence are gathered in more indirect ways and the overall weight of evidence is assessed. This reality of having to look at a variety of essentially indirect measures to assess health risk is one reason why environmental health risks are so often described with qualifying language. Each hazard is presented in terms of the relative level (or strength) of evidence. For example, for carcinogenicity, where assessment of evidentiary strength is best developed, the body of evidence for any given substance or agent (from both epidemiological and animal studies) may be described as sufficient, limited or inadequate to conclude whether a chemical causes cancer in people (Wigle, 2003).

For environmental exposures a weight of evidence approach is increasingly used to describe health risks as well as to determine regulatory responses about a substance or group of substances. The scientific evidence can include studies on humans, sometimes from occupational exposures or accidents or epidemiological evidence of health effects in a specific population. Evidence from studies on animals comes from laboratory experiments where it is possible to study specific effects at varying exposure levels. These *in vivo* (or “in the animal”) studies look

at effects in laboratory animals under controlled conditions. Additional laboratory testing can be done *in vitro*, (literally, “in glass” experiments, that is, in a test tube or Petri dish using tissue or cell cultures). These experiments can look at specific reactions between chemical substances and specific animal or human tissues. Animal data can also be gathered from studies of wildlife experiencing chemical exposures typical of general environmental levels or specific contaminated environments. The ability to state whether a particular substance is known or suspected of contributing to a particular health effect in humans comes from evaluating a wide range of results from these diverse investigations. Notably, it is extremely rare that such investigations include, or are even capable of considering, the combined effect of multiple chemical exposures.

The above is a simplified description of how the health risks of environmental contaminants are assessed, both by scientists and regulatory agencies. A more thorough description would include the many variations on how these broad categories of scientific inquiry are conducted. It would also describe how the overall risk assessment process functions in terms of weighing the scientific evidence. Whether these more specific details are included or not, the central fact remains that scientific evidence can never provide a full answer to the complex questions raised. Unlike clinical drug trials, the human health effects of environmental exposures are not within the realm of scientific experiments that can be carefully controlled in a laboratory. They are rarely simple to evaluate. Also unlike drug trials, the “experiment” cannot always be stopped once the environmental releases have occurred or, in many cases, are continuing.

In describing this situation with respect to children’s chemical exposures, Herbert Needleman, a professor of psychiatry and pediatrics and well-known expert on lead, has stated “we are conducting a vast toxicological experiment in which our children and our children’s children are the experimental subjects” (Needleman, 2001). This chilling statement challenges the approach of requiring scientific proof of harm before taking action to reduce or prevent exposure to environmental toxicants. It also prompts precautionary responses, which include making choices to reduce or prevent exposures before full knowledge is available. Such choices can also include substitution of inherently safe alternatives for situations or exposures of suspected but not fully understood (and likely never fully understandable) risk.

CHAPTER TWO – CHILDREN AT RISK

2.1 More Vulnerable and More Exposed

In the field of child health and the environment, experts will often say: “children are not little adults.” Although parents need little convincing of this fact, a great deal of scientific research confirms it.

Numerous differences exist between adults and children in their susceptibility and exposure to environmental risks. These differences in children begin with the recognition that a consideration of “childhood” needs to encompass several distinct phases in life. Although some differences exist between the elderly and other adults, for the most part, the factors that constitute normal physiology and that contribute to good health are fairly consistent across the adult population. In children, development of physiological processes and mechanisms begins at conception and continues until the age of about 20 years. The broad stages of development therefore include preconception, prenatal, childhood and puberty. Across all these stages, children can be more susceptible and more exposed to harmful substances in the environment compared to adults, (NRC, 1993; Chance and Harmsen, 1998). As well, the reasons for concern vary a great deal during different life stages.

2.1.1 Windows of Vulnerability

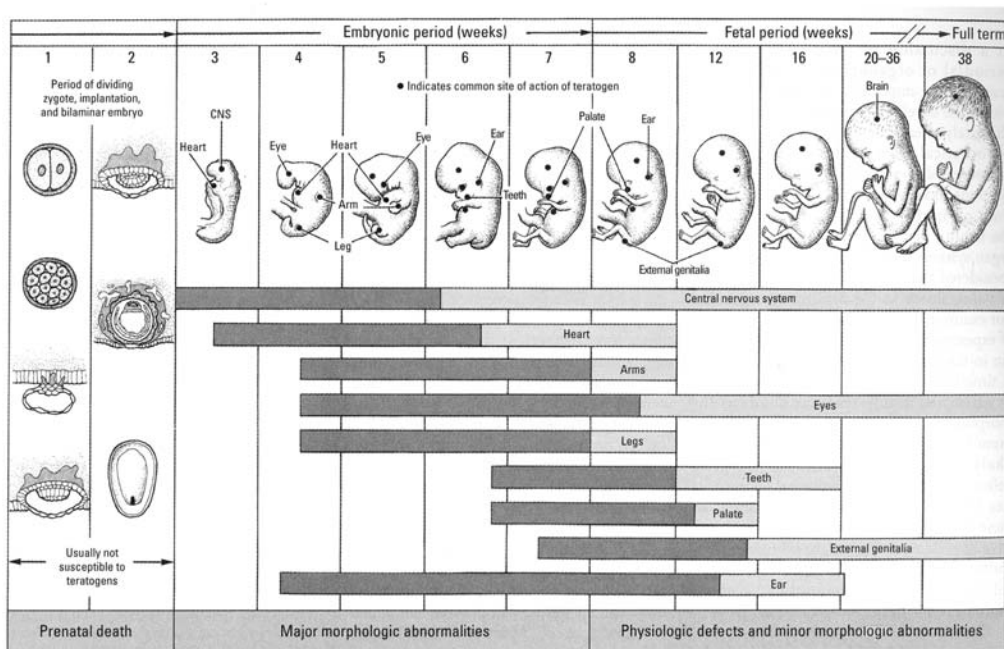
Evidence suggests that vulnerability or susceptibility to chemical exposures can be greater during certain periods of development (Selevan et al., 2000). Identifying time periods when a system is most susceptible to specific effects is important to understanding the relationship between exposures and outcomes.

When the timing of exposure is the key reason for a substance exerting a toxic effect, the sensitive time period is referred to as a ‘window of vulnerability’. Susceptibility to exposures may not be uniform across this window. A related concept in scientific literature is the ‘critical window of development’. Rather than being identified in terms of the timing of exposure alone, these windows are recognized by key developmental events or processes (Lemasters et al., 2000). This window is indicative of situations where chemical or other exposures can interfere with the interactions of normal chemicals in the body that orchestrate and regulate developmental stages, and reflects the discrete time period when there is the greatest risk of potential effects associated with an exposure. For example, the differentiation of embryonic cells into more specialised cell types is a critical window of development (Pryor et al., 2000).

Understanding of the developmental windows of vulnerability is evolving. Information on the precise timing of these windows of vulnerability and how they differ across body systems is limited. Much of the existing research focuses on prenatal exposures, even though developmental processes continue into adolescence and in adult males, are ongoing in the production of sperm. Limited attention is given to the effects of exposures occurring during puberty and adolescence. As well, although there is a growing interest in the impacts of child health for lifelong health, research into the consequences in adulthood of environmental exposures during development is largely lacking (Selevan et al, 2000).

What is known is that windows of vulnerability are specific to each developing body system. For example, as illustrated in Figure 2.1, the most sensitive period to chemical exposure for the morphological (physical) development of the heart is different from that for the palate. Also, in the case of exposures to reproductive toxicants, the windows may be sex-specific as males and females develop differently (Lemasters et al., 2000).

Figure 2.1 - Critical windows of vulnerability



There is greater understanding of critical windows during the prenatal period as illustrated in the graphic above. However, although less well-studied, windows of vulnerability also exist in early childhood and through puberty as discussed further in this section.

Source: Moore & Persaud, 1973 as cited in Selevan et al, 2000

Several noteworthy examples during the past fifty years have contributed to understanding this notion of “windows of vulnerability” during prenatal development. These include effects from the drugs thalidomide and diethylstilbesterol (DES) and two metals, mercury and lead.

Thalidomide, a drug taken during early pregnancy to alleviate symptoms of morning sickness, interfered with development of major morphological features, the most well-known examples of which were the impact on normal development of the fetal arms and legs. If taken at other stages of pregnancy however, the effects did not occur. DES, another drug taken during pregnancy, affected the development of the reproductive system. In their teens and twenties, daughters of these women had higher rates of unusual cancers known as clear cell adenocarcinomas of the cervix and vagina, and both sons and daughters had higher rates of genital birth defects. DES illustrated a situation of effects that were both immediate (such as birth defects) and delayed (such as early life cancer).

“Minamata disease” refers to methylmercury poisoning that was first documented in Minamata, Japan. Prenatal consumption of fish and shellfish that were heavily contaminated with methylmercury due to mercury emissions from a local chemical plant caused severe brain damage, cerebral palsy, and other neurodevelopmental outcomes among children born to exposed women. The mothers generally showed no ill effects themselves.

Lead is another neurotoxic substance and one of the most well studied neurotoxicants. The effects at high levels of exposure in children or adults are obvious and can include severe brain damage, coma and death. At lower levels of chronic exposure such as occurred from the widespread use of lead in gasoline, the neurotoxic effects are subtle and would not necessarily prompt a visit to the doctor. In fact, it took more than thirty years of research for the medical and scientific community to show conclusively the effects of relatively low-level lead exposure on the developing brain *in utero* and during early childhood. This long scientific journey towards understanding “windows of vulnerability” is more typical than occurred with prenatal use of thalidomide.

The traditional approach to toxicity testing is that “the dose makes the poison.” In other words, the toxicity of a substance to an organism is dependent on the amount taken into the body. It is recognized that many substances can be considered toxic if the exposure dose is high enough. As is illustrated through the examples above, when exposures are high, such as those that occurred in Minamata, and clear clinical effects are present, as was the case with thalidomide, assessment of the risk is relatively straightforward. However when exposure to contaminants is lower and/or effects or developmental outcomes are more subtle, such as can occur with exposures to low levels of lead and other pollutants or

contaminants, scientific understanding and assessment is much more elusive or can take a very long time. Section 2.2 further examines what is known about developmental susceptibility to environmental exposures.

2.2 Factors Contributing to Greater Vulnerability

The fetus, infant and toddler are at greatest risk to potential adverse effects from environmental exposures. This susceptibility is due to biological and behavioural differences. Awareness of the susceptibility of the fetus and infant to derailed or altered development from environmental exposures has advanced over the past decade (Barker, 1995; USEPA, 2003a, 2003b, 2003c).

The stages from conception through gestation, birth, infancy, childhood and adolescence involve a series of genetically controlled molecular processes that are highly complex and not fully understood. Throughout each stage (with some stages more sensitive than others) there are opportunities for chemical substances to interfere with, or disrupt, these natural processes and cause irreversible structural and/or functional deficits.

The human body has systems in place to mitigate toxic exposures but these systems are still developing *in utero* and often into early childhood. For example, infant kidneys do not have the adult capacity to excrete certain toxicants until about 16 months of age. Nor do the liver and biliary excretion systems have the adult capability to metabolize and excrete toxicants until at least six months of age (Bell et al, 1989). The lungs are similarly immature at birth. Growing lung tissue increases during childhood and adolescence from an internal surface area of 3 square metres (m²) to 75 (m²). A child's lung tissues are more sensitive to air pollution than lung tissues in an adult. Part of this sensitivity is related to the substantial, ongoing lung growth and development during early childhood. In fact, the human lungs continue to develop until about 18 or 20 years of age (Dietert et al, 2000).

Across the literature, including results of research funded by pesticide and other chemical industry associations, there is agreement about the particular vulnerability of the prenatal and early postnatal (early infancy up to 6 months) periods of development (Scheuplein et al, 2002; Dourson et al, 2002; Wigle, 2003; USEPA 2003a and b). This period extends up to about age 2 to 3 years when studies on the neurotoxicity of low-level lead exposure are considered (Wigle, 2003; Canfield et al, 2003). The vulnerability of older infants, children and adolescents is less understood (with the exception of respiratory effects, see Chapter 3). Throughout the rest of childhood, concern exists due to the opportunity of substances to interfere with ongoing brain development and the

many hormonally-regulated processes of reproductive development that continue through adolescence (Selevan et al, 2000).

Once exposed, children may absorb and also retain more contaminants in their bodies than adults for a variety of reasons. Skin permeability tends to be higher in young children than in adults (Bearer, 1995a). Children can also more effectively absorb certain ingested contaminants. Another difference between adults and children relates to differences in the blood-brain barrier. This barrier functions rather like a semi-permeable membrane that controls access to the brain of substances circulating in the blood. This barrier is not fully developed until about age 6 months and remains permeable to small lipid-soluble agents such as many pesticides and other lipophilic substances (i.e., substances that bind to fat molecules) (Rodier, 1995). The result may be greater delivery of contaminants to the brain of a child than to an adult.

Another simple difference is that children have a longer life expectancy than adults. Chemicals with latent (delayed) effects are more capable of exerting these effects in adults exposed during childhood or *in utero* (Landrigan et al, 1998).

Adverse effects in the vulnerable tissues of the fetus or infant may be permanent and irreversible; therefore, preventing adverse exposures to pregnant and nursing women and to infants is a key concern (Bearer, 1995b; Selevan et al, 2000).

Finally, there may be genetic differences among groups of children that increase vulnerability. Within the genetic blueprint provided from the combination of human germ cells (sperm and egg) there can be much variability. Genetic “polymorphisms” or alternate forms of genes (sometimes called genetic variants), will determine differences in human form and function. For example, genetic variety gives rise to differences in eye colour, skin pigmentation and can include the presence, or predisposition to some diseases (e.g. asthma) or the ability to break down toxicants in the body. Certain genetic traits can interact with factors in the environment (infections, chemicals, nutrients, etc.) and result in adverse health effects. For example, it has been hypothesised that the genetically inherited disease cystic fibrosis appears to be worsened by exposure to environmental tobacco smoke, although this research is inconclusive (Beydon et al. 2002; Smyth et al 2001).

Polymorphisms exist in different frequencies among various sub-groups of the human population and can result in different susceptibility to certain environmental hazards. For example, a study of leukemia risks and exposure to pesticides among children in Quebec, showed that leukemia risks were higher in children who carried specific genetic mutations and were exposed to pesticides prenatally (Infante-Rivard et al, 1999). These mutations likely alter the biological

activity of the cytochrome P-450 enzymes, which normally transform or metabolize foreign substances in the body, including pesticides. The role of these enzymes in pesticide metabolism or the reason that specific mutations modify cancer risks have not been well studied (Infante-Rivard et al, 1999).

Genetic polymorphisms may also be associated with varying susceptibility to health effects from lead. Studies suggest that at least three different genes alter the absorption and toxicokinetics of lead in people (Lidsky & Schneider, 2003). For example, two forms of ALAD (δ - aminolevulinic acid dehydratase), the enzyme product of one gene, bind with differing strengths to lead. While this results in increased blood-lead levels, it appears paradoxically to decrease clinical or subclinical symptoms associated with the same exposure to low levels of lead because lead is kept out of other tissues (Bellinger et al, 1994). Two other genes (involved in calcium and iron absorption, respectively) may also influence the absorption of lead in the body, though further research is needed to understand their impact (Lidsky & Schneider, 2003).

Ethnic differences in susceptibility to environmentally linked health outcomes may also represent interactions with genetic influences. For example, researchers from Columbia University in New York City found that chromosomal damage in cord blood from prenatal exposure to carcinogenic air pollutants was more frequent among the African-American women as opposed to the Dominican women in their sample despite similar exposures (Bocskay et al 2005). The Environmental Genome Project of the U.S. National Institute of Environmental Health Science (NIEHS) supports research to identify polymorphisms of environmental disease susceptibility genes in the U.S. population (NIEHS, 2001a).

2.3 Factors Contributing to Greater Exposure

Greater exposure is an important aspect of the greater vulnerability of children. Among the general population, children's exposures are unique, more varied and clearly different from those experienced by most adults (acknowledging that high occupational exposure can exist for some adults). As well, the nature of children's exposure changes over time.

Before conception, the environmental and/or occupational exposure circumstances of both parents may contribute to adverse outcomes in children that are not otherwise indicated in the parents.

The mother's stored body burden of contaminants contributes to the first exposure environment for her fetus. In the womb, the mother and child's blood circulation

is shared through the placenta. The fetus can be exposed to the mother's ongoing exposure to contaminants (such as pesticide residues on food) as well as to stored contaminants (such as PCBs in fat or lead in bone) that can be liberated during pregnancy. Once thought of as an impermeable barrier, the placenta, like the blood-brain barrier, functions somewhat like a semi-permeable membrane although this is an oversimplification as metabolic processes are also involved. Nevertheless, the placenta is equipped to filter large molecules or to kill any incoming bacteria with specialized immune cells. But it allows the passage of metals such as lead or mercury or other small molecules dissolved in fats such as pesticides or PCBs. Hence, substances in maternal circulation can be readily transported across the placenta with fetal levels in most cases comparable to maternal levels. Indeed, for methylmercury, the placenta actually serves to magnify levels in the umbilical cord blood to levels about 70% higher than occur in maternal circulation (Stern and Smith, 2003).

After birth, the breastfed infant may experience further exposure to the mother's body burden and ongoing intake of contaminants via her breast milk. Or, if fed with infant formula, a different range of contaminants may be present. A fuller discussion of infant food sources as a route of exposure and the implications for women's decisions to breastfeed are found in Section 2.4.

Children's smaller body mass and generally higher metabolic rates and activity levels can result in their being proportionately more exposed. Children take in (by breathing, ingesting or absorbing) more air, water, food and soil per unit body weight and consequently, more of the contaminants found in these media (NRC, 1993; Selevan et al, 2000; USEPA, 2003c). Likewise proportional to body mass, a child's brain is larger and receives more blood flow, than occurs in an adult. For example, the newborn has a surface area to body mass ratio that is 2.7 times that for adults and this relationship changes as children grow (Selevan et al, 2000). Per unit of body weight, infants consume proportionately double the amount of water and have breathing rates more than 65 times those of adults (Selvan et al, 2000). Some of these differences between children and adults are summarised in Table 2.1.

Table 2.1 Differences between Children and Adults

Criterion	Life Stage/Age Group			
	Newborn	Young child	Older child	Adult
Surface Area:Body Mass ratio (m ² /kg)	0.067	0.047	0.033	0.025
Respiratory ventilation rates	Infant			Adult
Respiratory Volume (mL/kg/ breath)	10			10
Respiration rate (breaths/min)	40			15
Ventilation rate	133			2
Drinking Water	<1 year	1-10 years	11-19 years	20-64 years
Mean intake (mL/kg/day)	43.5	35.5	18.2	19.9
Fruit Consumption (g/kg/day)	<1 year	3-5 years	12-19 years	40-69 years
Citrus fruits	1.9	2.6	1.1	0.9
Other fruits (including apples)	12.9	5.8	1.1	1.3
Apples	5.0	3.0	0.4	0.4

Source: Selevan et al 2000

Children also consume far greater amounts, per unit body weight, of certain foods compared to adults. Two differences are at work here. The first is the simple difference in physical size and the resulting difference in ratio between food intake and body weight as shown in Table 2.1. The second is behavioural. Although not the case for all children, it is often true that children have strong preferences for only a limited range of food types. A child's diet tends to include relatively large amounts of milk, fruits and fruit juices. When food choices are limited, any toxicants, including pesticide residues in these foods, will contribute disproportionately more to a child's exposure. For instance, childhood consumption of milk, combined with their greater thyroid uptake of iodine, caused them to accumulate far greater thyroid radiation doses from fallout of radioiodide nuclides after the Chernobyl nuclear reactor explosion. The latter

caused a major epidemic of thyroid cancer among exposed children (as reviewed by Wigle, 2003).

Because children are smaller in stature, their breathing zone is closer to the ground. Some contaminants, such as pesticides, mercury vapour, components of automobile exhaust and heavier particles of environmental tobacco smoke (ETS), are typically present in a vertical gradient, settling above the ground, therefore closer to a child's breathing zone (Fenske et al, 1990; WHO-EEA, 2002).

Additional behavioural characteristics among children, which vary at different developmental stages, also account for greater contact with and hence, greater exposure to, environmental contaminants or substances in consumer products. This greater exposure in young children is particularly relevant if soil or house dust are contaminated. Studies have shown that exposure varies inversely with age and this fits with the model that the greater hand-to-mouth activity of the very young enhances intake (Kissel et al, 1998; Health Canada, 1998a; Freeman et al, 2001). Additional contributing factors in children are crawling and/or toddling, frequent falling or deliberate contact with the ground, floors, and other surfaces, play patterns, alongside frequent hand-to-mouth behaviour. Children also play at ground level and raise dust that may be inhaled or ingested by mouthing contaminated hands or objects.

2.4. Breast milk - Still the optimal infant food

Researchers have known for some time that human breast milk carries environmental contaminants. Of particular concern are the persistent organic pollutants (POPs) which can persist in the environment, build up in the food chain, concentrate in breast milk and which can be associated with a variety of health effects (as discussed further throughout Chapters 3 and 4). In the early 1950s, the organochlorine pesticide, DDT was first detected in breast milk samples in healthy U.S. women (Kimbrough, 1991). Other POPs measured in breast milk include PCBs, dioxin and PBDEs. Following the widespread restrictions placed on PCBs and DDT in the late 1970s, the levels of these two POPs have steadily decreased in breast milk although PCBs and the metabolite of DDT are still detected in breast milk. Dioxin is also found in breast milk but levels have been dropping steadily as changes in certain industrial operations and additional regulatory controls have contributed to reduced environmental emissions. PBDE levels in breast milk have been steadily increasing across the 1980s and 1990s (Hoover, 1999; Ryan et al, 2002; Schecter et al, 2003; NEW, 2004). Exposure to POPs is discussed further in Section 4.3.2.

Contaminants in breast milk ultimately result from a woman's lifetime cumulative exposure, largely dietary and particularly through foods with a higher animal fat content such as some fish, meat, eggs and dairy products. Lipophilic contaminants become stored in body fat, a mechanism that acts to protect body tissues. During lactation, maternal fat stores provide about 60 percent of the fat that goes into breast milk whereas 30% comes from mother's diet and 10% is newly synthesized in the breast (Schreiber, 2001).

A number of studies (further reviewed in Section 3.2.3) have examined the health impacts from exposure to contaminants (mainly PCBs) in early life. One important observation is that it is difficult to distinguish between the effects from exposure to PCBs through breast milk versus the effects from exposure while in the womb (Ayotte et al, 2003). The evidence from four different cohort studies is consistent with the hypothesis that prenatal exposure (rather than the transfer through breast milk), during the time when the fetal nervous system is developing, is largely responsible for the subtle effects observed (Jacobson and Jacobson, 2001; Landrigan et al, 2002a; Ayotte et al, 2003). This conclusion underscores the truism that a mother's body is the "first environment" for every individual. As well, data from two separate cohorts of PCB-exposed children indicate a pattern of distinctly stronger and statistically significant cognitive effects associated with the prenatal PCB exposure only among the children who had *not* been breastfed (Jacobson and Jacobson, 2002). While the reason for this apparently protective effect of breastfeeding in these cohorts remains under discussion, it may be related to specific constituents of breast milk, or as the researchers speculate, the quality of intellectual stimulation that mothers who breastfeed provide for their children (Jacobson and Jacobson, 2002).

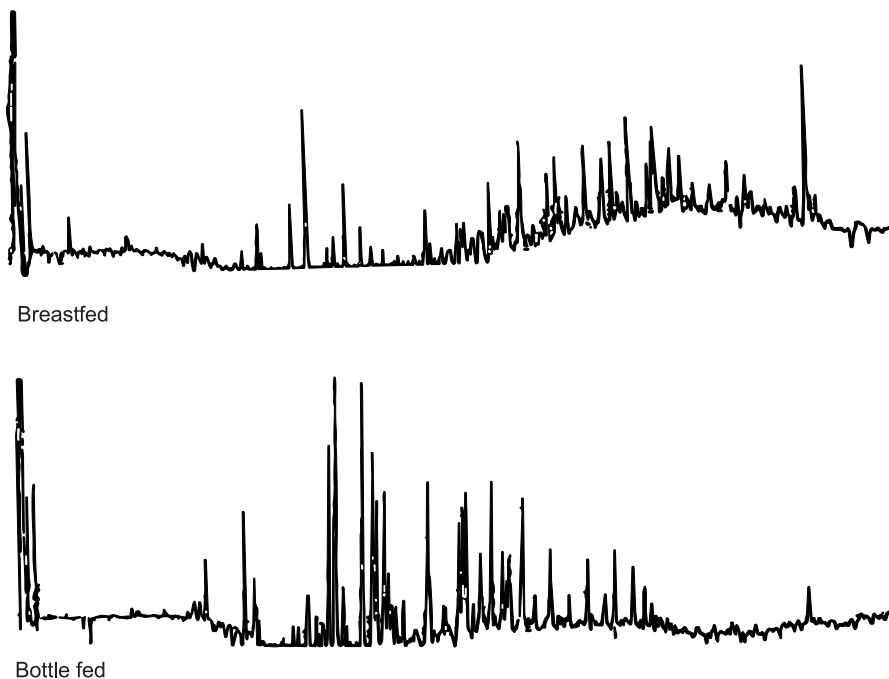
The unclear risks of these early exposures do not diminish the multiple, proven benefits (to both mother and child) from breast milk and breast-feeding. Breast milk (also referred to as "human milk") is an unsurpassed infant food, providing the precise nutrient composition to support optimal growth of the human infant. Recent research reveals as well that breast milk contains many different molecules and biologically active factors that confer a variety of physiological, developmental and protective benefits beyond nutrients and calories (German et al, 2002). Breast milk is also an economical and sustainable foodstuff for infants at a vulnerable time in their lives.

Contaminant profiles of infant formula and breast milk are slightly different with breast milk generally containing higher levels of the persistent substances that accumulate in the food chain. In general, infant formula, like any food, may contain trace levels of contaminants. For example, metals (e.g., lead, aluminum and cadmium), POPs (e.g., PCBs and dioxins) and phthalates have been found in infant feeding products, though again, not necessarily in amounts that pose a risk

to the infant (Health Canada, 2003a; USFDA, 2002; UKFSA, 2003, 2004; UKMAFF 1996). Often reconstituted with tap water, infant feeding products are also subject to contamination by any toxic substances or pathogens present in the water used (OPHA, 2004a).

The paired graphs below are gas chromatographs showing results of monitoring for contaminant exposure via the urine of two one-year old infants. The traces indicate that even at this young age, both breast fed (upper trace) and bottle fed (lower trace) babies were exposed to many different contaminants.

Figure 2.2 - Gas chromatograph analyses of urine of breastfed versus bottle fed infants



Peaks reflect different PCB congeners or pesticides identified. Heights of peaks indicate amount detected.

Source: Bush et al, 1990 as cited in Carpenter et al , 1998

Breastfeeding undeniably provides immense health benefits to both baby and mother. Mothers who breastfeed have immediate postpartum health benefits such as decreased bleeding, more rapid return of the uterus to its pre-pregnancy size and shape and reduced menstrual blood flow (AAP, 2005). Breastfeeding mothers also have long-term health benefits, such as reduced risk of conditions like osteoporosis and premenopausal ovarian and breast cancers (Cumming &

Klineburg, 1993; Newcomb et al, 1994). Breastmilk has been found to contain an array of substances that promote immune system development and regulate some aspects of the immune response in infants (Field, 2005). Breastfed babies gain protection against different childhood illnesses both in the short and long-term. For instance, breastfeeding has been correlated with significantly decreased incidence and severity of many infectious diseases (AAP, 2005). Breastfeeding is associated with reduced risks of postneonatal mortality and breastfed infants have lower rates of Sudden Infant Death Syndrome (SIDS) in the first year (Chen & Rogan, 2004, AAP, 2005). Breastfeeding also appears to confer lifelong benefits to health including lowered risks of asthma, certain cancers (e.g. lymphoma, Hodgkin's disease, acute leukemias in childhood), types 1 and 2 diabetes, obesity and overweight and hypercholesterolemia (AAP, 2005). There is also evidence of a positive association between duration of breastfeeding and cognitive and academic ability (Grantham-McGregor et al, 1999; Mortensen et al, 2002; Anderson et al, 1999; Jain et al, 2002).

These findings collectively reinforce the conviction that the tangible benefits from breast-feeding (for both infant and mother) compensate substantially for the potential risks from exposure to contaminants in mother's milk. Breastfeeding is therefore vigorously supported for its broad-reaching health protective effects. Many health organizations including Toronto Public Health, the Ontario Public Health Association, Health Canada, the Canadian Paediatric Society, the American Academy of Pediatrics and the World Health Organization continue to support the value and importance of breastfeeding, even in an environment contaminated with toxic substances (Frank & Newman, 1993; Schreiber, 2001; OPHA, 2004a; AAP, 2005; WHO, 2003a; PHAC, on-line).

Research by Toronto Public Health (TPH, HI&P 2005a) shows that the estimated breastfeeding initiation rate in 2003 among new mothers in Toronto is quite high (94%). The decision to breastfeed correlates with mother's age, education and income level; older mothers with higher levels of education and income are most likely to breastfeed their infants. Breastfeeding initiation is only one indicator and data show that duration of breastfeeding declines over the first year of an infant's life. Data from 2001 indicate that a substantial proportion of Toronto mothers (55%) had stopped breastfeeding after six months and most (about 75%) stopped breastfeeding by 12 months. Although Health Canada, Dietitians of Canada and the Canadian Pediatric Society (PHAC, on-line) all recommend exclusive breastfeeding for the first six months, fewer than one in five Toronto mothers exclusively breastfed their infants for six months.

2.5 Additional Risk Factors – Poverty, Poor Nutrition, and Lack of Information

Well established as a key determinant of health, poverty is also associated with greater likelihood and opportunities for exposure to harmful substances. Poverty is also linked with greater susceptibility to harmful effects from those exposures further compounding health disparities. The literature documenting the relationships between socio-economic status, environmental exposures and health is more extensive and has been more widely debated in the United States than in Canada (see e.g., Brown, 1995; Olden, 1996; Friedrich, 2000). In the U.S. it has attained significance in the research paradigm and social movement known as “environmental justice” (or equity) (USEPA, undated; NIEHS, 2001b; Lee, 2002). This perspective recognizes that the poor, those from visible minorities or Aboriginal communities, are more often downhill, downwind and downstream of harmful environmental exposures (CICH, 1997; Thompson, 2003).

Of particular relevance to children’s environmental health, environmental justice aims to eliminate the inequities in exposure according to income, minority status or ethnicity. The cycle of child poverty, higher exposure and vulnerability, lack of parental awareness and so on, requires urgent attention. Especially in the case of children’s exposure to neurotoxic substances such as lead, mercury or PCBs, the resultant compromise to cognitive and behavioural development can result in lifelong deficits, reduced likelihood of reaching their full potential and therefore possible perpetuation of a cycle of disadvantage environmentally, medically and economically, across generations (Friedrich, 2000).

At the international level it has also been well documented that in many nations, children living in extreme poverty, tend to be disproportionately exposed to environmental hazards (WHO-EEA, 2002). Canadian data and explicit attention to the environmental justice paradigm are limited although environmental inequities arguably underlie most of the major environmental issues of concern in Canada (Hegan, 1997; Thompson, 2003). Summaries have been compiled but there has been only limited research and less attention within the Canadian environmental health agenda (e.g., Hegan, 1997; Chaudhuri, 1998, Cooper, 2001; Jerrett et al, 2001; Finkelstein et al, 2003). Information on the diversity of child poverty in Toronto is already noted in Section 1.3. However, data to clarify the relationships among poverty, ethnicity, exposure, susceptibility and health disparities among children across Toronto are clearly lacking.

The poverty-related risk factors relevant to children in urban environments include the age, physical condition and location of their homes and

neighbourhoods, and the occupation and household practices of parents (or landlords).

If neighbourhoods (including schools, play areas as well as homes) are close to high-traffic roadways, children can experience increased exposures to traffic-related air pollutants such as benzene, a cancer-causing chemical added to gasoline, or to traffic noise, which can harm their ability to learn (Wigle, 2003). Limited Canadian data agree with the more extensive U.S. data indicating that the environmental health risks from industrial facilities, hazardous wastes disposal facilities and major roadways are more likely to affect low income areas, minority or Aboriginal communities (including reserves) (Thompson, 2003).

Indoors, rental units and low-income units, particularly apartments and those with frequent turnover in occupancy, can become more readily afflicted with cockroach infestations and therefore, more frequently sprayed with pesticides. Studies in the US note that inner city, minority populations are high-risk groups for indoor pesticide exposure (Perera et al, 2003; Landrigan et al, 1999).

Any dwellings built before 1976 when the lead content of paint was initially and only partially regulated, present a potential lead exposure hazard to children, pregnant women, and women of child-bearing age. Homes built prior to 1950 will have the highest levels of lead in old paint. Parents in low-income households may not have the resources or knowledge to adequately contain or repair flaking or deteriorating paint. By the same token, landlords that allow dwellings occupied by low-income tenants to fall into disrepair, also contribute to lead exposure among poorer children. Hence, in lower income, older dwellings, children may have greater exposure to lead in house dust. Data from Toronto's South Riverdale community at the time when lead contamination was being addressed, indicated that there was an inverse relationship between socioeconomic status and blood-lead levels which agrees with historic population level data from the U.S. (Chaudhuri, 1998; Lidsky and Schneider, 2003). While poverty can contribute to the deteriorated condition of older painted surfaces, it is important to also recognize that renovation practices are a hazard for high lead exposure for any socio-economic group.

Dampness, another significant issue for indoor environments, can be worse in poorly maintained buildings (such as can exist under conditions of poverty) but can also arise from "tight" buildings that have been sealed too effectively for the sake of energy efficiency. Dampness can contribute to house dust mites (HDM) and mould growth as well as provide hospitable environments for cockroaches and rodents. Excessive moisture may also initiate chemical emissions from building materials and furnishings (IOM, 2004). While this field is complicated, there is increasingly solid evidence that the HDM and mould growth encouraged

by indoor dampness can contribute to allergies and respiratory problems in children, including asthma episodes.

Low income is often associated with greater frequency of smoking and therefore, greater exposure to ETS inside the home among poorer children. ETS exposure is more likely among disadvantaged households that is, those with a single parent or that are of low-income (as reviewed by PS-FC, 1999). For example, 51% of Canadian children whose parents are low-income are regularly exposed to ETS, compared to 18% of children in the highest-income families. ETS is another significant risk factor for respiratory and neurodevelopmental problems as well as creating exposure to the over 40 known cancer-causing chemicals in ETS.

There also appears to be a relationship between low socioeconomic status and greater susceptibility to harmful effects from toxic environmental exposures. For example, data from Hamilton, Ontario indicate that the effect of air pollution on mortality is more pronounced in areas of low social status (Jerrett et al, 2002). In Toronto, poorer children appear to be particularly vulnerable to general respiratory health effects from air pollution and are more likely to be hospitalized from respiratory conditions as discussed further in Section 3.2.2. (Toronto, 2000). As well, research on lead suggests that SES has an independent effect on the neurotoxicity of lead, though the mechanism is not entirely known (Lidsky and Schneider, 2003). One hypothesis (supported by animal studies) is that impoverished as opposed to socially stimulating and enriched environments dampen the expression of certain genes that influence neural growth and development (Lidsky and Schneider, 2003). Parallel findings in a study of postnatal PCB exposure support the corollary of this hypothesis in that harmful effects were partly offset for children living in a stimulating home environment (Walkowiak et al, 2001).

Inadequate nutrition is clearly a key condition that predisposes children of lower income homes to greater risks to health in general as well as from environmental exposures. Pregnant women, specifically the developing fetus, are particularly at risk. Poor nutrition, including deficiencies in protein, calcium or iron can compound absorption of and adverse effects from, toxic exposures. For example, poor nutrition results in children having greater uptake and impaired biological capacity to deal with lead exposure (Bruening et al 1999, Mushak and Crocetti, 1996, and Mahaffey, 1995). Studies have confirmed that a child's digestive system already absorbs more lead than an adult's (50% compared to 10%). The rate of lead absorption in the digestive system of pregnant women is similar to what occurs in children (Wigle, 2003). As well, in both children and pregnant women, absorption will be even greater where dietary calcium or iron is deficient. The result can be even greater lead exposure during highly sensitive stages of fetal, infant and early childhood brain development. The corollary may also be

true, in that certain aspects of good nutrition mitigate the effects from contaminants as has been suggested with studies of methylmercury (Clarkson and Strain, 2003; Passos et al, 2003).

Toronto's Nutritious Food Basket survey determined that for 2004 the average monthly cost of food for a family of four was about \$525, an increase of over 13% since 1999 (TPH, 2004a). These rising food costs, along with rising rents and decreasing social assistance rates mean that the nutrient and energy needs of children living in low income families are less likely to be met now than they were five years ago. By inference, their ability to withstand adverse effects from physical and social environmental stressors is likely compromised.

Finally, lack of information can contribute to greater exposure. There is a wide range of possible sources of potentially toxic substances in the air, water, food, soil, indoor dust and consumer products. It is extremely challenging for parents and caregivers as well as pregnant women, to be aware of all such risks much less to have a sense of relative importance or the means of avoiding exposure or otherwise taking precautions. Available information can also be alarming and conflicting. For example, concerns about pesticide exposure for children might be expressed by a family doctor and then refuted by a university scientist.

Toronto Public Health has some limited data on the information needs of parents in Toronto. In 2002, as part of implementing one recommendation of the Needs Assessment Framework Study, TPH conducted a survey of parents to assess awareness and knowledge about environmental health issues and children (TPH, 2002c). While Toronto parents with the lowest reported household income often took extra measures to protect their children compared to those with higher income levels, the lower education group was less likely to feel they could do a lot or even a moderate amount to protect their children from environmental hazards. (Further details on the awareness of Toronto parents are summarized in Chapter 5.) Another survey was done in 2000 of knowledge, behaviour and attitudes among Toronto residents with respect to pesticides used indoors (TPH, 2004b). Additional information about public concerns comes from the annual Health Hazard Statistics, collected by Healthy Environments, the TPH inspections division. Discussion of the results of these surveys and statistics is provided in Chapter 5.

CHAPTER THREE - HEALTH OUTCOMES OF CONCERN

3.1 Overview of Environment and Health Issues of Concern in Children

There is increasing evidence of associations between health outcomes and various pollutants to which all children are exposed. These outcomes include both developmental and functional endpoints. For some contaminants such as lead, ozone and particulate matter (and possibly mercury) it is widely believed that there is likely no exposure threshold below which children are unaffected (Canfield et al, 2003; Landrigan, 2000; Burnett et al, 1994; Health Canada and Environment Canada, 1999; Rice, 2004).

The broad categories of health outcomes to be discussed in this chapter for which exposures in children have been linked to some environmental pollutants are: birth defects, low birth weight and altered fetal growth, asthma and other respiratory effects, cancers occurring during childhood or young adulthood, cognitive and behavioural outcomes related to a range of impacts on fetal and childhood brain development, and reproductive and other developmental effects.

Known associations, or areas where there is scientific “proof” of cause and effect relationships between specific human health outcomes and specific environmental exposures, are limited to a small range of substances. For a much larger range of exposures, evidence is more often suggestive of associations since it derives from a more limited range of studies, often in animals. In addition to the health outcomes noted above, there is emerging evidence pointing to effects in the immune and endocrine systems. This Chapter concludes with a summary of recent research that attempts to calculate the monetary impact of the health effects in children of some key environmental exposures.

3.2 Large Numbers of Children Affected

Two health outcomes that are observable in large numbers of children include respiratory conditions, particularly asthma, and a range of conditions related to cognitive and neurobehavioural functioning. Substantial evidence demonstrates associations between respiratory effects and indoor and outdoor air pollutants. This evidence includes numerous studies, including in Canada, showing associations between increased air pollution and increased respiratory illness among vulnerable populations including children.

For effects and related outcomes in the developing nervous system, the situation is less well understood. There is strong evidence that low-level lead exposure can contribute to developmental neurotoxicity (damage to the brain or nervous system during developmental stages) and suggestive evidence for neurotoxic effects of low-level prenatal maternal exposure to PCBs and related toxicants. There is also a very large number of children in Canada (nearly one third) affected by one or more learning or behavioural problems. Linking the latter to low-level environmental contaminants other than lead is currently impossible. However, these trends among children and concern about suspected neurotoxicity of a large number of substances are contributing to greatly increased research in this area. Each of these lines of evidence is described below.

Large numbers of children also live in poverty. As previously noted, nearly 20% of children living in Canada live in poverty but this level rises to 30% of children in Toronto. This constitutes a large number of children who are at risk for hazardous exposures and associated ill health effects, particularly when housing quality is poor or inadequate, or children are malnourished or homeless.

3.2.1 Respiratory Conditions and Air Pollution

Short-term exposure to outdoor air pollution has been clearly and consistently associated with increases in premature deaths, hospital admissions for respiratory and cardiovascular diseases, respiratory infections, asthma symptoms, respiratory irritation, and reductions in lung function among adults and/or children (Bates 1995; Environment Canada 1999a, 1999b; Wigle 2003).

Childhood exposure to air pollutants is associated with deficits in lung growth, up to and including deficits that result in below-normal lung development (Gauderman et al, 2004). Adult lung cancer and cardiopulmonary mortality have been linked to long-term exposure to air pollutants (Pope et al., 2002).

While an increasing body of evidence demonstrates that air pollution can affect all members of society, children, the elderly and those with predisposing health conditions are particularly vulnerable. For example, a study conducted in Toronto demonstrated that a 45 parts per million (ppm) increase in ozone levels was associated with a 19% increase in respiratory hospital admissions among adults and a 35% increase in respiratory hospital admissions among children less than 2 years of age (Burnett et al., 2001).

Data linking increased hospital admissions to air quality indicate that ozone and particulate matter can cause demonstrable health effects even at very low levels and that there is likely no threshold for such health effects (Burnett et al 1999).

This finding and those summarized above are confirmed in the recent publication of the “Children’s Health Study,” a multi-year epidemiological investigation of air pollution impacts in children in Southern California (Peters, 2004; Gauderman et al, 2004). This longitudinal study of 3,535 children in 12 communities found associations between breathing polluted air in Southern California (including at levels below current ambient air standards) and significant chronic deficits in lung function among adolescent children. Three to five times more children living in communities with high outdoor air pollution had clinically significant deficits in lung functioning compared to children in lower pollution communities.

Indoor and outdoor air quality contributes to the burden of illness from asthma. Exposure to pollutants in air can trigger and exacerbate respiratory symptoms among asthmatic children. An interesting “natural experiment” in Atlanta, Georgia during the 1996 Summer Olympic Games, highlights how improvements to outdoor air can mitigate impacts on asthma in children. The decision to significantly limit downtown Atlanta traffic for the 17-day duration of the games resulted in decreased traffic density and lower daily peak ozone levels. These reductions were associated with significantly lower rates of childhood asthma events severe enough to require medical attention (Friedman et al, 2001).

Although exposure to outdoor air pollution has long been associated with exacerbating existing asthma, studies have shown that exposure to high levels of ozone in children engaging in high activity outdoor sports may contribute to the risk of developing new-onset asthma (McConnell et al, 2002). Regardless of activity levels, an increasing body of evidence is suggestive of a causal relationship between incident asthma and outdoor air pollutants, especially ozone (as reviewed in Wigle, 2003 and updated on-line at Wigle, 2004). The Southern California Children’s Health Study found that the incidence of new asthma cases was associated with heavy exercise in communities with high levels of ozone in the air (McConnell, 2002; Peters, 2004). A study of Japanese children found proportionately greater risk (that is, a dose-response relationship) between incident asthma the closer the home was to main roads (Shima et al, 2003). A study of asthmatic children in France found similar associations between early exposure to air pollutants from traffic (measured as cumulative traffic density) and onset of asthma (Zmirou et al, 2004). Although the available studies are relatively few, evidence is viewed as being suggestive for a causal association between living close to high traffic areas and the prevalence/incidence of asthma symptoms (Binkova et al, 2004). There is a clear need to assess this relationship further, to determine which specific air pollutants and the critical exposures times (that is, prenatal, early infancy etc.) that pose the greatest risks for development of asthma.

In addition to respiratory conditions, both the Southern California study and another longitudinal study at Columbia University in New York City have found associations with adverse developmental outcomes. Prenatal maternal exposure to air pollutants has been linked to increased risks of reduced birth weight and preterm birth (Ritz and Yu 1999; Ritz et al. 2000; Liu et al. 2003; Srám et al, 2005) and some birth defects (cardiac birth defects) (Ritz et al. 2002). The Columbia University prospective study also found associations between prenatal maternal exposure to high levels of polycyclic aromatic hydrocarbons (PAHs) in urban air pollution and low birth weight (Perera et al., 2003). More recent findings note associations between prenatal maternal PAH exposure and chromosomal aberrations in cord blood a predictor for increased cancer risks. These findings are described in more detail in Section 3.3.3, Effects of Toxic Substances on Development.

The Contribution from Indoor Air Contaminants

A number of indoor air contaminants have been associated with respiratory infections, aggravated asthma symptoms, and/or the development of asthma. Contaminants include: the constituents of environmental tobacco smoke (ETS) (i.e., second-hand smoke); inhalable particles and gases released from fireplaces, wood-burning stoves and gas stoves; volatile chemicals emitted from furniture, cleaning products and household furnishings; and biological allergens from moulds, pet dander, house dust mites and cockroaches (Lanphear et al, 2001; Courage, 2002; IOM, 2000). The evidence linking early-life exposures to the development of new-onset asthma is particularly strong for ETS and house dust mite antigen while there is suggestive evidence for wood smoke, exhaust, pesticides, pet dander and cockroach antigen (Salam et al, 2004; IOM, 2000; Lanphear et al 2001). Indoor exposures are of particular importance since children spend most of their time indoors (as discussed in Chapter 4).

It is worth highlighting that there is an enormous body of literature that examines the health effects from childhood exposure to ETS, as well as from maternal smoking during pregnancy. Children who are exposed to secondhand smoke are at increased risk of suffering from a variety of respiratory and other health conditions including frequent episodes of respiratory illness and infections, middle ear infections, asthma initiation and exacerbation, recurrent pneumonia, developmental and learning delays, sudden infant death syndrome (SIDS) and cancer in adulthood (Fergusson et al, 1980; Etzel et al, 1992; CPS, 1986; Taylor and Sanderson, 1995; Sandler et al, 1985; ALA, 1994; IOM, 2000 and Courage, 2002). There also appears to be an additive effect of maternal prenatal exposure to ETS and urban air pollution resulting in adverse birth outcomes. In the ongoing investigations at Columbia University looking at child health and environment

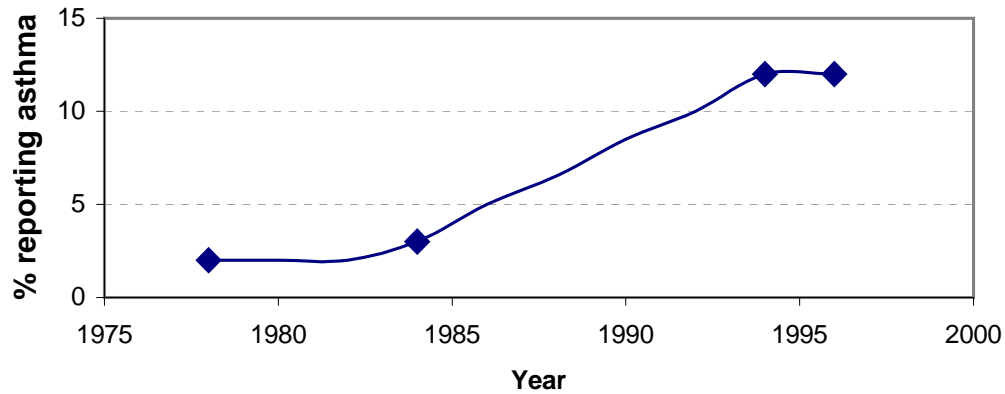
interactions, researchers have found a significant interaction between ETS and polycyclic aromatic hydrocarbons (PAHs) in urban air pollution such that the combined exposure had a significant multiplicative effect on adverse fetal development including deficits in birth weight and head circumference (Perera et al, 2004).

Paradoxically, household cleaning products are also believed to contribute to indoor air contamination, as they can contain chemicals such as formaldehyde. Increased risks of asthma and related symptoms have been associated with occupations classified as ‘cleaners’ compared to ‘office workers’ (Zock et al, 2002). Significant associations have also been found between cleaning products and the development of respiratory conditions in children. One study found a dose dependent effect between maternal use of household chemical products during pregnancy and persistent wheeze during the pre-school years in the resultant children though it was not possible to conclude whether this was associated with exposures occurring *in utero* or continued use of products postnatally (Sherriff et al, 2005).

3.2.2 Respiratory Effects – Available Data on Trends in Children

Statistics on asthma incidence in children in the US and Canada are similar. In the US, the prevalence of asthma among pre-school-aged children rose throughout the 1980s and 1990s. Recent figures indicate that 13% of US children under 18 have been diagnosed with asthma at some point in their lives with low income children more frequently affected (USDHHS, 2005). Likewise in Canada, the prevalence of childhood asthma stood at 2.5% of children aged 0 to 14 years in 1983 and by 1995 had risen dramatically to 11.2% (MOHLTC, 2000). The most recent prevalence data come from the National Population Health Survey (1996-97) which indicated 12.2% of children had been diagnosed with asthma (Health Canada, 1999).

Figure 3.1 Reported Asthma Prevalence, Birth to 19 Years Canada, 1978-1996



Notes: 1984 Survey included children 0 to 14 years only

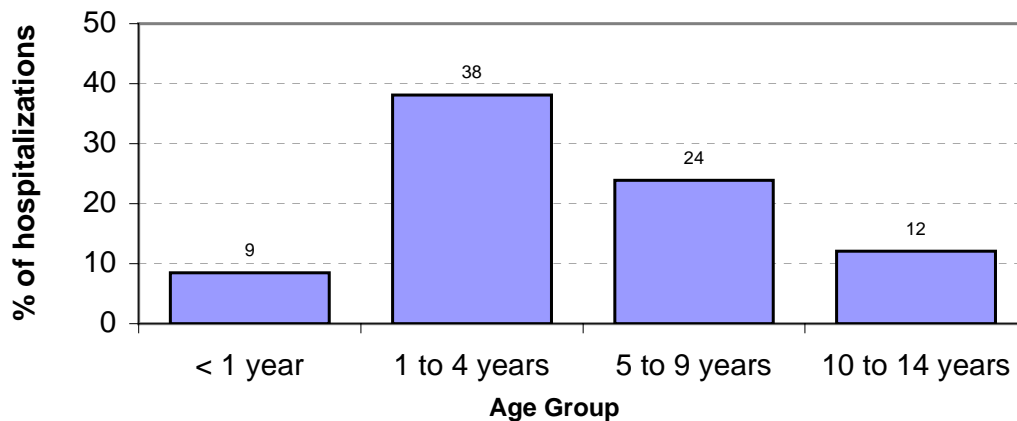
Percentages are weighted estimates

Source: Health Canada 1999

The number of asthma-related hospital admissions of young children in Canada increased by 28% among boys and 18% for girls between 1980-81 and 1989-90 (Dales, 1994). In Ontario, from 1980-1994, hospital admissions for asthma, bronchitis, pneumonia, and croup in children under two years increased by 27% to 45% when ozone levels increased (Burnett et al 2001). In London, England, another study found that exposure to nitrogen dioxide from car exhaust nearly doubled the severity of viral-induced asthma attacks (Chauhan et al, 2003).

Overall, respiratory disease is the leading cause of hospitalization among young children in Toronto. As Figure 3.2 demonstrates, children under 5 are most likely to be hospitalized for respiratory conditions, with acute bronchitis as the leading respiratory cause for hospitalization among children under 1 year, and asthma as the leading cause among children 1 to 4 years in age.

Figure 3.2: Percentage (%) of Hospitalisations Attributable to Respiratory Conditions, 0 to 14 years, Toronto, 1997-2001



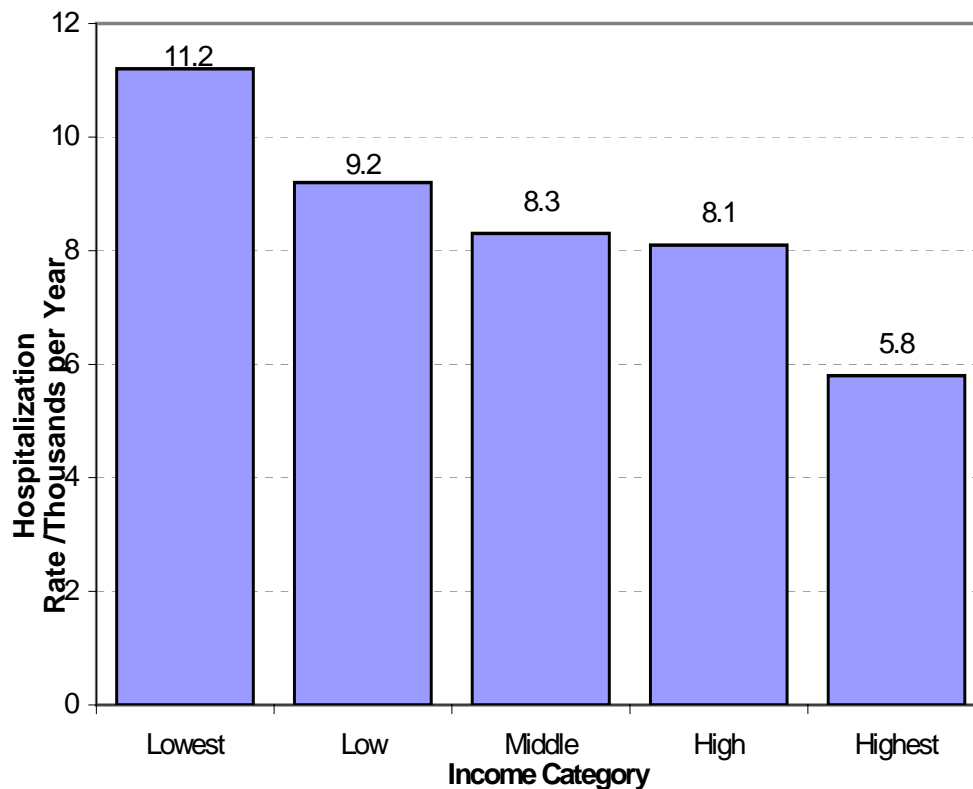
Source: Hospital Inpatient Data, Provincial Health Planning Database (PHPDB)

Hospitalization is only a partial measure of population of asthma since studies estimate that only one in ten children who report having asthma are hospitalized (Toronto, 2004c). Estimates for the year 1997 were that about 15,000 children suffered chronic bronchitis episodes and many more suffered exacerbations of asthma due to air pollution (TPH, 2000). Physicians report that they treat children for acute and chronic respiratory symptoms more than any other health complaint (Toronto, 2000). Although many such children have respiratory infections, air pollution increases children's susceptibility to such infections.

Finally, as discussed in Chapter 2, studies have shown that socioeconomic status is linked to the severity of asthma experienced and that poor children are more likely to suffer from disabling asthma (i.e., asthma attacks that have to be treated in hospital and that force them to miss school) (Halfon and Newachek, 1993). Toronto data from 1996 to 1999 indicate that poorer children may be particularly

vulnerable to general respiratory health effects from air pollution. For example, hospital admissions rates for respiratory conditions in children (asthma, croup, bronchitis and pneumonia) ages 0 to 14 years were nearly double among children from the lowest income areas (11.2 hospitalizations per 1000 children per year) compared to those from the highest income areas (5.8 per 1000 per year) (Toronto, 2000). These data are summarized in Figure 3.3. This pattern is consistent with recognized links between income and effects from outdoor air pollution (Finkelstein et al, 2003; O'Neill, et al. 2003).

Figure 3.3 Respiratory Hospitalization*, Children 0 to 14 years, by Income Category, Toronto, 1996-1999



Sources: Provincial Health Planning Database, Hospital Inpatient Data, Ministry of Health and Long Term Care; Statistics Canada Census, 1996

*due to asthma, croup, bronchitis or pneumonia

It is not possible to link these hospitalizations directly with air pollution since other causal factors will be involved. But the trend is consistent with the known links between income and indoor/outdoor air pollution and should be cause for concern and inform further research and preventive action, particularly for the most vulnerable children.

3.2.3 Effects of Toxic Substances on the Nervous System

A great deal of evidence has emerged in the last decade about the toxic effects of pollutants on the developing nervous system. At the same time, scientists have uncovered the enormity of our ignorance about this field. Understanding how the human brain develops is no small task. Likewise, understanding the reasons for learning or behavioural disabilities or conditions is equally complex. Part of the complexity is that effects on the brain can include both developmental and functional health outcomes. Brain development and functioning is the result of complex interactions among genetic, environmental and social factors. Nor are health outcomes in this area easily defined or diagnosed (such as with a simple blood test) or consistently identified. Rather, physicians and parents are faced with a broad spectrum of disorders and behaviours. Of the diverse factors that influence brain development and functioning, many parallel the determinants of health described in section 1.2. However, this issue-focussed report examines exposure to neurotoxic contaminants in the environment as only one of many of these influencing factors.

Evidence of developmental neurotoxicity is best understood for only a handful of substances. These include metals like lead and mercury, nicotine (from maternal cigarette smoking and ETS exposure), dioxins and polychlorinated biphenyls (PCBs), radiation, and some pesticides and solvents (including alcohol) (Wigle, 2003; Schettler et al, 2000). Associations have been found between these kinds of toxic exposures and neurodevelopmental disorders such as learning disabilities, intellectual deficits and attention deficit/hyperactivity disorder (AD/HD) and propensity to violence. Among environmental contaminants, the evidence is strongest for lead and PCBs (Weiss and Landrigan, 2000). There is a much larger number of substances for which neurotoxic effects are suspected but for which the information base is poor. Environmental links have been postulated for dyslexia and autism but there is little supporting evidence (no large well-conducted studies have been done yet).

Known Developmental Neurotoxins

Lead

Health effects from low-level lead exposure are largely asymptomatic or sub-clinical. The cognitive and behavioural effects include developmental delays, deficits in intellectual performance and neurobehavioural functioning, and reduced attention span (Wigle, 2003, Cooper, et al 2000). Low-level lead in utero exposure is also linked to various developmental effects such as greater risk of prematurity, effects on birth weight and decreased stature. Sensorimotor effects, such as diminished hearing acuity, are also evident from exposure to lead at low levels (Wigle, 2003).

Adverse neurological and neurobehavioural effects of relatively low-level lead exposure encompass a variety of measured and observed effects including:

- deficits in IQ or deficits in comparable/age appropriate tests of intellectual functioning;
- deficits in speech and language processing;
- deficits in perceptual-motor function and integration;
- deficits in reaction time;
- reduced attention span;
- non-adaptive classroom behaviour;
- deficits in reading, spelling and mathematics scores;
- poorer handwriting;
- significant increase in the risk for learning disabilities, as measured by the need for remedial education in reading, speech and math;
- sevenfold increased risk of failure to complete high school;
- sixfold increased risk for reading disability;
- poorer vocabulary and grammatical reasoning scores; and
- poorer hand-eye coordination. (Summarized from Needleman and Bellinger, 1991)

Blood-lead levels of 0.5 micromoles per litre ($\mu\text{mol/L}$) (Canadian metric nomenclature) or 10 micrograms per decilitre ($\mu\text{g/dL}$) or higher are now widely considered by health authorities as too high. [Note: Since the majority of scientific literature on lead reports blood-lead measures in micrograms per decilitre, this report steps away from Canadian metric nomenclature conventions in describing blood-lead levels.] As an “intervention level,” $10\mu\text{g/dL}$ is a trigger for taking precautionary action to investigate and remove lead sources (AAPH, 1998). However, recent research has found health effects below $10\mu\text{g/dL}$, with no discernible lower threshold (or level below which there are no effects) (Canfield

et al, 2003). Several studies have also found larger effects on IQ at blood-lead levels below 10 µg/dL than appeared at higher levels (Lanphear et al 2000; Canfield et al 2003; Bellinger and Needleman 2003; Wasserman et al 2003). Finally, the effects of low-level lead exposure in children may be irreversible.

Mercury

Mercury exists in many forms, methylmercury being the most toxic. Damage to various organs and body systems will vary depending on the amount and form of mercury exposure. The neurological effects of high-level mercury poisoning were learned unexpectedly, via the tragedies of Minamata, Japan (also described in Chapter 2), and unintended but widespread poisoning in Iraq from consumption of bread made from methylmercury-treated seed grain. In addition to many deaths and serious health effects among children in Minamata and Iraq, there was evidence of the greater sensitivity of children. Harmful effects from prenatal exposure included mental retardation, lack of coordination, blindness, seizures, and inability to speak. Children poisoned by methylmercury were also found to develop kidney damage (as reviewed in Wigle, 2003).

Low-level exposure to methylmercury from maternal consumption of fish, has been associated with deficits in attention, fine-motor skills, language development, visual-spatial abilities and verbal memory among the children exposed *in utero* (USEPA, 2000; NRC, 2000a). Debate about low-level effects continues in the scientific literature with some longitudinal studies showing neuropsychological effects of low-level exposure (for example, among Faroe Islanders - Grandjean et al, 1998, and in New Zealand - Crump et al, 1998) and others finding no effects (for example, studies in the Republic of Seychelles - Myers et al, 2003, Huang et al, 2005). The recommended intake levels for mercury in food, particularly in fish, have come under review in recent years as new evidence has emerged. Despite the scientific debate, experts conclude that the issue is not whether methylmercury poses a risk but rather making a determination about the appropriate dose that would provide prudent protection for the most vulnerable individuals in the population (Stern et al, 2003).

PCBs

Polychlorinated biphenyls, or PCBs, include a group of over 200 compounds used for many different purposes until they were widely restricted in the early 1970s. Manufacturing was discontinued in most countries by the late 1970s. Ongoing use occurs in sealed units such as electric transformers, large stockpiles remain and these compounds continue to circulate in the environment. PCBs are extremely stable compounds at high temperatures, highly soluble in lipids (or fats), and

resistant to biodegradation. Although useful, these properties are the reason that PCBs are highly persistent in the environment. PCBs are also particularly toxic to the developing nervous system and are linked to a wide range of additional health effects (discussed in subsequent sections of this chapter).

Pre-natal exposure to high levels of PCBs is associated with developmental delays and cognitive deficits in infants (Kimbrough, 1991). At lower exposure levels, there is suggestive evidence of deficits in neurologic development but, like the investigation of lead, subtle effects are difficult to demonstrate conclusively. One of the most well-known investigations is among children in Michigan whose mothers regularly ate PCB-contaminated fish from the Great Lakes during pregnancy (and during the six years prior to pregnancy). In a series of prospective studies these children have shown neurological effects and neurodevelopmental deficits at birth, during infancy, through early childhood (age 4) and at age 11 years. The effects are linked to prenatal PCB exposure and have been observed, though not always consistently, in other studies in other locations around the world. Specific effects include abnormal reflexes among newborns, reduced motor skills among infants, cognitive deficits at age 4 years, and, in the Lake Michigan study, full-scale and verbal IQ deficits at age 11 years. (Jacobson and Jacobson, 1996; Jacobson et al 2002; and as reviewed in Wigle, 2003). Whether postnatal exposure, especially via breast milk, is also associated with these effects is not clear. Although one German study (Walkowiak, et al, 2001) found associations between postnatal (i.e. through breast milk) PCB exposure and learning deficits, most have not (as reviewed in Wigle, 2003). However, the findings in the German study are consistent with animal results where low-level postnatal exposure in monkeys has been associated with learning deficits (Rice, 1997).

Pesticides

Pesticides include a wide range of different substances with many different “pest-killing” purposes and modes of action. Concern exists about those pesticides with various types of toxicity in experimental animals including neurotoxicity (mode of action is interference with the normal functioning of the nervous system), carcinogenicity (capable of causing cancer), teratogenicity (capable of causing birth defects) and endocrine disruption (interference with normal endocrine function). Epidemiologic studies of potential health effects of pesticides in children have been limited mainly to acute poisoning and childhood cancer.

Despite the fact that most insecticides (pesticides used to kill insects) are neurotoxic, there are very few experimental animal studies and even fewer epidemiological studies that have assessed developmental neurotoxicity (DNT) or

the ability of pesticides to harm the developing nervous system. Moreover, research has shown that DNT tests are *the* most sensitive health end-point in experimental animals for the majority of pesticides (Makris et al, 1998). DNT tests are more sensitive than the standard animal studies (such as tests for developmental or reproductive toxicity, or adult neurotoxicity tests).

Some have predicted that human prenatal exposure to neurotoxic insecticides may contribute in humans to social and emotional deficits, up to and including, at the most extreme end of the spectrum of nervous system disabilities, conditions like autism, cerebral palsy, and mental retardation (Goldman and Kodura, 2000). Many years of study, such as occurred to demonstrate the neurotoxic effects of lead and PCBs, would be necessary to assess the potential role of pesticides in these developmental conditions.

While not all scientists draw the same conclusions (e.g., Maurissen et al, 2000, Sheets, 2000), growing evidence from animal and cell culture studies shows that the developing animal nervous system is particularly vulnerable to neurotoxic effects from some insecticides, such as those from the organophosphate and carbamate classes (Eskenazi et al, 1999, Rice and Barone, 2000). There is very little human research on neurotoxic effects from exposure to these types of pesticides but some possible links have been drawn between children highly exposed to organophosphate pesticides and neurodevelopmental and neurobehavioural effects (Guillette et al, 1998).

Emerging data on other substances

As noted in Chapter 2, extremely sparse information exists to evaluate the neurotoxic effects, particularly developmental neurotoxicity, of most substances. Indeed, the depth of ignorance about whether substances are neurotoxicants pales by comparison to how little scientists know about whether substances are also or specifically toxic to the *developing* nervous system.

According to a literature scan done by scientists working for the US-based non-governmental organization (NGO) Environmental Defense, there are over 1100 substances, including several groups of substances, in commercial use in the US for which there is scientific evidence of suspected neurotoxicity (Scorecard, online). Whether or not these substances are also or specifically toxic to the developing nervous system is largely unknown. Of these 1100 substances, nearly 800, are in use in Canada. Of these 800, over 300 are on a preliminary list (created by Health Canada) of substances that the agency considers present the greatest potential for human exposure. The Health Canada list excludes pesticides (unlike the Environmental Defense list). Hence, a larger number of suspected

neurotoxins present exposure risks in Canada if pesticides are included in this preliminary list given what is known or suspected about the neurotoxicity or developmental neurotoxicity of some pesticides (Cooper, 2004).

In dealing with the backlog of large numbers of unregulated and inadequately studied substances, lessons can be learned from groups of substances like PCBs. One lesson is that substances like PCBs are similar in important ways to a wide range of other groups of compounds for which the scientific evidence of exposure and health effects is limited or in some cases is completely lacking. These similarities can include properties such as environmental persistence and also common mechanisms of toxicity. PCBs can be included among a wider group called polyhalogenated aromatic hydrocarbons (PHAHs). These are semi-volatile chemicals that are stable at high temperatures, highly soluble in lipids, and resistant to biodegradation. PHAHs can be chlorinated, brominated, or mixed halogenated (i.e. depending on which specific halogen molecule is attached). They are similar in structure and toxicity but widely variable in potency (Wigle, 2003). Most are also considered Persistent Organic Pollutants or POPs. Major sub-groups include polyhalogenated biphenyls, diphenylethers, dibenzo-*p*-dioxins, dibenzofurans and naphthalenes (see Table 3.1).

Table 3.1 PHAHs: Types and Acronyms

	Polychlorinated	Polybrominated
Dibenzo- <i>p</i> -dioxins	PCDDs	PBDDs
Biphenyls	PCBs	PBBs
Dibenzofurans	PCDFs	PBDFs
Diphenylethers	PCDEs	PBDEs
Naphthalenes	PCNs	PBNs

Source: Wigle, 2003

Most of these substances are suspected of being capable of a wide range of toxic effects including neurotoxicity. The dioxins and furans are considered among the most toxic of all substances. Of particular concern currently are the PBDEs (polybrominated diphenyl ethers) that are widely used as flame retardants. They are routinely found in indoor dust and data on rising levels in human breast milk has prompted regulatory action in Europe and some US states. Bans are being put in place on what appear to be the most persistent and toxic members of this group of substances. Exposure to PBDEs and other Persistent Organic Pollutants (POPs) is discussed further in the Chapter Four.

To summarize, concerns about substances that may be toxic to the developing brain can be viewed in the following context:

- The developing brain is vulnerable. Health outcomes result from many factors and can be developmental and/or functional. Environmental causes apart from lead, methylmercury, PCBs and ETS are largely unknown and their investigation must occur within a multi-disciplinary and evolving field of inquiry;
- There are large numbers of substances (and quantities of emissions or other means of exposure) suspected of having the same kinds of neurotoxic effects as the small number of substances that have been well studied; and
- There are large numbers of children affected by learning and behavioural problems that may have environmental links (but limited research to date does not prove direct links to environmental exposures).

3.2.4 Nervous System Effects – Available Data on Trends in Children

Trend data exist in both the US and Canada concerning a broad spectrum of neurological and neurobehavioural outcomes. These data are available from national and state- or provincial-level investigations. The prevalence of these conditions is comparable in the US and Canada. Where Ontario and Toronto-specific data were found, they appeared to fit within this larger trend information. However, the research for this review found very little in the way of Toronto-specific data.

In the United States, it is estimated that:

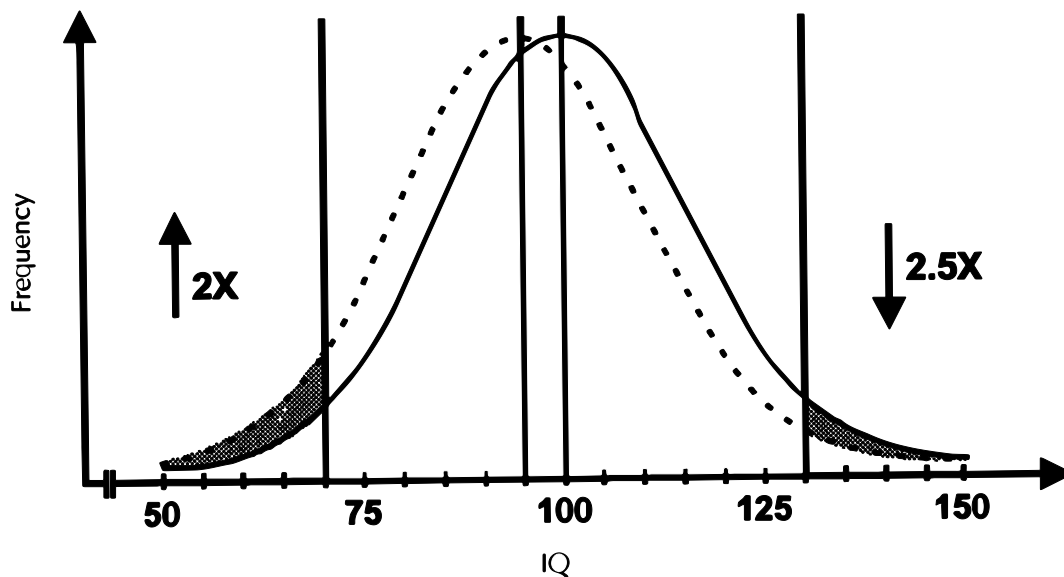
- 3 to 8% of live-born infants have, or go on to develop, neurodevelopmental disabilities including dyslexia, Attention Deficit Hyperactivity Disorder (AD/HD), major cognitive deficits, and autism (Weiss and Landrigan, 2000);
- 12 million children in the United States (17% of children) have one or more developmental, learning or behavioural disabilities or problems (Boyle, et al, 1994);
- Within the above estimate of 12 million are included the 5 to 10% of all children estimated to have learning disabilities (Parrill, 1996; Kavale and Forness, 1992). US statistics for the year 2003 show that almost 5 million US children 3-17 years of age (8%) had a learning

disability; 9% of boys had a learning disability compared with 6% of girls (USDHHS, 2005);

- Almost 4 million children in the US, 3-17 years of age (6%) had AD/HD in 2003. Boys were more than twice as likely as girls to have AD/HD (9% versus 4%) (USDHHS, 2005);
- About 1% of US children are mentally retarded* (Am Psych Assoc, 1994); and
- Programs for children in the US classified with learning disabilities increased nearly two-fold from 1977 to 1992 (Kavale and Forness, 1992).

* Note that the term “mental retardation” is generally viewed as objectionable in Canada. It is still used in older texts and in the current US literature. Because most of the references used in this report are taken from the US literature, the authors have used the term mental retardation so as not to misinterpret others’ reports. The preferred term in Canada is “intellectual disability.” Mental retardation is often defined in terms of an individual having an IQ < 70 (see figure 3.4) but other criteria, beyond intellectual capacity, are also considered.

Figure 3.4 - Distribution of IQ and changes with a 5 point downward shift



The figure shows how the difference between the normal distribution of IQ values in a population with a mean of 100 (shown by the solid line) and what happens when there is an overall population IQ shift downward by 5 points (the dashed curve). In the population with lower average IQ, if you consider the shaded tails of the new distribution, there are 2.5 times fewer individuals with IQ's greater than 130 and double the number of persons with IQ's less than 70 which is the clinical definition of mental retardation.

Source: Rice, 1998

Trend data for children living in Canada are similar to the US. According to data collected in 1994 for the National Longitudinal Study on Children and Youth (NLSCY):

- 26% of children living in Canada (age 6-11 yrs) have at least one, identifiable learning or behavioural problem;
- Delayed vocabulary skills were found in 16% of children living in Canada aged 4-5 years;
- 14-16% of children living in Canada had cognitive deficits, and another 17-22 % had "behaviour problems" defined as hyperactivity and AD/HD (Landy and Tam, 1998); and
- According to school principals who participated in the first cycle of the NLSCY, an average of 12% of children in their schools had a learning disability (Statistics Canada, 1997).

The Ontario Ministry of Education tracks the number of students for which teachers have prepared an Individual Education Plan (IEP) and those who are receiving special education programs and/or services (MOETCU, on-line). An IEP is prepared for students who have some form of neurological or behavioural exceptionality. The Ministry has established categories of "exceptionality" describing behavioural or other conditions among students most of which present some form of challenge to learning such as behaviour, hearing deficits, learning disabilities, speech and language impairment, intellectual, developmental and physical disabilities, autism, deafness, etc.

An IEP is prepared for gifted students as well although those students are excluded from the calculations made here. Some students, though not all, for various reasons, go on to be "formally identified" by an Identification, Placement and Review Committee as having one or more of the categories of exceptionalities.

Whether formally identified or not, the IEP provides eligibility for support services from special education teachers. For 2000-2001, excluding children considered gifted, 9.5% of the Ontario student body had an IEP. This number is calculated as the percentage of elementary and secondary students with an IEP within the total student enrollment in all school boards in the province (LDAO, 2004).

During 2001-2002, Toronto District School Board (TDSB) Special Education teachers were providing services to approximately 13% of enrolled students. Of these, approximately 23,500 students had been formally identified and approximately 14,000 had not been formally identified but had been placed in the "special needs" group so as to receive support from special education teachers.

Among the identified students, those with learning disabilities or behaviour disorders comprise 4.4% of the total in this group.

This figure of 13% for the TDSB is considered an underestimate. Students with learning disabilities and those with behaviour disorders would also be among the group that has not been formally identified. Also, when students are identified with other exceptionalities (e.g., hearing deficits or speech impairment), they often have behaviour problems as part of the identifying characteristics of their exceptionality. However, their identification is with the primary exceptionality such as learning disability, mild intellectual disability, multiple exceptionalities, etc., rather than with the related behaviour problems (Ellerker, TDSB, pers comm., March, 2002 and March, 2005).

Finally, diagnosis of autism is problematic and variations of reported prevalence over time and place may reflect differences in detection and reporting methods. Numbers from Europe and Asia note a prevalence of 2 per 1000 children with some form of autism. A similar prevalence level is reported from a study in Atlanta. Higher levels have been found in a New Jersey study where a prevalence of 4 per 1000 children was found for autism and 6.7 per 1000 for autism spectrum disorders. According to the US Centers for Disease Control and Prevention (US CDC), national trend data are not available (CDC, on-line).

Twenty years ago, autism was estimated to occur in one of every 10,000 children in Canada. In recent years, according to the Autism Society of Canada, one in every 200 children have an autism spectrum disorder (or 50 out of every 10,000 children) (ASC, 2004). Despite uncertainties about the true prevalence of autism, this condition is about four times more common in boys than girls and has been found throughout the world in families of all racial, ethnic and social backgrounds (ASC, undated).

In summary, the prevalence of learning disabilities, AD/HD, autism and other neurobehavioural deficits appears similar among data from the US, Canada and Ontario. For the limited Toronto-specific data noted, Toronto appears to be on the higher end of the scale with about 13% of enrolled students with at least one or more learning or behavioural deficit of concern.

Although the apparent increase of these varied conditions may be influenced by more aggressive diagnostic practices, the burden of disabling conditions is very high nonetheless. Although not universally accepted, there is limited evidence that prevalence rates for most of these conditions have increased in recent years (Rowland et al, 2002, Centre for Learning Disabilities, 2001). The Greater Boston Physicians for Social Responsibility (Schettler, et al, 2000) refer to neurodevelopmental conditions as a problem of epidemic proportions. Although

they discuss these trends alongside the evidence about suspected associations between environmental exposures and neurotoxicity, they also recognize that not enough information exists to definitively tie such exposures to these trends. The social costs are considerable as discussed further in section 3.6.

3.3 Rare and Often Severe Effects

3.3.1 Effects of Toxic Substances on the Reproductive System

Many factors affect reproductive health including nutrition, the environment, socioeconomic status, lifestyle and stress. Reproductive toxicity includes adverse effects of substances on sexual maturation, gamete production and transport (sperm in males, oocytes/ova in females), the female reproductive cycle, sexual behaviour, fertility, gestation and lactation (Wigle, 2003; USEPA, 1996).

Information is limited about the influence of chemical exposures as contributing factors in these reproductive effects in humans. The lines of evidence are often closely linked to investigations of effects on prenatal development, discussed separately below. As well, human reproduction and development are the result of complex interactions among the hormones of the endocrine system. There is emerging evidence that reproductive and developmental effects may result from endocrine system-mediated effects (discussed in Section 3.4, Emerging Issues).

Men and women can develop reproductive disorders as a result of chemical exposures experienced prenatally, in early childhood, and as adults. Pre-conception exposures to substances that are mutagenic can affect developmental outcomes when such mutagens involve DNA changes in germ cells (the gonadal cells that give rise to eggs or sperm). If such changes cause permanent alterations in the DNA of germ cells, there can be multigenerational effects. Chemical contaminants are in direct contact with gonadal cells as evidenced by their detection in follicular fluid, and in semen (Silbergeld, 1986; Younglai et al. 2002; Arbuckle, et al 1999).

Several studies suggest that a number of reproductive disorders observed among men living in industrialized countries may reflect increasing exposure to anthropogenic (man-made) chemicals over the last three to four decades (as summarized in Wigle, 2003; USEPA, 1996; Schettler et al, 1999; Sharpe and Irvine, 2004). For example, some studies have demonstrated an association between environmental (i.e. background level) exposure to phthalates and PCBs (as measured in urine and blood serum, respectively) and some measures of sperm quality (Duty et al 2003; Hauser, et al, 2003). Organochlorine pesticides are also implicated in these findings of reduced sperm quality. As well, there are fairly

consistent reports of increasing rates of testicular cancer in several countries (including Canada as discussed in section 3.5.6 below regarding cancer). Closely related are increasing rates of some birth defects of the male reproductive system, discussed in the next section with respect to developmental effects.

Females are born with all the ova they will produce in their lifetimes and so are influenced by any prenatal exposures. A male's sperm are produced continuously and often only a few days or even hours before conception and so may be influenced by recent paternal occupational and other exposures in adulthood. While it has long been recognized that offspring can be affected by maternal exposure to toxic chemicals during pregnancy or lactation, it is more recently understood that children can be affected by paternal exposures as well (USEPA, 1996). Aside from the drug DES (discussed in Chapter 2) there are only a few known human reproductive toxicants, and these have been identified in studies of occupationally exposed persons, mainly men. They include lead and lead compounds, linked to reduced male fertility (Sallmen 2000), the pesticides 1,2-dibromo-3-chloropropane, chlordecone, and ethylene dibromide, and certain solvents (reviewed in Wigle, 2003). The literature review summarized on the www.scorecard.org website notes 106 individual substances that are recognized reproductive toxicants and an additional 248 that are suspected reproductive toxicants (Scorecard, on-line).

3.3.2 Reproductive Disorders – Available Data on Trends

Available data on reproductive disorders are limited to what is known about maternal age-specific birth rates (an indirect indicator of fertility) and very limited non-population-based data on rates of infertility. Aggregated national data on maternal age-specific birth rates are available from Statistics Canada. These data show that the birth rate across the Canadian population declined substantially from 1979 to 1999 particularly among women aged 20 to 24 (40% decrease) and those aged 25 to 29 (25% decrease) while rates increased among women aged 30 and over. This overall drop in birth rates has much to do with societal changes affecting women's choices for whether and when to have children, including many economic and employment issues as well as improvements in contraception.

The data for birth rates among women in Toronto are comparable to the national picture. In general, there is an overall trend towards women putting off having children until after the age of 25 and more still until after the age of 30. During 2000, Toronto's age-specific fertility rates were lower than those for the rest of Ontario in all age groups between 20 and 34 years of age. The largest difference was in the 25-29 age group where the birth rate for Ontario women outside

Toronto was 37% higher than in Toronto. The age-specific rates for older women in Toronto (aged 35 to 49 years) were higher than the rest of the province (TPH, HI&P, 2005b).

It has been estimated that 7.9% of all couples in the U.S. in 1988 were infertile and that 5 million women have endometriosis, a painful disease of the uterus that may cause infertility (U.S. EPA, 1996).

One Canadian source states that infertility affects over half a million Canadian women and men (IAAC, on-line). However, although medical professionals can point to an increase in consultations to treat infertility, there appear to be no clear data or statistics to be able to say whether infertility is genuinely increasing (Buckett, undated). Other researchers note that while there is a general perception that the prevalence of infertile couples has increased in recent years, studies of infertility rates in Canada and the US do not demonstrate an increase. The increase in medical consultations to treat infertility may be a factor of more women delaying childbearing (when the ability to conceive declines normally), as well as more couples being aware of and having the means to try new treatments. Also, it is not possible with currently available data to discern whether the observed tendency for reduced sperm count is a contributing factor. In Canada, researchers at McMaster University have shown that semen quality is lower in some regions than in others (Younglai et al, 1998). However, no studies have explored possible causal factors. Moreover, there has been little research examining the links between exposure (using direct measures of exposure) and changes in semen quality (University of Ottawa, emcom.ca, undated) Also, there are major challenges in standardized semen quality measurements. Comparability of data between studies or across regions and time is far from certain.

3.3.3 Effects of Toxic Substances on Development

Developmental biology is yet another extremely complex field of inquiry. Understanding of the many stages of human development, and the possible influence of a myriad of toxic substances on development, requires multi-disciplinary coordination of the fields of pharmacology, toxicology, embryology, molecular and developmental biology and epidemiology. There are gaps in information and understanding within the field of developmental biology itself and far greater ignorance about the influences of chemical substances on the many stages of development (NRC, 2000b).

A number of types of chemical substances (including lead, mercury and arsenic, some pesticides, and industrial chemicals, notably PCBs) and physical agents (such as ionizing radiation), have been shown, through animal studies and/or

epidemiological studies, to be capable of producing developmental effects in children. The literature review summarized on the www.scorecard.org website notes 579 individual substances as recognized developmental toxicants. This list includes numerous entries from groups of metal compounds (such as lead, mercury and arsenic compounds) as well as numerous individual PCBs and specific pesticides. The scorecard literature review notes an additional 220 individual substances that are suspected developmental toxicants (Scorecard, on-line).

Contributing exposures can occur to parents prior to conception, and among children who are exposed prenatally or in childhood (USEPA, 1991; EPA, 1997). As discussed in Chapter 2, the placenta, like the blood-brain barrier, functions somewhat like a semi-permeable membrane. In most cases, the placenta readily allows the passage of metals and other toxic substances from the mother to the fetus. Indeed, as noted in Chapter 2, the placenta actually magnifies methylmercury levels resulting in higher levels in cord blood than in maternal circulation (Stern and Smith, 2003).

Developmental toxicants are agents that can cause preimplantation loss, fetal death, impaired growth and structural and functional abnormalities. Four categories of effects are identified including (as reviewed in Selevan et al, 2000; NRC, 2001; IPCS, 2001; USEPA, 1991; Wigle, 2003):

1. prenatal or early postnatal death (e.g., spontaneous abortions (early fetal deaths) or stillbirths (later fetal deaths));
2. structural abnormalities (e.g birth defects such as orofacial clefts, heart, lung blood vessel defects, genotoxic effects (chromosome abnormalities));
3. altered growth (e.g. decreased head circumference and body length at birth, intrauterine growth retardation (IUGR) (defined as birth weight below the 10th percentile for gestational age and gender), preterm delivery); and
4. functional deficits (e.g., visual and hearing deficits, cerebral palsy (congenital), mental retardation.

The causes and mechanisms of developmental health effects are largely unknown but are considered to arise from a combination of genetic and environmental factors. As with the other health effect types already discussed, developmental effects are similarly multifactorial in nature. Research reveals that maternal nutritional and health status can influence development of the fetus and embryo. For example, low levels of maternal folic acid intake are associated with greater

risk of neural tube defects. Maternal thyroid disorders are associated with higher risk of fetal heart defects (Vohra and Koren, 2001). In addition, birth outcomes are important both, as indicators of the health of newborns and infants, and as indicators of individuals in later life. Low birth weight, intrauterine growth retardation (IUGR), and impaired growth in the first years of life are associated with increased mortality and morbidity in childhood and an elevated risk of hypertension, coronary heart disease, and non-insulin-dependent diabetes in adulthood (Sram et al, 2005).

The evidence concerning associations with exposure to chemical substances includes human, animal and other laboratory studies that range from weak to robust. Like the health effect areas discussed so far, knowledge is limited to a small number of substances and effects are suspected for many more. Ongoing research tends to confirm and expand upon what is known about critical windows of exposure and vulnerability (described in Chapter 2) during developmental stages. Structural defects are generally better understood and studied compared to functional deficits. Mechanisms of developmental effects vary. For example, ionizing radiation can involve mutations in DNA of germ cells which can then lead to abnormal development of the embryo.

Again, high-level exposure to a few well-studied substances can cause adverse developmental effects. These include lead, methylmercury, ETS (and maternal cigarette smoking during pregnancy), PCBs contaminated with dioxins and furans and ionizing radiation (X-rays or radioactive fallout from either atomic testing or nuclear accidents).

Maternal body burdens of accumulated persistent toxic substances, such as PCBs stored in fat, or lead stored in bone, may be mobilized to the fetus during pregnancy (Hu et al, 1995). For example, Japanese women who prenatally consumed cooking oil contaminated with high levels of PCBs, gave birth to infants who were small for gestational age, had dark pigmentation of the skin and nails, early eruption of teeth, and swollen eyelids and gums. In follow-up studies, the children continued to be small and had lower IQs (Tilson et al, 1990).

Alongside these more extensively studied substances, the information base is growing about organic solvents, a number of pesticides, and some air pollutants.

The Motherisk Program, a joint project of Toronto's Hospital for Sick Children and the University of Toronto, has conducted several investigations into effects from occupational exposure to organic solvents during pregnancy. Organic solvents are another large and diverse group of substances. Common characteristics are low molecular weight, volatility (ability to give off airborne vapours at room temperature) and their ability to dissolve other organic

substances (properties that would enable these substances to readily cross the placenta). Organic solvents are very common in industrial settings and in the home (e.g., houses with attached garages may have relatively high benzene levels if vehicles or gasoline-powered machines are stored therein).

Categories and Examples of Organic Solvents include:

- Aliphatic hydrocarbons (mineral spirits, varnish, kerosene);
- Aromatic hydrocarbons (benzene, toluene, xylene);
- Halogenated hydrocarbons (carbon tetrachloride, trichloroethylene);
- Aliphatic alcohols (methanol);
- Glycols (ethylene glycol);
- Glycol ethers (methoxyethanol);
- Constituents of common products (in vapours from gasoline, lighter fluid, spot removers, aerosol sprays and/or paints); and
- Occupational exposure settings: dry cleaning, hair salons, working with paint removers, thinners, floor and tile cleaners, glue and laboratory reagents.

In laboratory animal studies, many organic solvents have demonstrated toxic effects on the developing embryo resulting in spontaneous abortion and teratogenicity (the ability to induce birth defects) (as reviewed in Till et al, 2001a). Evidence of such effects in humans is more limited due to difficulties in being able to accurately assess the specific solvents or the associated dose. However, in a meta-analysis (a study that combines and analyses the results of many studies on the same topic) of epidemiological evidence, maternal occupational exposure to organic solvents was associated with increased risks for spontaneous abortion (McMartin et al, 1998). Researchers at Toronto's Hospital for Sick Children also found a statistically significant association between maternal occupational exposure to solvents during pregnancy and major malformations in their infants (Khattak et al, 1999).

Additional, follow-up studies have found associations between prenatal exposure to organic solvents and neurodevelopmental effects. Specifically, these effects include an increased risk of impaired color vision and visual acuity in these children (Till et al, 2001b). Tests of neurocognitive and neuromotor functioning, as well as language and behavior, indicated poorer performance in the solvent-exposed offspring several years after birth (Till et al, 2001b; Laslo-Baker et al, 2004). Further study continues to confirm these effects noting as well that the damage could be irreversible (Till et al, 2003). While more research is needed, there is an increasing weight of evidence that would support the prudent avoidance of prenatal exposure to organic solvents during pregnancy. Paternal

exposure to solvents may also contribute to adverse pregnancy outcomes (Daniell and Vaughan, 1988).

Some studies (mainly of workers) suggest that maternal exposure to pesticides used in agriculture and gardening during early pregnancy is associated with increased risks of certain birth defects, such as cleft lip and palate, spina bifida, defects of limbs or digits, or undescended testes (Arbuckle and Sever, 1998, Arbuckle et al, 2001; Curtis et al, 1999; Garcia, 1998, Garcia et al, 1999, Nurminen, 1995; Sever et al, 1997, Kristensen et al, 1997, Garry et al, 1996, Shaw et al, 1999, Bell et al, 2001 and Weidner et al, 1998). Although less well studied, reproductive effects may also be observed from pesticide exposures in men (Sever et al, 1997). For instance, children of male pesticide applicators on farms were found to have a higher rate of birth defects or developmental disorders in the first three years of life (Garry et al, 2002). Higher risks for miscarriage and prematurity have been found with direct paternal exposure to certain agricultural pesticides among a study of Ontario farmers (Savitz et al, 1997).

Several studies have reported on pesticide exposure from pest control in urban and indoor settings, mainly organophosphate insecticides (Berkowitz et al, 2003). For example, associations were found between maternal exposure to the pesticide chlorpyrifos and a significant but small reduction in offspring head circumference (Berkowitz et al, 2004). Columbia University's ongoing prospective study also found associations between maternal chlorpyrifos exposure and lower birth weight and reduced birth length (Perera et al, 2003). The pesticide diazinon was also implicated in these impaired fetal growth effects. Moreover, in looking at time trends in these pesticide exposures and birth outcomes, these researchers found a correlation between lower exposures (after regulatory action was taken to limit chlorpyrifos and diazinon) and the weakening of associations with impaired fetal growth. The authors note that the findings provide support for regulatory action phasing out the residential use of these insecticides (Whyatt et al, 2004).

Summarizing the research on pesticides, the overall weight of evidence suggests that there is an elevated risk of reproductive and developmental effects from direct exposures to some pesticides prior to conception, or during prenatal or postnatal periods.

Over the last decade or more the impact of air pollution on birth outcomes and reproduction has been examined. Biological markers, such as increased levels of DNA adducts in maternal and placental circulation, confirm that maternal exposure to ambient air pollution does result in exposures to the fetus that may affect birth outcome.

A recent weight-of-evidence review of the studies directed at ambient air pollution and adverse birth concluded that:

- There is sufficient evidence to infer a causal relationship between particulate air pollution and respiratory deaths in the postneonatal period;
- The evidence suggests a causal relationship between air pollution and birth weight; and
- There is insufficient evidence to infer a causal relationship between air pollution and premature births, IUGR, or birth defects, but that more study is justified for all three outcomes (Sram et al., 2005).

Intrauterine growth retardation (IUGR) was strongly linked with exposures to polycyclic aromatic hydrocarbons (PAHs) in two studies (Sram et al., 2005).

A number of studies suggest biological mechanisms by which air pollution may affect birth outcomes. For example, increases in DNA adducts have been positively associated with a number of birth outcomes including IUGR, lower birth weight and decreased head circumference. Toxicological studies indicate that PAHs can increase rates of mutagenesis and may interfere with the endocrine system which directs placental growth and fetal development (Sram et al., 2005).

3.3.4 Developmental Disorders and Effects – Available Data on Trends

National data on developmental outcomes are collected by the Canadian Perinatal Surveillance System (CPSS). CPSS is a coordinated initiative between Health Canada, other federal and provincial agencies and health professionals and researchers. National data are collected and analyzed for 27 perinatal health indicators including many aspects of maternal, fetal and infant health. The CPSS data present overall trends as well as regional differences. However, concerns about the quality of Ontario's vital statistics data have meant that information for Ontario has been excluded from the national picture summarized by CPSS. Ontario data is reported separately (CPSS, 2003 and Appendix G, Ontario Vital Statistics Data).

Over the past fifteen years, CPSS reports that leading indicators of fetal and child health reveal steadily improving trends. They note an overall reduction in the rate of fetal and infant mortality and reductions specifically in terms of most causes of death such as stillbirth, SIDS or congenital anomalies.

Other indicators of adverse pregnancy outcome and fetal growth, as measured by rates of pre-term birth (births that occur before gestation week 37) or small-for-gestational age babies, show a steady temporal decline alongside an increase in large-for-gestational age live births. The national rate for pre-term birth in 2000 was 7.6 per 100 live births. Ontario reported a rate of 7.3 per 100 live births. The national rate for small-for-gestational age in 2000 was 7.9 per 100 live births while Ontario reported a rate of 12.7 per 100 live births. Calculating the rate of small-for-gestational age is a relative measure that includes births in the lowest 10th percentile (of weight) that would be expected at specific gestational periods.

Low birth weight (commonly defined as a live infant weighing less than 2500 grams at birth) is a more commonly used measure and more useful for the trend comparisons being made in this report. Low birth weight is clearly associated with poor health experienced later in life (TPH, 2004d). Low birth weight may be caused by pre-term birth, intrauterine growth restriction or both factors. Low birth weight is also more common among twin and multiple births. Intrauterine growth restriction (also referred to as small for gestational age) indicates a birth weight below 2500 g at term (gestation week 40) or below the 10th percentile for gestational age (the latter outcome is also known as intrauterine growth retardation).

Trend data for low birth weight (LBW) are not entirely reliable, particularly in Ontario, due to an increasing problem of unregistered births, a problem that is more pronounced in Toronto. In 1999, the total LBW rates (which include both singleton and multiple births) for all of Ontario, for Ontario excluding Toronto and in Toronto itself were 5.8, 5.6 and 6.5% respectively. Statistics Canada reports the national total LBW rate as 5.6% in 1999, therefore similar to that for Ontario excluding Toronto (Statistics Canada, on-line). Data for Ontario reveals that the singleton LBW rate is consistently higher in Toronto (5.3% in 1999) compared to the rest of Ontario (4.2% in 1999). Toronto Public Health has updated these data to look at trends across the time period 1997-2002. They found that the total LBW rate for Toronto was also consistently higher than the rate for the rest of Ontario from 1997 to 2002. For this six-year period, the total LBW rate in Toronto was 6.6% while it was 5.6% in the rest of Ontario (TPH, HI&P, 2005c). Moreover, the total LBW rates vary significantly across the city, from 3.8% to 6.9% across the 15 smaller planning areas where data were collected, for reasons that have yet to be identified. Socio-demographic factors including neighbourhood income, maternal country of birth and maternal age possibly contribute to this variability in total LBW rate across the city (TPH, 2004c).

Trend data appear roughly comparable when looking at somewhat dated information for the United States. 1991 estimates for liveborn infants in the U.S. are that 7.4% were low birth weight, 3% had one or more congenital birth defects

at birth, and 3% had serious developmental deficits after one year (USEPA, 1991).

Congenital anomalies, birth defects or congenital malformations are interchangeable terms. They can refer to abnormalities of structure, function or metabolism that are present at birth (but may not be diagnosed until later in life). The CPSS annual report for 2002 focuses on congenital anomalies (CPSS, 2002) noting a national birth defect rate of 2-3% (or 7000 to 10,500 births) each year. The prevalence data reported are limited to five categories of birth defects. The total number of cases of limb reduction deficits, oral facial clefts, neural tube defects and Down Syndrome were quite low (123, 366, 195 and 366 cases, respectively, among all live births in Canada, except Ontario during 1999). Congenital heart defects are much higher at 3518 cases among all live births in 1999. However, these five categories only tally up to just over 4600 cases. No additional details are provided as to the types or prevalence of other birth defects in Canada (CPSS, 2002). While the total number of cases may be considered small, or rare across the entire population, most affected infants survive with varying degrees of disability and the cumulative prevalence of such persons at any one time would be in the order of several hundred thousand.

Several studies suggest the following birth defect increases in populations in industrialized countries within the last three or four decades:

- Inconsistent reports of increasing rates of cryptorchidism (birth defects involving late or non-descent of the testicles) in some industrialized countries; and
- Reports of increasing rates of hypospadias (an abnormal opening of the urethra on the lower surface of the penis) in the United States (Wigle, 2003; USEPA, 1996).

Toronto Public Health collects data on infant mortality rates, birth rates and, as noted above, low birth weight (among other measures less related to the developmental issues under consideration here). Infant mortality rates decreased slightly in Toronto from a rate of 7.8 infant deaths per 1000 live births in 1989 to 5.8 infant deaths per 1000 live births in 2000 (TPH-HI&P, 2005d). During this period, the Toronto infant mortality rate exceeded the rate for the rest of the province for most years. The leading causes of infant deaths are conditions related to pregnancy or delivery (e.g. pre-maturity, low birth weight, complications of labour and delivery) and congenital anomalies. It is not clear from the data how many infant deaths could have been prevented.

As noted above with respect to low birth weight, there are some limitations to note with respect to these data. Registration fees initiated in some areas in 1996

may have reduced the numbers of infants registered, especially for low income and/or adolescent parents. Since the number of live births is used in the calculation of the Infant Mortality rate, any difference between Toronto and the rest of Ontario in the numbers of unregistered births will impact comparisons using this rate.

In 2002, the crude birth rate in Toronto was 12.1 live births per 1000 persons, which was higher than the rate of 11.0 per 1000 for the rest of Ontario and represents a decline since 1997 figures (TPH HI&P, 2005e). Crude Birth Rate is influenced by the age structure of the population, specifically by the proportion of women who are in their childbearing years.

Like the other health effects discussed thus far, causes are related to many, often inter-related and not entirely understood factors. There is insufficient information to link these trend data with particular environmental exposures.

3.3.5 Cancer

Cancers that occur in the young are another rare but severe type of health impact. Research indicates that both genetic and environmental factors contribute to cancer risk to varying degrees depending on the specific type of cancer (Anderson et al 2000). However, environmental factors and the interaction between genetic and acquired susceptibility, are believed to account for over 80% of all cancers (Hoover, 2000; Perera, 1997). Most adult cancers are caused by a few factors including smoking, diet, occupational exposures, intense sun exposure and certain biological agents such as viruses (Krieger et al, 2003). However, the specific causal agents, mechanisms and critical exposure windows for most cancers in children and young adults are not well understood (Anderson et al 2000; Olshan et al, 2000).

The scientific evaluation of substances for their carcinogenicity in experimental animals is probably more advanced than for any other category of health effects. For many years, investigation of cancer risk was a major focus in the evaluation processes applied by regulatory agencies. This situation remains largely true in the sense that testing for carcinogenicity of chemicals remains as a core requirement.

Comparatively little is known about the environmental links to childhood cancer because chemicals used or generated for many applications in society have not been assessed for their ability to produce cancer in children, as distinct from carcinogenic effects in adults. Governments and funding agencies have not given sufficient priority to research on childhood cancer. Health Canada started a

childhood cancer epidemiologic research program in 1992 but dedicated funding was cancelled a few years later. Childhood cancers are relatively rare compared to adult cancers and more difficult to study (because of the need to include enough cases to have reasonable statistical power). There are no generally used testing protocols for cancers in young animals, including those that may originate from parental exposure or where developmental exposure may increase susceptibility to carcinogen exposure in the adult (USEPA, 2003d).

The USEPA has noted two different types of cancer concerns related to early-life exposures, including preconception and *in utero*. There is the concern that:

- Early-life exposures can increase the rate of cancer among children; and
- Early-life exposures can increase the rate of certain adult cancers (USEPA, 2003d).

There are few established environmental risk factors for cancer that occurs in the young. Ionizing radiation, extremely low frequency electromagnetic fields, ETS, some air pollutants (such as, PAHs and benzene), pesticides and certain chemicals are among the few agents where evidence is strongest (Van Larebeke et al, 2005; Wigle, 2003; Perera, 1997; Zahm and Devesa, 1995). Epidemiological studies, though not conclusive, suggest that exposure to pesticides may contribute to moderately increased risks of certain cancers in children (Olshan and Daniels, 2000, Zahm and Ward, 1998). Pre-conception, prenatal and early childhood exposures to pesticides are associated with increases in childhood brain tumours, leukemia, non-Hodgkin's lymphoma and neuroblastoma (Daniels et al, 1997, Leiss and Savitz, 1995, Infante-Rivard and Sinnett, 1999, Buckley et al, 2000, Meinert et al, 2000, Daniels et al, 2001, Pogoda and Preston-Martin, 1997). These are the cancers that are most often seen in children in industrialized countries (as discussed further in the next section). Home use of pesticides (particularly indoor use of insecticides) appears to account for the greatest risk of these cancers (Ma et al, 2002; Daniels et al, 1997; Leiss & Savitz, 1995; Infante-Rivard et al, 1999).

Certain childhood cancers may be related to parental (largely occupational) exposure to pesticides (Kristensen et al, 1996; Hardell and Eriksson, 1999; Feychting et al, 2001; Infante-Rivard and Sinnett, 1999). A number of studies have also demonstrated that childhood cancers such as brain cancer and leukemia may be associated with parental exposure before conception, prenatal exposure, or exposure in early-life to petroleum products, benzene and other solvents, or combustion by-products such as dioxins and PAHs (Gouveia-Vigeant et al, 2003).

A few studies suggest that some childhood cancers are linked with outdoor air pollution. For example, California researchers found elevated rates of leukemia

among children living in regions with higher air levels of hazardous air pollutants such as benzene, perchloroethylene, and 1,3-butadiene, emitted from industrial, mobile and area sources (Reynolds et al, 2003). Other studies have found weak associations between proximity to high traffic and childhood leukemia and other childhood cancers (Pearson et al 2000; Savitz and Feingold, 1989; Feychting et al, 1998; Raaschou et al, 2001; and as reviewed in Wigle, 2003). A French study has also shown a link between living near a gas station or mechanic's garage and higher risk of acute leukemia in children (Steffen et al, 2004). As well, recent results from the Columbia University prospective study (discussed in Section 3.3.3. Effects of Toxic Substances on Development) indicated an association between maternal prenatal PAH exposure and chromosomal aberrations in cord blood cells. Because chromosomal aberrations are predictive of increased cancer risk from prospective studies in adults, it suggests that exposure to PAHs in the womb has implications for cancer risks later in life (Bocskay et al, 2005).

There is also evidence of associations between increased risks for some childhood cancers with specific enzyme polymorphisms when they are exposed to certain environmental contaminants (as reviewed in Wigle, 2003).

Although the mechanisms and relevant exposure windows for childhood cancer are not all identified or understood, the evidence described here is suggestive that exposures *in utero* or during early postnatal life are the most important in determining risks for cancer in children (Olshan et al, 2000).

Childhood exposure to carcinogens also has implications for the development of cancer in adulthood. The US EPA notes that, in the case of well-studied mutagenic carcinogens (e.g. vinyl chloride), early-life exposures can also increase the number of tumours observed among those later exposed when they are adults. Early life exposure appears also to reduce the latency period or time between exposure and tumour development. In other words, evidence (in both animals and humans) suggests that with early-life exposures, cancer may develop earlier and the probability may be higher that a given exposure leads to cancer (Van Larebeke et al, 2005). For example, it has been shown that exposure to ionizing radiation during childhood increases the lifetime cancer risk by about double the risk that results from similar exposure during adulthood alone (Wigle, 2003). Ionizing radiation is a known thyroid carcinogen (Wild and Kleinjans, 2003).

In light of evidence suggesting that the cancer latency period is shorter when exposure occurs in childhood, concern is also warranted about the impact of early life exposures on development of cancers in young adults. Some cancers among young adults are on the rise for reasons that are not fully understood and some environmental factors are suspected as discussed in Section 3.3.6.

The evidence for cancers in adults that are linked to early life exposure to environmental agents is limited. There is good evidence that ozone depletion has increased ultraviolet radiation exposure, which in turn has increased the risks of certain skin cancers, such as melanoma, in adulthood. Episodes of seasonal sun exposure during childhood and adolescence that cause severe sunburns substantially increase the risk of melanoma in adulthood, although adult exposures increase risk as well (Weinstock et al, 1989; Corona et al, 2001; Pfahlberg et al, 2001; Whiteman et al, 2001; van Dam et al, 1999).

Evidence suggests that childhood or early life exposure to other substances, such as ETS or pesticides, may also contribute to lifetime cancer risk (AAP 2003; Zahm and Ward, 1998).

Study of the environmental links to cancers of certain reproductive tissues (such as, breast, prostate and testicular cancers) has looked for associations with exposure to persistent organic pollutants. However, conclusions have been inconsistent when these studies used levels of contaminants measured in adult tissues as an estimate of exposure. Experts on the topic of endocrine disruptors hypothesize now, however, that exposures to these hormonally active agents (HAAs) in the prenatal period may alter the development of reproductive tissues and increase sensitivity to carcinogens later in life. The *in utero* exposure to diethylstilbestrol (DES) mentioned in Section 2.1.1. led to increased risks of clear-cell adenocarcinoma of the vagina when exposed females were teenagers or young adults (Anderson et al, 2000). This strong epidemiological evidence supports the hypothesis of links between fetal exposure to HAAs and increased cancer risks in adulthood.

3.3.6 Cancer Trend Data

US and Canadian Trends in Childhood Cancer

Childhood cancer is still quite rare although it remains the leading cause of illness-related death among children in Canada over one year of age. Canadian cancer statistics for 1997-2001 show that an average of 1,285 children every year were diagnosed with cancer and an average of 287 children died each year from the disease. Annual statistics since 2001 reflect a similar pattern. For 2005, the Canadian Cancer Society and the National Cancer Institute of Canada estimate that 1,250 children in Canada will be diagnosed with some form of cancer and that 190 children will die of the disease (NCIC, 2005). The most common forms of cancer among children living in Canada are leukemia (about 26% of new cases), brain and spinal cancer (about 17% of new cases), and lymphoma (about 16% of new cases) (NCIC, 2005).

In the United States, like Canada, the death rates from cancer among adults and children are declining. However, incidence of certain forms of cancer is on the rise among some populations. In Canada, childhood cancers increased during the period 1974 to 1984, but have not increased since that time (Morrison, 1997). In young adults (age 20 to 44 years) in Canada, incidence rates increased for several cancer types during the 1990s. Excess cancers of the reproductive system (such as breast and cervical cancers) in the last decade explain why incidence rate increases were greater among women than young men (NCIC, 2002). These trends in cancer in young adults are discussed further below.

In the United States, with a much larger population with which to discern trends, the incidence of all cancers in children increased nearly 21% between 1975 and 1998. This is an increase of about 1% per year over two decades (as reviewed in Gouveia-Vigeant and Tickner, 2003). Steady increases occurred in the same cancers seen in children in other industrialized countries including leukemia, brain cancer and non-Hodgkin's lymphoma (NHL). In the case of NHL in the US, the incidence rate in children has increased by 30% since the 1970s (Zahm and Ward, 1998).

There is an ongoing debate about the issue of increasing incidence of childhood cancer. Some experts consider earlier diagnosis and improved case reporting as sufficient explanations for the increases found in childhood cancer incidence rates. Others consider that additional factors, including environmental contaminants, may be involved. Analysis of temporal trends is challenging. Conclusions differ depending on the data sample analysed, the time frame compared and the analytical techniques used, among other factors. These differences are summarized elsewhere (Vanderlinden and Cooper, 2002) and it is fair to say that the jury is still out on the exact contribution of improved diagnosis as opposed to environmental exposures to increased childhood cancer incidence. Both factors could be at play. European data also appear to show a steady increase in childhood cancer over time. In a recent study looking at 30 years worth of data from 35 European countries, researchers found a 1% per year increase in childhood cancers (Steliara-Foucher et al, 2004).

Cancers in Young Adults

Cancer is primarily a disease of the elderly. Cancers among young adults, while occurring less frequently than in older adults, still account for a greater proportion of cases (8.7% in 1996) compared to cancers in children 0 to 19 years (~1% in 1996). Similar to cancers in childhood, the types of cancer found most frequently in young adults also differ from those in older adults (NCIC 2002). For adults in

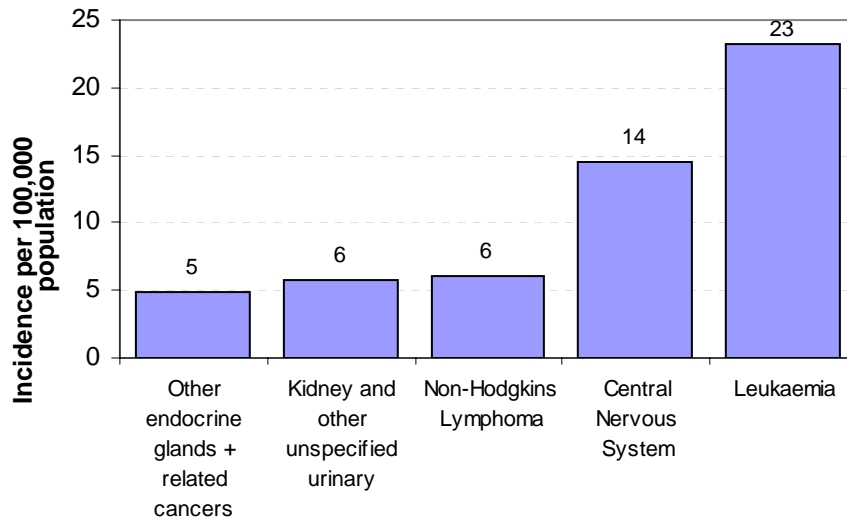
Canada aged 20 to 44 years in age, the incidence of all cancers combined increased gradually over the twenty-six years between 1969 and 1996 (NCIC, 2002; Marrett et al, 2002). Over the most recent decade analysed (1987 to 1996), it is estimated that the increase was at 0.7 % per year in young men and 0.5% per year in young women (NCIC, 2002). Increases in the incidence of a few cancers however, were particularly strong. These include thyroid cancer in men (2.8% per year), non-Hodgkin's lymphoma in both sexes (2.7% per year among women and 3.7% per year among men), lung and brain cancers among women (1.9 and 2.0% per year, respectively) and testicular cancer among men (2.7% per year) (Marrett et al., 2002). For other cancers in young adults there were substantial, but non-linear increases such as, lung cancer in women and melanoma in both sexes. Recent trends in melanoma and breast cancer show a leveling of incidence (Marrett et al, 2002).

While the possible reasons for increased rates of lung cancer and melanoma have been established, for many other cancers (such as testicular, NHL and thyroid), the explanations have not yet been uncovered (Marrett et al, 2002). The latency period for cancer in young adults also would be relatively short compared to cancer at older ages and could be related to the issue of shortened latency periods from early-life exposures discussed above. Contributing exposures may have occurred during childhood or *in utero*.

Cancer in Children - Toronto Data

In Toronto, 234 cases of cancer in total were diagnosed among children 0 to 14 years, in the five-year period from 1996 to 2000. This equates to a rate of 55 new cancer cases per 100,000 children (ages 0 to 14 years) over the time period. Leukemia and cancer of the central nervous system were the leading child cancer diagnoses, at 23 and 14 cases per 100,000 children respectively in Toronto between 1996 and 2000 as shown in Figure 3.5 (Ontario Cancer Registry, 2002).

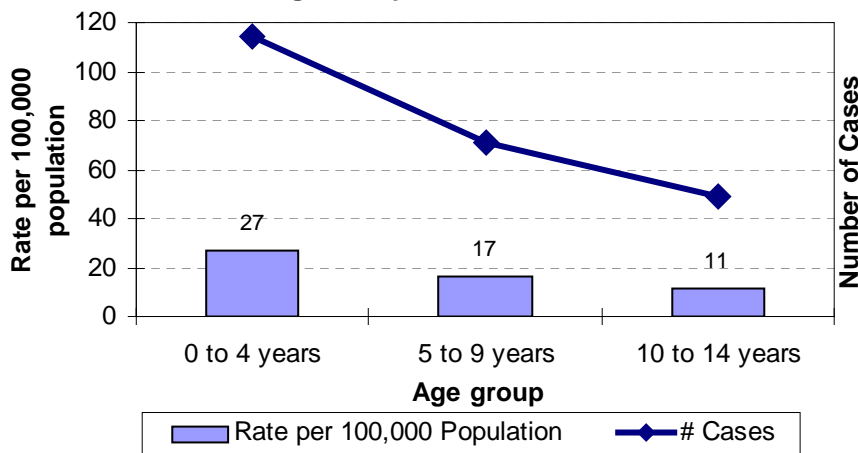
Figure 3.5 Incidence of Selected Childhood Cancers, 0 to 14 years, Toronto, 1996-2000



Source: Ontario Cancer Registry, 2002

As shown in Figure 3.6, childhood cancer incidence in Toronto is greatest among children under 5 years of age, with an incidence rate that is 1.5 to 2.5 times that for older children.

Figure 3.6 Incidence of 5 Major Childhood Cancers by Age Group, Toronto 1996-2000



Source: Ontario Cancer Registry, 2002

3.4 Emerging Issues

Researchers continue to identify the potential for disruption of normal endocrine, reproductive and immune system function from exposures during development to numerous chemicals that are present in the environment and in consumer products.

Several other issues of global proportion that are troubling to health and environmental scientists are also identified as having an impact on the health of the young. Among those most widely cited are the global cycling and transport of persistent organic pollutants (POPs), depletion of atmospheric ozone and climate change.

With respect to climate change, experts note that children (and the elderly) will be more seriously affected by hot, stagnant weather and related increases in smog levels (WHO, 2003b). Higher temperatures cause more volatile organic compounds (VOCs), such as solvents and other air toxics resulting from vehicle emissions, to volatilize into air, increasing exposures, and increasing the levels of ground-level ozone, a key component of smog.

Increasing local temperatures will also extend the range and breeding seasons of insect species. Diseases such as West Nile virus and malaria are known to be spreading northward and westward beyond more typical tropical climates. Figures for incidence of West Nile virus indicate that children are at lowest risk in the population. However, for other serious diseases, such as malaria, the highest rates of morbidity and mortality are among children. Other climate change-induced effects include the potential for higher concentrations of water contaminants under circumstances of lowered water tables from heat and drought episodes.

3.4.1 Immune System Effects

As research into children's health and environmental issues has advanced, investigators have noted that the ability to test substances for immunotoxicity (ability to create toxic effects in the immune system) is both limited and not applied during regulatory evaluations of chemicals (NRC, 1993).

In the general child population, several immune-system-related diseases such as type 1 diabetes, asthma, and allergies are on the rise. Evidence for immunotoxicity of environmental contaminants exists mainly for PCBs and dioxins (Tryphonas, 1998) but additional concerns arise for other persistent

substances such as organochlorine pesticides and other compounds that are similar to PCBs and dioxins, such as PBDEs.

The immune system defends the body against attack by external invaders such as disease, bacteria and allergens, using a complex array of organs, tissues and cell types. The immune system is immature in human infants and not fully functional. Breast milk plays an important role in both imparting immunity from the mother's immune system and in supporting the development of the immune system in the infant (as reviewed in Steingraber, 2001; Newman, 1995). However, transplacental transfer and breast milk also enable fetal and infant exposure to persistent toxic substances for which studies have suggested immunotoxic effects.

For example, there is an ongoing study of Inuit babies and children in Nunavik where maternal body burdens of organochlorine compounds and therefore, prenatal exposure to POPs, is higher than for the general Canadian population. This research has found possible associations with maternal organochlorine levels and the incidence in offspring of some acute infections (e.g. lower respiratory tract, gastrointestinal tract and ear infections and meningitis) early in life (Dewailly et al, 2000; Dallaire et al, 2004). In general, associations have not been found when breast milk exposure alone was considered.

Poor diet can compromise the immune system and its ability to detoxify and excrete pesticides (Keusch, 1994, Forget, 1991). Some pesticides have been shown to have a larger effect on the immune systems of children if they are iron-deficient (Sadikova, 1990). In a study on rats given a protein-deficient diet, they were more susceptible to liver damage from the organophosphate insecticide malathion and showed depressed immune function with exposure to the organochlorine pesticide DDT (Bulusu and Chakravarty, 1984; Bannerjee, 1999).

3.4.2 Endocrine System Effects

The endocrine system is the body's "communication" system allowing it to function as an integrated whole. This communication occurs via signaling between many different naturally occurring chemicals in the body. Concern exists about interference with the endocrine system by environmental contaminants that are variously called endocrine disruptors, endocrine modulators or hormonally active agents.

Using hormones, and operating in conjunction with the other two regulating systems, the nervous and immune systems, the endocrine system integrates and regulates development, growth, reproductive structure and function, metabolism, and glandular, muscle and nervous system functions in the body (USEPA, 1997).

There is some limited evidence of direct toxicity to the endocrine system from chemical exposures. But, most evidence in this area has to do with effects in other systems (reproduction, nervous, etc.) that appear to be mediated through altered chemical signaling of the endocrine system.

Health effects that may be caused by chemical exposures interfering with the hormones of the endocrine system have been observed in wildlife, laboratory animal, and *in vitro* studies. Some limited evidence exists from the few relevant investigations conducted to date in humans. This growing body of evidence supports the plausibility of the hypothesis that certain chemicals can interfere with the normal functioning of the endocrine system. Plausible effects include (see reviews in USEPA, 1996; Wigle, 2003; WHO, 2003c; IPCS, 2002, Boisen et al, 2001).

- Spontaneous abortion and stillbirth;
- Changes in sex ratio (fewer male babies conceived lowering the male:female sex ratio);
- Testicular dysgenesis syndrome (TDS) – a range of conditions affecting development of male reproductive systems including cryptorchidism (undescended testicles), hypospadias (birth defect in male urinary tract) and later conditions of decreased sperm count/quality;
- Testicular cancer - hypothesized as part of TDS since cryptorchidism is a known risk factor for late development of testicular cancer;
- Premature menarche (onset of menstruation) and precocious (unusually early) puberty;
- Polycystic ovary syndrome (irregular or absent menstrual cycle, elevated androgens and small cysts in the ovaries);
- Shortened lactation (reduction in breast milk supply unrelated to behaviour choices about duration of breastfeeding);
- Neurobehavioural effects (by *in utero* interference with thyroid hormone function; essential for normal fetal and child growth and development, especially central nervous system in development);
- Endocrine-mediated immunotoxicity; and
- Cancer promotion at endocrine-mediated sites (breast, endometrial, testicular, prostate, and thyroid) by synthetic chemicals with hormonal activity (prenatal/perinatal or otherwise early life exposure).

Most of the effects noted above are known or suspected from a very small number of chemicals. Using various levels of evidence, the European Commission has identified 564 substances as hormonally active agents; 146 of these were identified as being in high production and/or highly persistent (reviewed in Wigle, 2003). Among those chemicals recognized or suspected as being hormonally

active agents are a number of organochlorine pesticides such as DDT, dieldrin and lindane, certain industrial chemicals or by-products such as dioxins, hexachlorobenzene, PCBs and phthalates, and several metals such as arsenic, cadmium, lead and mercury. Food is the principal medium of exposure to these substances (with the exception of lead), including in breast milk.

As discussed in Section 2.1.1 above, the theory of endocrine system disruption via chemical exposures challenges the long-held view that “the dose makes the poison.” Hormonal signalling, such as signals that influence the course of development, may be occurring at very low doses, in the parts per trillion, and parts per billion. For example, in a study of rats, weight gain in adulthood, decreased motivation, and hormonal changes were seen resulting from a single exposure to dioxin at environmental levels (Markowski, et al 2001).

Hormonally-mediated effects are also atypical in that they can be non-monotonic. In other words, effects may be more pronounced at low doses and lessen at higher doses, indicating the complexity of enzyme and hormonal systems (Welshons et al, 2003). Considerable public health concern exists for such exposures because these are substances to which low-level exposure is nearly ubiquitous and may be capable of exerting the endocrine and reproductive effects of concern.

3.5 Multiple Exposures, Multiple Effects

Despite the preceding discussions looking at separate body systems, it is important to recognize that the human body functions as an integrated whole. It is equally important to recognize the “real world” situation of multiple exposures to mixtures of substances. Also, chemical exposures, either individual substances or mixtures, can often be capable of exerting multiple effects. While such a perspective on chemical risks should be obvious, it is a perspective that has barely begun to be applied in measuring, evaluating and regulating these substances. Rather, in both the scientific investigation of effects and evaluating whether and how exposure occurs, it is much more typical that the focus is on one effect or one contaminant at a time and generally in isolation from other effects or exposures.

This singular focus on one chemical at a time is part of good scientific technique. To evaluate cause and effect relationships, scientific investigations carefully control for as many variables as possible in efforts to isolate exactly whether one substance can cause a specific effect. In animal experiments, such isolation of variables, including limiting exposures to specific substances being studied, is much simpler than the evaluation of effects in humans who are constantly exposed to chemical mixtures.

A major part of these investigations involves research to isolate the “mode of action,” meaning the specific mechanism(s) by which toxicity occurs. The mode of action for a single substance is rarely simple, either in terms of how it manifests or in the scientific investigation seeking to understand it. Substances will often act at multiple sites in the body on different cell types or may exert multiple actions within the same cell type. Sometimes there is plenty of evidence of harm even when the mode of action is not understood. For example, knowledge of the neurotoxicity of lead in children was identified long before the mode of action was fully understood. Also, different mode of action results can occur at different locations in the body all with different disease results. Another variation occurs when the toxic mode of action occurs at sites in the body that regulate other organs or cells or developmental processes contributing to disease in sites in the body quite removed from the location where the toxic mode of action occurred (Carpenter et al, 2002). An example of this latter point is the neurodevelopmental toxicity in children that can result from chemicals exerting toxic effects on a mother’s thyroid gland during pregnancy.

Hence, not only does the one-chemical-at-a-time approach arise from traditional scientific methods, it occurs for purely practical reasons. Adding the additional complexity of multiple exposures to substances, especially when those substances can exert multiple effects, introduces an almost infinite number of combinations

of chemicals to be studied. Questions then arise about where to focus in terms of chemical combinations, dose ranges or biological endpoints (Carpenter et al, 2002).

Concern about exposure to mixtures however is very serious. It is reasonable to expect that mixtures of substances, especially those with a common mode of action, can have additive, interactive or synergistic effects (Carpenter et al, 1998). Without getting into the complex details, including the many uncertainties of these possible combined effects, the point to note is that the isolated evaluation of a single substance is not likely to reveal its full potential for adverse health effects given the reality of routine exposure to mixtures of substances.

More important, some evidence has been gathered documenting situations of additive and synergistic effects of exposure to mixtures. These investigations also reveal some of the circumstances and reasons for finding non-additive effects, underscoring the complexity involved in trying to understand the many variations that are possible. As is the case with many of the health effects already discussed in preceding sections, this evidence comes from some of the most intensively studied substances including lead, PCBs, dioxin, some of the organochlorine pesticides and polycyclic aromatic hydrocarbons (PAHs) (as reviewed in Carpenter et al, 2002). Some of these results have been discovered since these substances, particularly PCBs and PAHs, are often complex mixtures themselves. Some researchers also point out the need to look at the combined effects of contaminant exposures and other health determinants, such as stress (Cory-Slechta, 2005).

It is also true that the ability even to measure multiple exposures, and evaluate multiple effects barely exists. This reality was emphasized in a landmark study on pesticides and children by the National Research Council in the US (NRC, 1993). That work prompted a great deal of subsequent investigation into the special vulnerability of children to environmental contaminants in general. The NRC study also contributed to important policy measures, subsequently incorporated into pesticide law in both the US and Canada, to evaluate entire groups of substances having a common mechanism of toxicity. Requirements were also put in place to aggregate exposures (from air, water, food, etc.) of the same substances to get a picture of total exposure. These techniques, for aggregating exposure, and evaluating common mechanisms of toxicity, are very new, not yet implemented, and tend to only address groups of similar substances. While they represent a start at addressing the real-world situation of human exposure, they do not even scratch the surface of measuring and evaluating the combined effect of many groups of dissimilar substances for which multiple low-level exposures are routine and multiple effects are suspected.

3.6 Economic Impact of Environmental Exposures

Research into the economic burden of disease and disorders from exposure to toxic substances suggests that exposure prevention could result in substantial savings in health care, human productivity and myriad social costs. For example, the impact of neurodevelopmental disorders such as AD/HD on children and their families is immense. Children with AD/HD are at greater risk for dropping out of school, delinquency, substance abuse, and suicide (Satterfield and Schell, 1997; Rasmussen and Almvick et al, 2001). It is estimated that in the US 3% of neurobehavioural disorders (dyslexia, AD/HD, diminished intelligence, autism and mental retardation) are caused directly by toxic environmental exposures, and that another 25% are caused by interactions between environmental factors (defined broadly), and genetic susceptibility of individual children (NRC, 2000).

A US-based health economics analysis estimated annual costs of paediatric environmental disease, focussing on four categories of illness in children: lead poisoning, asthma, cancer and neurobehavioural disorders (Landrigan et al, 2002). The study estimated that the total annual environmentally-attributable cost of these illnesses is 54.9 billion USD: 43.4 billion for lead poisoning; 2.0 billion for asthma; 0.3 billion for childhood cancer; and 9.2 billion for selected neurobehavioural disorders (mental retardation, autism and cerebral palsy) from toxicants other than lead. These estimates are deemed low because the analysis only considered four categories of illness, did not include related costs to families, or later complications due to these diseases or disorders. Also, the study was based on our current incomplete knowledge of the role of environmental contaminants in child health conditions.

Analysis by Canadian researchers of the cumulative costs of four health outcomes (diabetes, Parkinson's disease, neurodevelopmental effects and hypothyroidism, and IQ deficits from neurodevelopmental effects) in Canada was \$46 to 52 billion a year (Muir and Zegarac, 2001). Prevention of the approximately 10 to 50% of these health outcomes that may be environmentally induced would result in substantial savings annually to the Canadian economy (Muir and Zegarac, 2001). Although this analysis was not focussed solely on paediatric disease, the results allow one to conclude that such costs warrant much greater government and academic attention to environmental health research, disease and exposure tracking, and prevention.

A groundbreaking U.S. economic benefits analysis indicated that the societal benefits of reducing population blood-lead levels by just one microgram per deciliter (1 µg/dl) were estimated at \$17.2 billion dollars a year to the US economy. These figures were based on cost calculations for the effects of lead-related reductions in IQ on years of schooling and earnings, as well as

cardiovascular effects from lead exposure (Schwartz, 1994). Schwartz noted that these benefit estimates are low, as other known subtle effects of lead - on behaviour, attention, hearing, balance, and reduced stature - have not been assigned a monetary value. This benefit was revised upward in a subsequent economic analysis (Salkever 1995) based primarily on labour-market changes and more recent data on the relationship of IQ with educational attainment and projected earnings gains.

A more recent study looks at the societal cost of lost productivity associated with reduced IQ resulting from methylmercury toxicity. The study estimates losses to the US economy of \$8.7 billion annually (range: \$2.2 billion to \$43.8 billion) of which \$1.3 billion (range: \$0.1 billion to \$6.5 billion) is attributed to mercury emissions from US-based power plants (Trasande et al, 2005). Concern about the high cost to these plants of pollution abatement was recently cited by the USEPA to justify slowing down regulatory progress towards reducing their mercury emissions.

In 2005, the Ontario Medical Association (OMA) released a new report in which it updates the health impact estimates associated with air pollution in Ontario, and the economic costs associated with those impacts. Using updated air pollution studies, health statistics, and air pollution data, the OMA estimated that air pollution associated with human activity will contribute to about 5,800 premature deaths, 16,000 hospital admissions, 60,000 emergency room visits and 29 million minor illnesses in Ontario in 2005. These health impacts are attributed collectively to six air pollutants – ozone, fine particulate matter (PM10 and PM2.5), nitrogen dioxide, carbon monoxide and sulphur dioxide.

The OMA estimates that these health impacts will cost Ontario's health care system about \$507 million in 2005 for institutional care and medication, and employers and/or employees about \$374 million in lost-time as patients or caregivers. These costs do not include those associated with visits to doctors' offices, which are expected to be significant. The OMA values the pain and suffering associated with these health impacts at \$537 million in 2005, and the loss of life at \$6,391 million. In total, the OMA estimates that the preventable health outcomes in 2005 will "cost" about \$7.8 billion in economic terms (OMA, 2005).

Toronto Public Health estimates that air pollution in Toronto contributes to about 1700 premature deaths and 6000 hospitalizations on an annual basis. The mortality estimate is based on the health risk associated with acute exposures to ozone, nitrogen dioxide, carbon monoxide and sulphur dioxide, as well as the health risk associated with chronic exposure to fine particles (PM_{2.5}), the latter also associated with increased mortality due to lung cancer (TPH, 2004c).

Alongside these estimates of the most serious health effects (hospitalization and premature mortality), Toronto Public Health also notes that less serious health outcomes (e.g., chronic bronchitis, emergency room visits and number of days that people experience asthma symptoms) affect tens of thousands of people in Toronto every year.

As noted in Section 3.2.2, the rate of hospitalization for children with respiratory problems is very high, particularly in low-income communities in Toronto. A recent report on the burden of illness from childhood asthma in Ontario suggests that the healthcare utilization of and costs for children with asthma is markedly higher than for children without asthma. Children with asthma cost approximately \$100 more per year in health care and all told, represent about one-third of the total Ontario Hospital Insurance Plan (OHIP) expenditures for the general population of children in this Province (To et al, 2004).

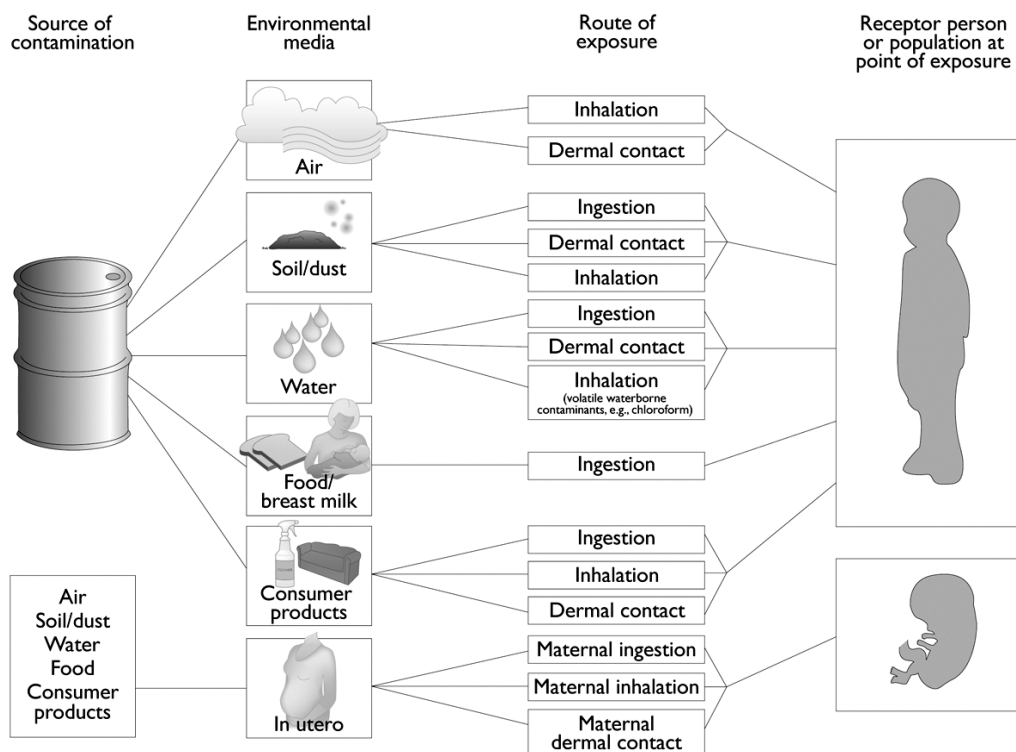
CHAPTER FOUR - EXPOSURE SOURCES AND SETTINGS OF CONCERN

4.1 Overview of Children's Exposure Issues

Like the health effects discussed in Chapter 3, describing the range of substances to which children are exposed is challenging. A discussion of exposure can include both a vast range of substances and, in many cases, multiple sources or settings for exposure. The gaps in information are even more pronounced for exposure data than for the scientific investigation of health effects.

This chapter elaborates on the concept that children are exposed via many potential pathways and they are often relatively more exposed to environmental contaminants compared to adults. Beyond the four main environmental media through which contaminants travel to people (namely air, water, soil and food), there are unique exposure media for children. These include the placenta, breast milk and non-food products, such as toys, carpets, floor surfaces, etc., all of which may harbour or transfer contaminants. The complexity of children's exposure sources and pathways is captured in Figure 4.1.

Figure 4.1 Major Pathways of Human Exposure to Environmental Contaminants



This multi-media nature of exposure is common for many environmental exposures. It is also the case that exposure will be highest, and of greatest concern, in certain media. In order to paint a complete picture of exposure to a specific substance, all media must be considered, including *in utero*, or transplacental exposure, as well as through breast milk.

A discussion of how children's exposures are assessed, including the evidence from recent tests of human tissues, is useful to set the stage for a fuller discussion of specific exposure circumstances.

4.2 Assessing Exposure

4.2.1 Overview of Methods of Determining Exposure

Since exposure to harmful substances can occur via several possible pathways, measurement of exposure can include various sampling techniques. To be thoroughly representative of an entire population, sampling ideally should be within the context of a longitudinal survey of exposure to a large group of people over time and across different media. Measurement should include concentrations or levels in environmental media (such as in air, food, water, etc.) as well as biomonitoring (further discussed in the next section) to measure the same substances in human biological samples including hair, blood, urine or breast milk. Unlike Germany and the United States, such biomonitoring has never been done systematically in Canada. Statistics Canada plans to conduct some national biomonitoring in 2006 with the Canada Health Measures Survey (Statistics Canada, on-line).

Unfortunately in Canada, we currently have less than ideal measures of exposure. Environmental levels are more commonly estimated by regional, or localized, environmental monitoring programs or from emission data. Levels in human tissues, if such information is available at all, may exist as so-called "convenience" samples, that is, measures of a particular substance (or substances) undertaken for a specific study. While informative, such results provide a one-time, cross-sectional picture of exposure, generally in a community or otherwise localized, non-representative sample. Such data are inadequate for the systematic characterization of population exposure, identification of high-exposure groups, changes in exposure levels over time and emerging exposure problems in Canada.

Assessment of exposure relies on both direct and indirect measurements. Several sources of data can be identified:

- biomonitoring, or measurement of contaminants in human biosamples (blood, urine, breast milk, etc.);
- environmental monitoring, or measurement of contaminant concentrations in environmental media such as air, water, food, soil and dust;
- estimates of emissions such as from industrial stacks, or automobile tailpipes; and
- information on amounts of products manufactured or sold (e.g., pesticides).

What matters in the end is the actual dose, or the amount of a substance that enters into the body of a child who is exposed. This is very difficult to determine. Biomonitoring can provide a direct measurement of the actual exposure. Many substances, including pesticides, will break down in the environment or be metabolized once in the human body. These breakdown products, or metabolites, may be of greater interest in monitoring than the original substance. For example, the body will metabolize many of the organophosphate (OP) pesticides, which can then be measured as six different urinary metabolites. The degree of exposure to OP pesticides can then be measured using the presence of these metabolites in urine (CDC, 2003). Biomonitoring can help evaluate trends in exposures over time and assess differences in exposures among groups of people. In some cases, for example lead, it is possible to relate levels in human tissues to health effects. Biomonitoring is further discussed in Section 4.2.2.

Environmental monitoring can provide data on the levels of a substance in the various media. Personal exposure monitoring, using data that is obtained when individual subjects wear portable devices that measure actual exposures throughout the day, is the most accurate. Most of the time however, environmental monitoring data are only available for certain points in time and space, which may underestimate the real exposure, since it will not factor in levels that are encountered in the actual places (microenvironments) a person spends time in over a day. For example, outdoor air quality data are available from only a small number of monitoring stations in Toronto. These outdoor air measurements cannot necessarily tell us what children's exposures are indoors where they spend up to 90% of their time, or when they are walking on a sidewalk near traffic.

In some cases, there may not be any suitable environmental monitoring information available. It may then be necessary to assess exposures using estimates of quantities of substances emitted, manufactured, sold or used. Such data introduces greater uncertainties in the assessment.

Once data are available on the levels of a pollutant in the environment, more calculations are needed to estimate the amount of the substance that is taken in by

a child. For each of the different ways a child can be exposed to a chemical the amount of a chemical that may be taken in has to be estimated. For example, to calculate the intake of air pollutants by a child's lungs, the measured level of contaminants at a certain location, or averaged over a certain time period, are combined with the volume of air inhaled and the internal surface area of the lungs. Further calculations may also be needed to estimate the true internal dose (the amount actually received by bodily tissues) taking into account what is known about uptake and absorption, including how this may be different in children than in adults.

When estimating exposures to substances in food, including in breast milk, information about the levels in food, the amount of the food consumed, and the expected uptake and absorption are combined. To obtain a more accurate estimate of a child's exposure, such calculations need to account for differences between adults and children in terms of:

- dietary preferences (a child who consumes little else but bananas, milk and apple juice for three days will get a much higher dose of associated pesticides than with a more varied diet);
- gastrointestinal absorption (which is generally higher in children than adults); and
- factors such as permeability of tissues like the blood-brain barrier (creating a much higher internal dose to a child's more sensitive tissues and organs).

Understanding the risk may in some cases involve additional considerations, such as, understanding how a substance behaves in the environment and estimating where it will end up. Persistent substances will tend to accumulate and can contribute to exposure via other media. Such substances can build up in the local, or even the global, food chain or end up in the soil that is handled (and partially consumed) by a child. For example, in Toronto, emissions that occurred for several decades from two secondary lead smelters contributed to contamination of local soil and house dust (Cooper et al, 2000). Likewise, several decades of burning leaded gasoline contributed to a reservoir of elevated lead levels in soil alongside major roadways in all major cities across North America, including Toronto (Mielke 1998; Cooper et al, 2000).

Many substances are found in more than one medium. For example, lead will be found in water, food, soil, house dust and in some consumer products. Measurement data for all of these media need to be aggregated to obtain a true picture of total exposure. This overall picture can also be useful to understanding which exposure sources are of greater concern. To complicate the situation further levels can change over time. Lead is again a useful example. The level of lead in paint was very high until the 1950s and lower, but still relatively high up to the

mid-1970s when regulatory limits were put in place in the US and Canada. Hence, a child's exposure to flaking paint, or the dust of deteriorating paint, is of greatest concern in any buildings built before the mid-1970s and even greater for those built before about 1950. Renovation practices can further change this exposure picture.

Sometimes the route of exposure is unexpected. For example, several chemicals within the group of substances called polybrominated diphenyl ethers (PBDEs) are widely used as flame retardants in numerous consumer products. These chemicals were found in human milk samples. When potential sources of exposure were being examined, they were found at high levels in indoor dust (Sharp and Lunder, 2004; Santillo et al, 2003; Stapleton et al 2004; and as reviewed in Hooper et al, 2004). They have also been found in indoor air and on the organic film collected from window surfaces (Santillo et al., 2003; Butt et al., 2004). This suggests that these substances are released into the environment during normal use of products containing PBDEs.

Across the board, the measurement of exposure to substances of concern in the indoor and outdoor environment is quite limited. This lack of data is a serious problem and undermines the ability of regulatory agencies to establish, evaluate and revise standards. Scientific and medical investigators need these exposure data to be able to understand dose-response relationships, that is, to understand what health effect or "response" can occur at what "dose" (or exposure level). This information is necessary to establish emission limits or other regulatory standards to reduce and or prevent hazardous exposures.

4.2.2 Biological Measures of Human Exposure - Biomonitoring

Biomonitoring is the direct measurement of substances (or their breakdown products) in human tissues, such as hair, blood, breast milk or urine. Biomonitoring studies conducted in recent years in the US, provide direct evidence that all children (and adults) are routinely exposed to environmental contaminants of which a variety can be measured in their bodies.

When done on a large scale, biomonitoring can provide a robust investigative technique. The United States has the longest history of biomonitoring with the National Health and Nutrition Examination Survey (NHANES). This survey, among many other measures of population health status, recorded the twenty-five year, nation-wide decline in children's blood-lead levels beginning in the 1970s as unleaded gasoline slowly replaced leaded gasoline and as lead was phased out in canned foods and certain other consumer products.

Biomonitoring data can also show exposure patterns within populations. For example, blood-lead levels have been consistently highest among urban-dwelling US children compared to their rural-dwelling counterparts and higher still among minorities (that is, those of Afro-American or Hispanic descent) and among those living in poverty (ATSDR, 1988). A similar urban to rural gradient in children's blood-lead levels was measured in Ontario in the early 1980s (Duncan et al, 1985). By 1995, after a multi-year phase-down of lead in gasoline and the national ban that took place as of 1990, this rural-urban gradient had disappeared (Smith and Rea, 1995).

The US Centers for Disease Control and Prevention (CDC) has expanded the ongoing NHANES biomonitoring of the child and adult population and in July 2005 released its third, biannual *National Report on Human Exposure to Environmental Chemicals*. The CDC's first such report provided measures of exposure for 27 chemicals (15 metals, 7 phthalate metabolites, and 6 organophosphate pesticide metabolites) in the blood and urine of the population (CDC, 2001; see also USEPA, 2003e). Urinary metabolites of organophosphate pesticides reflect fairly recent exposure and were found in all children assessed, consistent with the widespread use of these pesticides in agriculture and domestic environments. Phthalates were present in a large percentage of the population. These substances are found in many cosmetics, such as nail polish and shampoos, in vinyl flooring and soft plastic toys, and soft plastic I/V bags and tubing. From animal studies, phthalates are suspected in hormonal effects, reproductive organ effects, and changes in sexual behaviour (ATSDR, 2002a; and reviewed in Wigle, 2003).

In its second biannual biomonitoring report (CDC, 2003), CDC included 116 chemicals including some of the persistent organic pollutants (POPs) like dioxins, furans and PCBs, as well as more metals, PAHs and disinfectants. The report presents data collected over the 2-year period from 1999 to 2000. The recent third biomonitoring report (CDC, 2005) presents data collected between 2001 and 2002 for 148 chemicals, 38 of which have not been tested for before. New additions to the chemicals tested include the pyrethroid insecticides, some organochlorine pesticides and additional phthalate metabolites.

In addition, biomonitoring studies on smaller numbers of people have been conducted by some large environmental organizations. For example, the Washington-based Environmental Working Group reported that an average of 91 different contaminants was found in individual, adult subjects (EWG, 2003). A small but important recent study indicated that on average there were over 200 different chemicals, pesticides and environmental pollutants detected in ten neonatal cord blood samples from the U.S. (EWG, 2005). The contaminants detected included mercury, PAHs, polybrominated and polychlorinated dioxins

and furans, PCBs, organochlorine pesticides, PBDEs, perfluorinated chemicals (PFCs) and polychlorinated naphthalenes (PCNs).

The Pesticide Action Network of North America (PANNA) has used the CDC's biomonitoring data to analyze population trends. Such trend analysis is not a particularly strong feature of the CDC reporting as yet. PANNA noted that 100% of those tested had pesticide residues in their bodies, from pesticides that are in current use, as well as from those widely restricted or banned in North America since the 1970s. Of note, 99% had detectable levels of DDE (the breakdown product of DDT). These results reinforce the knowledge about the persistence of some substances, substantially phased out since the 1970s, such as PCBs and DDT, but which are still commonly found in human tissues and in other biota.

For the neurotoxic organophosphate insecticide chlorpyrifos, 93% of the CDC sample had detectable levels of its breakdown product (3,5,6-trichloro-2-pyridinol or TCP) (PANNA, 2004). Regulatory action in the US and Canada placed limits on the use of chlorpyrifos in the home or school setting due to concerns about exposure to children. Chlorpyrifos uses were substantially restricted in both countries as of December 2001. These limits were based on the evidence of possible developmental neurotoxicity but also because of earlier NHANES findings of chlorpyrifos residues in the urine of over 80% of a representative sample of U.S. adults (MacIntosh et al, 1999). The results of this earlier sampling of adults, combined with the CDC biomonitoring data, strongly suggest that children were experiencing widespread exposure to chlorpyrifos. Although it is not yet possible to detect the effect of the 2001 regulatory restrictions on uses of chlorpyrifos, it is likely that in future CDC reports the chlorpyrifos exposure prevalence of the U.S. population should decrease.

These CDC results offer lessons to be learned for other persistent, but still widely used substances, such as brominated flame retardants or PBDEs. Recent research analyzing a range of biomonitoring results, concludes that given present trends, a significant proportion of the North American population could already be (or could be within as little as three years) exposed to toxicologically relevant concentrations of PBDEs, (Muir, 2004).

Although this monitoring has found that people in the US of all ages have measurable body burdens of many contaminants, or their metabolites, further research is necessary to assess the implications to health. Work in Europe has continued apace with attempts to develop health-based reference values for these human biomonitoring values (Ewers et al, 1999). In the meantime, this ongoing data collection is extremely valuable to estimate the proportion of the population with body burdens of substances above established health-based reference levels (such as have been established for lead and mercury in blood). It also helps to

establish reference ranges for physicians and scientists to better understand exposure results in specific individuals or exposure circumstances.

4.3 Environmental Exposures by Media and Setting

4.3.1 Introduction

Children in Toronto, like all children, are more highly exposed to environmental contaminants than adults are for the reasons explored in Chapter Two. Their daily routines, activities, behaviours and food preferences place them at greater risk than adults. Toronto's children have specific differences in exposure compared to many other children in Canada because they live in a densely populated urban environment.

The rest of Chapter 4 scopes down the rather complicated discussion of exposures that are a concern to children by focussing on those related to the health effects of concern identified in Chapter 3. Namely, information on exposure to substances in the environment that are associated with effects on respiratory function, neurological development and functioning, reproduction and development, as well as childhood cancers, is presented. Priority is given to the exposures where evidence is strong or highly suggestive. These include air contaminants (outdoors and indoors), metals, pesticides and POPs. Physical factors, such as UVR, noise and heat, are also described.

Looking at exposure in terms of the settings where children (and pregnant women) spend their time provides a useful framework. It reveals the reality of multiple exposure sources and ideally highlights the settings where exposure is greatest. Ultimately this will assist with assigning priorities for preventive action.

4.3.2 The Settings of Exposure for Children

Children spend time outdoors in many settings: around the home; in the neighbourhood, often on streets with motor vehicle traffic of varying intensity; at school; in parks, playgrounds and outdoor recreational facilities; and when travelling, in cars and buses. When they play outdoors, they are generally more active and inhale more air per unit body weight compared to adults (Fenske, 1992). Timing matters as well. When children come home from school or child care centres, levels of key air pollutants like ozone are usually peaking (Bates, 1995).

Environmental threats for which some information exists to estimate or characterize the degree of exposure include air pollutants, pesticides, several metals, heat and UV radiation.

The information and data, where available, about outdoor exposures summarized here includes information about air pollutants, including smog-related pollutants and air toxics or hazardous air contaminants, soil contaminants and physical agents. Outdoor exposure is also discussed with respect to particular settings including, school grounds, parks, playgrounds and recreational facilities. The outdoor exposure section also addresses circumstances of exposure (such as travelling in vehicles) as well as aspects of the neighbourhood (such as proximity to traffic or to industry).

Children in Canada spend over 80% of their time indoors (Leech et al 1996). Clearly, exposures inside homes, schools and other indoor environments will have a key influence on children's environmental health. While there are reasonably good data to track trends in outdoor air pollution, there are no comparable data to describe trends in indoor air. Adoption of energy efficiency measures can have an impact on indoor air quality.

Children's indoor environments include the home, schools, child care centres or recreational facilities (swimming pools, arenas, etc.). Major contaminants or exposures of concern indoors, for which there is reasonably good information and/or exposure data, include air pollutants, biological agents such as moulds and other allergens, pesticides, some metals, PAHs, and POPs. Recent studies also identify a broad range of substances that originate from consumer products such as flame retardants or PBDEs, and a variety of other substances often found in indoor air or dust.

The media by which exposure to these substances may occur indoors include water, food (including breast milk), dirt/dust and in some cases, direct contact with consumer products. While eating and drinking can obviously occur outdoors, and being indoors is not a defining feature of such exposures, they are grouped here with indoor exposures for the sake of organizing a vast and complex topic. Exposure to toxic substances often occurs through more than one medium indoors or outside. This is the case for substances such as lead or pesticides as will be discussed towards the end of Chapter 4.

Since many sources can contribute to total exposure from any given substance and recognizing that a complete picture of exposure is indeed rarely available, the following discussion focuses on exposures where the information base is largest, such as for outdoor and indoor air and food exposures. In many instances, data specific to exposure of Toronto's children are not available, but where possible

regional, provincial or national level exposure information that may be indicative of the situation in Toronto is summarized.

4.3.3 Outdoor Air Pollution

Urban air contains numerous substances that are harmful to children's health. Combustion of fossil fuels, particularly diesel fuel, used in the transportation sector – cars, trucks and buses – is responsible for much of the emissions of key contaminants that contribute to urban air pollution. Fossil fuel combustion by industries, including coal-fired power plants, is also a contributor. During episodes of particularly poor air quality across Southern Ontario, a substantial portion of the pollution arises from transboundary sources (from the United States) including from coal-fired power plants in the US Midwest (MOE, 2005a).

Common Air Pollutants

Smog is a mixture of air pollutants dominated by ground-level ozone, nitrogen oxides, other irritating gases and particulate matter. Ozone is created when nitrogen oxides and volatile organic compounds (VOCs) combine in the presence of sunlight, hence smog can be worse in summer. Fine particles, or PM_{2.5}, are produced in many ways. They can be the result of chemical reactions in the atmosphere or a direct result of fossil fuel combustion. Fine particles can elevate smog levels during all months of the year.

Particulate matter (PM) is a complex and toxic mixture of sulphates, nitrates, ammonium ion, elemental carbon, polyaromatic hydrocarbons (PAHs), other toxic organic compounds as well as metals (reviewed in Wigle, 2003). As particle size decreases, surface area increases, which can result in greater concentrations of the adhered toxicants. Particle size also influences how deeply they are inhaled into the respiratory tract and lungs. Coarse particles are inhaled to the upper respiratory tract where they may be coughed up or perhaps swallowed, while fine and ultra-fine particles are taken deep into the lungs.

For some air pollutants such as fine particulate matter and ground-level ozone, evidence suggests there may be no lower threshold of exposure for the observed health effects (Environment Canada, 1999a, 1999b).

There are a large number of additional pollutants in air, many of which contribute to the problem of smog, and others that are part of the overall air pollution mix. One overall category of pollutants is volatile organic compounds (VOCs).

In urban areas, the major sources of VOCs from human activities are from industrial processes and motor vehicles, either as hydrocarbons resulting from incomplete combustion or evaporative emissions from fuels. Over 600 VOCs have been identified. In addition to transportation and other fossil fuel combustion (such as for power generation) other human activity-generated sources of VOCs include steel-making, petroleum refining, fuel-refilling, industrial and residential solvent use, paint application, manufacturing of synthetic materials (such as plastics and carpets), food processing, agricultural activities and wood processing and burning.

VOCs are a key part of the chemical mixture that creates smog because of the way ozone reacts with nitrogen oxide to produce NO_2 and oxygen. In the absence of VOCs, this ozone and nitrogen oxide reaction reaches a state of equilibrium. When VOCs are present, they react with nitrogen oxide and cause a net increase in ozone levels. As well, in the presence of sunlight, VOCs are oxidized (photochemical oxidation) which produces highly reactive hydrocarbon radicals that react with other smog components to form the reactive chemicals peroxyacetyl nitrate (PAN) and aldehydes. Alongside the smog pollutants discussed earlier, these additional substances, created during photochemical reactions with VOCs, add to the burden of reactive chemicals that contribute to eye and respiratory tract irritation and inflammation (reviewed in Wigle, 2003).

Local Data on Air Pollutants

Air monitoring programs are conducted by the Ontario Ministry of Environment, often in conjunction with Environment Canada's National Air Pollution Surveillance (NAPS) program. Monitoring occurs at 37 monitoring stations across Ontario, which form the Air Quality Index (AQI) network (MOE 2004). Six of these AQI sites are currently located in Toronto. Environment Canada's (or the MOE's) monitoring activity for air toxics is far from comprehensive. Monitoring is best for VOCs.

Air quality in Toronto, and in the Province as a whole, improved during the 1970s, in response to regulations placed on major emissions sources. However, progress on air pollution control has stalled during the last 20 to 25 years. Pollution levels from local and transboundary sources, particularly for the main ingredients of smog (ozone, nitrogen dioxide and fine particulate matter) are either on the increase or are not improving (TPH, 2000, MOE 2001, TPH, 2004c). In 2005, the province experienced a smog alert in February, the first ever officially recorded in the winter months (reflecting the addition of $\text{PM}_{2.5}$ to the AQI). With 41 smog alert days (through to August 31), the year 2005 is on track to be the smoggiest ever on record (TPH 2005 online).

Toronto's air is typical of that for other large cities in the world (ranking 16th out of 27 cities when comparing ozone levels) (as summarized in TPH, 2004c). However, Ozone concentrations in downtown Toronto continue to rise (MOE, 2004). The second highest annual mean levels of nitrogen dioxide occur in Toronto compared to all other communities in the Province where monitoring occurs (MOE, 2004). The major sources of man-made nitrogen dioxide emissions are high-temperature combustion processes such as those that occur in motor vehicles, other types of diesel engines and power plants.

The Ministry of Environment reports that over the 23 years from 1980 to 2003, the mean summer and winter ozone concentration in Ontario has increased (MOE, 2004). In a report on air quality in Ontario (with data for the year 2001), the Ministry's monitoring continues to show ground-level ozone exceeding the one-hour ambient air criterion routinely throughout the province, including in Toronto (MOE, 2001).

Published analyses of NAPS data show modest downward trends in overall VOC emissions across Canada during the 1990s. Environment Canada notes that urban levels for these contaminants are typically higher than for rural areas (Environment Canada, on-line). Benzene levels have fallen markedly in urban ambient air (65% between 1990 and 2002) in step with the lowered benzene content in gasoline (Thompson et al, 2004).

University of Toronto researchers have studied the "films" of outdoor volatile air toxics that collect on the surfaces of man-made structures, particularly impervious surfaces like glass. This is another source of and provides an indirect indication of the airborne material that children may inhale. In addition, since these contaminants are also deposited on many other surfaces (such as pavement, exterior walls, fences, outside plastic toys and playground equipment) the films are indicative of a potential *ingestion* exposure route (with hand-to-mouth activity) for children. These surfaces appear to become both a site of deposition and an ongoing source of these semi-volatile organic contaminants to the urban environment, that is, to the air and water.

Research has found a decreasing urban-rural gradient of these atmospherically-derived organic surface films and an indication that they derive from both localized sources and from long-range deposition. A range of PAHs, total PCBs and other compounds, including organophosphate pesticides, have been found in the films (Diamond, et al, 2000; Gingrich et al, 2001). One study looking at PBDEs on indoor and outdoor window surfaces in Toronto and other areas of the GTA, concluded that the source of PBDEs in both urban outdoor air and in the outdoor regional environment is urban indoor air (Butt et al, 2004).

4.3.4 Other Outdoor Air Exposure Circumstances

Proximity to Traffic

Within the large body of evidence demonstrating associations between air pollution from automotive traffic and respiratory health effects, there are studies showing that exposure to harmful pollutants increases with greater urbanization and traffic density (Rijnders et al 2001). This relationship has been shown over time, with emission reductions and presumably lower exposure correlated to reduced respiratory effects in children (Frye et al, 2003). A spatial relationship is apparent as well, with less severe effects seen in those further removed from dense traffic (and less exposed) compared to those living nearer high density traffic (Pandya et al, 2002, English et al, 1999, Oosterlee et al, 1996).

Travelling

The air inside cars and buses constitutes a microenvironment that can be a significant exposure source of vehicular emissions. A study of Connecticut school buses looked at children's exposure to diesel exhaust. Concentrations of PM_{2.5} inside the buses were frequently five to ten times higher than average levels measured at stationary monitoring stations (EHHI, 2002). In-car benzene concentrations can exceed roadside levels by up to four-fold and carbon monoxide levels can be 10 ten times higher. In fact, motor vehicle interiors have the highest average carbon monoxide levels of all "microenvironments" (Wigle, 2003). Likewise in subways and in buses, levels of PM₁₀ may be two to four times those in outdoor air (Wigle, 2003). There are some data to suggest that lowering the window may improve the levels of some in-vehicle pollutants (e.g. benzene and toluene, especially in older cars, or PAHs in diesel-fuelled buses), although it can also increase the exposure to other substances like ozone (Jo and Park, 1999; Sabin et al, 2004).

There are known hazards from the pollutants in diesel exhaust that are harmful to children and that affect air quality inside and outside school buses, as well as inside nearby buildings. Both the Toronto District School Board (TDSB) and the Toronto Catholic District School Board (TCDSB) are attempting to address the problem of children's exposures to pollutants from diesel fuel use in school buses. For example, the TDSB has implemented a pilot project using biodiesel in four of its buses. Biodiesel fuel is purchased at a 60% premium over the cost of regular diesel fuel. The TCDSB has a high percentage of its students bussed to school and applies a cleaner fuel policy to address the issue. Retrofitting buses with other new technologies that result in cleaner emissions is another option to be

considered. Cost is definitely a factor limiting the ability of school boards to implement more aggressive measures to reduce exposure to diesel pollution.

Travelling brings with it other risks that are not the focus of this report, but important to the overall picture of child health, such as injuries from traffic accidents (the leading cause of accidental death among children in Canada over one year in age). There is also great concern for greater frequency of car use and/or dependency, rather than walking (particularly to school), and possible links to the dramatic rise in obesity among children and youth in Canada.

4.3.5 Physical Factors

Physical factors in outdoor settings can include noise, heat, ultraviolet or other forms of radiation and electric, magnetic or radio frequency fields.

Low-level but chronic noise from moderate traffic can stress children and raise their blood pressure, heart rates and levels of stress hormones (Evans et al, 2001) and is linked to poor health (Passchier-Vermeer and Passchier, 2000). Exposure to aircraft noise has been linked to reduced reading levels in children (Evans and Maxwell, 1997, Evans et al, 1998, Haines et al, 2001).

Radio frequency (RF) fields are produced from a multitude of sources. Cellular telephone services, radio, television, pager services, and police and ambulance emergency communications, all depend on the use of radio waves.

There is uncertainty related to the health effects of low level RF fields. Adverse effects that have been observed in scientific studies in laboratory animals include tumour promotion, the ability to affect the blood-brain barrier, the potential to influence the body's pain control mechanisms, and the ability to affect sleep patterns (TPH, 1999b). Children may be particularly vulnerable to the effects of RF fields produced by cellular phones because of their developing nervous system.

Extremely Low Frequency (ELF) magnetic fields are produced from the flow of electrical current through power lines. Common electrical appliances produce identical fields, although smaller in magnitude. Concerns about a possible association between ELF magnetic fields and human health have been raised. To date, the strongest available evidence supports an association with childhood leukemia. An extensive review of the literature by the International Agency for Research on Cancer (IARC) has resulted in the classification of ELF magnetic fields as a 'possible human carcinogen', based on limited evidence of childhood leukemia in humans (IARC, 2002).

Heat and UV radiation (UVR) are often related issues. Lack of shade outdoors, at school, at home or in recreational settings, will increase children's risk of over exposure to UVR as well as contribute to heat-related stress. While sunscreen products are important to reduce the risk of sunburn, experts caution that the Sun Protection Factor or SPF should really be called a *sunburn* protection factor to highlight the fact that sunscreens only reduce the severity of skin damage from sun exposure rather than provide true protection (Gasparro, 2000). It is preferable to reduce and even avoid exposure during the peak exposure time for UVR (11 a.m. to 4 p.m.) by wearing protective clothing and/or seeking shade, rather than to rely on sunscreens alone (Koh, 1995).

Recognizing that children and youth are particularly vulnerable to exposure to high levels of UVR, and that skin cancer is largely a preventable disease, Toronto's Medical Officer of Health has reported on a proposed shade policy and technical considerations for the City of Toronto. The policy was developed by a multidisciplinary group comprised of appropriate City of Toronto departments and community partners in conjunction with the Ultraviolet Radiation Working Group of the Toronto Cancer Prevention Coalition. The report recommends that when planning for and developing future city-owned facilities such as a new park or public space, or when refurbishing old city-owned and operated facilities and sites such as a childcare facility, the provision of shade be considered as a protective measure against ultraviolet radiation (TPH, 2004e). Providing shade in areas where children are most likely to be in attendance was underscored as a priority. The Toronto Board of Health has twice endorsed the shade policy, most recently in May 2005.

The Toronto District School Board (TDSB) and Evergreen produced a guide for schools to increase shade on school grounds for the sake of preventing children's (and staff) overexposure to UVR and for cooling school buildings to reduce energy needs and create a comfortable environment. The guide is part of the series of guides in the EcoSchools Program and provides ways to assess needs, complete a site audit, plan and design for shade and to involve the community in the process. The guide focuses on shade provision via greening the school grounds and recommends starting with planting native trees and shrubs (TDSB and Evergreen, 2004).

The TDSB also has practices in place to protect children from temperature extremes. For example, children are to be kept indoors for recesses due to risk of frostnip and frostbite whenever temperature and/or the wind-chill factor indicate minus 28C or lower. The Board monitors the heat and smog alerts issued for the City by TPH and advises that students and staff be vigilant about their level of

activity and ensure they remain adequately hydrated under these conditions (TDSB, 2001).

Climate change is expected to continue to create summer and winter temperature extremes and children are at greater risk than healthy adults for the effects of intense heat, humidity or cold.

Children, especially very young children, can also experience stress and psychological trauma from the climate change-induced severe storm events that are occurring with greater frequency and at times causing significant damage to homes, property and large trees. Some data suggest also that stress experienced by pregnant women during such extreme weather events may impact on the functioning of the children who were so exposed while in the womb (Laplante et al, 2004).

4.3.6 Arsenic-Treated Wood Structures

Arsenic can be found on the surfaces and in the soil underneath playground equipment and other outdoor structures built from pressure-treated wood containing the pesticide chromated copper arsenate (CCA). The production of CCA-treated wood for residential uses was phased out as of January 1, 2004 in both Canada and the United States. However, this wood has been a popular choice for building decks, fences and play structures, and it appears to slowly leach arsenic for years (Riedel et al, 1990; USEPA, 2001). Existing structures therefore remain an arsenic exposure source of concern for parents of young children.

In humans, chronic arsenic exposure causes cancer, is toxic to the nervous system and is linked to skin lesions (including skin cancers) and reproductive problems. It is also known to be toxic to the immune system in experimental animals (as summarized in Ursitti et al, 2004). Precautionary steps should be taken to avoid exposure, especially of children.

Tests done by the City of Toronto revealed arsenic levels above the federal guideline in soil directly below 15% of a total of 217 structures made of CCA-treated wood on city-owned playgrounds. On the wood surface, dislodgeable (that is, available) arsenic was detected on most play structures although only 15% had surface arsenic levels above the interim action level established by TPH. (There are as yet, no health-based standards for “acceptable” levels of dislodgeable arsenic on wood surfaces.) One Canadian study has determined that children playing on structures made with CCA-treated wood had higher levels of arsenic on their hands than children who played on non-wood structures. The resulting

intake or effects in children from exposure to these levels of arsenic is not fully understood (Kwon et al, 2004).

The U.S. EPA and Health Canada have undertaken a risk assessment looking specifically at this question and so far have published draft findings (USEPA, 2003f). In 2003, the U.S. Consumer Product Safety Commission (CPSC) determined that exposure to arsenic from play structures might increase the risks of certain cancers in the long-term. With TPH guidance, the City removed and replaced soil, sealed the wood or did both, wherever arsenic was elevated. In addition, there is a long-term plan for continued monitoring and risk management of these play structures. While the City of Toronto has recognized this problem, taken mitigation steps and developed public educational materials, it is likely that the many backyards fences, decks and play structures on private property are a potential, ongoing arsenic exposure source for children.

In response to the information sent by Toronto Public Health to school boards in August 2002, the boards have taken a number of actions with respect to structures built with CCA-treated wood. The Toronto District School Board (TDSB) specifies that any new wood playground equipment is made from wood treated with the available non-arsenic-based preservative. Both the Toronto Catholic District School Board (TCDSB) and the TDSB have undertaken to regularly seal any existing CCA-treated structures with appropriate penetrating sealants. At one TDSB site (where a problem with vandals burning CCA-wood structures prompted removal of many of these structures in 1999) wood chip re-surfacing was carried out where soil test results indicated arsenic exceedances (David Percival, e-mail communication, 2003).

4.3.7 Other Outdoor Use Pesticides

Reducing childhood exposure to pesticides through diet and from household and institutional use has gained much attention in recent years among policymakers in North America. There is a range of media and sources by which children may come into contact with pesticides including applications in and around their homes, day care facilities and schools and, on lawns, parks and family pets. Children are fairly likely to encounter low-level exposure to pesticides indoors and outdoors from their use as treatments for pests or to control lawn and garden weeds or insects (TPH, 2002d). Indoor application of pesticides and exposure through water and diet are discussed later in this chapter.

Spray applications of pesticides increase the chance that there is inhalation exposure to the applicator and any by-standers. Lawn-care pesticide residues may also be tracked into the home (Nishioka et al, 1996, Nishioka et al, 1999,

Nishioka et al, 2001). Children are more likely to be exposed by inadvertent ingestion of, or dermal contact with pesticide residues on treated turf or objects in their environment (Lewis et al, 1994, Bradman et al, 1997, Freeman et al, 2001).

Only limited information on the exposure of children in Canada to outdoor use pesticides exists. For example, L'Institut national de santé publique du Québec (INSPQ) conducted a small study to monitor pesticide exposure in children from suburban areas of Quebec. Pesticide exposure was not detected if neighbours had used lawn chemicals. But, where families reported applying a phenoxy herbicide to their own lawn, these pesticides were detected in urine samples of most of the family's children (INSPQ, 2004). As well, exposure to insecticides was evident where these pesticides had been used to spray fruit trees in gardens.

Despite many gaps in the information and limitations to the research available, at the very least, the data on health effects associated with pesticides warrants continued efforts to raise awareness of the need to limit exposure of the young and of pregnant and nursing women to pesticides used around the home (TPH, 2002d). A decision to be prudent in light of these scientific data and uncertainties underscored the choice made by Toronto City Council in 2003 in establishing a by-law to limit "cosmetic" or nonessential uses of pesticides. The pesticide by-law does allow for pesticide uses that are related to the protection of public health. The Toronto pesticide by-law, like similar by-laws in other Canadian municipalities, applies to both public and private property. The extension to private property is a new direction for the City but the decision to restrict the use of pesticides on public property is longstanding. The Toronto by-law came into effect on April 1, 2004 and enforcement is being phased in, along with intense public educational efforts, over a 3-year period.

Across Canada, many municipalities, parks departments and school boards have steadily reduced their "cosmetic" use of pesticides in the last fifteen years. Both the Toronto District School Board and the Toronto Catholic District School Board prohibit application of any herbicides on school properties and focus on traditional horticultural methods for weed control and grounds maintenance. While the TCDSB has established a written policy in this regard (TCDSB, 2001a), the TDSB does not have a written policy, but has not used herbicides on school grounds for many years. Like the Toronto pesticide by-law, this TDSB approach does allow for the use of pesticides where health impacts are a concern such as for control of poison ivy, a rare occurrence (Richard Christie, TDSB, pers comm).

Use of personal insect repellents represents another type of outdoor, personal pesticide exposure. The spread of West Nile virus (WNV) in North America has prompted public health concerns along with the increased use of and scrutiny

towards personal insect repellents, particularly their safety for children. In the past, regulatory agencies did not recommend use of N, N-diethyl-m-toluamide (DEET) on children under 2 largely due to concerns about neurotoxic effects shown after over-exposure (from incorrect use) (USEPA, 1998; Reigart and Roberts, 1999; PMRA 2002). With the risk of mosquito-borne illness increased in recent years, the “acceptable” use of DEET has been extended to children 6 months to 2 years (in 10% or lower concentration, one application per day), but it is still not recommended for infants under 6 months of age (PMRA, 2004; CPS, 2003). Some still suggest that use of DEET on children should be avoided altogether (Briassoulis, et al. 2001). As part of its a public education materials, TPH recommends that people only use products containing 10% or less DEET or that approved alternatives be used (according to label directions) (TPH 2005a).

DEET provides a good example of how the determination of risk is reflected in the product label. If the label instructions are not followed, the “safety” of the product, as calculated by the risk assessment, is no longer valid. A recent study in the U.K. determined that about 45% of a sample of parents felt that pesticide product labels were hard to understand and did not provide all the information they would need (Grey et al, 2005).

4.3.8 Contaminants in Recreational Water

The need to post warnings on beaches during hot summer weather has been a problem for many years in Toronto. Water contamination by *E. coli* is routinely monitored. *E. coli* bacteria, found in animal and human waste, can cause diarrhea and complications including severe kidney damage. Less severe, more typical impacts can include ear or throat infections and skin rashes (Health Canada, 1998). The exposure concern relates less to dermal contact than to accidental ingestion of water or exposure to the ears, nose and throat from swimming underwater. Those most susceptible to *E. coli* infection include young children, the elderly, and those with depressed immune systems (Wigle, 2003). Measurement of *E. coli* is used as a surrogate for estimating risks from a wider range of potential waterborne pathogens. When *E. coli* reach unsafe levels (greater than 100 *E. coli* per 100 millilitres of water), beaches are posted as unsafe for swimming.

During hot weather and particularly after summer rain and thunderstorms, the sewage outflows to Toronto’s rivers and to Lake Ontario can become overloaded with bacteria. A combination of factors is at work including the age and type of sewers. Recent improvements in beach water quality are reflective of progress in modernizing sewers in older parts of the City and the construction of large holding tanks for stormwater so that it may be treated after storm events. For

example, two large holding tanks near Toronto's Eastern Beaches trap storm water so that the main sewage treatment plant can process the extra flow following storm events. The Wet Weather Flow Management Master Plan, approved by City Council in June of 2003, endorsed construction of a similar but larger system, to increase capacity. Called the Western Beaches storage tunnel, it is a series of underground tanks, tunnels and outfall treatment of three storm outfalls and seven combined sewage outfalls and has been operational for two years. This storage tunnel and associated system should contribute to improved beach water quality on the western side of the waterfront as has been the case in the East.

There is some research to suggest that other contaminants in local recreational water may present an exposure risk to people, including children, who swim in these waters. Health Canada researchers have concluded that people, including children, who swim in Great Lakes water are potentially exposed (both orally and dermally) to low levels of contaminants of concern found in Great Lakes water sediment (Moody and Chu, 1995). Among the chemicals known to contaminate the Great Lakes waters are PCBs, organochlorine pesticides, metals (including lead and mercury), PAHs, VOCs and phthalates (Moody and Chu, 1995).

4.3.9 Contamination of Soil and Surfaces

Soil contamination from industrial operations and vehicle emissions presents a reservoir of potentially significant exposures of concern to children. Much site-specific work has been done over the years in Toronto to evaluate soil contamination levels at industrial facilities, generally as a result of redevelopment activity. Provincial rules are in place governing levels of contaminants that are allowed when land uses change. These levels are more stringent if land use changes from industrial to residential use.

During the 1970s, after 50 years of the use of lead in gasoline, and the operation of two secondary lead smelters in two older Toronto neighbourhoods, soil lead concentrations reflected a clear pattern. Soil lead concentrations were highest near the two industrial point sources. Levels were slightly lower but still greatly elevated along the busiest traffic corridors and levels decreased with distance away from both traffic arterials and the downtown core, and then from the urban area generally (Sharpe et al, 1974, reproduced in Wallace and Cooper, 1986).

Automotive lead emissions ceased in the early 1990s. However, the gradient of soil lead contamination in the urban core and along heavy traffic areas will continue for decades into the future. Such urban concentrations of lead are routinely above the guideline for residential soil removal of 200 parts per million

(MOEE, 1993). However, this removal guideline is only applied in the context of site remediation around industrial facilities or during redevelopment. Applying this guideline to the entire city would mean digging up the soil along every major traffic artery and in many additional downtown locations, an impossible and impractical response. Rather, this situation calls for ongoing awareness and education. For example, it is important to recognize that healthy ground cover reduces exposure risks and that dust control practices are necessary during any activities that disturb roadside soils.

Soil contamination from the historical use of leaded paint on older homes also warrants concern and ongoing awareness. Many studies from the United States, particularly in areas where dwellings tend to be older, indicate that soil lead levels can be extremely high in the area immediately surrounding the house and below areas where walls and other surfaces have been painted. Children should not play in this soil. Considerable care is also needed during demolition, renovation or the treatment of these older surfaces in preparation for re-painting to avoid contributing to the lead burden in the immediate area, including indoors, on porches or other outdoor surfaces, and in the surrounding neighbourhood (Farfel et al 2003, Mielke et al, 2001, Mielke, 1998). For example, in one study, power sanding of old painted siding and window frames (in a 1920s-era home in New Orleans) contributed to lead levels inside the home that were *billions* of times above the US EPA's Tolerable Daily Intake for young children (Mielke et al, 2001). Even though an alternative dry scraping method did not contribute to increased lead around the exterior of the home or in indoor dust in this study, extensive care and cleaning was deemed important to reduce the likelihood that children are exposed to bits of old lead paint during re-treatment.

Across Canada, just over 60% of homes were built before 1976, 39% were built before 1965 (NRCan, 2000). In Ontario, it is estimated that just over 2 million homes built before 1970 could be affected by lead-based paint (MOEE, 1993), a large proportion of which will be in many of the older neighbourhoods of Toronto. Data from the 1991 census indicates that over two-thirds of the housing stock in the City of Toronto (former Metropolitan Toronto) was constructed between 1946 and 1980 and nearly one quarter of Toronto homes were constructed prior to 1946, a time when paint lead levels were extremely high (Campbell and Lee-Han, 1996). These figures suggest there is potential for lead exposure in many Toronto homes when buildings are in poor or deteriorating condition and especially during renovations if proper precautions are not followed.

Any such reservoirs of toxic substances in soil and dust in the outdoor environment present an ongoing source for indoor exposures via shoes, pets,

bicycles, stroller wheels, and other means of tracking soil and dirt indoors (as discussed further in the next section).

4.3.10 Indoor Air Pollution

Indoor air quality is a largely unregulated source of exposure to a variety of contaminants. Scientific experts acknowledge that poor indoor air quality is a significant environmental health issue requiring further study and monitoring (WHO 1997; CARB, 2005).

One environmental threat where evidence of harm from indoor exposure is strong is environmental tobacco smoke (ETS). The scientific evidence of health and developmental effects associated with exposure to ETS justifies continued efforts to eliminate smoking around the young and to counsel pregnant women to avoid smoking or exposure to ETS (Di Franza et al, 2004).

As discussed in Section 4.2.1 concerning outdoor air pollutants, volatile organic compounds (VOCs) can be an indoor air pollution problem as well. VOCs can be released from building materials including pressed wood used in furniture, shelving and wall materials, glues, caulks and adhesives, paints and sealants, and floor coverings including the adhesives used in them and to attach them. Such compounds will also be released from the use of cleaning products, indoor pesticide use and paints (USEPA, on-line and USEPA, 1995).

In any building, the heating, ventilation and, where present, air-conditioning (HVAC) systems can circulate these compounds throughout the building. The gains made from improvements in building energy efficiency can often result in these compounds being trapped indoors. Levels of VOCs, such as those emitted by the use of latex paint, will drop significantly within a few weeks while those in wood products and carpeting can persist indoors for years underscoring the need to ensure ongoing ventilation and better still, the initial choice of low-emission products (ALA, USEPA, CPSC & AMA, 1994).

Exposure to various indoor air contaminants is believed to increase the risks of developing asthma or other respiratory irritation and health problems (IOM, 2000). The indoor exposures of concern in these health outcomes can include inhalable particles from combustion products (fireplaces or wood-burning stoves), VOCs and biological allergens (such as moulds, pet dander, house dust mites and cockroaches). In addition, poorly vented gas furnaces and especially unvented gas stoves (particularly in winter months) can produce substantial amounts of nitrogen dioxide indoors (USEPA & CPSC, 1995).

Research into the different factors that increase the risks of asthma and allergies in children continues to expand knowledge of the indoor exposures of concern to children's respiratory health. Residential factors such as exposure to ETS, use of a gas stove or oven for heat and presence of a dog in the home are reported to account for approximately 39% of the physician-diagnosed cases of asthma in the U.S. (Lanphear, et al, 2001). As well, exposure in infancy to house dust mite (HDM) allergens (enzymes present in mite feces), that are found in bedding, soft furnishings, carpets and other indoor sources, increases the chances of developing asthma and allergies later on in life (Warner, 2000). As discussed in Section 3.2.1, household-cleaning products can contribute to indoor air contamination. Significant associations have been found for wheezing and other asthma symptoms after exposure to cleaning products in adults working as cleaners and among children prenatally exposed (Sheriff et al, 2005).

Finally, the air of indoor recreational facilities can present exposure risks. Researchers have found an association between inhalation exposure to certain chloramines in indoor chlorinated swimming pools and development of occupational asthma among some swimming pool workers (Thickett et al, 2002). Effects on the lung epithelium of children were found to be associated with regular attendance at an indoor chlorinated pool (Bernard et al, 2003).

Schools and Child Care Facilities

Many of these indoor exposures are equally relevant in the indoor air of schools and child care facilities. As in the home environment, VOCs can be released from furniture and building materials, glues, caulks and adhesives, paints and sealants, and floor coverings, as well as from the use of some cleaning products, pesticides (discussed further below), markers (e.g. dry-erase markers for white boards), paints and other art or science supplies in schools (USEPA, on-line and USEPA, 1995).

The Toronto Catholic District School Board (TCDSB) has in place a wide range of written policies or guidelines that aim to protect children from indoor environmental exposures. The Toronto District School Board (TDSB) holds to many of the same procedures as are evident in TCDSB policies.

In the TCDSB a general guiding principle states that maintenance schedules be adapted to fit the school schedule, so that some activities only take place when children (and most staff) are not in the building. More specific policies set out requirements for construction, renovation and maintenance activities including, for example, ensuring dust and noise control, applying low emissions or low odour paints and conducting renovations during periods of extended school

breaks, with appropriate ventilation. Precautions are applied with respect to lead paint repair including assigning priority of repair to areas with visible peeling paint depending on the accessibility of that paint to students and the degree of supervision of students in the area. As well, the TCDSB reports that it uses only low VOC cleaning products, does not allow use of bleach and is switching away from use of deodorizers that contain paradichlorobenzene.

Both the Toronto District School Board (TDSB) and TCDSB have aggressively dealt with the issue of mould in portable classrooms. The TCDSB Standard Operating Procedure for mould outlines a sequence of steps in assessing and remediating mould, beginning with visual inspection, air testing and swab sampling, comparison to the Health Canada benchmark, followed by invasive testing and repair where the benchmark is exceeded. Monthly inspections of the facilities are conducted by a Joint Health and Safety Committee and portables are thoroughly inspected during the summer. At the recommendation of an expert advisory committee the TCDSB removed carpets from portables. The current TCDSB policy is that where mould is found all occupants are relocated.

Despite policies that are in place, indoor air quality problems can surface, testament to the reality that this issue requires greater proactive attention. An innovative pilot project occurred at the TDSB's Blake/East Alternative School of Toronto (EAST). Working with the school's administration, the local trustee, TDSB Facilities staff, teachers, caretakers, the parent council and South Riverdale Community Health Centre (SRCHC), the Blake/EAST Indoor Air Quality Project identified and worked to correct indoor air quality problems at Blake Street Junior Public School. This two-year project mobilized the community and developed its own checklist for helping diagnose potential sources of indoor air problems. As well, SRCHC's Indoor Air Working Group is currently developing an instructional video and associated print material that can be used by any community in applying the same assessment techniques for diagnosing and remedying indoor air problems (Ann Phillips, SRCHC, pers comm).

As a result of the work on the Blake/EAST Indoor Air Quality Project, the TDSB recently affirmed a commitment to work with its schools and the community to improve the learning, teaching and working environment by improving indoor air quality (TDSB, 2004). This commitment will be achieved by targeted assessments of indoor air for three schools in each ward, using the Blake/EAST Indoor Air Quality Project checklist as a guide.

4.3.11 Indoor Pesticide Use – Home and Schools

Pesticide exposure can be unsafe when use occurs in the indoor environment. It is because of this potential for overexposing children that limits have increasingly been placed on the use of pesticides in areas frequented by children.

Whether applied directly indoors or tracked indoors from outdoor use, the biodegradation of pesticides that generally occurs outdoors (via sunshine, wind or rain) slows down dramatically out of the elements. One study found measurable levels of up to twenty pesticides in household indoor air and dust (Rudel et al 2003). Some surfaces, particularly carpeting, will retain residues more readily than others, such as smooth flooring. Given that children spend so much time indoors, the US EPA estimates that for most people up to 80% of pesticide exposure occurs from the indoor use of pesticides (USEPA, 2004). EPA researchers have determined that the pesticide residues measured on children's hands corresponded to the type and concentration of the pesticides most often found in carpet dust (Lewis et al, 1994).

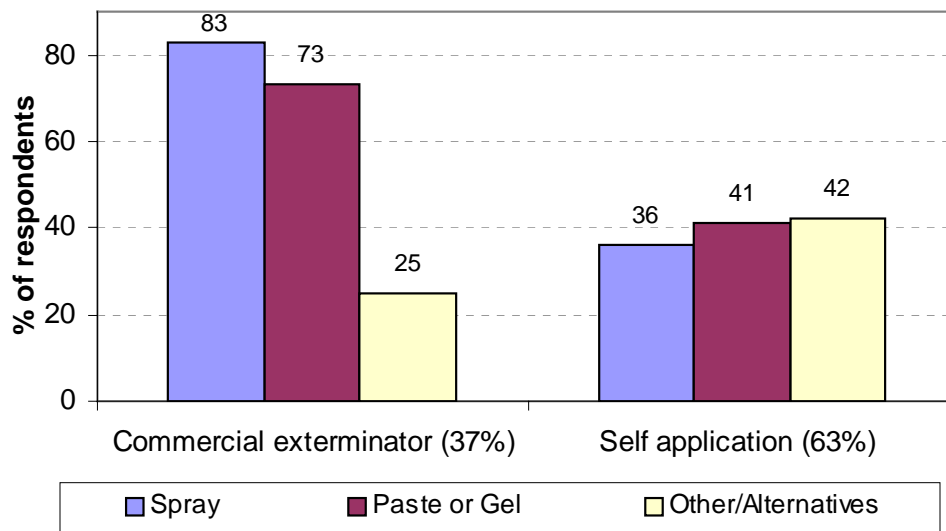
A dramatic illustration of this indoor pesticide exposure comes from a study that looked at a standard residential indoor application of the insecticide chlorpyrifos. Residues persisted and secondarily accumulated in toys, pillows and on hard surfaces for over two weeks, long past the safe period for re-entry (Gurunathan et al, 1998). The levels measured in plush toys or pillows suggest that these objects provide an ongoing source of exposure for children. The range of estimated exposure doses (depending on exposure scenario and frequency of mouthing, etc.) were from 21 to 120 times the current US EPA reference dose (RfD) of 3 µg/kg/day (Gurunathan et al., 1998). Chlorpyrifos is a neurotoxic organophosphate pesticide for which home and school use (but not agricultural use) has been substantially restricted due to child health and exposure concerns.

Toronto Public Health has surveyed the extent of indoor pesticide use and encourages use of non-toxic alternatives. A 1990 survey of residents in the former City of Toronto found about half used some form of indoor pest control (Sly et al, 1991). The 2002 survey of parents with children aged 0 to 12 found only 17% indoor usage, leading to a possible conclusion that parents tend to avoid indoor pesticide use for the sake of reducing their children's exposures (TPH, 2004c, 2004d). Much higher use rates are reported in the US. Based on recent national survey data, the US EPA estimated that 75% of households used at least one pesticide indoors within the last year (USEPA, 2004). A comprehensive study of indoor pesticide use in New York State found 69% of survey respondents applied pesticides in their own homes, and 33% did so at least once a week. Eight out of ten housing developments surveyed reported routine prophylactic indoor use of

pesticides and also using restricted pesticides (those only for use by a licensed applicator). Likewise, individual respondents reported using illegal pesticides and 12 of 73 stores surveyed were found selling illegal pesticides (NYS EPB, 2002).

Awareness of the risks of indoor pesticide use appears to be much higher in Toronto. In another survey, conducted during 2000, Toronto Public Health surveyed 471 apartment dwellers due to the high concentration of City residents living in apartments and the evidence of a greater prevalence of indoor pesticide use among apartment dwellers (TPH, 2004b). Most respondents had not used pesticides indoors in the last two years. Respondent experiences, and likely their pesticide exposure (in the control of cockroaches), differed depending on whether they took action themselves or called a commercial exterminator as shown in Figure 4.2 below.

Figure 4.2 Comparison of indoor pest control methods used to control cockroaches



Source: Toronto Public Health, 2004

The majority of respondents (63%) took action themselves and were more likely to use chemical paste or gels (41%) or non-toxic alternatives (42%). Chemical sprays were chosen by 36% of respondents. For those who used a commercial exterminator, 83% used a spray, often in combination with other control methods like chemical paste or gels. Just over half of those using the commercial exterminator were given information about removing food, dishes and utensils from cupboards before the pesticide was sprayed. Only one-third were given

information about leaving the home while the pesticide was being sprayed and waiting at least 8-10 hours before re-entering. It seems clear from these survey results that the combination of the much greater likelihood of using chemical sprays, as well as perhaps not being informed of, understanding, or taking precautions to avoid exposure would mean that those choosing an exterminator will experience greater pesticide exposure.

Another result of this survey included respondent knowledge about risks from pesticides including risks to children. A majority (60%) knew that children are more likely than adults to be exposed to pesticides used indoors and a large proportion (79%) knew that children are more likely than adults to be harmed by such exposures. A minority (39%) recognized that pesticide sprays constituted a major factor in causing indoor air pollution (TPH, 2004b). The majority of the sample population (85%) also felt that chemical sprays should be a last resort after other methods had failed.

TPH's "Roach Coach" pilot project was initiated in 1998 to assess the effectiveness of an Integrated Pest Management (IPM) program for controlling cockroaches in apartment complexes (Campbell et al, 1999). The project came about due to concerns regarding the use of pesticide sprays indoors and because cockroach infestations result in the greatest use of pesticides in Toronto homes and apartments. Participants in the pilot showed improved knowledge and practices, shifting to lower toxicity and lower exposure risk methods for controlling and preventing cockroaches after the intervention which consisted of an education session and booklet.

In Toronto schools, in addition to the prohibition of herbicide applications on school grounds, the TCDSB has an IPM-based pest control program based on preventing pest habitat, applying barrier methods, responding to problems according to careful assessment and monitoring and matching the response to the degree of the problem. Pest control contractors are only called in the event of an infestation and are to visit schools on Friday evenings, assess the problem, apply non-toxic or low toxicity controls (for example, boric acid paste injected into baseboards for cockroach control) and are not allowed to spray without obtaining prior approval from the Occupational Health and Safety Department (TCDSB, 2001b).

Finally, another not uncommon avenue of children's exposure to pesticides indoors is through their use in insecticidal lice treatments. The active ingredients used in shampoos for treating lice are the same as those found in other insecticides, however, lice shampoos (pediculicides) are regulated as drugs (i.e. they have a Drug Identification Number or DIN), not as pesticides. Over the counter pediculicides now almost exclusively contain either synthetic or natural

derivatives from the chrysanthemum plant (pyrethroids or pyrethrins, respectively), which are generally less toxic than their predecessors (such as, lindane or malathion).

The persistent organochlorine pesticide lindane is currently still approved for use in lice shampoos in Canada, although it is only rarely used, such as for resistant cases or where there is sensitivity to other pediculocidal products. The Canadian Paediatric Society notes that lindane-based lice treatments are not recommended for infants or young children (CPS, 2004).

Permethrin, a pyrethroid insecticide, is the pesticide found in most over the counter lice treatment shampoos (e.g. *Nix* and *Kwellada-P*). A Motherisk study of a permethrin-based product suggests there is no increased risk of major malformations in the infant from its use by pregnant women (Kennedy et al, 2005). Both pyrethroids and pyrethrins have relatively low toxicity profiles and appear to be only minimally absorbed through the skin (CPS, 2004). EPA researchers recently acknowledged, however, that there is a need for greater study of the potential for developmental neurotoxicity from pyrethroid, the synthetic version of these insecticides (Shafer et al 2005). TPH's pediculosis policy suggests that pyrethroids not be used as an anti-lice treatment on children under two (TPH, 2005b).

TPH endorses the use of manual methods (such as the Lice Meister® fine-toothed comb or use of sticky tape. Regarding alternative, low toxicity substances (e.g. vinegar, petroleum jelly, mineral oil, baby oil, cooking oil, tea tree oil or other alternatives) TPH agrees with a number of authorities in finding that there is insufficient scientific evidence of efficacy and of toxicity (CPS, 2004; US NPA, online undated; HSPH, 2000). Some pure oils such as tea tree oil, may be contraindicated for babies, young children, pregnant women and pets (NPA online, undated). In some cases, combining substances, such as vinegar, with lice treatment shampoos can reduce their efficacy. Though a new generation of lice-treatments such as the dry-on, suffocation-based pediculicides (DSPs), offer the promise of non-toxic treatment with a different mode of action, more research on efficacy is needed (see Pearlman, 2004).

A study in the UK notes that 85% of a sample of parents stated they would use the “bug busting”, non-pesticide method for lice removal first, before using the chemical anti-lice shampoos. The authors suggest this is because of negative information on the chemical methods, via the schools and the media, plus the fact that the schools generally promote the bug busting method, illustrating the influence of educational strategies (Grey et al, 2005).

4.3.12 Food Exposures

Pesticide Residues

The Canadian Food Inspection Agency (CFIA) monitors for pesticide residues on imported and domestically-produced food in Canada. The agency has aggregated monitoring results for the four year period from 1994 through 1998 (CFIA, 1998). More recent data, up to the end of the 2003 - 2004 monitoring program, is available on-line (CFIA, on-line) and is summarized in the agency's annual report (CFIA, 2003).

Monitoring is conducted on a wide range of foods, with the largest number of samples being fresh and processed fruits and vegetables. About 80% of monitoring of fresh fruit and vegetables occurs on samples of imported foods reflecting the degree to which imported fresh produce is consumed in Canada.

For the period 1994-1998, almost 25% of the total samples contained detectable levels of pesticide residues. The violation rate (those residues exceeding the federally-established Maximum Residue Levels - MRLs) was low - 1.2% for domestic samples and 1.94% for imported samples. Imported produce contained residues of both permitted and banned pesticides including organochlorines, such as aldrin, heptachlor, lindane and DDT and its metabolites, but rarely at above MRLs.

More recent monitoring results are comparable. The rate of violation of MRLs is similarly low and the proportion of samples with no detectable level of pesticides, at least on the largest sampling category of imported fresh fruits and vegetables, appears to have dropped to about 12% (CFIA, on-line). Pesticide residue detection is reported as generally in compliance with regulatory limits.

The CFIA monitoring also includes results of testing where multiple pesticide residues are found in a single sample. For example, in the 1994-1998 results, 84 samples of both domestic and imported fresh fruits and vegetables had residues of three or more different pesticides and residues of two pesticides were found 124 times (CFIA, 1998). The data reported for the 1999-2004 period indicate that there were 43 food samples where residues of three or more pesticides were found and 61 samples with two pesticides (CFIA, on-line).

Of particular note, the processed food monitoring program surveyed infant and junior baby foods for residues of five pesticides. Twenty-five percent of the sampled products were imported, while the remaining were domestic. Of 412 samples, 383 had no detectable residues. Residues were found in 7.7% of the

regular domestic samples and in 6% of the "Organic" products, one of which (an imported product) was in violation of the MRL (CFIA, 2004).

Testing for pesticide residues is also done by the Ontario Ministry of Agriculture, Food and Rural Affairs (OMAFRA). In 2000, OMAFRA tested almost 800 domestically-produced fruit and vegetable samples and found 28 cases (3.5%) where chemicals exceeded acceptable limits, in some cases by as much as 80 times the limit. In response to concerns raised by the Provincial Auditor, OMAFRA has improved its notifications to growers and retailers about test results and has implemented a risk-based hazard evaluation system (PAO, 2003). In what appears to be the only published reporting of this additional evaluation, an investigation of pesticide violations on peaches, routine exceedances of the MRL for two pesticides were found. Commercial washing of the fruit before packaging significantly reduced the pesticide concentrations but some violations still occurred due to the high initial concentrations (Ripley, 2003).

From these data, it is difficult to know either the extent, or the implications to children's health, of these combined exposures to multiple pesticide residues on food, either at detection levels or for those found, albeit fairly rarely, in excess of MRLs. However, most MRLs are in need of re-evaluation in light of the particular exposure circumstances and greater vulnerability of children to pesticides. Nor are multiple exposures yet considered in these calculations of regulatory compliance or in revisions to MRLs. These data paint a picture of fairly strong regulatory compliance and could lead to a conclusion of very low exposure. It seems essential to combine such data with actual biomonitoring results to know whether a record of minimal exceedances of MRLs is reflective of safe or low exposure levels.

Recent biomonitoring data from Quebec and the US paints a somewhat different picture. The small study by L'Institut National de Sant  Publicque de Quebec (INSPQ) found that 98.7% of suburban Quebec children's urine samples tested indicated exposure to organophosphate (OP) insecticides. These amounts were inexplicably higher than those reported for samples of children in the U.S. (INSPQ, 2004). The researchers suggest that food may be an important source of this exposure. While none of the urinary levels was above the No Observed Adverse Effects Level (NOAEL), some did approach the NOAEL for the alkylphosphate metabolites (breakdown products of OPs). These results provide further justification for more comprehensive biomonitoring nationally and locally, to better understand the nature of children's exposure to pesticides and also indicate the prudence of reducing exposure wherever possible (INSPQ, 2004).

University of Washington researchers found a significantly lower concentration of the breakdown products from organophosphate insecticides in the urine of

children eating organic food compared to those eating a diet of conventionally grown foods. They conclude that consumption of organic produce appears to provide a direct way for parents to reduce children's exposures to organophosphate insecticides (Curl et al, 2003). Although this study has not been replicated by other researchers it provides information on the differences in exposure to pesticides that relate to dietary choices.

CFIA inspection results are also provided for meat and meat products, fish and seafood, processed foods and dairy products. Across the board, chemical, metal and pesticide residues are generally within regulatory limits.

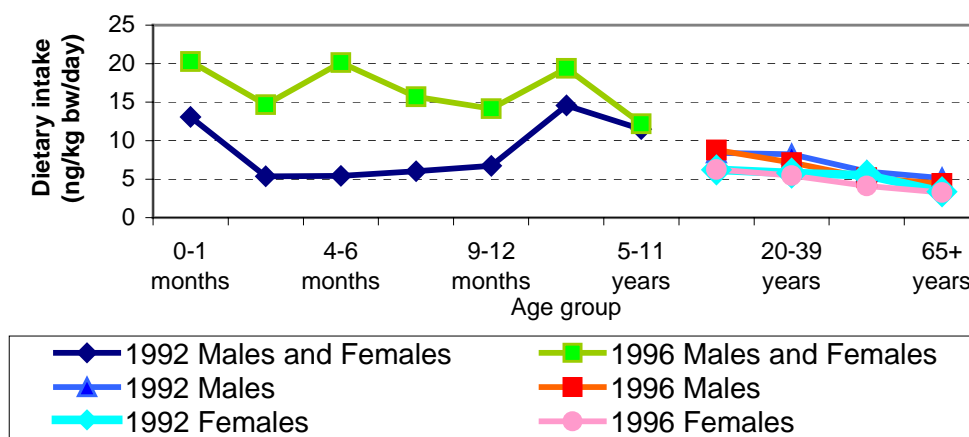
Persistent Organic Pollutants (POPs)

The most important route of exposure to Persistent Organic Pollutants (POPs) is through food. These substances circulate in the global ecosystem and due to their chemical nature and persistence, continue to build up in the fatty tissue of organisms. They are suspected or known to cause multiple health effects of particular concern to the developing fetus and young children. Safe levels of POPs are practically unattainable due to their environmental persistence and since toxicity is often associated with relatively very low doses or, depending on the effect in question such as carcinogenicity, with no discernible lower threshold.

Health Canada's Total Diet Study surveys a sampling of food items found in the average Canadian diet and includes tests for several POPs. The Total Diet Study has been an ongoing surveillance program since 1969 conducted as multi-year surveys of selected cities to estimate the levels of chemicals to which Canadians in different age-sex groups are exposed through the food supply. The fifth cycle of the program began in 2000. Although testing is done annually and major cities across the country are included, not all cities are tested every year. For example, Toronto was part of the Total Diet Study in July 1992 and January 1996 (Health Canada, Canadian Total Diet Study, on-line). Over the years these studies have provided chemical residue results for a number of metals, pesticides, PCBs, dioxins, PBDEs, phthalates and other substances. Each survey does not provide data on all of the monitored substances.

Figure 4.3 below illustrates the dietary intakes of PCBs in Toronto in 1992 and 1996 as determined by this study. Results for PCBs show pervasive contamination but also a steady downward trend in PCB levels in food.

Figure 4.3 Dietary intakes (ng/kg bw/day) of PCBs in different age-sex groups, Total Diet Study, Toronto, 1992 and 1996



Source: Health Canada 2003b and 2003c.

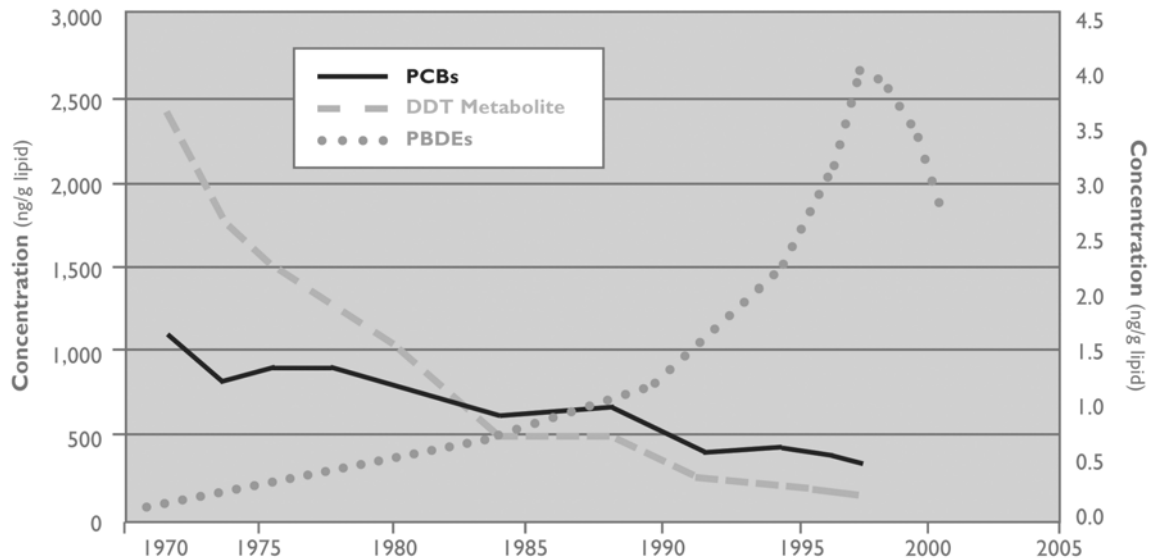
In Ontario, an OMAFRA study surveyed trace chemical contaminants in meat and milk in samples taken in the 1990s and confirmed a similar downward trend of organochlorine pesticides, and their breakdown products, often to below the detection limit. Compared to results from the 1970s and 1980s, trend lines for DDE (the DDT breakdown product) dropped steadily downward. No PCBs were detected in the meat or milk samples (Burchat and Ripley, 2003).

PCB levels in breast milk have also decreased over time since being substantially phased out in the 1970s (Craan and Haines, 1998). Though the levels of breast milk POPs have declined, earlier estimates indicated that exclusively breastfed infants may be exposed to PCBs and dioxins in amounts that approach or exceed the federal guidelines for Tolerable Daily Intake (Health Canada, 1998b; Hoover, 1999).

In contrast, PBDE levels are rising rapidly in the North American environment and in breast milk. American women have the highest levels in the world, with those for women in Canada a close second (Ryan et al, 2002; Schechter et al, 2003; NEW, 2004). In Sweden, where the most comprehensive sampling of breast milk has been conducted, it has been shown that PBDE levels in breast milk doubled every 5 years from the early 1970s until the late 1990s. The impact of Swedish regulatory action to phase-out certain PBDEs is apparent in a subsequent lowering of PBDE levels in breast milk (as reviewed in Hooper and She, 2003). These trends are illustrated in Figure 4.4. As discussed in Section 2.4, health experts

and authorities continue to uphold breast milk as the best choice for infant feeding and agree that the unclear health risks from these early exposures are still outweighed by the substantial and proven benefits to both the infant and the mother.

Figure 4.4 Trends in Chemicals in Breast Milk, Sweden



Trends in time for PCBs, DDE (declining) versus PBDEs (increasing then decreasing post-regulation) in Sweden. Source: Adapted from NRDC online and Hooper and She, 2003.

Monitoring continues to find that the substances listed under the Stockholm Convention are circulating in the Canadian environment and in food, though downward trends are discernible for banned substances, as the OMAFRA food residue data also demonstrate. Emissions from incinerators, and any uncontrolled burning of plastic, constitute an ongoing source of dioxins and furans. Recent recommendations made by a number of countries seek to add several substances to the Stockholm Convention including PBDEs, polychlorinated naphthalenes, used for cable insulation, and short-chained chlorinated paraffins used in metal working and leather finishing (EC, 2004).

Mercury

Although mercury exposures can occur in several ways, such as from spills in the home (e.g., from a broken thermometer), from certain spiritual and cultural rituals, in some work environments and from some medical uses (vaccines and amalgam fillings), generally these exposures are relatively rare or much smaller

compared to the continuing exposure from consumption of fish over a lifetime (Copes et al, 2004). As an aside, there has been concern about exposure to mercury in both vaccines and dental fillings. Health Canada states that the mercury in dental amalgam does not generally pose a risk to human health. However, the agency also recommends that pregnant women should avoid having mercury fillings removed and the primary teeth of children should be filled with non-mercury fillings. Health Canada also encourages anyone needing to replace a filling to consider non-mercury alternatives (Health Canada, 2004). Thimerosal, the mercury-containing preservative used in some vaccines, has been removed from all vaccines intended for infants and children in Canada. Thimerosal is still used in the flu vaccine but contains a less toxic form of mercury than methylmercury (Copes et al, 2004).

There are three main forms of mercury found in the environment: the metallic form, inorganic mercury salts and the organic compounds, which include methylmercury. Estimates vary but it is generally accepted that about 70% of the environmental burden of mercury is due to human activity (UNEP, 2002). In Canada, environmental emissions, mainly of various forms of elemental mercury, arise from diverse industrial settings including mining, manufacturing facilities, and electricity generation (from coal-fired power plants). The latter contributed up to 25% of Canadian mercury emissions in 2000 (Environment Canada, online). Mercury enters the environment during the use, disposal or incineration of products such as switches, batteries, fluorescent lamps, etc. and from dental and medical facilities. With the exception of coal-fired power plants, environmental mercury emissions have dropped steadily in recent years (Environment Canada, online). But, emissions remain significant, and like most persistent metals, once in the environment mercury binds to particles in air and falls out to surfaces. Mercury resulting from human activities then cycles in the environment in the same way as occurs with natural releases of mercury. Mercury is transformed through microbial action to methylmercury (organic mercury) that can then concentrate up the food chain, particularly in the aquatic food chain.

The most important and largest source of methylmercury exposure to people is from fish and shellfish consumption (Mahaffey 2004). Methylmercury content in fish varies depending on species and the size of the fish. Subpopulations in Canada consuming greater than average quantities of fish or marine mammals are more heavily exposed. This includes but is not limited to recreational anglers, Aboriginal populations, communities living in areas of concern (e.g. around the Great Lakes), and certain ethnic groups (e.g. Asian Canadians). One study found that European-Canadians and Asian-Canadians living in communities around the Great Lakes consumed on average 14 and 27 fish meals per month respectively (Cole et al 2004). In comparison, most Canadians consumes an average of about 3

fish meals per month which illustrates the wide range of potential exposures among the Canadian population (Statistics Canada, 2004).

Exposure to methylmercury is of special concern during pregnancy. Recent reviews conclude that *in utero* exposure to methylmercury among populations that consume fish and marine-mammals has been associated with harmful neurodevelopmental effects on the young (NRC 2000a; Stern et al, 2004). The U.S. EPA estimated that 6% of women of child-bearing age in the United States are exposed to mercury from fish at levels in excess of the intake considered acceptable (Jones et al, 2004). Comparable data collection or related estimates have not been done in Canada. Minimizing exposure to methyl mercury in fish is important for children and women in their childbearing years.

The *Guide to Eating Ontario Sport Fish* provides detailed information (with biannual updates) about sampling results for a range of different contaminants found in fish from lakes throughout the province (MNR, on-line). Fish consumption advisories have generally been prompted by concerns about a handful of contaminants, and most of these have been related to methylmercury content. The Guide provides sport fish consumption advice based on species, size, and location (water body) of fish. The Guide further recommends specific limits on fish consumption by women of child-bearing age and children under 15 to reduce exposure to methylmercury.

There are also several commercial fish for which health authorities issue consumption advisories to women of child-bearing age, pregnant and nursing mothers and children. Advisories for these sub-populations differ slightly between the US and Canada. The US advisories are more conservative and recommend not eating shark, swordfish, King mackerel, or tilefish (USDHHS and USEPA, 2004). They also recommend limiting fish consumption to two meals a week (up to 12 ounces or about 350 grams) of those seafood species that are low in mercury such as shrimp, canned light tuna, salmon, pollock and catfish. Recent changes in US advisories recommend limiting consumption of canned albacore or “white” tuna and tuna steaks (fresh or frozen) to one meal or six ounces (about 170 grams) per month (US FDA and EPA 2004). Health Canada and the Canadian Food Inspection Agency currently recommend limiting the consumption of shark, swordfish, and fresh and frozen (but not canned) tuna to no more than one meal per month (Health Canada – online 2002). Canadian agencies are currently conducting a review of the data on methylmercury in fish (Bueckert, G & M, 2004).

There is some controversy about recommendations on eating canned tuna. Although canned tuna does not exceed the Health Canada guidelines on average, because it is affordable and widely available (including in local food banks), it

can contribute substantially to mercury intake for the average person, particularly for children. This is especially so if people frequently or preferentially consume canned white (that is, albacore) tuna, which is relatively higher in mercury content compared to light canned tuna.

In setting guidelines great care is taken by regulatory bodies to balance the risks of mercury with the nutritional benefits of fish and shellfish. These food sources are high in protein and contain omega-3 fatty acids, which have been associated with benefits to the developing nervous system among other health benefits (Mahaffey 2004). At this stage the evidence is not clear about how much of the essential fatty acids supplied by fish are needed for optimal neural development. Nonetheless, while ensuring that pregnant and lactating women and infants are not overly exposed to methylmercury, fish consumption advisories and recommendations must take both sides of the issue into account. Toronto Public Health is currently investigating the issue of amounts, portions and species of fish that can be safely consumed to minimize exposure to methylmercury among sensitive sub-populations.

4.3.13 Contaminants in Drinking Water

Lead can enter drinking water from lead service lines, from solder containing lead or from brass fixtures. Older plumbing, in homes built prior to the 1950s may include lead pipes. Generally the lead plumbing that remains in these buildings is the municipal supply line from the street. Municipalities, including Toronto, are gradually replacing these older supply lines but some remain. Newer homes will have primarily copper pipes joined with lead-based solder. The use of lead solder for incoming water pipes was banned in Ontario in 1989 (MTTHU and SRHC, 1995). In homes with lead-soldered copper pipes, mineral build-up in the pipes over time helps to make the lead less available. An exception to this tendency towards mineral build-up will be under conditions of acidic water. Acidic or 'soft' water will both reduce the mineral build-up and make the lead in the solder more likely to leach into the tap water. Toronto's water tends to be hard or non-acidic unlike areas with water of naturally high acidity such as on the Canadian Shield. The latter circumstance is an issue for some summer cottagers. In addition, some brass plumbing fixtures (faucets and taps) can contain lead. The risk of lead exposure can be minimized so long as flushing occurs to avoid consumption of water left standing in either solder-based plumbing or plumbing that includes brass fixtures.

Both the Toronto District School Board and the Toronto Catholic District School Board have a practice of flushing drinking fountain and water taps on a regular basis to reduce lead in drinking/washing water. For example, the practice at

TCDSB is to recommend daily flushing of all water sources, particularly drinking fountains (for 20 minutes), sinks, showers and other taps (for 10 minutes).

Another category of chemical contaminant of concern to child health in drinking water is created as a result of the disinfection process or the use of chlorine in cleaning products. Called disinfection by-products (DBP), these are substances created by the reaction of chlorine with naturally-occurring organic material in raw water, either from groundwater or surface water supplies. Trihalomethanes (THMs), such as chloroform (a suspected carcinogen), are a subset of DBPs. THMs are the more commonly known contaminants resulting from drinking water disinfection.

Generally, the higher the level of organic matter in the source water, the higher will be the resulting level of DBPs in the treated water. Toronto's water source, Lake Ontario, has a relatively low level of organic material and hence low levels of DBPs. In comparison, communities where the water supply is from rivers, especially those that are shallow or that flow through areas of heavy agricultural activity, will often be treating water with high levels of organic material. Trihalomethanes (THMs) a type of DBPs have been decreasing in Toronto's water and are well below the interim Maximum Allowable Concentrations (MAC) for THMs (100 µg/L) in drinking water according to the Canada Drinking Water Guidelines. The average THM concentration in Toronto's drinking water over the last ten years has been below 20 µg/L and 1999 it was 11.5 µg/L (TPH, 2001b).

DBPs can also be created in a dishwasher from the reaction of chlorine-containing dishwashing detergents and the organic matter in the food being washed off the dishes. DBPs are thus released to indoor air in the steam from the dishwasher. DBPs in tap water are similarly released in the steam during showering. Exposure during showering or bathing can occur via inhalation, dermal absorption and perhaps ingestion (Health Canada, 1998). Swimming pools, especially indoors, can also be a significant exposure source, via inhalation and dermal absorption, of DBPs (ATSDR, 2003).

Although definitive conclusions are not possible, there is some moderate evidence that certain DBPs are associated with increased risks of spontaneous abortion, low birth weight and some birth defects (such as neural tube defects) in humans (as reviewed in Wigle, 2003). Cancer is also of concern with some evidence linking DBPs to childhood cancer and some additional cancers in adults (Wigle, 2003). McGill University researchers have preliminary data to suggest that individual genetic variation plays some role in susceptibility to effects from exposure to DBPs (See e.g. Infante-Rivard 2004; Infante-Rivard et al, 2002).

Additional chemical contaminants such as pesticides, metals and industrial chemicals can occur in drinking water. Toronto's water typically contains quite low or non-detectable levels of such substances often because those of greatest concern tend to be hydrophobic and either bind with particles or are lipophilic (tending to bind to fatty molecules) or both. Traditional municipal water filtration methods, or secondary filtration, cannot filter chemical residues. Tertiary methods that employ carbon filtration or other advanced methods are necessary. Alternatively, point-of-use filters on in-home drinking water taps can apply tertiary filtration methods. Considerable care is needed to change such filters since they can trap bacteria and, if not changed regularly, can secondarily contaminate drinking water. Insufficient maintenance and changing of filters can also result in the accumulated contaminant load in the filter being released into the water. The result can be a potentially very large exposure "dose" if such pockets of contaminants break through the filter.

Toronto Public Health conducted a detailed review of bottled water in the late 1980s and found that both aluminum and lead were lower in bottled water than tap water and THMs were not present in bottled water (Toronto, 1990). That said, Toronto's tap water has consistently been found to contain low levels of such contaminants but it is also clear that water filters bring levels down further (so long as filters are properly maintained). Survey results (explored in more detail in Chapter Five) also indicate that a large percentage of the Toronto parents choose alternative water sources. Nearly 60% of parents reported using either filtered or bottled water for their children.

Finally, drinking water can sometimes be contaminated with infectious microbes including bacteria (e.g., *E. coli*) protozoa (e.g., cryptosporidium or giardia) and viruses (e.g., rotavirus or hepatitis A and E) all of which can be a serious concern to child health. Since all Toronto residents can obtain treated drinking water, these contaminants are not relevant with respect to drinking water sources in Toronto and were not included in the scope of this review.

4.3.14 Consumer Products

Within the vast uncertainty that exists about the effect and circumstances of chemical exposures, recent investigations have chosen to look indoors for evidence of exposure to substances that are already known or suspected of health impacts in the literature on environmental contaminants. As discussed in Section 4.3.9, a great deal of information exists about indoor air pollution sources, including indoor combustion, ETS and some cleaning products and solvents.

Indoor exposures may also originate from a range of consumer products that contain substances of emerging or increasing concern. Many different chemical substances in products often confer some useful property within or related to the product. For example, substances like PBDEs are incorporated into consumer products in high volumes to prevent fire and save lives. In some products like foam or computer casings, PBDEs make up 30% or more by weight of the product. PBDEs, or other such substances, then turn up as contaminants in indoor dust. Because of their ubiquitous use in products, exposure sources in the home and workplace and other indoor environments are pervasive. Toronto researchers recently published data on estimates of exposure to PBDEs through indoor and outdoor air, soil, dust and food and concluded that unintended ingestion of indoor house dust can account for 90% of a toddler's exposure to PBDEs and is the major exposure pathway for all life stages except infancy (Jones-Otazo et al, 2005). Infants currently receive their greatest exposure from breast milk.

People are widely exposed to Bisphenol A which is used in the manufacture of polycarbonate plastics, resins, dyes, flame retardants, dental sealants and is found in many products such as food and pop cans (the protective inner lining) and hard plastic food and drink containers (including baby bottles). Repeated washings of these products or contact with heat, acidic or basic substances, all increase the rate at which Bisphenol A leaches. Consequently, exposure is believed to be continuous and occurs through multiple sources. US CDC scientists recently showed that 95% of people sampled had exposure to BPA at levels that approach or exceed those causing adverse effects in animal and *in vitro* laboratory studies (Calafat et al, 2005).

A number of other substances found in consumer products are causing scrutiny of the indoor environment. For example, concern also exists about exposure to artificial musks in air fresheners and to phthalates used as softeners in PVC plastics such as toys and vinyl flooring. Phthalates are also used in some cosmetics such as hair sprays and nail polishes. Organotins are used in carpet linings, vinyl flooring, PVC gloves and some shoe insoles. Alkylphenols and alkylphenol ethoxylates are used in detergents, paints, glues, lubricating oils, and some shampoos and shaving foams. Finally, chlorinated paraffins are used in some plastics, paints and rubbers.

For many of these substances there is concern about persistence and bioaccumulation. As well, laboratory research indicates effects on animal development or reproduction that may result from endocrine-mediated alterations with exposure to these substances. Overall, there is not enough evidence to understand the potential for harm in humans. However, it does appear that the exposure to chemicals in the environment is substantially influenced by consumer product choices and indoor exposures.

It has been known for some time that contaminants such as PAHs, lead, and PCBs are often found at higher concentrations in house dust than in soil near the foundation or on home walkways (Roberts and Dickey, 1995).

A study of lead and other metals in the indoor dust and outdoor soil of Ottawa homes found lead at much higher levels indoors than outside (Rasmussen and Subramanian et al, 2001). The levels of lead (and the other metals) appeared to correlate to indoor rather than outdoor sources. Paint (interior, but particularly exterior) manufactured before 1976 contained high levels of lead. The Canadian regulation put in place in 1976, allowed for 5000 parts per million of lead, now considered to be a dangerously high level. This level was only recently revised to 600 parts per million, the same level established in the US in 1976 (Canada Gazette, 2005). Fortunately, the paint industry standard for North America has followed the lower regulated limit from the US since 1976.

Lead in outdoor or household dust is likely the most important route of lead exposure in young children, especially those exhibiting pica (a tendency to eat soil or other non-food items) and frequent hand-to-mouth behaviour (Roberts and Dickey, 1995, Calabrese et al 1997). There is empirical evidence indicating that lead levels in indoor dust also correlate to blood lead levels in toddlers (Rhoads et al, 1999). When outdoor dust sources are tracked indoors accumulation can occur on plush surfaces, most dramatically on older carpeting. For example, it has been estimated that the amount of dust in each square metre of an older carpet may be 400 times greater than levels found on a bare floor in the same house (Roberts et al, 1987).

The second of two major clean-up operations in Toronto communities affected by lead contamination from secondary lead smelters (See Section 4.3.8) included clean-up of indoor house dust. Monitoring of children's blood-lead levels occurred for most of the 1980s and into the early 1990s in one of these communities, South Riverdale. The combination of clean-up activities (both targeted soil replacement and professional housecleaning services) and community awareness resulted in a more accelerated decline in blood-lead levels in South Riverdale compared to control samples and by 1992, South Riverdale blood-lead levels were no longer significantly different from the control group located elsewhere in the city. These results reveal the value of the educational efforts in the community and the impact that personal measures can have on reducing in-home exposures (Langlois et al, 1996).

Recent studies looking at indoor dust in the US, UK and other European countries (Santillo et al, 2003; Rudel et al, 2003; Sharp and Lunder, 2004) have found unexpectedly high levels of PBDEs and other substances that are originating from

consumer products. Since PBDEs are semi-volatile at room temperature, concern also exists about their presence in ambient indoor air.

Throughout the 1990s a steady stream of consumer products have been found to contain high levels of lead including crayons from China, painted zippers on some children's clothing, plastic mini-blinds, some toy figurines and some candle wicks. Most recently, lead has been commonly found at extremely high levels (ranging from 10% to 50% pure lead) in costume jewellery not intended for children. This distinction is largely irrelevant as these products are widely available, very inexpensive and attractive to children. Almost without exception, these products have been imported from developing countries where rules for occupational and environmental health are weak or do not exist (Cooper et al, 2000).

Regulatory response in Canada to the problem of lead in jewellery has been slow and has exempted one of the largest sources of the problem. After eight years of discussion, regulations that were promulgated on June 1, 2005 apply only to "jewellery that is produced, sized, decorated, packaged, advertised or sold in a manner that appeals to a child under 15 years of age." This regulation, and its (draft) enforcement guideline will not apply to costume jewellery. Such jewellery has been widely available in stores across Canada for several years but it does not fit within this artificial distinction of being "intended for children." Moreover, the exemption of costume jewellery will mean that women of child-bearing age and mothers of young children will still be able to buy and wear leaded jewellery which may then be easily accessible to their children.

Indoor air or dust is not subject to traditional environmental regulation and there are no legal requirements in Canada to provide information about the use or levels of most of these substances on product labels.

4.4 Multimedia Exposures

Many substances are found in more than one medium. Measurement data for all of these media need to be aggregated to obtain a true picture of total exposure. This overall picture of the environmental sources and fate of different substances can also be useful to understanding which exposure sources are of greatest concern and possible strategies to reduce total exposure.

Lead as a Multimedia Exposure

Lead remains a major contaminant of concern for children and lack of awareness can create greater exposure. As discussed throughout this chapter, people can be exposed to lead via soil, household dust, food, drinking water and air. Since the phase-out of lead in gasoline and many industrial sources, levels of lead in Toronto's air are negligible. Also levels of lead in food have decreased with the voluntary reduction of lead solder in can seams that began in 1988 and with improvements to food processing (MTTHU & SRCHC, 1995). This has resulted in an overall reduction in the dietary intake of lead (MOEE, 1993). Use of leaded crystal or lead-glazed ceramics for food serving or storage can still contribute to significant exposure to lead. In the US there have been efforts to address lead exposure from candies imported from Mexico.

As noted with respect to outdoor exposures, outdoor soil and dust can be a reservoir of lead from current or historical deposition due to industrial activities, from past use of leaded gasoline as well as weathering of old outdoor paint, all of which can become sources of lead that can be tracked indoors (Cooper, et al 2000; Mickle, 1998). It has been shown that metals like lead will accumulate indoors (Rasmussen and Subramanian et al, 2001).

The main sources of lead exposure for children in Canada are via indoor dust (from multiple sources) and drinking water from some older plumbing (Rasmussen and Subramanian et al, 2001; MTTHU & SRCHC, 1995). Unregulated levels of lead in consumer products (for example, PVC plastic mini-blinds and some metal costume jewellery) can also create significant lead exposures (as reviewed in Cooper et al, 2000).

A Toronto study conducted in 1996 revealed lack of awareness about lead exposure risks. As illustrated in Figure 4.5, many respondents incorrectly assume that lead exposure occurred primarily from drinking water, and many were unaware of an important exposure pathway, lead in dust (Campbell and Lee-Han, 1996; Rasmussen and Subramanian et al, 2001). Some empirical evidence suggests that children's blood lead levels can be affected by lead in indoor dust and regular dust control measures combined with education efforts will at least partially reduce blood lead levels in children (Rhoads et al, 1999).

Figure 4.5 Perceived Major Sources of Lead Exposure for Children

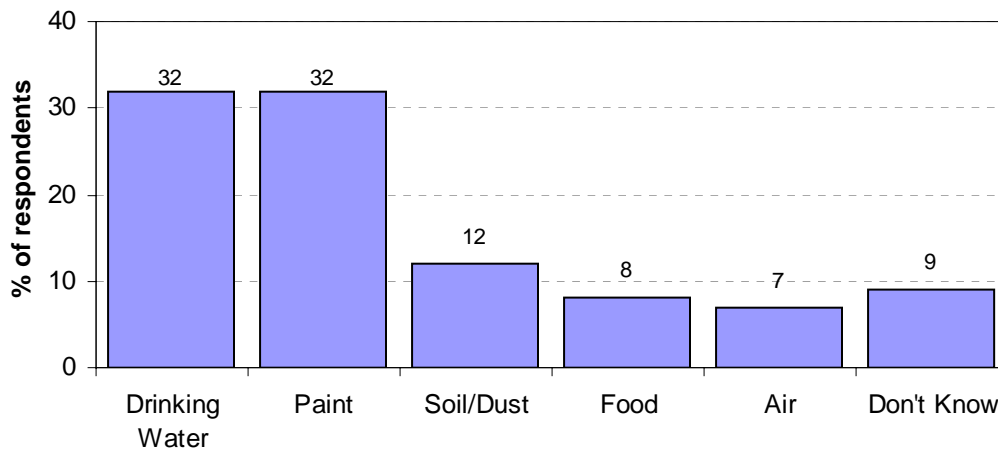


Figure shows low awareness for soil/dust exposure pathway

Source: Campbell and Lee-Han 1996

Although data are not current, the majority of children in Canada likely have blood-lead levels below the “level of concern” that is, 0.48 micromoles (μmol)/L or 10 micrograms (μg)/dL as established by the US CDC in 1991. Canada does not have routine blood-lead screening, however data from periodic testing of children in Ontario suggests the average blood-lead level has a “floor” of about 2-3 $\mu\text{g}/\text{dl}$ below which the values for urban Ontario children under the age of 6 years do not fall (Langlois et al, 1996). However, as discussed in Section 3.2.3, it is increasingly agreed among experts on lead that there is no real threshold for its neurotoxic effects, as they can be demonstrated down to blood-lead levels of 5 $\mu\text{g}/\text{dL}$ (0.24 $\mu\text{mol}/\text{L}$) or lower (Canfield et al, 2003).

With the concerns raised surrounding lead exposure in children in this chapter and Chapter 3, several important needs can be identified. There is a need for more current information on the blood lead levels of children in Canada. There is also a need to increase education and awareness of ways to minimize exposure to children that focus on practices in the home (Rasmussen and Subramanian et al., 2001).

Pesticides as a Multimedia Exposure

The preceding discussions highlight that children’s exposure to pesticides also occurs through more than one pathway and through different routes. Pesticide

exposure can occur in house dust, in water or in food, depending on its original use and specific chemical characteristics. A child's breathing zone is much lower than an adult's. Research has found that some pesticides settle closer to the ground and are in much higher concentrations in an infant's breathing zone (Fenske et al, 1990). A child's hands may pick up a pesticide residue via direct contact with treated surfaces or as tracked-in (on shoes, pets, wheels) contamination in homes, schools or recreational facilities.

When parents who work with pesticides do not take proper precautions, pesticides can be brought into the home or they may be absorbed by the parent, potentially leading to effects in their offspring.

Pesticides found in soil can be washed into surface waters by run-off of rain and snowmelt and may contaminate ground water. While not an issue for children in Toronto, pesticide contamination of ground and well water is not uncommon in agricultural areas of Canada. Depending on the water source used for infants or children, there may be exposure to pesticides from these drinking water supplies.

Children may ingest minute amounts of pesticides via the residues that remain in fruits and vegetables (NRC, 1993, Neidert and Havelock, 1998), as discussed earlier. While CFIA data suggest that exceedances of Maximum Residue Limits are low, many of these regulatory limits require re-evaluation to ensure allowable limits are appropriate for protecting children. In addition, although we have only limited Canadian data, results from population biomonitoring studies indicate that despite strong regulatory compliance, children are still widely exposed to a number of pesticides used in agriculture (INSPQ, 2004; CDC, 2003, 2005).

In Toronto, public opinion surveys of the general population and of parents indicate there is high awareness of the risks to children's health from exposure to pesticides and that parents alter their pesticide use practices appropriately in order to protect their children.

The 2002 survey of parents with children aged 0 to 12 indicated that the majority (62%) of parents felt that it was "very harmful" for children to come into contact with pesticides used on lawns and gardens. Figures for outdoor pesticide use can be compared to those for the general population suggest that parents in Toronto tend to avoid outdoor pesticide use for the sake of reducing their children's exposures (TPH, 2004c, 2004d, 2002e). As well, only 17% of parents reported using pesticides indoors. Toronto Public Health has been very active in its efforts to shift public awareness and knowledge through education and resources. This outreach activity combined with the regulatory backing of the Toronto pesticide by-law may further reduce reliance on outdoor use pesticides and shift behaviour towards the use of non-toxic alternative methods. Preliminary analysis of survey

data on self-reported use of outdoor pesticides gathered by TPH since the pesticide by-law came into effect suggests that exposure will continue to fall with phase-in of the by-law as people in Toronto move towards the use of permitted products. Limited research indicates that regulation to reduce pesticide exposure can lessen health impacts in children. For example, an ongoing prospective study by Columbia University researchers found that after federal government restriction to limit home uses of chlorpyrifos and diazinon in 2000, there were lower exposures and no longer an impact on fetal growth compared to before the government restrictions (Perera et al, 2003; Whyatt et al, 2004).

A small but not insubstantial proportion (20%) of Toronto parents also reported that *all* or most of the food (vegetables, fruits, dairy products and meat) they purchased for their child was organic (TPH, 2002c). To date, only one study has examined the relationship between dietary choices and pesticide exposure, showing that exposures to organophosphate pesticides in children on diets of organic foods were significantly lower than those on diets of conventionally grown produce (Curl et al, 2003). This study concluded that consumption of organic produce appears to provide a direct way for parents to reduce children's exposures to organophosphate insecticides.

Similar to lead, the other multimedia contaminant discussed here, research that continues to explore the pathways of exposure to pesticides for children supports efforts (both through education and regulation) to shift awareness and behaviour such that children are less exposed.

CHAPTER FIVE – WHAT DO TORONTO PARENTS UNDERSTAND ABOUT ENVIRONMENTAL IMPACTS ON CHILDREN’S HEALTH?

5.1 Introduction

The goal of safeguarding children’s health is well served by raising awareness of the issues and allowing parents and caregivers to make informed decisions. Public health staff are well placed to play a leading role in educating and working with those who influence the health and well-being of children including parents, caregivers, health professionals, educators and policymakers (Campbell and Shulman, 1998). In September 2000, Toronto Public Health embarked on a needs assessment to strengthen the child health focus in its work (2002a).

Public opinion surveys indicate that people generally have great concern for the effects of environmental factors on child health. Globally, the majority of people feel that the health of future generations will be affected a great deal by environmental problems (Enviroics, 1999; Globescan, 2003). Better than 90% of health, environmental and education professionals and organizations surveyed in Canada agree that children are vulnerable to environmental contaminants (CICH, 1998). A similar proportion of the Canadian public is worried that environmental hazards influence their children’s health (Ekos, 2000 as cited in NRTEE, 2001). National and international surveys reveal that people have a strong sense that children’s health can be affected by environmental conditions. Canadians also appear to appreciate how some specific environmental conditions (e.g. poor air quality) are likely to affect their own child’s health. However, it is not clear how specific that awareness is, or whether awareness translates into people taking effective protective measures to avoid harmful exposures to children. Determining what Toronto parents know and may need to know was identified as an important action.

Toronto Public Health staff therefore undertook to assess public knowledge, practices and beliefs surrounding the environmental risk factors that influence children’s health. A public survey is an expedient and reliable way to determine information needs among a representative sample of Toronto’s parents. Although there have been a few surveys assessing knowledge among professionals or organizations (e.g. in the U. S. see Chai et al, 2001; in Canada, see CICH, 1998) to date, there has been rather limited research, in either Canada or the United States, to assess public awareness of the array of environmental threats to children’s health. Little information exists as well to identify the behaviours and practices of parents that can increase or mitigate adverse exposures. The latter

information is particularly important in assisting public health program planning and health promotion activities.

The specific aims of the Toronto parents/caregivers survey were:

- Collecting empirical, baseline data for a representative, random sample of Toronto parents;
- Describing and measuring the attitudes and perceptions of parents to environmental risks on children's health;
- Assessing behaviours and practices that impact on children's risk of environmental exposures;
- Observing associations between respondent characteristics and knowledge base or practices; and
- Generating hypotheses and identifying areas of concern to be targeted by future activities of Toronto Public Health.

5.2 Methods

It is generally preferable to use a pre-tested, established instrument, given that designing an effective survey tool is a considerable challenge. Therefore, a search for appropriate existing tools was conducted via Internet searches and a broadcast e-mail message through the U.S. Children's Environmental Health Network listserv. This search uncovered several examples of environmental health and safety assessment surveys used in other communities or public health jurisdictions in the U.S. While most of these surveys were in some way instructive and provided example question material, none was entirely suited to the specific aims of the Needs Assessment Study. It was necessary therefore to develop a unique quantitative survey instrument.

Content for the questionnaire was informed by consulting recent scientific literature reviews on children's environmental health and via key informant interviews (See TPH, 2002c). These consultations indicated that a microenvironment or settings approach is important and particularly, that factors of indoor environments and indoor air quality needed greatest attention. As a result, the survey questions focused largely on indoor and home exposures and was restricted to topics for which there is at least probable (or strongly suggestive) scientific evidence of an association with health effects in children. Specific indoor environmental factors probed for parental awareness were mould, pesticides, cleaning products, pet allergens, dust, woodsmoke, elements in food, lead in water and paint and environmental tobacco smoke. In addition, the survey asked about practices to protect children from indoor exposures, such as cleaning, shoes-off policy and so on. One section also explored awareness and practices

around food and water safety. Because Toronto Public Health has extensive programs related to smoking, sun safety and smog alerts, the survey probed for household practices in these areas as well.

The tool was extensively modified and refined, drawing on feedback of a multi-stakeholder panel of project advisors. A professional survey firm (Smaller World Communications - SWC) helped further refine the instrument to ensure it was of high quality. SWC researchers assessed individual questions and the overall tool for five factors, including, necessity of questions, wording and language level, neutrality of language, consistency of response categories and logical flow. The questionnaire was pilot tested on a random sample of respondents. (The final questionnaire can be found at www.toronto.ca/health).

The target sample was parents or primary caregivers of children from birth to age twelve inclusive. It was not feasible to address the range of issues for other important stages in child development, such as the prenatal period and adolescence, because the questions posed would need to differ substantially.

The survey was conducted in English only, using random digit dialling and an advanced Computer Assisted Telephone Interviewing (CATI) system. The pilot version of the tool was comprised of 21 closed-ended and three open-end questions keeping the interview length to about 10 minutes per respondent.

The pilot test showed a very low incidence (~35%) of the target population in Toronto with completely random sampling. Therefore, a modified random sampling strategy was taken to maximize chances of reaching the target audience. This strategy focussed on randomly selecting respondents from the 50 (out of 121) Forward Sorting Areas (FSAs) in Toronto that had the highest ratio of children to adults according to the 1996 Census data. The 50 FSAs were geographically distributed across the City suggesting, although not proving, that the survey findings may be generalized to the entire population of Toronto parents of children 0 to 12 years old.

The survey was run in February 2002 and interviews were completed with 452 respondents. The sampling error for a survey of 452 residents is three to five percent 95 times out of 100. This allowed adequate confidence that the sample should be representative of most parents of young children in Toronto.

Aside from generating frequency counts and percentages for categories of response, cross tabulations of data were also performed to determine if two variables were related (for example, if reported use of pesticides is related to education). Chi-square analyses were used to test the strength of the relationship between two related variables. A significance level of $p \leq 0.05$ or less was used as

a criterion to determine the strength of the relationship between two variables. Only statistically significant findings are reported in the results section. Verbatim comments from open-ended questions were analyzed for themes.

5.3 Results

The survey response rate (75%) was higher than for others conducted on behalf of Toronto Public Health in recent years. For example, the average response rate for the telephone-administered Rapid Risk Factor Surveillance System (RRFSS) monthly survey is 56% (J. Phillips, TPH, HI&P, pers comm., February 2005). Closest in comparison is the Toronto Perinatal and Child Health Survey which had a response rate of 66.63% (W. Ng, TPH, HI&P, pers comm., February 2005). Historically, response rates to Canadian health surveys (national and provincial) have been between 70 and 100% (Choi, 2004). Therefore the response rate for this survey was high compared to other TPH sponsored surveys, but comparable to other large-scale national or provincial health surveys. Among the factors that affect response rates to telephone surveys are sponsorship, purpose and importance of the information being gathered, nature of the population sampled, survey length and confidentiality issues (Hox & de Leeuw, 1994). Most of these issues and concerns are explicitly addressed in the introductory script, as was the case for this survey. The better than expected response rate for this survey is likely explained then by the non-random nature of the target sample (i.e. parents of young children) and the respondents' perceptions of the importance and legitimacy of both the sponsoring agency and the information being gathered.

5.3.1 Sample Characteristics

Two-thirds of the sample respondents were female ($n = 300$) and one-third were male ($n = 152$). This skewed sex ratio may reflect a gender bias in either willingness or availability (depending on when calls were made) to participate in a telephone survey. Another strong possibility is that knowledge pertaining to children's health likely more often resides with the female parent. Nearly 90% were between 25 and 44 years of age ($n = 376$). Compared to available 2001 census data for parents of children 0 to 6, there are only small differences in the age make-up of respondents to this survey although the age categories available for comparison are quite broad. The age distribution of survey respondents is provided in Table 5.1.

Table 5.1. Age of Survey Respondents

Age Category (Years)	Survey Responses (Parents of children 0-12 years) %	2001 Census (Parents of children 0-6 years) %	Difference
18 to 24	4.4	4.3	+0.1
25- 34	35.6	38.0	-2.4
35- 44	47.6	58.1	+1.9
45 – 54	12.2		
55 and up	0.2		
Total	100	100	

Source: Toronto Public Health, 2002b and Statistics Canada, Census 2001

As seen in Table 5.2 below, an almost equal percentage of respondents reported that their youngest child was between 18 months and 5 years (38.3%) and 5 to 12 years of age (39.4%). Twenty-two percent (22.3%) of respondents had a youngest child less than 18 months.

Table 5.2. Age of respondents' youngest child

Age of Youngest Child	Percent (%) (n = 452)
0 to 18 months	22.3
18 months to 5 years	38.3
Over 5 years	39.4
Total	100

Source: Toronto Public Health, 2002b

More than half (59%, n = 267) of all respondents had a university, college or postgraduate degree and only 6% (n = 29) did not have a high school certificate.

Compared to 2001 Census data for the City 38% of parents (with children 6 years or younger) have a post-secondary certificate or diploma, but 27% have less than a secondary education. Therefore, although the age range of children from the available comparison census data is smaller, this parent/caregiver sample appears to have a higher level of education. An almost equal percentage of respondents indicated their total household income before taxes to be under \$40,000 (32%) and between \$40,000 and \$80,000 (35.6%). Over twenty percent (21%) of respondents reported their household income as being more than \$80,000. Fifty-four respondents (12%) did not disclose their total household income. The average household income for Toronto from 2001 census data is \$69,194. For families with children age 14 and younger, the comparable statistics are provided in Table 5.3. The available 2001 census data for this variable includes household income for parents of children with a slightly wider age range (0 to 14 years) than for the respondent sample here. It appears that the current survey sample, while comparable in representation for the bulk of the income categories (that is, from over \$20,000 to under \$100,000), it is underrepresented for parents in both the lowest and highest income categories.

Table 5.3 Reported Total Household Income of Respondents

Reported Household Annual Income	Survey Responses (Parents of children 0-12 years) %	2001 Census (Parents of children 0-14 years) %	Difference
< \$20,000	9.2	18.6	-9.4
\$20,000 to \$39,999	22.3	22.3	0
\$40,000 to \$59,999	19.9	19.8	+0.1
\$60,000 to \$79,999	15.7	13.6	+2.1
\$80,000 to \$100,000	9.0	8.3	+0.7
\$100,000 and up	11.7	17.4	-13.4
Missing	11.9	NA	
Total	100.0	100.00	

Source: Toronto Public Health, 2002b and Statistics Canada, Census 2001

5.3.2 Parents' Attitudes and Beliefs

Parents were asked to comment on how harmful (from “very”, “somewhat” to “not at all harmful”) they felt it was for children to come into contact with a series of items listed by the interviewer as follows:

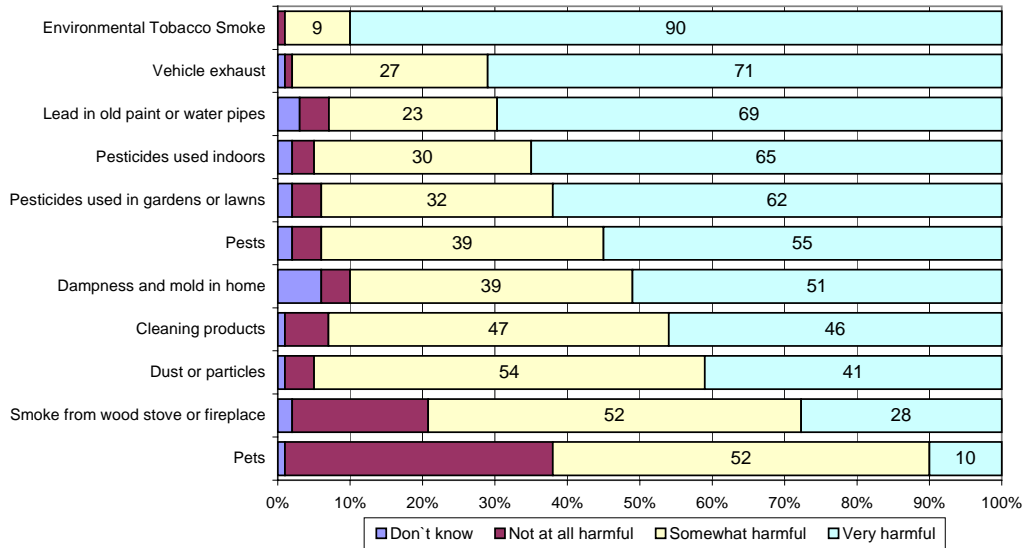
- “Second hand smoke” from cigarettes or tobacco;
- Smoke from a wood stove or wood-burning fireplace;
- Common household cleaning products;
- Dampness and mould in the home;
- Dust or particles from renovations or older homes;
- Lead in old paint and water pipes;
- Pets like dogs, cats or birds;
- Pests such as cockroaches and mites;
- Pesticides used indoors for killing insects; and
- Pesticides used in the garden or lawn.

Environmental tobacco smoke (ETS) (or the less technical name, “second-hand smoke” as it was termed in the survey questionnaire) was recognized as “very harmful” by nearly all parents. Overall, 90% of respondents felt that it was “very” harmful and an additional 9% felt it was “somewhat” harmful for their child to come into contact with ETS.

The percentage of respondents who felt it was “very” harmful for their child to come into contact with exhaust from vehicles, lead in old paint or water pipes, pesticides used indoors, pesticides used in gardens or lawns, pests, dampness and mould in the home, cleaning products, and dust or particles ranged from 41 to 71%. These results indicated that there is generally good awareness of the potential for harm from common environmental agents.

In contrast, there was lower awareness for lesser-known environmental exposures, such as wood stove or fireplace smoke which affects indoor air quality and is a risk factor for asthma or respiratory problems (USEPA, 1995; AAP, 2003). Only 28% of respondents felt that it was “very” harmful, and an additional 52% felt it was “somewhat” harmful for their child to come into contact with smoke from a wood stove or fireplace. Although some responses varied according to household income, no statistically significant differences were found across age of respondents, age of youngest child, and education in this question. Figure 5.1 summarizes the range of responses for this set of questions probing awareness.

Figure 5.1 Parents Perceptions of Harm to Children from Select Environmental Elements



Responses to the question: "How harmful is it for children to come in contact with these items?"

Source: Toronto Public Health 2002b

Respondents were also asked unprompted to name three things in the environment in Toronto they felt were most harmful to children’s health. Table 5.4 presents summary data for the 12 items most frequently mentioned in response to this open-ended question.

Table 5.4 What Things in the Environment may be Harmful to Children's Health?

Comments by Main Themes	Percent (%) of respondents mentioning*
Air pollutants	76.8
Bacteria & micro-organisms in water	25.4
Pesticides, chemicals & minerals running off into the water	18.1
Quality of drinking water	13.6
Pesticides & chemicals in food	10.3
Chemicals & pesticides used in garden or on lawn	8.2

*Percent calculated as n of mentions over total respondents for each theme (since respondents were allowed three mentions), therefore, percents do not add up to 100.

Source: Toronto Public Health, 2002b

Air pollution was identified most frequently (by 77% of respondents) as the main environmental exposure that can be harmful to children followed by water quality (~57%), which included concerns about biological organisms and chemicals such as pesticides in water. Concerns about pesticides (in water, food and on lawns and gardens) were mentioned by just over a third of respondents (35%).

Parents' top of mind responses reflected high awareness for environmental impacts that are high profile, highly visible or are well covered in the media or in public education activities. For example, the mention of air pollutants was not unexpected given that this is a priority focus of Toronto Public Health and that the summer of 2001 had the highest number of smog advisories since statistics were recorded. As well, although drinking water quality in Toronto is very good, it was not surprising that micro-organisms in water were mentioned with second greatest frequency since the Walkerton tragedy, having occurred less than two years before the survey was conducted, was likely still fresh in peoples' minds. Pesticides may have been top of mind because the City's public consultation about reducing outdoor pesticide use was underway and garnering media attention at that time as well.

The different information coming from open versus closed question types is illustrated well by a comparison of responses to these first two questions assessing awareness. The high agreement that ETS is very harmful to children contrasts with the low unprompted number of mentions (n = 17) for ETS as a harmful

environmental substance for children in Toronto for the open-ended question. This unexpectedly low number of unprompted mentions may be explained by the public's interpretation of the word "environment", i.e. that the public may not include the built environment in that understanding. Indeed, in the question prompting, interviewers mentioned, "by environment, I mean the air, water, soil, food, etc." As well, it is important to note that this sample reported low rates of smoking among householders, although clearly they recognize ETS exposure as harmful to children's health in general. There is of course substantial health promotion work centred on limiting exposure to ETS, as well as ongoing work by the City of Toronto to regulate smoking in public places.

When asked how much they could do "to protect their child from things in the environment", about 80% of the respondents felt they could do "a great deal" or "a moderate amount". Only one percent of respondents felt they could do "nothing at all" to protect their child from things in the environment. Parents' perceptions of how much they could do varied significantly with their level of education. Parents without a high school certificate were significantly less likely (55%) to feel they could protect their children from things in the environment compared to those with high school level or higher education (81% or more) ($X^2 = 12.48$, $p = .002$). These findings suggest that in planning education programming there should be adequate attention to ensure that knowledge and resources are translated to plain language and an appropriate literacy level. It may also highlight the need for non-written resources such as videos, pictorials or oral information sessions.

Although the majority of parents felt they themselves could do a great deal to protect their child's health, clearly they felt that the City played an important role as well. In another open-ended question, parents were asked what they believed was the single most important action that the City should take (i.e., respondents could make only one mention). Again, the responses here were in line with their "top of mind" thoughts on environmental factors that can be harmful to children's health. That is, most respondents felt that the City should ensure protection of air and water quality and limit the use of pesticides. They also felt that the City should apply rules, policies and by-laws, as well as education and awareness raising to ensure improvements in the environment. (See Table 5.5 for detailed response information.) The top five themes were comments related to:

- Air quality (23.5%)
- control emissions from industry and vehicles;
- improve air quality; and
- reduce pollution;
- Measures to ensure quality and safety of water (13.7%);
- Education, awareness and dissemination of information (13.2%);

- Development and enforcement of rules and policies (9.8%); and
- Limiting the use of pesticides (7.3%).

Table 5.5 Most important actions City of Toronto can take to protect children from harmful things in the environment

Theme	Percent (%) (n = 392)
Measures to ensure quality and safety of water	13.6
Education, awareness, and dissemination of information	13.1
Control emissions from industry and vehicles	11.6
Development and enforcement of rules & policies	9.4
Limit the use of pesticides	8.5
Improve air quality	6.8
Reduce pollution	4.8
Limit the use of vehicles	4.1
Clean up the city and community	3.9
Improve and increase utilization of funds	3.1
Improve school environment	3.1
Ban or limit smoking tobacco	2.7
Improve public transportation	2.2
More efficient collection of garbage and litter	2.2
Ensure building inspections	1.9
Regulate chemical used in food industry	1.7
Waste disposal and waste management	1.7
Limit use of chemicals	1.5

Themes reflect those with more than five mentions.

Source: Toronto Public Health, 2002b

5.3.3 Behaviours and Practices

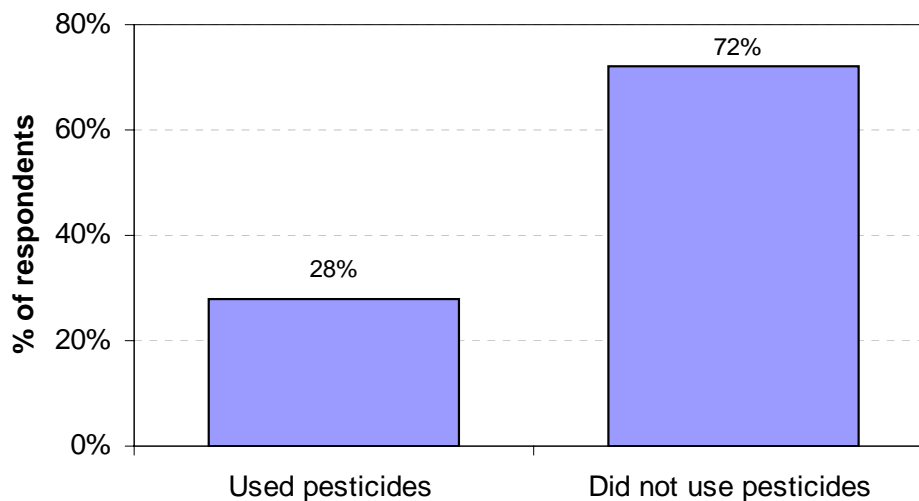
This section highlights what the survey findings indicate about behaviours or practices in and around the home that might influence or mitigate children's exposures.

Nearly one-quarter of the sample indicated that they owned a cat or dog and of this group, over 40% said that their pet slept in the child's room at least occasionally. Because research suggests that exposure to pet allergens

(particularly in infancy) is a risk factor for childhood asthma, this is potentially an important influence on a child's health and one that could be highlighted in health promotion work around asthma prevention (Lanphear et al, 2001).

Parents were asked about the use of pesticides (by themselves or someone else in the household or by professional applicators) in the previous 12 months (See Figure 5.2). As expected, the decision to use pesticides is influenced by residence type and location of the pest problem (that is, indoors versus outdoors). Reported use of pesticides indoors during the previous year was 17% overall and higher among apartment dwellers (27.4%). Respondents living in homes (that is, single, semi-detached, townhouse or an apartment in a house) were less likely to report that pesticides had been used outdoors on lawns and gardens (28%) compared to those living in apartments (39%). Overall results indicate that for the year 2001, the application of chemical pesticides in City of Toronto households with children is more prevalent outside the home (29%) on lawns and gardens compared to inside the home (17%).

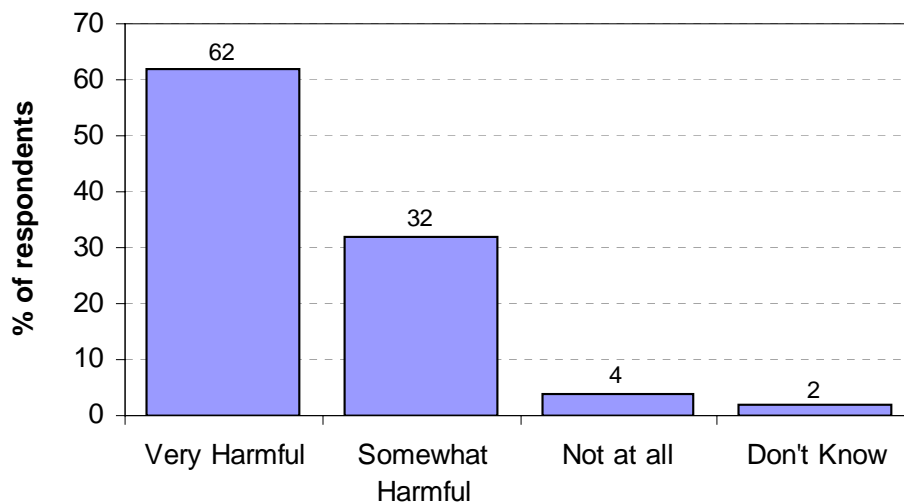
Figure 5.2 Self-Reported use of Outdoor Lawn and Garden Pesticides over the Past 12 Months



Source: Toronto Public Health 2002b

Although other surveys in Toronto are not entirely comparable, figures for pesticide use outdoors among this sample appear to be lower than those for the general population in the City of Toronto. For example, in a 2001 survey of homeowners (those who made lawn care decisions), 45% reported pesticide use outdoors in the previous two years (TPH, 2002d). Among Toronto residents with a lawn (not restricted to homeowners) responding in 2002, 38% reported pesticide use outdoors in the previous two year period (TPH, 2002e). The results reported for the present survey suggest that households with young children are less likely to use pesticides than the general adult population, particularly if they live in a home rather than an apartment and therefore have potentially more say in decisions about pesticide use. These findings agree well with those above for perceived harmfulness of pesticides, where 62% of parents offered that it was “very harmful” for children to come into contact with pesticides used on lawns and gardens (as shown in Figure 5.3).

Figure 5.3 Perceived Harmfulness of Children Coming Into Contact with Pesticides Used in Lawns or Gardens



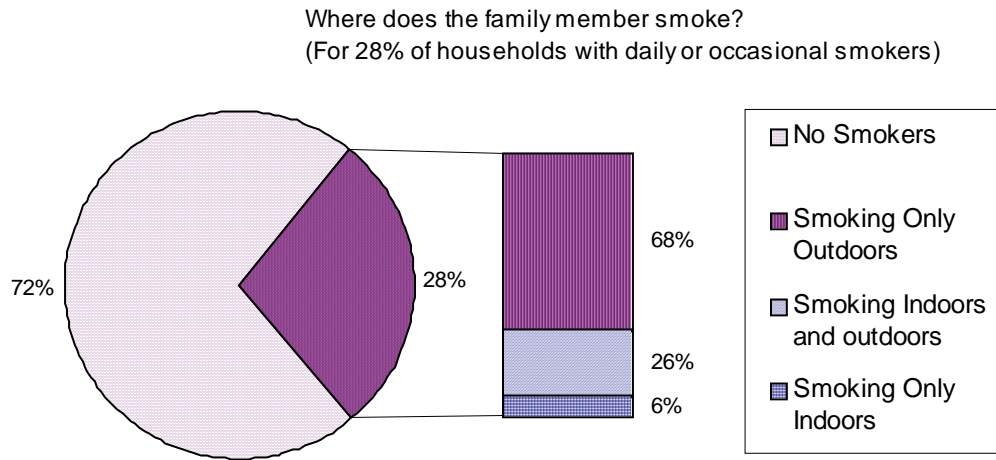
Source: Toronto Public Health 2002b

The City of Toronto pesticides by-law that came into effect April 1, 2004 restricts the use of pesticides on homeowners' lawns and gardens to a list of permitted lower risk products. Traditional chemical pesticides may only be used in cases of insect infestations, or if a pest, including a weed, poses a health risk. TPH is delivering a comprehensive education program including print and web-accessible materials, workshops and presentations, all promoting natural lawn and garden care and integrated pest management to prevent pest problems proactively.

Data for indoor pesticide use among the general population are limited to the 1990 survey of residents in the former City of Toronto which found about half used some form of indoor pest control (Sly et al, 1991). It is encouraging in the current survey that the figures for indoor pesticide use in households with young children is very low (17% overall). Clearly there is still room to reduce indoor pesticide use, especially among apartment dwellers and owners of apartment buildings.

There was high awareness and perception of the harmfulness of children's exposure to ETS as noted in section 5.3.2 above. When asked about smoking by household members, 73% of respondents reported that none of the family members smoked cigarettes. Sixteen percent indicated that there was a regular smoker in the household. Because of sample differences it is not possible to say precisely how this figure for households with smokers in the Toronto parents/caregiver survey compares to provincial and City-wide figures for proportions of adults who smoke. The 1996/97 Ontario Health Survey (OHS) determined that 21% of Ontario adults are regular smokers and that adult smoking rates were lowest in Toronto at 17% (PHRED, 2000). However, the most recent data for Toronto adult smoking rates show that 20% of adults in Toronto are smokers (TPH, 2004f).

Respondents with at least one smoking family member were asked where that person (or persons) smokes when at home. The majority of these respondents (68%) indicated that the smoking was done outdoors only, while an additional 25% indicated that smoking was both indoors and outdoors. Six percent of respondents reported that the family member(s) smoked indoors only. The responses indicate therefore, that nearly a third (31%) of those households with a smoker reported smoking was done indoors (either exclusively or at least part of the time). Figure 5.4 shows these data graphically.

Figure 5.4 Exposure to Environmental Tobacco Smoke

Source: Toronto Public Health, 2002b

Based on the respondent data, whether or not the smoking household member smoked indoors or outdoors was related to the age of the youngest child. In the households with a regular smoker, smoking indoors was less common (23%) where the youngest child was five or under as compared to households with a child over five years old (45%).

Again, it is instructive to compare the results for where smoking occurs in the present survey to data from other surveys. Data from the 1996-97 National Population Health Survey (NPHS) are most comparable and indicate that just under a third (~30%) of children under twelve in Ontario were regularly exposed to ETS at home at that time (PS-FC, 1999). The NPHS data indicate also that exposure is more likely among disadvantaged households that is, those with a single parent or that are of low-income.

The comparisons above suggest that households in Toronto with children 0 to 12 years of age have low prevalence of smoking behaviour (16%). As well, it is good news that in total, only 9% of this sample of children was regularly exposed to ETS inside the home which is substantially less than the figure of 30% reported for a comparable sample at the national level from 1996-97 (PS-FC, 1999). It appears also that even where there is a regular household smoker, most make the effort to smoke outdoors, especially if there are children under 6 years old in the home.

Continued efforts to help parents understand the importance of not allowing smoking indoors are warranted, especially in light of the large body of research

which links children's pre- and postnatal exposure to ETS with a range of health and developmental effects. Some of these effects include altered fetal growth contributing to risk of low birth weight, decreased lung growth, increased risks for respiratory and ear infections as well as, sudden infant death syndrome and neurocognitive and behavioural effects (Di Franza et al, 2004).

Respondents were asked whether household members and visitors remove their shoes when entering the respondent's home. Over 80% of respondents indicated that household members and visitors are asked to remove their shoes "always" (55%) or "most of the time" (26%). Eight percent of respondents indicated that they "never" ask household members and visitors to remove their shoes when entering the respondent's home.

Respondents were asked about the number of times the floor surfaces where their child spends most of the time playing are cleaned (swept, damp mopped or vacuumed). Over 40% of respondents reported cleaning the child play areas "almost every day" or "every day or more", whereas 23% said "several times a week". Thirty-five percent reported cleaning "once a week" (29%) or "less than once a week" (6%). Significant differences were found across various groups of respondents with regards to the reported frequency for cleaning floor surfaces. Those reporting the most frequent cleaning tended to be younger (18 to 34 years), with household income of less than \$40,000 and had children under five years old.

The above two questions indicate that a high percentage of respondents follow simple ways to minimize children's exposure to environmental contaminants indoors such as asking people to remove shoes in the home and frequent cleaning of indoor surfaces. Carpet and floor dust can be important reservoirs for contaminants that make their way into the home or result from the use of products inside. As noted in Section 4.3.2, with respect to lead exposures, the amount of dust in each square metre of an older carpet may be 400 times greater than levels found on a bare floor in the same house (Roberts et al, 1987). Contaminants such as pesticides can be tracked indoors (where they take longer to degrade) on footwear and pet paws (Nishioka et al, 1996, 1999, 2001).

Using a doormat or removing shoes are two ways to reduce the tracking indoors of contaminants. As well, parents and caregivers can also reconsider the use of carpeting in children's play areas. It is especially important to practice these and other dust control measures in homes with young children who may spend more time playing on the floor and have greater hand-to-mouth behaviour (Roberts & Dickey, 1995; Ott & Roberts, 1998).

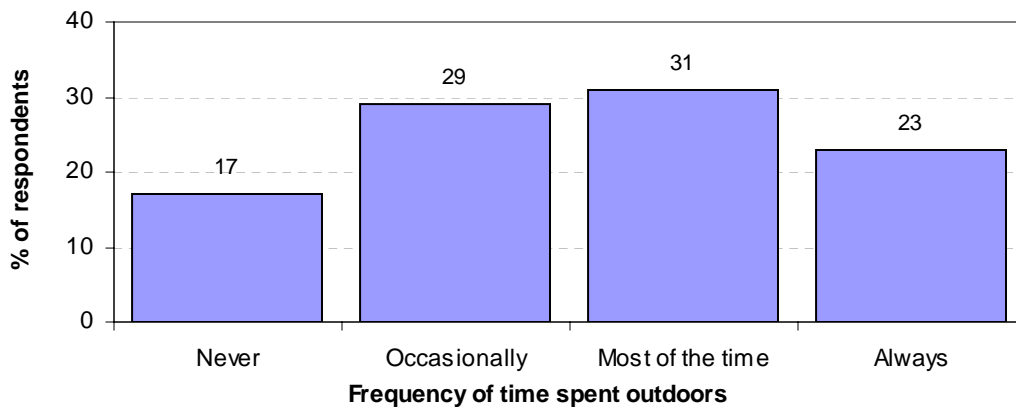
Figure 5.5 Parents' practices that reduce child exposures inside the home



Source: Toronto Public Health 2002b

Over three-quarters of the respondents (77%) reported being aware of the City’s smog alerts or air quality advisories from the previous summer (2001). While over half of the sample (54%) said they limited the time their child spent outdoors during such alerts always or most of the time, large proportions did so only occasionally (29%) or never (17%). The responses for these questions are depicted in Figure 5.6.

Figure 5.6 Time spent outdoors during smog alerts (among respondents aware of the smog alert)



Source: Toronto Public Health 2002b

TPH is currently conducting with Health Canada and Environment Canada a Personal Exposure Monitoring Study that seeks to characterize pollution levels in a variety of indoor settings, such as homes, schools, day cares, shopping centres and sports and recreational facilities, in comparison with outdoor pollution levels. This study will help develop policies (and verify current messages) regarding *physical activity* on days with poor outdoor air quality and will enable the general public and especially parents and sensitive populations, to take protective actions when Air Quality index (AQI) values increase sufficiently to trigger air-pollution related symptoms (TPH, 2003). The advice to monitor symptoms and moderate physical activity appropriately during smog alerts represents a shift in thinking since the time that the survey was conducted. In particular it reflects a growing awareness and uncertainty about the impact of indoor air quality for some people. It also denotes an appreciation for the importance of encouraging physical activity, particularly among children (Campbell et al, 2005).

Respondents were asked to tell how they protected their children from sun exposure when they were in the sun for more than 30 minutes the previous summer. They were probed for up to three practices. The overwhelming majority (nearly 90%) reported relying on sunscreen as their main sun safety measure. This figure is similar to data from a 1996 national survey on sun exposures and protection, where in Ontario, 83% of parents reported using sunscreen (OSSWG, 1998). Only about one quarter of Toronto parents reported having their child wear a hat and only 12% mentioned the use of protective clothing to avoid sun exposure. These figures are well below those for the Ontario sample where 74% mentioned wearing a hat and 56% mentioned wearing protective clothing (OSSWG, 1998).

Although very few reported doing nothing to protect their child, sun avoidance was far less commonly mentioned with 5% or less indicating that they protected their child by staying in the shade, using an umbrella or limiting the child's time outside. These figures are well below those reported for the 1996 Ontario telephone survey data mentioned earlier where 36% reported avoiding the sun and 35% reported seeking shade (OSSWG, 1998). The comparison should be taken with caution however, as the question formats are different between the Toronto and Ontario surveys. This open-ended question in the Toronto parents survey probed for three different practices whereas the Ontario survey asked parents how frequently did they take each of seven different protective actions for their children. Yet another view of sun protection practices among Canadian parents comes from an observational study of parents and children at freshwater beaches in British Columbia. That study reported parent actions to protect their children from the sun in proportions very similar to those from the telephone survey of Toronto parents (Shoveller et al, 2002).

Recently, there has been disagreement as to whether risk factors during childhood alone (that is, the pattern of exposure to UV radiation and/or the number of severe sunburns) are the most critical in predicting risk for adult development of melanoma or basal cell carcinoma (See for example, Corona et al, 2001; Pfahlberg et al, 2001; Whiteman et al, 2001; van Dam et al, 1999). Regardless, early exposure prevention is clearly important for establishing lifelong protective habits. However, a review by the International Agency for Research on Cancer (IARC) concluded that sunscreens may not provide complete protection from all skin cancers (Vainio et al, 2000; IARC. 2001). This suggests the need to encourage parents to practice other protective behaviours, particularly sun avoidance, seeking shade and wearing UV protective clothing where necessary.

5.3.4 Water Practices

Respondents were asked what water source was used most often for their child for drinking (including baby formula or drinks made with water). Most parents (more than 80%) reported using some sort of “treated” or alternative water source. A significantly larger proportion of parents whose youngest child was under 18 months reported using “boiled tap water” (39%) versus those with a youngest child over 18 months (19%). This likely reflects practices related to preparing infant formula however, since there was no question asking about infant feeding practices this assumption cannot be verified.

The survey results suggest that a sizeable segment of the parent population in Toronto uses alternatives to tap water for their children, such as bottled water or point-of-use (POU) filtration devices. This finding corresponds with the high awareness and concerns from survey respondents about drinking water quality as being very important for children’s health. Different choices for drinking water source come with different risks that should be clarified for parents. City of Toronto drinking water quality reports indicate that any substances of concern are either not detectable or present only at levels well below federal and provincial guidelines (Toronto WES, 2003). For example, levels of trihalomethanes (THMs), including chloroform, which is linked to bladder and colon cancer, are consistently less than 0.02 mg/L, well below drinking water guidelines which currently are 0.1 milligrams per litre (mg/L) (Toronto WES, 2003; NTP, 2004). Although bottled water is only recently undergoing greater regulatory attention in Canada it tends to have lower levels of aluminium and lead compared to tap water (Toronto, 1990). Lead levels are also typically lower in POU filtered water compared to plain tap water reflecting the fact that lead may be present in tap water from older plumbing pipes or from lead solder in homes built before 1990 (Toronto, 1990; MTTHU and SRCHC, 1995). Finally, those using POU filtration devices must be aware that containers should be cleaned and filters changed

regularly to maintain proper water quality, as discussed in Section 4.3.2 under Contaminants in Drinking Water.

5.3.5 Food Practices

Respondents were asked to categorize how much of the vegetables, fruits, dairy products and meat that they purchased for their child's consumption was "certified organically grown" (COG). Five percent said that all of the foods they purchased for their child were organic and 15% stated that most was organic. The fact that more parents (31%) who reported household income under \$40,000 reported giving their children organic produce most or all of the time (compared to those with incomes over \$40,000 - 13 to 15%) was an unexpected and interesting finding. Although not statistically significant, there appeared to be a decrease in the percent reporting the use of COG foods with increasing age of the youngest child. This may reflect a lower perceived need to protect older children, although it may also indicate a conscious choice governed by economic considerations, as COG foods are generally more expensive than commodities that are not organic.

Recent research (published since the time of this survey) testing for pesticide metabolites in the urine of children on primarily organic versus traditionally grown diets suggests that exposure to pesticide residues is significantly reduced with the choice of organic foods (Curl et al, 2002).

The majority of parents (nearly 90%) reported that their children (or their youngest child if more than one child was present) usually washed their hands before eating. The parents of households with a lower income were more likely to report that their child always washed their hands in this case. Handwashing is a generally good public health practice that reduces a child's exposure to contagions (such as cold germs, microbial agents, etc.) as well as to environmental contaminants. This is particularly so in young children whose frequent hand-to-mouth activity means greater likelihood of exposure to any substances picked up on the hands.

5.4 Variability in Practices

Overall, results show that parent or caregiver behaviour changes based on ages of the children in their care. A few examples confirm this observation.

- Significantly more households with young children (under 18 months old) reported very frequent cleaning (48.3%) compared to households with children over five (33.1%) ($X^2 = 12.54$, $p = .05$);

- Although not statistically significant, there is a general decrease in the reported use of certified organically grown foods with the increase in the age of respondents' youngest child. For instance, 25% of respondents with a youngest child under 18 months bought "most" or "all" certified organically grown foods, while this was the case for 21% of those with a youngest child between the ages of 18 months and 5 years, and 14.6% for those with a youngest child over 5 years of age; and
- A significantly smaller percentage of family members smoke outdoors when the youngest child is over 5 years (54.7% vs. 78% when child is under 5 years of age) ($X^2 = 10.72$, $p = .03$).

It is reassuring to see the attention that Toronto parents place on protecting their children when they are infants, toddlers or pre-schoolers, but there is a need to encourage parents to sustain the protective behaviours in children as they get older.

As well, there were associations observed between those with lower household income doing more to protect their children. This was seen in the reported purchases of certified organically grown foods, floor cleaning frequencies, source of water used for their children and in the reported frequency of their child's handwashing behaviour. Parents of lower income households were by contrast, less likely to report an awareness of smog alerts called in the City of Toronto. Paradoxically, parents with a low level of education (often a proxy for income) were less likely to feel they could do something to protect their children from things in the environment. The association between income and awareness is supported by survey data from Washington State where parents of the lowest income category were more likely to say that the environment plays a large role (87% for those under \$25,000) in how healthy people are compared to those in higher income levels (e.g. 73% for \$75,000 or higher) (Thurston County Public Health and Social Services Department, 2004).

5.5 Survey Limitations

Telephone-administered surveys by definition are limited in not being able to include respondents from households that do not have telephones. In Ontario, only 1.1% of households has no phone (Statistics Canada, 1997). The possible exclusion from the survey of households without phones is not expected to have an impact on the results.

A second limitation is the potential for self-selection bias. In this study, the refusal rate was only 6%. Although it is possible that those individuals who

refused to participate in the survey had different opinions than those people who did respond to the survey the high rate of participation lends confidence to the ability to generalise the findings. Although the readily available 2001 census data for comparison was for parents with children 0 to 6 years, it appears that the current sample is over-represented by those with a high level of education, but underrepresented in the highest level of annual household income (\$80,000 or more).

For reasons of cost the study could only be conducted in English, consequently it did not adequately allow for exploring the opinions of parents who do not speak English at all, or possibly of those whose first language is not English. The 2001 census indicates that 40% of Toronto children were born outside Canada (Toronto, 2003). In addition, nearly 30% of Toronto residents speak a language other than English or French at home (Statistics Canada on-line data, TPH, 2001a). However, 85% of Toronto adults report being able to speak, write and understand English (2001 Census). It is not clear to what extent the results represent the information from parents with limited or no skills in speaking English.

In order to maximize the sampling process respondents were randomly selected from areas with the highest proportion of children as determined from the 1996 Census. Therefore the results from this study may be generalised to parents and caregivers with at least one child (0-12 years) living in those areas (50 FSAs) included in the study. However, it is not clear if this sample adequately represents all of the parents and caregivers with at least one child (0-12 years) living in the City as a whole. The distribution of the selected FSAs appeared to be relatively uniform across all regions of the City. Again, the refusal rate was very low. Although it does not seem logical that parents and caregivers of children 0 to 12 years living in the areas with low ratio of children to adults (that were not sampled) have different attitudes and behaviours from respondents included in the study, it is not possible to confirm this assumption.

Owing to the vast nature of the topic area, the survey could not cover all the environmental issues related to child health. Questions were limited to those that might give results most useful for current programming initiatives in Toronto Public Health. Some topic areas were eliminated if too many questions were needed to elicit meaningful responses. For example, in trying to ask parents about mould in the home it would be necessary to ask about previous water damage, moisture problems, clean-up procedures, and so on. As well, there were uncertainties about whether respondents could adequately distinguish between mould and mildew. Some questions were eliminated where there was a good chance the response would be influenced by a social desirability bias. For example, would parents be likely to admit they did not wash fruits and vegetables

given to children? This limitation may affect responses to other questions that were retained, such as child handwashing and floor cleaning as well.

In addition, it was apparent that question wording might have limited the data gathered in some cases. For example, few respondents felt it was harmful for their child to “come into contact with” pets. Although the intention was to ask parents about the harmfulness of pet *allergens*, in keeping question wording to an appropriate literacy level, the tool avoided using the word allergen. It is not surprising then that parents reported low levels of concern for children coming in contact with pets, as clearly most people see pets as having a positive impact on children’s health and well-being.

These limitations aside, the survey provided useful information on what parents in Toronto know or may need to know about the relationship between child health and the environment.

5.6 Summary

Toronto parents have high awareness of the harmfulness of exposures to children’s health for issues that are high profile, frequently reported in the media or are already well covered in TPH health promotion work (e.g. air pollution, water quality, pesticides). Parents also felt these same areas were priorities for action on the part of the City. While most parents feel they can do a fair amount to protect their children themselves, there is a need to enhance that sense among parents with lower levels of education by providing information at appropriate literacy levels and considering non-written resources.

The good news is that most Toronto parents or caregivers are already practising or ensuring they take some simple measures that may reduce their child’s exposures in and around the home such as:

- shoe removal when entering the home;
- frequent floor cleaning;
- child’s hand-washing;
- food choices;
- providing smoke-free homes;
- minimal pesticide use;
- awareness of smog alerts; and
- use of sunscreen.

As well, fewer parents or primary caregivers of young children report practices or the use of products that are harmful to children when compared to data for the general adult population. Two examples from this survey are:

- less frequent use of pesticides inside and outside of households with children; and
- lower proportions of smokers in households with children.

The survey identified however, some areas requiring further public education:

- further reducing use of indoor and outdoor pesticides;
- wearing hats/protective clothing and avoiding the sun when children are outdoors during peak UV times;
- avoiding smoking indoors entirely;
- avoiding pet sleeping in a child's room; and
- informing parents about the benefits of extending protective, cautionary behaviors to all children, regardless of age.

This survey has increased understanding of the perceptions and awareness of Toronto parents regarding children's environmental health. These data should inform the development of more effective risk communication and allow for addressing parents' concerns more appropriately (Chai et al, 2001). The results provide greater clarity regarding the variability in parent concerns, awareness and practices, which is important to assist program planners in development of resources and implementation of programs. Specifically, they will allow for resources to be relevant, appropriate and tailored to the needs of Toronto parents, with attention to literacy levels, format and medium for resources and by ensuring content that fills information gaps.

Clearly there is room to further explore the awareness and practices around children's environmental health in Toronto. For example, the current survey does not shed light on what parents with children over twelve know (or what the children themselves know), nor does it reveal practices and awareness with respect to prenatal exposures. The information needs of prospective parents was addressed in part from survey work done in 1996 with women attending TPH prenatal classes. These women identified pesticides, lead, paints and solvents, plastics, cleaning products, electromagnetic fields, asbestos and mercury as areas of concern. Nevertheless, the current survey stands as an important first step in characterizing parental knowledge and practices for children in the youngest to middle age categories.

CHAPTER SIX – ADDRESSING ENVIRONMENTAL RISKS TO CHILDREN IN PUBLIC POLICY

Among environmental and health agencies at all levels of government in Canada and elsewhere, there is increasing consensus about the need to address the children's environmental health issues discussed in this report. Recognizing the large areas of uncertainty or simply non-existent information, the main areas of consensus are with respect to the vulnerability of the developing fetus, infants and young children alongside the evidence associated with a fairly small number of well-studied substances.

As a result, policy reforms have been initiated or recommended to address child health concerns during the assessment of substances that pollute the environment or from evidence of human exposure. The following sections briefly summarize the range of responsibilities across the international, national, provincial and municipal levels. The role of community networks and schools is also discussed.

6.1 International Commitments

Numerous international agreements have been signed by Canada recognizing the vulnerability of children and committing to policy and related efforts to address these risks. The 1989 United Nations Convention on the Rights of the Child laid a strong foundation for a series of further international commitments towards protecting children from environmental harm. The 1997 "Miami Declaration on Children's Environmental Health by the G8 Environment Ministers" promotes research into understanding children's unique vulnerability to environmental hazards and the development of protective policies to prevent childhood disease and adverse health effects from known and emerging environmental pollutants. Additional international commitments have been signed by Canada concerning the broad issues of children's environmental health as well as targeting specific issues such as lead pollution. National governments, particularly in the United States, have made similar overarching policy commitments to the protection of children from environmental threats. Alliances of non-governmental organizations have drawn up similar declarations. A detailed listing of these various international declarations is available on the website of the Canadian Partnership for Children's Health and Environment (CPCHE) at: http://www.healthyenvironmentforkids.ca/english/special_collections/fulltext.shtml?x=703

Canada was the first country to sign The Stockholm Convention on Persistent Organic Pollutants (POPs), an international treaty initiated in 2001 and ratified in 2004. The Stockholm Convention seeks to have all ratifying countries implement

plans to phase-out and ultimately ban a group of persistent toxic substances that are often called the “Dirty Dozen”. These twelve substances include dioxins, furans, PCBs, hexachlorobenzene, and eight organochlorine pesticides including mirex, aldrin, DDT, chlordane, dieldrin, endrin, heptachlor and toxaphene. Increasing evidence suggests that a number of other persistent toxic substances should also be considered for this international phase-out. For example, PBDEs (discussed in Chapters 3 and 4) are similar to PCBs in terms of both environmental persistence and toxic effects (Birnbaum and Staskal, 2004) but are not currently included in the Stockholm Convention.

In May 2005, a number of substances, including octa and penta forms of the polybrominated diphenyl ethers (PBDEs), perfluorooctanyl sulfonate (PFOS) and its salt, lindane, hexachlorobutadiene, polychlorinated naphthalenes and short-chained chlorinated paraffins, were nominated by other jurisdictions including the European Commission, Norway, Mexico and Sweden as possible additions to the Convention. A Persistent Organic Pollutants Review Committee (POPRC) responsible for evaluating prospective chemicals to be added to the convention, was established. Parties to the Convention, including Canada, will discuss recommendations from this committee. Among the first five chemicals to be reviewed is the penta form of the PBDEs.

6.2 Federal Activities

Since the mid-1990s, it has been standard practice by the federal (and Ontario) governments, to consider key aspects of the greater vulnerability of children in evaluating the human health impacts of commercial substances (pesticides, industrial or automotive emissions, etc.).

An important example of progress in federal legislation includes the *Pest Control Products Act* (PCPA) which governs the regulation of pesticides in Canada. During 2005, it is anticipated that the federal government will be ready to proclaim a revised PCPA. The new law contains several child-protective features to be applied to the registration of new pesticides or the re-evaluation of older ones. For example, it stipulates that the risk assessment process include an additional safety factor to ensure the adequate protection of infants and children. The cumulative effects of pesticides with similar modes of action, and potential effects of aggregate exposures from residues on foods and domestic uses must also be considered. These changes give legal force to what has been customary procedure in recent years, but also include several elements that will, if implemented, apply a more precautionary approach to regulating pesticides. In particular, the onus is shifted to registrant to persuade the regulator that the health and environmental risks of their pest control products are acceptable. There is a

provision for the obligatory reporting of adverse effects associated with registered pesticides, and there are mechanisms that facilitate product re-evaluation, including a mandatory 15-year re-evaluation of all products.

While this is encouraging, much remains to be done. The *Canadian Environmental Protection Act, 1999* (CEPA 1999) is the main federal legislation that manages toxics in Canada. Under CEPA, toxics are managed through a process that considers chemicals individually. Core requirements for such evaluations are not comprehensive. For example, within the federal system of evaluating toxic substances or pesticides, such evaluations do not require, although they may optionally include, assessments of effects on the developing brain. Only if other evidence points to the need for such studies are they then required. One difficulty with this approach is that there is an overwhelming dearth of studies that might prompt this optional evaluation. As well, evidence indicates that neurotoxic effects in the developing organism may not be readily predicted by studies of neurotoxicity in the adult (USEPA, 1999). It is of serious concern to scientists and health professionals that, alongside most pesticides, most commercial chemicals have not been tested for their toxicity to the developing nervous system in animals (Cooper, et al, 2000, Wigle, 2003).

Also, to date few comprehensive evaluations have been conducted as required by CEPA. Only 69 substances, or groups of substances, (out of 23,000), have been fully evaluated and about 50% of the active ingredients in pesticides still require re-evaluation for their effects in children. The figure of 23,000 is the number of substances on the Domestic Substances List (DSL), a list created in the mid-1980s to catalogue the full range of substances in commercial use in Canada. Since the DSL was created, several thousand additional substances have been introduced as well. The backlog of untested substances remains enormous. According to a deadline of 2006, Environment Canada and Health Canada will have categorized the 23,000 substances on the Domestic Substances List according to a “worst first” approach. The prioritization exercise seeks to identify those substances that appear to be persistent, bioaccumulative or toxic, and for which there is a high potential for human exposure. Following this short-listing exercise, these substances will be subject to either regulatory action, or more likely, further detailed assessment to support various risk management choices. The latter may or may not include regulatory action. The forthcoming review of CEPA would benefit from legislative amendments that incorporate precautionary and mandatory child-protective measures into this assessment process.

The example of PBDEs, frequently mentioned throughout this report as a category of substances of emerging concern for children’s health, illustrates a fundamental limitation with the current regulatory approach which prompts reaction only once evidence of widespread contamination is available. Recently, Health Canada and

Environment Canada completed screening assessments of the PBDEs to determine risks to human health or the environment. The agencies recommended, primarily due to environmental considerations, that PBDEs as a group be added to the list of Track 1 “toxic” substances under CEPA 1999 and targeted for virtual elimination (Canada Gazette, 2004). This proposal has been met with approval from environmental groups. However, they also suggest that the federal Minister of Environment impose an interim ban to escalate phase out of PBDEs in Canada and explore ways to incorporate greater precaution in the regulation of toxic substances in the environment before they become a problem (Sierra Legal Defence Fund, 2004).

An important example of progress in Canadian legislation is the *Pest Control Products Act* (PCPA). The PCPA, revised in 2003, is projected to receive final government proclamation in 2005. The new law contains several child-protective features to be applied to the registration of new pesticides or the re-evaluation of older ones. These changes give legal force to what has been customary procedure in recent years but also include several elements that will, if implemented, apply a more precautionary approach to regulating pesticides. Noteworthy among these are: the provisions to apply greater margins of safety for products used around homes or schools to protect children; provisions for reporting adverse effects from pesticides; mandatory re-evaluations every fifteen years; as well as, placing the onus of demonstrating that any associated risks are acceptable on the individual registering the pesticide (CELA 2002). The health-protective revisions to this law were largely due to concerns about pesticide risks in children.

The timelines for re-evaluating older pesticides are following timelines that are largely established under the *Food Quality Protection Act* in the US and in line with the NAFTA objective to harmonize pesticide standard setting. Health Canada’s Pest Management Regulatory Agency (PMRA) has come under repeated criticism for the slow pace of both re-evaluation of existing pesticides and expedited review of lower risk products (OAG, 1999, OAG, 2003, Standing Committee, 2000).

As pesticides are re-evaluated, the resulting decisions may range from an unchanged registration to amending label instructions, to modifying existing Maximum Residue Limits (MRLs), to elimination of some or even all uses. While there are safeguards built into this process to amend MRLs in light of a reassessment of toxicity data, it remains the case that when pesticide residues are found on foods, it is not uncommon to find multiple pesticide residues. Historically, MRLs (and therefore most MRLs currently on the books) have been generally based on good agricultural practices and not on safety with regard to potential human health effects. When the PMRA reviews MRLs it assesses human health safety by estimating daily intakes from food residues and toxicity data from

animal studies. There are significant underlying assumptions that the animal data can be extrapolated to humans and that this can be done one pesticide at a time and still assure human health safety. Experts note that this practice should be subject to ongoing external in-depth peer review by independent university-based scientists including epidemiologists with public reports on their findings (D.Wigle, University of Ottawa, pers comm, October 2004).

Increasing evidence points to consumer products as either contributing to or being the major source for certain contaminant exposures. Recent examples of hazards in products abound, including lead in jewellery, perfluorochemicals in non-stick surfaces of cookware, PBDEs in electronics and home furnishings, phthalates in toys and food wrap, Bisphenol A in resins and dyes, and nonlyphenol in detergents. However, Canada's 36-year old *Hazardous Products Act* is inadequate for regulating toxic substances that are found in consumer products and pose risks to child health. It is typically reactive, not structured to prevent problems before they occur and regulates on a product-by-product basis. Important deficiencies include: a lack of pre-market assessment for these products, extremely limited labelling requirements that focus only on situations of very high hazard and/or acute health effects, no provision for the government to legally issue product recalls or demand the removal of products from store shelves once problems are identified and, if regulatory action is taken, it is extremely slow. The incomplete and obsolete nature of Canada's regulation of potentially hazardous substances in consumer products is therefore another area of particular concern.

For some key areas of environmental or health regulation, the federal, provincial and territorial governments co-ordinate activities within the terms of various accords signed by the Canadian Council of Ministers of the Environment. The Committee on Health and Environment (CHE) is a relatively new Federal / Provincial / Territorial (F/P/T) established in 2003 by the Deputy Ministers of Health and Environment. At its inaugural meeting in 2004, the Committee identified children's health and the environment as one of three priority themes for F/P/T action. The Children's Task Group (CTG) of this Committee is pursuing three projects. These include: 1) an inventory of children's health and environment initiatives in governments and other organizations in Canada, 2) an inventory of blood lead level studies and review of the recent science for the blood lead intervention level and strategies, and 3) development of indicators to address the status of children's environmental health in Canada (J. Maxwell letter dated March, 2005). This latter project is being co-ordinated within Health Canada by the Office of Children's Environmental Health.

Health Canada's Office of Children's Environmental Health (OCEH) carries out its stated mandate to advance the protection of children's health in Canada from

environmental risks by collaborating with various government agencies, non-governmental organizations, academics and the community. The OCEH is the designated lead for co-ordinating activities on children's environmental health and has a stated objective to "catalyze action to manage environmental risks to child health" (Health Canada, online <http://www.hc-sc.gc.ca/hecs-sesc/oceh/>). However, in contrast to its counterpart in the US EPA, the Office of Children's Health Protection, Health Canada's OCEH is a small and under-resourced unit.

The US EPA's Office of Children's Health Protection (OCHP), established in May 1997, supports the EPA as it implements both President Clinton's 1997 Executive Order on the Protection of Children from Environmental Health Risks and Safety Risks as well as the National Agenda to Protect Children's Health from Environmental Threats. The Executive Order requires all federal agencies to place a high priority to addressing health and safety risks to children. The OCHP is a dedicated office, which has institutionalized child health protection within the US federal government. It works with internal and external partners to improve scientific understanding of children's environmental health issues. Its contribution to children's health protection in the US and worldwide is noteworthy with substantial, far-reaching work in the areas of research, regulation, outreach and education. For example, the OCHP has been key in establishing 12 Centers for Children's Environmental Health and Disease Prevention Research and reviewing, improving or developing health-based standards that specifically address impacts on children's health. The OCHP has also supported the development of Pediatric Health Fellowships and Pediatric Environmental Health Specialty Units (PEHSUs). There is an urgent need for a similarly strong political mandate, clear accountability and resources devoted to children's environmental health activities at the federal level in Canada. A comprehensive integrating mechanism for activities both within the government and beyond, such as might be achieved by designating children's environmental health with program status, is clearly required.

In spite of the lack of such a body to coordinate activities, the Canadian federal government has made some important, though modest, contributions to ongoing research into the human health effects, such as through addressing contaminants in the Great Lakes basin and in arctic populations. The various monitoring activities described in Chapter Four are complementary to these research efforts and to the implementation of federal law and policy regarding toxic substances and pesticides in general.

Throughout this report and indirectly in this chapter, discussions have highlighted the need for enhancing Canadian research on children's environmental health, specifically to improve understanding of environmental impacts on the developing brain and nervous system.

One important initiative in the U.S. bears consideration as having the potential to lay the groundwork for a new generation of studies that will help answer many questions on how exposures at different times affect child health and health throughout life. Several U.S. federal agencies recently begun implementation of the National Children' Study – a longitudinal cohort study that will track the health of 100,000 American children from in utero to adulthood (Branum et al, 2003). This 21-year endeavour is estimated to cost 2.7 billion USD (Check, 2004). Researchers will collect information relevant to describing exposures from pre-pregnancy and in early pregnancy. Data collected will include biological samples from the mother and child, as well as from air, water, dirt, and dust in the child's environment. As well, the study will gather information on the children's genetics. The study intends to examine the possible impacts from exposures together with consideration of how environment and genes interact with each other (NCS online 2004).

In the planning phases, the American agencies sought participation from Health Canada and Environment Canada in the study. Although Health Canada and Environment Canada held national consultations to determine the feasibility for a Canadian arm of the study, ultimately, the substantial costs of the proposed study have thwarted attempts for Canadian participation. A Canadian arm of the National Children's Study would help gather valuable data and benefit from the collaboration with US researchers. This effort to study and understand the long-term impacts of the environment on the health of a representative cohort of Canadian children is a unique and important opportunity that should be supported by the Federal government.

6.3 Provincial Actions and Canada-Wide Standards

Jurisdiction over the environment in Canada is shared between the federal, provincial and territorial governments. Between these levels, some areas are exclusively federal jurisdiction, some areas overlap. Pesticides, for example, are regulated by the federal government in terms of the evaluation process that determines whether a pesticide can be registered for use in Canada, among other matters, such as product labeling and MRLs on foods. Provinces then focus mainly on regulating the conditions of pesticide use, often applying a range of categories that spell out where individual pesticides can be used, by whom and under what conditions (including sales and availability to consumers).

In June 2004, the Ontario government announced a "Five-Point Plan" to address air pollution. This plan extended the emissions trading framework for sulphur dioxide and nitrogen oxides from the electricity generation sector to other

industrial sectors. The plan proposes emission caps for NO_x and SO₂ for seven new industrial sectors. While TPH supports the capping of emissions from the industrial sector, the proposed caps are not sufficiently stringent (TPH, 2005c). The plan also proposed improvements to the modelling tools used to estimate the impacts of air emissions from an industrial source, a risk management framework for the implementation of “health-based” standards and new or revised standards for 29 substances. While these proposals are an improvement, they still do not address the situation completely. For examples, sources of emissions are evaluated on a site by site basis, and do not adequately take into consideration the cumulative impacts of sources on air quality. Recently the Ministry has promulgated the new air standards that will place stricter controls on 29 hazardous air pollutants (MOE, 2005b). Toronto Public Health remains engaged in discussions with the Ministry of the Environment on these issues.

For toxic substances, the federal departments of Health and Environment share responsibility for evaluation and regulation, or other risk management activities, with respect to whether toxic substances may be used in Canada. Toxics are also regulated at the provincial level, often in a more specific or detailed manner, and generally at the “end-of-the-pipe” or once these substances become emissions or wastes. For example, provincial laws in Ontario include standards for contaminant levels in surface waters, drinking water and in sector-specific effluent emissions to waterways. Standards are also established for air pollutants, either as emission limits from stationary or mobile sources, or as ambient air quality criteria or “point of impingement” standards around industrial facilities. There are also laws and regulations controlling the transfer and disposal of hazardous wastes, municipal waste, and guidelines governing the clean-up and redevelopment of contaminated land.

For some key areas of environmental regulation, the federal, provincial and territorial governments coordinate activities within the terms of various accords signed by the Canadian Council of Ministers of the Environment. Key among these accords is agreement that provinces, territories and the federal government will establish Canada-Wide Standards (CWS) on certain pollutants. It is often the case that the federal and provincial levels of government refer substances to the CWS process when the level of concern, and controversy, is very high and potential impacts of regulatory or other management decisions are far-reaching. Hence, governments have referred to the CWS process the issue of mercury (in fluorescent lighting tubes and dental amalgam waste, from incinerators and base metal smelting, and from electric power plants), dioxins and furans (from waste incinerators and conical waste burners, and from several other industrial operations), and three key air pollutants: benzene, ground level ozone and fine particulates. In all of these cases, very large commercial interests and/or control or clean-up costs are at stake. As well, for most of these substances, there is

considerable concern about increasing evidence of toxicity at exceptionally low exposure levels up to and including the reality, in some cases, of no lower threshold for toxic effects.

The Ontario Minister of the Environment recently announced her intention to eliminate mercury emissions from Ontario's remaining coal-fired generating plants by 2010 as part of a strategy to meet the new Canada-wide standard (CWS) on mercury.

A number of other province-wide initiatives offer the opportunity for enhancing health promotion work in children's environment health. The Best Start Resource Centre (Ontario's Maternal, Newborn and Early Childhood Development Resource Centre) is a key program of the Ontario Prevention Clearinghouse and is funded by the Government of Ontario. This resource centre seeks to enhance the capacity of service providers to implement effective health promotion programs for expectant and new parents (including both men and women), newborns and young children. Noteworthy among the Best Start Resource Centre initiatives is the recent "Health Before Pregnancy" social marketing campaign to increase awareness in women and men ages 20 to 35 about the importance of preconception health (Best Start, 2005 online). The associated "Health Before Pregnancy" workbook includes a section on the environment along with a companion "Environmental Checklist" (Best Start, 2005). These are available online and are distributed to Ontario Public Health Units and interested community groups as well.

In addition to the Best Start Resource Centre, the Ontario Early Years program is establishing community-based Early Years Centres across the province to provide services to parents and prospective parents.

Finally, in November 2004, the Ontario Ministry of Children and Youth Services announced the Best Start Plan, a separate initiative from the Best Start Resource Centre. This initiative focuses on child care as well as children's healthy development and well being. Important elements of the plan include: universal newborn screening and ongoing screening and services to identify needs and provide vital developmental supports, a province-wide comprehensive developmental assessment for every child in Ontario at the 18 month old well baby visit, and the establishment of Best Start neighbourhood early learning and care hubs that provide one-stop services for families.

It is apparent that these discrete activities at the provincial level have the potential collectively to advance the children's environmental health agenda in Ontario. However, similar to the situation discussed earlier regarding federal initiatives, there is an equivalent need for political leadership, resources and integration of

activities within Ontario. A new Ontario Children's Environmental Health Initiative would strengthen provincial legislation and regulations, establish comprehensive surveillance programs to better understand exposure trends and health risks, and expand public education and outreach in Ontario. Particular attention needs to be directed at coordinating the activities of the Ministries of Environment, Health and Long-Term Care, and Children and Youth Services into a comprehensive cross-cutting provincial program.

6.4 Municipal Involvement

Shared jurisdiction also exists between provincial and municipal governments on health matters. Under Ontario's *Health Protection and Promotion Act* a range of activities is mandated and their delivery occurs within public health units across the province. The Mandatory Health Programs and Services Guidelines, issued by the Ministry of Health and Long-Term Care (MOHLTC), set out minimum requirements for core programs and services targeted at disease prevention, health promotion and health protection. The provincial rules establish the programs and services eligible for provincial funding and local health units can then tailor the programs for local circumstances.

There are many possible actions that can be taken at the municipal level on environmental health issues, including those that are intended to benefit children's health. Many of these actions occur as a result of municipal implementation of provincial laws, often via related provincial funding arrangements. The *Healthy Babies, Healthy Children* program is one of many examples of activities that stem from the municipal role in implementing the provincial *Health Protection and Promotion Act*, specifically the Mandatory Health Programs and Services Guidelines (MOHLTC, 1997). Another example is the Toronto Public Health mandate to provide Reproductive Health programming with the goal to support healthy pregnancies.

The Mandatory Health Programs and Services Guidelines are under review. In 2003, Toronto Public Health provided comments to urge that greater focus on environmental health issues be injected into the Child Health, Reproductive Health and Chronic Disease Prevention Programs. Revised guidelines have yet to be released. In June 2004, the Ontario Public Health Association (OPHA, 2004b) also called for revisions to the Mandatory Health Programs and Services Guidelines to incorporate a distinct Environmental Health Program with cross links to other program areas such as Child Health. OPHA noted for example, that currently the Guidelines make almost no mention of air quality except as it relates to environmental tobacco smoke.

Municipalities are also empowered by the provincial *Municipal Act* to establish by-laws for a range of specific purposes. This law provides a more general power as well to establish by-laws in response to local concerns and for purposes related to the health, safety and well-being of the inhabitants of the municipality. For example, this latter power was used to establish Toronto's by-law concerning the non-essential use of pesticides. In terms of their activities and law-making powers, municipalities in Canada are sometimes described as "creatures of the provinces" in the sense that they undertake activities for which the Province provides both enabling legislation and in many cases, direct funding. Flexibility needs to be built into these enabling laws to reflect the diversity of municipalities across the Province and the need to respond to local circumstances. In Toronto, a number of progressive municipal bylaws have been enacted to better protect health and/or the environment and thereby, the health of children as well. Notable among these are bylaws with respect to pesticides, anti-idling, sewer use and smoking.

Like all municipalities, Toronto can establish guidelines and policies, including by-laws, to reduce exposure to environmental contaminants in general, with benefits to children, or in some cases with the intention of specifically targeting reductions in children's exposures.

Two overarching documents adopted by Toronto City Council in 1999 are the Toronto Children's Charter and the City's Strategy for Children. The Charter draws mainly on the central tenets of the United Nations Convention on the Rights of the Child and situates the provisions of the Convention in Toronto's local context.

The City's Strategy for Children is a related document that notes a shared responsibility, with families, to improve children's well-being as a legitimate part of the City's public service agenda. Key components of the Strategy include a holistic response to issues affecting children, the setting of benchmarks to measure progress and the reporting of progress. Additional components include the promotion of equity of access and responsive service approaches to children through integrated service planning at the neighbourhood level. Further, the City's actions will be informed by current theory and research and the City will promote innovation and aggressively pursue policy and program improvements, to work with, strengthen, and lever support for a children's agenda from other levels of government and community partners. Finally, the Strategy prompts the City to focus media attention on children's issues, advocate for necessary change and celebrate achievements using incremental success as steps towards achieving the broader vision for children.

6.4.1 Working in Partnership

Local health units are encouraged to work in partnership with community-based organizations to enhance the health of the public, including children. Toronto Public Health has a history of working on children's environmental health issues with community partners who deal with health or environmental matters. Partnership activities in the area of children's health have brought about exciting and productive new working relationships. To address information needs of women attending TPH prenatal classes, TPH, the South Riverdale Community Health Centre (SRCHC) and the Sunnybrook and Women's College Hospitals' Environmental Health Clinic (SWC-EHC) collaboratively developed "Hidden Exposures", a fact sheet series for prenatal educators (to be made available online in 2005 at www.srchc.com). The fact sheets cover information on reproductive health risks and ways of minimizing exposure to these agents of concern to Toronto women. The fact sheets are used in a "training trainers" format and are not written for a general public audience. Health promoters from TPH's community partners on this project worked to train a substantial number of the prenatal educators in TPH and other health units. Those public health nurses who have received the training incorporate the information into their prenatal education sessions and also distribute a brochure that introduces the attending couples to basic principles, suggestions for exposure reduction and resources. The importance of the Hidden Exposures resource has been acknowledged implicitly by the Best Start Resource Centre's initiative to develop an environmental checklist as part of its Health Before Pregnancy Workbook (Best Start Resource Centre, 2005).

During the preparation of the Needs Assessment Framework Study (TPH, 1999a; TPH, 2002a), community partners acting as project advisors recognized a need to explore ways to sustain and coordinate the work on CEH for all groups. The assembled partners successfully secured development funding from the Laidlaw and Trillium Foundations and in September 2001, the Canadian Partnership for Children's Health and Environment (CPCHE) was formed.

CPCHE's overall goals include:

- Moving children's environmental health (CEH) issues into mainstream decision-making in Canada;
- Promoting CEH issues among caregivers, health professionals and the public;
- Increasing awareness about how to prevent harm by adapting and expanding the "childproofing" concept for a major health promotion campaign; and
- Coordinating activities and creating efficiencies among the partners.

From seven core founding organizations, including Toronto Public Health, CPCHE has grown to eleven partners representing national, regional and local health and environmental organizations. An important product of the partnership has been a web portal which outlines the activities of members, assembles an extensive resource library and, over time, will provide a means of engaging a broader network of organizations and individuals interested in keeping abreast of children's environmental health issues in Canada. See: <http://www.healthyenvironmentforkids.ca>

6.4.2 Initiatives in Schools

From the age of four until nearly the end of adolescence, children spend a substantial part of each weekday at school. The school environment (outside and indoors) is therefore an important influence on a child's health.

A healthy physical environment is one among four core components (along with curriculum, services and a supportive social environment) in the Comprehensive School Health Model (CSHM), a model endorsed by the Toronto Board of Health in 2000 that guides the delivery of school health services by Toronto Public Health.

The CSHM follows on efforts elsewhere (such as, through the World Health Organization Global School Health Initiative or the Ontario Healthy Schools Coalition) that advocate comprehensive approaches to school-based health promotion. More importantly, these efforts support the "vision that every child will have the opportunity to be educated in a 'healthy school'" (See OHSC, online). A healthy school is described as one that "promotes the physical, mental, social and spiritual health of the whole school community and constantly strengthens its capacity as a healthy setting for living, learning and working" (OHSC, 2002). Toronto Public Health sees the CSHM as strengthening the ability of school communities to take action on health issues, with public health services as a support. Of relevance in the context of this report, the CSHM outlines specific, key elements of a healthy physical environment in schools as including acceptable air quality and ventilation, safe water, low allergen environment and safe playground equipment, among others.

Throughout this report, exposures of greatest concern, including many that children might encounter in a school setting, have been identified. In addition, this report describes many of the progressive policies and procedures that both the

Toronto District School Board (TDSB) and the Toronto Catholic District School Board (TCDSB) have put in place to address environmental health issues for children at school.

Using the school setting as one example, Table A.1 in Appendix One, briefly summarises both outdoor and indoor exposures at school and some best practices for addressing the concerns. The range of issues in the school setting is quite broad and warrants a more focussed examination than was possible in the present report. Nonetheless, the information throughout provides schools and school boards with a reference point against which to assess the need for further change. From a public health perspective, it is important for the school boards to reaffirm a commitment to improving the school environment by continuing to make progress towards implementing safeguards that address the exposures outlined. The approach taken in preparing Appendix One can be applied to other settings in subsequent work at Toronto Public Health.

While many issues are raised in Table A.1 in Appendix One, it is important to highlight the fact that indoor air quality is probably the most significant issue facing schools in terms of both the potential for harmful exposures, and cost to remediate. The indoor air quality in Toronto schools is influenced by three factors: 1) type of ventilation, 2) maintenance of existing systems equipment, and finally, 3) contaminant or emissions sources present inside or around school buildings. Improving indoor air quality relies on addressing all of these factors where needed (See Table A.1, Appendix One).

During 2004, widespread media attention focused on the badly deteriorating condition of many school buildings across Ontario, particularly in Toronto, in both the TDSB and the TCDSB. After many years of inadequate funding for routine maintenance and major repairs, schools are literally crumbling, especially those buildings that were built decades ago, including some that have even passed the century mark. A litany of problems exists from leaking roofs to badly deteriorating electrical and plumbing systems, antiquated heating systems, peeling paint, broken windows, poor ventilation and more.

Many elementary schools built before the 1980s lack true ventilation systems, having only an exhaust vent system. About three-quarters of the TCDSB schools rely on windows and doors alone to provide adequate ventilation. With the dramatic rise in energy prices of the last 30 years, particularly in the 1970s and again in recent years, some remedial work has occurred on these old buildings to accomplish energy efficiency gains. However, these efforts have mainly focused on sealing up the spaces around windows and doors which, architecturally in the older buildings, were incorporated as the main routes for ventilation air intake. The TDSB has researched the feasibility of installing air-to-air heat exchange

units to resolve ventilation system issues, but with a need for one unit per classroom, at a cost of \$3000 per unit, this solution is entirely out of reach.

Looking at the full range of repair issues (i.e., beyond the ventilation issue) the two Toronto Boards estimate that catching up on necessary repairs and, in some cases, rebuilding, would cost \$1 billion. Estimated costs for the entire province have been close to \$6 billion. In response, the Ontario government established the Good Places to Learn Initiative to address these as well as additional costs related to specialized teaching spaces such as libraries, gymnasias, technical shops and science labs. The province committed \$2.1 billion towards these costs in addition to continued annual maintenance budgets. While this additional money may not address all the issues of concern, it represents an enormous opportunity to make some strategic investments in schools and children's health. Given the solid scientific evidence of harmful effects of poor indoor air quality and the high burden of respiratory illness among children, it seems clear that high priority should be given to improving indoor air quality as school boards make investment decisions about the infrastructure and maintenance of Toronto's schools.

CHAPTER SEVEN – SUMMARY AND RECOMMENDATIONS

7.1 Summary of Key Findings

Children are at Risk

- The health of Toronto’s children, like all children in Canada, is at risk from environmental contaminants. Children in Toronto are disproportionately affected by poverty, compared to children living in neighbouring regions in the GTA. Poverty is a known risk factor for poor health, greater susceptibility and greater exposure to environmental contaminants;
- The developing fetus, infants and young children up to age three can experience greater exposure and greater vulnerability than adults to substances in the environment. Multiple exposures of uncertain risk occur during pregnancy and continue throughout the course of child development;
- Scientific evidence exists of associations between environmental hazards and asthma, cancer, learning, behaviour and developmental effects, low birth weight and birth defects. Emerging evidence exists that raises concerns about additional, equally serious, health effects such as impaired functioning of the immune system and interference with the hormones of the endocrine system. Hundreds of environmental contaminants are suspected of contributing to serious health outcomes in children although only a small number of them have been fully evaluated for their effects on prenatal and child development; and
- In Toronto, asthma, learning disabilities, cancer, low birth weight and birth defects occur in the child population at rates that are similar to or in some cases, higher than, rates that occur among children in the rest of Canada and in other industrialized countries. Understanding the role, if any, of environmental contaminants in such health outcomes is extremely complex. Not only is the information base about environmental exposures limited, but it is well-established that multiple determinants of health (including biological, social and physical) and multiple factors within these determinants, influence each of the health outcomes of concern mentioned here.

Health Trends Across the Population

- Although infant mortality and early childhood diseases have dropped significantly and life expectancy has substantially increased in the last century, some chronic diseases and other debilitating conditions, including several having suspected or known associations with environmental exposures, are on the rise among children;
- Cancer rates have been rising among children in the US and countries in Europe for many years but such increases are not apparent in children in Canada. Although still rare, cancer remains the leading cause of illness-related death for children in Canada older than one year of age. Moreover, cancer rates among young adults (aged 20 - 44 years) in Canada have increased gradually since the 1970s. For certain cancers, such as thyroid and testicular cancer in men, brain cancer in women and non-Hodgkin's lymphoma in both men and women, incidence rates increased by more than two percent per year or just under 20 percent per decade. Causes for these increases are largely unknown but given the long latency period for most carcinogens, early childhood, prenatal or parental preconception environmental exposures, especially during windows of vulnerability, could be contributing factors;
- Two health outcomes are observable in large numbers of children: respiratory conditions, particularly asthma, and a range of conditions related to cognitive functioning and behaviour. Substantial evidence demonstrates associations between respiratory effects, including asthma, and indoor and outdoor air pollutants. Evidence is increasing of many other serious effects from air pollution in the developing fetus and child;
- Among children in Canada, 12% have asthma and Toronto physicians report treating children for acute and chronic respiratory symptoms more than any other health complaint. Poorer children in Toronto may be particularly vulnerable to air pollution as hospitalization rates for those living in the poorest areas of the City are nearly twice (93% greater) the respiratory hospitalization rate for children living the highest income areas, a trend that is consistent with recognized links between income and effects from outdoor air pollution;
- Effects on the developing nervous system are well documented for a small number of well-studied substances including lead, mercury, dioxins, PCBs and some solvents. Although these contaminants can

also adversely affect several organ systems in the body, it is common for effects on the nervous system to be observed at lower doses than are required to affect other body organs. Concern is increasing about nervous system effects of environmental tobacco smoke (ETS), the organophosphate insecticides and polyhalogenated compounds such as the flame retardants, PBDEs;

- The prevalence of learning disabilities, AD/HD, autism and other neurobehavioural deficits in Toronto children appear similar to their prevalence in the US, Canada and Ontario. Although data are limited, Toronto appears to be on the higher end of the scale with about 13% of enrolled elementary and secondary students (in the Toronto District School Board) with at least one or more learning or behavioural exceptionalities of concern;
- The apparent increase in recent years in North American children of various neurocognitive and neurobehavioural conditions may be influenced by more aggressive diagnostic practices. Nonetheless, the burden of disabling conditions is high enough for some US-based physicians to refer to the problem as having reached epidemic proportions; and
- Research into the economic burden of the diseases and disorders of concern with respect to toxic exposures suggests that exposure prevention could result in substantial savings in health care, human productivity and myriad social costs.

Understanding Exposure

- Measurement of exposure is limited. Many of the substances of greatest concern are associated with multiple effects and multiple pathways of exposure including during critical windows of vulnerability throughout prenatal development and the many stages of childhood;
- Biomonitoring data collected from the US population show that people of all ages have measurable body burdens of many contaminants, or their metabolites, at levels that in most cases are of uncertain health significance;
- Monitoring of pesticide residues on food in Canada paints a picture of fairly strong regulatory compliance. However, at the same time, US-

based biomonitoring data demonstrate that pesticides and their metabolites are extremely common in people's bodies. Home and garden use of pesticides creates greater exposure risks for children than through pesticide residues on foods;

- Indoor air quality is a largely unregulated source of exposure to a variety of contaminants. Exposure to various indoor air contaminants is believed to increase the risks of developing asthma or other respiratory irritation and health problems. Children's exposures in schools and child care facilities are as much a part of the indoor exposure picture as occurs in the home environment. In addition, contaminant levels in indoor dust are of increasing concern and present a significant exposure pathway for children;
- Exposures occur through many media with some far more significant than others. As a result, there is a need for good public educational materials that raise awareness about where risks are greatest and the necessary precautionary response and practices. This includes highlighting how choices, whether in foods, water source, consumer products and related behaviours, can influence exposure, positively or negatively; and
- The toxicity of certain persistent organic pollutants (POPs) is well understood. This knowledge can be extended to those POPs of emerging concern. PBDEs in particular appear to require the same response as has occurred in the past with similarly persistent and potentially highly toxic substances. The rapid build-up of persistent substances in the environment, and in the case of PBDEs, in the indoor environment and in breast milk, necessitates swift regulation, industry withdrawals and phase-out.

What Parents in Toronto Know About Environmental Risks to Children

- Survey results show that Toronto parents have high awareness of the degree of harm to children's health for certain well-known exposures (e.g. air pollution, water quality, pesticides) and want the City to take action on these exposures, through policies, by-laws, other measures that protect environmental quality, or through education activities;
- Most parents or caregivers are already practicing simple measures to reduce their child's exposures such as removing shoes at the door,

frequent cleaning of areas where children often play, encouraging handwashing, providing smoke-free homes, minimizing pesticide use and using sunscreen to protect from ultraviolet radiation;

- The survey results also indicate areas requiring further public education to extend protective, cautionary behaviours to all children not just to infants and toddlers, as well as encouraging protective behaviours among all groups of parents and child caregivers; and
- The survey also highlights that resources should be developed with particular attention to literacy levels, format and medium and by ensuring content that fills information gaps. The results should allow for risk communication and the preparation of children's environmental health resources and programs that are relevant, appropriate and tailored to the needs of Toronto parents.

The Policy Response

- For most environmental exposures, control measures are often delayed or opposed until solid proof of harm is obtained. This approach will continue to place the developing fetus and child at unnecessary and preventable risk;
- Some progress has occurred in terms of revising federal and provincial regulatory approaches to take children's health into account. These new approaches must be applied to the enormous number of substances in commercial use, or that result from industrial emissions, that have never been fully evaluated for toxicity during prenatal and childhood life stages;
- Advocates for changing traditional approaches to environmental hazards call for a precautionary approach. This approach speaks directly to the reality of never having absolutely definitive or conclusive evidence of harm. It denotes a duty, on all members of society, to prevent harm, when it is within our power to do so, even when the evidence is uncertain or not readily attainable;
- There is an urgent need for strong political leadership and clear accountability and resources for children's environmental health at both the federal and provincial level. There must be greater integration across departments where policies and programs can minimize exposure to environmental hazards. At the provincial level,

particular attention needs to be directed at coordinating the activities of the Ministries of the Environment, Health and Long-Term Care, and Children and Youth services into a comprehensive cross-cutting provincial program.

- Some municipalities have exercised leadership in applying precautionary action to recognized risks through the passage of progressive bylaws and other actions. Toronto has been in the forefront of this activity as have Toronto school boards. However, both the municipal ability and that of school boards to apply progressive environmental controls is limited by funding constraints, their respective arenas of policy and regulatory authority and influence, and the magnitude of the issues needing to be confronted.

7.2 Recommendations – Strategic Directions for Future Action

The public health mandate of disease prevention and health promotion is fundamentally one of applying a precautionary approach. The following recommendations encompass measures that will assist the City of Toronto in applying a precautionary approach to reducing and preventing children's exposure to harmful substances in the environment. Priorities for action are guided by the need to address exposure risks that are: a) preventable; b) have the potential to affect large numbers of children, including children whose health status is compromised by other circumstances such as poverty; and c) associated with serious or irreversible health effects or with long-term consequences.

The recommendations outline measures that will enhance protection of child health from environmental threats in Canada and that will ultimately assist the City of Toronto in reducing and preventing exposure to harmful environmental substances among Toronto's children. The strategic actions recommended address the gaps that identified in this report in the areas of policy, research and education.

7.2.1 Policy

- (1) It is recommended that the federal Minister of Health and the Premier of Ontario take a leadership role in protecting children from environmental threats to health by:

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- (a) At the federal level, establishing a comprehensive Children's Environmental Health Program to oversee federal resources, research and surveillance initiatives, and to propose new policies and regulations; and
 - (b) At the provincial level, creating a new Children's Environmental Health Initiative to strengthen provincial legislation and regulations, establish comprehensive surveillance programs to better understand exposure trends and health risks, and expand public education and outreach.
- (2) It is recommended that the federal Minister of Health:
- (a) Require that testing for developmental neurotoxicity be included within the mandatory, core testing requirements for evaluations of chemical substances, including pesticides;
 - (b) Revise the Hazardous Products Act and associated regulations to incorporate a similar level of precautionary and mandatory child-protective measures as found in the revised Pest Control Products Act (PCPA) such that:
 - (i) Children's exposure to toxic substances used in consumer products is prevented; and
 - (ii) Requirements for labeling and disclosure of ingredients in consumer products are improved.
- (3) It is recommended that the federal Ministers of Environment and Health:
- (a) Support the addition of newly identified persistent toxic substances including octa and penta forms of the polybrominated diphenyl ethers (PBDEs), perfluorooctane sulfonate (PFOS) and its salt, lindane, hexachlorobutadiene, polychlorinated naphthalenes and short-chained chlorinated paraffins, to the list of substances targeted for global phase out and ban under the Stockholm Convention on Persistent Organic Pollutants;
 - (b) Strengthen Canada's National Implementation Plan under the Stockholm Convention through a commitment to identify, on a separate list, all substances on the Domestic Substances List that meet the Convention criteria of persistence or bioaccumulation and inherent toxicity and, nominate these for consideration by the POPs Review Committee established under the Stockholm Convention;
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- (c) Ensure that such substances are made subject to control under CEPA;
 - (d) Ensure that the forthcoming review of the *Canadian Environmental Protection Act* incorporate legislative amendments that provide a similar level of precautionary and mandatory child-protective measures as found in the revised *Pest Control Products Act*; and
 - (e) Ensure final proclamation of the Pest Control Products Act (PCPA) by the end of 2005.
- (4) It is recommended that the Minister of Health and Long Term Care:
- (a) Revise the Mandatory Health Programs and Services Guidelines to include Environmental Health as a separate, expanded program area that replaces the Health Hazard Investigation Program; and
 - (b) Ensure that there is enhancement of other Mandatory Health Programs, such as Child Health and Reproductive Health, to include strategies for protecting preconception, prenatal and children's health from environmental threats.
- (5) It is recommended that the Toronto District School Board and the Toronto Catholic District School Board, Conseil scolaire de district catholique and Conseil scolaire de district du Centre-Sud-Ouest and, where appropriate, independent/private schools in the City:
- (a) Commit to a process of detailed evaluation and prioritization of policies, procedures and pilot projects that address indoor and outdoor environmental exposure risks in the school environment;
 - (b) Choose strategic investments when applying the new provincial monies under the Good Places to Learn Initiative to the maintenance, renovation or reconstruction of Toronto's schools;
 - (i) to address indoor air quality problems; and
 - (ii) achieve important benefits such as energy efficiency gains and provision of shade in school grounds; and
 - (c) Continue to work in partnership with Toronto Public Health to increase awareness about measures to protect children from environmental exposure risks in the school environment.
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7.2.2 Research

- (6) It is recommended that the federal Minister of Health:
- (a) Fund a Canadian arm of the U.S. National Longitudinal Children's Study that will assess exposures and health of a cohort of children from birth through to the end of adolescence as a way to improve research and surveillance on environmental threats to children's health in Canada;
 - (b) Request the Canadian Institutes of Health Research (CIHR) to support independent research on environmental threats to children's health in Canada by:
 - (i) Consideration of a separate research institute or integrating mechanism devoted to children's environmental health; and
 - (ii) Placing high priority on funding research into environmental impacts on brain development, including longitudinal studies; and
 - (c) In collaboration with appropriate federal government departments (such as, Health Canada, Statistics Canada and Environment Canada), ensure that:
 - (i) The biological sampling component of the Canada Health Measures Survey is implemented;
 - (ii) The biological samples are tested for full range of contaminants proposed by Statistics Canada; and
 - (iii) Sampling is further expanded into an ongoing, comprehensive biomonitoring program mirroring that conducted by the US Centers of Disease Control and Prevention.
 - (d) Working with the Ontario Minister of Health and Long-Term Care, ensure that targeted testing for blood lead among children ages 0 to 10 is implemented; and
 - (e) Request the Public Health Agency of Canada to expand the Canadian Integrated Public Health Surveillance Program (CIPHS) to include data collection and analysis of trends in neurodevelopmental and neurobehavioural outcomes in Canadian children.

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- (7) It is recommended that the Ontario Minister of Children and Youth Services, through the Best Start Plan, explore the possibility that the information gathered from the enhanced developmental assessments of every 18-month old child in Ontario be centrally collected and analysed for trends in developmental abilities as an additional way to improve surveillance of children's exposures and health impacts.

7.2.3 Education

- (8) It is recommended that the Medical Officer of Health:
- (a) Continue to pursue opportunities within existing Toronto Public Health programs for integrating environmental awareness and supportive, preventive practices for parents-to-be, pregnant and nursing women, infants and children with particular emphasis on those with increased risk;
 - (b) Continuing to identify opportunities within the City of Toronto to disseminate educational resources, such as through Parks, Forestry and Recreation, Toronto Public Libraries and Children's Services; and
 - (c) Continue to work with the Canadian Partnership for Children's Health and Environment to:
 - (i) Create, as needed, new educational resources directed to parents, caregivers, and practitioners in different settings such as child care and recreational facilities, that identify and provide preventive advice on environmental threats to children's health; and
 - (ii) Disseminate educational resources through key organizations involved in promoting the health of children including school-based parent groups, environmental and community groups and health-care practitioners and organizations, particularly Community Health Centres.

APPENDIX ONE – ADDRESSING ENVIRONMENTAL HEALTH ISSUES IN SCHOOLS

Children spend their time in many different settings, at home, in early learning and child care facilities, outdoors in their communities and in the school environment. The table below focuses on one setting - the school environment - as an example of health risks and possible remedies. It summarizes a range of potential exposures and related action steps that can be taken in the school environment. It has been prepared as a template for application to other settings.

Note that specific actions noted in column three may be governed by specific provincial or federal law, regulations or guidelines, or industry codes of practice. There may be specific requirements noted in regulatory, policy or guidance documents for dealing with potentially hazardous circumstances (such as mould abatement or during renovations) or for dealing with hazardous materials/substances (such as lead, asbestos, pesticides, etc).

Table A.1: Potential Environmental Threats in Schools: Concerns and Actions for Prevention

Indoor Exposures		
Potential Concern	Why it is a Concern?	Action to reduce the concern.
Mould	Mould growth is related to moisture problems (e.g. high humidity, water damage). Mould growth in portables has been a particular problem in the past. Health effects include allergic reactions and respiratory symptoms.	<input checked="" type="checkbox"/> Proactively address moisture problems (e.g. flooding, leaks) ensuring no area remains damp for more than 24 hours. <input checked="" type="checkbox"/> Prevent mould by keeping ventilation systems dry and clean <input checked="" type="checkbox"/> Remove identified mould growth promptly

Indoor Exposures		
Potential Concern	Why it is a Concern?	Action to reduce the concern.
Indoor pesticide use	Pesticides, in particular the organophosphate insecticides, have been associated with a wide range of health effects including some cancers and possible impacts on developing brain and nervous system. Indoor pesticides are particularly of concern because they can present a greater exposure risk, due to poor ventilation and slower biodegradation.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> Adopt an Integrated Pest Management (IPM) approach focusing on prevention (e.g. caulking cracks, eliminating pest habitats and food sources) <input checked="" type="checkbox"/> If pesticide use becomes necessary for health or safety reasons, choose non-toxic or least toxic products. If non-toxic alternatives are not available, seek out options that present the lowest exposure risk, such as baits, gels or pastes, rather than sprays. Apply pesticides only at times when children are not in school (e.g. weekends, holidays). Ensure that children are not allowed to enter any locations where pesticide applications have been necessary.
Cleaning products	Studies have associated cleaning products with a range of health effects including decreased respiratory function. Many such products contain volatile organic carbons (VOCs) such as formaldehyde.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> Adopt and follow a purchasing policy, choosing only non-toxic or least toxic products (e.g. low VOC products) <input checked="" type="checkbox"/> Ensure products are stored appropriately and are not accessible to students.
Classroom materials (e.g. Arts & Science supplies)	Arts (e.g. markers, paints, glues) and science (e.g. solvents, acids, compressed gasses) supplies may release potentially harmful substances during their use and storage. For example, mercury has been found in some U.S. science classrooms where there was inadequate clean-up after thermometers broke.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> Adopt and follow a purchasing policy, choosing only non-toxic or least toxic products <input checked="" type="checkbox"/> Ensure supplies are stored according to manufacturer's instructions and that storage areas are separate from classrooms and properly ventilated

Indoor Exposures		
Potential Concern	Why it is a Concern?	Action to reduce the concern.
Lead Paint	Schools built before 1976 (but especially before 1960) are likely to have painted surfaces that contain some lead. Lead is a known neurotoxin. Lead dust can be released from normal decay of old painted surfaces. Renovations that involve old painted surfaces can generate substantial amounts of lead dust indoors.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> Wipe painted surfaces with a damp cloth to clean dust (do not dry dust or scrape the paint). <input checked="" type="checkbox"/> Assume all old paint contains lead. Intact painted surfaces are better dealt with by painting over them with new, lead-free paint that seals in the old lead paint. <input checked="" type="checkbox"/> Check for peeling paint in areas where children might gain access. <input checked="" type="checkbox"/> Dry scrape but never power sand to remove any flaking and peeling paint. Use HEPA-filtered vacuum cleaners for cleaning up paint flakes and dust. Dispose of old paint flakes or dust as hazardous waste. <input checked="" type="checkbox"/> During renovations, thorough dust control measures should also be in place.
Lead in Drinking Water	Schools built before the 1950s are more likely to have plumbing pipes made of lead. Schools in older areas of the City may have municipal water supply pipes made of lead. Since the 1950s, lead solder was used on copper plumbing. Lead solder was banned in the late 1980s. Water that has been sitting in pipes for several hours may contain dissolved lead. Lead is a known neurotoxin.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> Ensure any school built before 1990 has, and is following, a policy on daily flushing (running the water for a few minutes every morning) of pipes. Priority should be given to drinking water fountains, or other sources where water is likely to be ingested (e.g. taps for filling water bottles). This practice gets rid of water that has been sitting in the pipes and may contain dissolved lead.

Indoor Exposures		
Potential Concern	Why it is a Concern?	Action to reduce the concern.
Building Materials	New materials, adhesives etc. may release volatile organic compounds (VOCs). Carpeted surfaces may harbour contaminants and dust mites and other allergens. Older carpets can contain up to 400 times more dust and associated contaminants than an adjoining area of bare floor.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> Choose products with low levels of emissions where possible. <input checked="" type="checkbox"/> When materials with potentially harmful emissions are used, increase ventilation and allow time for off-gassing before the area is reoccupied. <input checked="" type="checkbox"/> Remove carpeting where possible. If carpeting is necessary, choose low VOC carpets that have not been subject to chemical treatments (e.g. stain resistance) <input checked="" type="checkbox"/> Vacuum carpets frequently using a High Efficiency Particulate Air (HEPA) filter
Maintenance and Renovation Activities	Renovations can release dust, asbestos, mould and lead from paint into the air. New materials may release VOCs. Concentrations of potentially toxic substances may diminish with time as the substance becomes diluted and ventilated from the building.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> Adopt and follow a policy that limits potential exposures (e.g. painting, renovations) to times when children are not in school (e.g. holidays, on weekends) whenever possible <input checked="" type="checkbox"/> Prior to renovation, ensure inspection of work areas for hazardous materials including asbestos, lead, mould, etc. <input checked="" type="checkbox"/> Ensure careful dust control and clean-up practices during all renovations

Indoor Exposures		
Potential Concern	Why it is a Concern?	Action to reduce the concern.
Ventilation systems	Ventilation systems play an important role in improving indoor air. Two approaches may be used. Passive systems rely on windows and air leaks, and are used by the older schools in Toronto. Mechanically-based systems may include central exhausts, or Heating, Ventilation and Air Conditioning systems (HVAC). Some HVAC systems can filter out particulate matter from the air, but may be costly. Improperly maintained HVAC systems may be the source or contribute to the distribution of contaminants.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> Install mechanical ventilation where feasible. <input checked="" type="checkbox"/> Ensure mechanical ventilation systems are properly maintained (i.e. free of mould growth, filters are routinely changed, air vents are unobstructed, no standing water). <input checked="" type="checkbox"/> Combine opening windows with the use of portable fans as an inexpensive way to draw air into the classroom.

Outdoor Exposures		
Potential Concern	Why it is a Concern?	Action to reduce the concern.
Pressure Treated Wood	Existing playground equipment constructed from wood treated with chromated copper arsenate (CCA) requires specific mitigation measures. CCA contains arsenic, a known human carcinogen. Small amounts of CCA may leach from the wood and be ingested by children via frequent hand to mouth activity. CCA wood was phased out for residential and commercial use and has not been available since January 2004.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> CCA-treated wood playground structures should be coated with an oil-based penetrating sealant every one to two years. Sealant surfaces should be monitored for wear and to assess need for re-sealing. <input checked="" type="checkbox"/> Encourage hand washing after playing outdoors. <input checked="" type="checkbox"/> Regularly till or refresh sand/soil or other base material underneath play structures made of CCA-treated wood.
UV Radiation	Exposure to UV radiation may cause sunburns, which can increase risk of skin cancers later in life. UV radiation is strongest between 11am and 4pm from May through September.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> Provide adequate shade in areas of active play on school grounds <input checked="" type="checkbox"/> Encourage the use of hats and sunscreen by children and staff.
Diesel Exhaust	Diesel exhaust contains known carcinogens and large amounts of fine particulate matter (FPM). Air quality inside buses can be worse than inside buildings. Children may be exposed while travelling in school buses.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> Retrofit school buses with appropriate technologies that result in reduced emissions and FPM. <input checked="" type="checkbox"/> Ensure that there is regular ventilation of air inside buses
Outdoor Air Pollution	Outdoor air pollution including fine particulate matter, ground level ozone and VOCs have been associated with decreased lung function. Smog alerts indicate when outdoor air pollution is high.	<ul style="list-style-type: none"> <input checked="" type="checkbox"/> Moderate children's play or activities outdoors during smog and/or alerts. That is, reduce the intensity of activity and allow for frequent breaks, adequate water intake, access to shade. <input checked="" type="checkbox"/> Or, if <i>air conditioned space</i> is available, schedule vigorous

Outdoor Exposures		
Potential Concern	Why it is a Concern?	Action to reduce the concern.
		<p>exercise or play activities indoors during smog alerts.</p> <ul style="list-style-type: none"><input checked="" type="checkbox"/> Ensure that a no-idling policy is enforced around schools.<input checked="" type="checkbox"/> Reduce vehicle emissions around schools by encouraging families to participate in a Walking School Bus program, where two or more families travel to school together.

GLOSSARY OF TERMS AND ABBREVIATIONS

AD/HD: Attention Deficit Hyperactivity Disorder.

Association: The relationship between an exposure and a disease. Such a relationship does not necessarily demonstrate a cause–effect relationship.

Birth defect: Any defect present in a baby at birth, irrespective of whether it is caused by a genetic factor or by non-genetic prenatal events. Common birth defects include heart defects, cleft lip or palate, Down Syndrome, spina bifida and limb defects. Birth defects are the leading cause of infant mortality in Canada.

Bioaccumulation: Some pollutants are excreted more slowly than they are absorbed and are thus stored in the body for long periods of time. Total pollutants in the body (the “body burden”) may increase if the organism is repeatedly exposed to bioaccumulating substances for a long period of time.

Biomarker: Biological materials, enzymes, hormones, etc., that change when exposed to contaminants. See also biomonitoring.

Biomagnification: Pollutants “biomagnify” when their concentration increases as animals eat contaminated plants or other animals. For example, when pollutants in plants are passed on to animals repeatedly feeding on the plants, the animals may accumulate higher levels of contaminants in their own bodies than were originally found in the plants. This is because the animals eat many plants and do not excrete most of the absorbed pollutants. Levels of contaminants can increase up the food chain.

Biomonitoring: Biomonitoring involves measuring and analyzing chemicals, hormone levels or other substances in biological materials (e.g., blood, urine, breath) to estimate exposure, or to detect biochemical changes in the exposed subject before or during the onset of adverse health effects. Biomonitoring sometimes refers to a specific indicator for a particular disease/functional disturbance (e.g., a blood test for lead).

Blood–brain barrier: A term that encompasses multiple mechanisms that control access of blood components to the brain; the fetal and neo-natal blood–brain barrier is more permeable than the adult barrier to small lipophilic molecules.

Body burden: The total amount of a chemical in the body. Some chemicals build up in the body because they are stored in fat or bone and/or are eliminated very slowly.

Carcinogen: A substance (e.g., a chemical) or an agent (e.g., ionizing radiation) that causes cancer.

Childhood cancer: Among all age groups, the most common childhood cancers are leukemia, lymphoma and brain cancer. As children enter their teen years, the incidence of osteosarcoma (bone cancer) increases. Sites affected are different for each type of cancer, as are treatment and cure rates. In almost all cases, childhood cancer appears to arise from non-inherited mutations in the genes of growing cells.

Contaminants: Substances foreign to a natural system or present at unnatural concentrations; unwanted substances that have entered the air, food, water or soil. Contaminants may be chemicals, living things (e.g., bacteria or viruses) or the products of radioactivity. Some contaminants are created by human (e.g., industrial) activities while others are the result of natural processes.

Cotinine: A major metabolite of nicotine and regarded as the best biomarker (in active smokers and in non-smokers) of recent exposure to environmental tobacco smoke (ETS).

DDT: Dichloro-diphenyl-trichloroethane. A persistent insecticide used worldwide until it was banned in most countries in the 1970s.

DEET: N, N-diethyl-m-toluamide. Personal insect repellent for mosquito control.

Developmental toxicants: Agents that cause adverse effects to the developing embryo, fetus or child. These effects usually result from maternal exposure to toxic chemicals before or during pregnancy but can also result from paternal exposures. Early postnatal contact with developmental toxicants can also affect normal development.

Dioxins and furans: Among the most toxic chemicals known. Numerous types in each group, these chemicals are by-products of combustion, degradation of other chemicals, and some industrial processes.

EBDCs: Ethylenebisdithiocarbamates are a group of non-systemic (surface acting) fungicides.

E-coli: Bacterial species (numerous strains exist) found in human and animal intestines.

Endocrine disruptors (also called hormonally active agents): Synthetic chemicals and natural plant compounds (and some pharmaceutical drugs) that

may affect the functioning of the endocrine system (the communication system of glands, hormones and cellular receptors that control the body's internal functions). Many of these substances have been associated with developmental, reproductive and other health problems in wildlife and laboratory animals. There is concern that they could affect humans in similar ways.

Epidemiology: The study of the frequency, distribution and determinants of disease risk in human populations. Also the field of medicine concerned with the determination of the specific causes of localized outbreaks of infection (such as hepatitis), toxic disorders (such as lead poisoning), or any other disease of known cause.

ETS: Environmental Tobacco Smoke (second-hand smoke). The smoke released by idling lit tobacco products (cigarettes, cigars, and pipes), and smoke exhaled by smokers after puffs. ETS contains hundreds of toxic chemicals including over 40 cancer-causing chemicals.

Fish advisories: Public notices issued by local, provincial or national environmental or health agencies that warn about the need to limit or avoid consumption of certain fresh or salt water species, generally those that are predator species at the top of aquatic food chains. Most advisories are directed at pregnant or nursing women and young children, as well as women of child-bearing age.

Food chain: The food chain is a series of organisms, each consuming the organism below them in the chain, and being consumed by the next higher organism. Green (chlorophyll-containing) plants are at the bottom of every food chain; green plants convert sunlight into food energy for the rest of the organisms in the food chain. Because organisms at each level of the food chain use up most of the energy they consume, energy is lost at each level, limiting the length of the chain. The extra buoyancy of water allows for some of this energy to be conserved thus allowing more levels in aquatic food chains. With more levels, contaminants can continue to concentrate (biomagnify). Hence the greater contamination of fish by methylmercury and the need for fish advisories. In nature, food chains usually combine to form food webs.

Food web: Found in any natural community, a food web contains many interlinked food chains. Humans are at the highest level of many food webs and their breastfed infants are higher still.

Incidence: The number of new cases of a disease occurring in a defined population within a specified period of time. Frequently presented as the number of new cases per 1,000 or 100,000 people per year.

Inorganic chemicals: Chemicals that do not contain carbon. Examples include metals like lead, mercury and cadmium, as well as salt and asbestos.

Intrauterine Growth Restriction (IUGR): A low birth weight infant with a birth weight below the lowest decile for gestational age. See also: low birth weight.

Ionizing radiation: A physical agent — ions — released during the spontaneous radioactive decay into small elements of radionuclides emitted during medical X-rays, the regular operation of nuclear power plants, and, in potentially massive amounts, as a result of nuclear accidents and the testing or use of atomic weapons.

Latency period: The period of time between exposure to a disease-causing agent and the first appearance of signs or symptoms of the disease.

Learning disabilities: Refers to a number of disorders that may affect the acquisition, retention, understanding or use of verbal or nonverbal information. These disorders affect learning in individuals who otherwise demonstrate at least average abilities essential for thinking and/or reasoning. Learning disabilities result from impairments in one or more processes related to perceiving, thinking, remembering or learning. They range in severity and may interfere with the acquisition and use of one or more of the following:

- oral language (e.g., listening, speaking and understanding);
- reading (e.g., decoding, phonetic knowledge, word recognition, comprehension);
- written language (e.g., spelling and written expression); and
- mathematics (e.g., computation, problem solving).

Lipophilic/hydrophobic: Literally “fat-loving” or “water-averse.” Used to refer to substances that bind to fat molecules, and as a result often concentrate up the food chain and reach their highest levels in high fat foods such as whole milk, cheese, fatty meats and oily fish.

Lipophobic/hydrophilic: Literally “fat-averse” or “water-loving.” Used to refer to substances that stay in solution in water and tend not to bind to either particles or fatty molecules.

Low birth weight: An infant that weighs less than 2500 grams at birth. See also: IUGR

Melanoma: Dangerous type of skin cancer.

Mental retardation: A disability characterized by significant limitations both in intellectual functioning and adaptive behaviour as expressed in conceptual, social, and practical adaptive skills. This disability originates before age 18 and is often more simply defined in terms of an individual having an IQ <70 but other criteria, beyond intellectual capacity, are generally considered.

Metabolism: Total biochemical and energy processes that maintain life in organisms. Includes the conversion of one compound into another, the building up of larger molecules from smaller ones (anabolism), and the breakdown of compounds (catabolism) to release life-sustaining energy.

Methylmercury: Organic mercury compound formed by moulds and bacteria.

Neurotoxicants: A biological or chemical substance or agent that has an adverse effect on the structure or function of the central (including the brain) and/or peripheral nervous system. Toxicants that exert adverse effects on the developing brain or nervous system are called developmental neurotoxicants.

Organic chemicals: Chemicals containing carbon, usually combined with hydrogen and other elements such as oxygen, nitrogen, or chlorine. Vegetable matter, petroleum, and plastics are examples of organic materials as are PCBs, DDT, and polyvinyl chloride.

Organochlorine compounds: A wide variety of synthetic organic compounds containing chlorine particularly notable for their persistence and stability. Some have been deliberately manufactured (e.g., several pesticides, including DDT, PCBs, though these are now banned or greatly restricted in use). Others are breakdown or reaction products like dioxins formed from incineration of products like PVC plastic. All are now widely distributed in the environment.

Organophosphates: Organic chemicals containing phosphorous, some of which are involved in cellular energy metabolism. Nerve gas (no longer manufactured) and organophosphate pesticides are based on the same chemistry and can interfere with important aspects of cellular energy metabolism in nervous system tissues, including in the brain.

PAHs: Polycyclic aromatic hydrocarbons include a large number of toxic chemicals, including several cancer-causing chemicals, created from the combustion of organic material, including fossil fuels. Exposure occurs via air pollution but most PAHs are adhered to fine particulate matter; unlike VOCs, they are not very volatile.

PBDEs: Polybrominated diphenyl ethers. Widely used in consumer products as flame retardants.

PCBs: Polychlorinated biphenyls. Manufactured for transformer cooling oil and numerous other applications. No longer manufactured and use is restricted because of carcinogenic properties and persistence in the environment.

Persistence: Refers to chemicals or agents that remain a long time in the environment. For example, lead and mercury persist in the environment because they are stable elements; PCBs are chemically stable compounds that resist degradation. Mercury and PCBs cycle between environmental media including air, water and food chains.

Phthalates: Compounds used to make some plastics soft and flexible. A plasticizer compound.

PM: Particulate Matter. Described in three categories: coarse, fine and ultrafine. Particle diameter generally noted in subscript. Hence:

- coarse - PM_{10} , or $PM_{2.5-10}$ (particle diameter between 2.5 and 10 microns);
- fine - $PM_{2.5}$ or $PM_{1.0-2.5}$ (particle diameter between 1.0 and 2.5 microns); and
- ultrafine – $PM_{1.0}$ (particle diameter less than 1.0 microns).

PMRA: Pest Management Regulatory Agency. Agency within Health Canada responsible for registering pesticides for use in Canada.

Polychlorinated naphthalenes: chemicals used for cable insulation, persistent and bioaccumulative and recommended by the European Commission (in 2004) as candidates for addition to the Stockholm Convention on Persistent Organic Pollutants.

POPs: Persistent organic pollutants. Chlorinated organic compounds characterized by resistance to natural breakdown, consequent persistence and bioaccumulation in the environment. Also often highly toxic.

Prevalence: The number of events (e.g. instances of a given disease or other condition in a given population at a designated time. Note this is a number not a rate. See also Rate and Incidence.

Rate: In epidemiology, an expression of the frequency with which a certain circumstance (e.g., asthma incidence in children) occurs in relation to a certain period of time, a fixed population, or some other fixed standard. The use of rates

rather than raw numbers is essential for comparison of experience between populations at different time or different places.

Reproductive toxicants: Chemical substances or agents that cause adverse effects on the male and female reproductive systems. Toxicity may be expressed as alterations of sexual behaviour, decreases in fertility, loss of the fetus or abnormal fetal genital development during pregnancy. Interference with sexual function may occur from puberty through adulthood.

SCCPs: Short-chained chlorinated paraffins. Chemicals used in metal working and leather finishing, and recommended by the European Commission (in 2004) as candidates for addition to the Stockholm Convention on Persistent Organic Pollutants.

SIDS: Sudden Infant Death Syndrome (also called Crib Death). Refers to the sudden and unexpected death of apparently healthy babies. Exact cause is unknown but risk factors include exposure to environmental tobacco smoke during pregnancy or after birth and sleeping on the stomach.

Teratogen: Any substance or factor that can cause structural or functional malformations of an embryo or fetus which are also known as congenital malformations or birth defects. Known teratogens include certain chemicals and viruses and ionizing radiation.

Toxic substances: Substances capable of causing harm to humans, animals or other living things. In common usage, the term refers to chemical substances that are capable of causing harm at very low levels of exposure, while providing little or no benefit.

USEPA: United States Environmental Protection Agency.

USFDA: United States Food and Drug Administration.

UV Radiation: Ultraviolet radiation, emitted by the sun's rays or used industrially (e.g., for sterilization purposes).

VOCs: Volatile organic compounds. Organic gases and vapours in the air. Examples of sources include the burning of fuels, dry cleaning operations, and the evaporation of organic compounds from solvents, paints, or other coatings.

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