Childproofing for Environmental Health: An Examination of Food Related Exposures

Dr. David McKeown
Medical Officer of Health

December 2009

TORONTO Public Health

Reference:

Toronto Public Health 2009. Childproofing for Environmental Health: An Examination of Food Related Exposures.

Authors:

Loren Vanderlinden, PhD
Liana Del Gobbo*, MSc
Josephine Archbold, MSc
Sudha Sabanadesan, MSc

*Formerly of Toronto Public Health

Acknowledgements: We thank Dr. Monica Campbell, Manager, Environmental Protection Office, Carol Timmings, Director, Planning & Policy, and Barbara Emanuel, Senior Policy & Strategic Issues Advisor, Toronto Public Health for comments and advice essential to the preparation of this report.

Distribution: This report and its accompanying Board of Health report are available at: *http://www.toronto.ca/health/hphe*

For more information: Environmental Protection Office
Toronto Public Health,
277 Victoria Street, 7th Floor
Toronto, Ontario,
Canada M5B 1W2
Tel: 416-392-6788
Fax: 416-392-7418

Executive Summary

This report summarizes information on key chemical exposures for children focusing on those largely related to food. It discusses a number of chemicals or categories of substances including lead, mercury, pesticides, dioxin-like compounds, bisphenol A, phthalates, polybrominated diphenyl ethers (PBDEs) and perfluorinated compounds (PFCs). Toronto Public Health's (TPH) review of research indicates that the levels of evidence for each of these substances is different. All are of concern to varying degrees; nonetheless all are worth reducing in the diet or from other routes of exposure where feasible. In light of incomplete information on risks to health, this report highlights relevant precautionary advice on ways to reduce or minimize exposure.

The effects on the developing child from some substances, such as lead and mercury, are quite well known. Lead is toxic to the brain and nervous system of the fetus, infant and young child at very low doses. Nowadays, lead exposure is much less from food than it was in the past when some cans had lead solder. However, traces of lead can still end up in food because it is a widespread environmental contaminant. Lead can also still be a problem in drinking water because of old lead pipes or plumbing fixtures.

Methylmercury, the most toxic form of mercury, is also harmful to the developing brain and nervous system. The most important source of methylmercury is from fish consumption. The critical period of vulnerability is largely during pregnancy although fish consumption messages typically advise caution for both pregnant women and children in consuming high mercury fish. Exposure to pesticides is also known to be associated with health impacts in children such as certain cancers or neurodevelopmental delays. However, the evidence suggests that exposures prior to conception or during pregnancy in parents who work with pesticides or exposure during pregnancy or in infancy to pesticides used in the home are more important than food exposure which is comparatively much lower. Effects from dioxin and dioxin-like compounds are also well studied in people who had high exposures or from low level prenatal exposure. While food is the most common source, the levels in food have declined in the last couple of decades.

On the other hand, health effects from substances such as BPA, PBDEs, phthalates and PFCs are known only or mainly from animal studies. Exposure studies indicate however, that most people carry traces of these compounds in their bodies beginning at an early stage of life and that the effects on living organisms or the environment are of potential or actual concern.

Food or food containers, packaging, processing and cookware contribute to varying degrees to children's exposures to these substances. For some of these substances, such as dioxin-like compounds, lead, phthalates or PFCs, their presence in food is likely from environmental sources and therefore it is challenging to reduce exposures with individual dietary choices. In some cases, such as for BPA and PFCs, not all the sources for children's exposures are known.

In the case of many of these substances, the critical exposure periods generally appear to be *in utero*, during early infancy and in some cases, during early childhood and adolescence. Therefore, the recommended actions can be taken in a child care setting or in the home. However, they may apply also, and in some cases more directly apply, to pregnant or

breastfeeding women, and to women who may become pregnant (that is women of childbearing age).

This review identifies a number of best practices, including several recommendations from *Eating Well with Canada's Food Guide*, that can minimize potential risks from food- or food-related exposures including: avoiding processed foods of low nutritional quality; reducing high fat meat, fish and dairy recognizing the nutritional need for full fat dairy for children under two years of age and the nutrition benefits of higher fat fish such as salmon; cooking methods that reduce saturated fat; serving fresh or frozen food items when feasible; and, avoiding heating food or drink in plastic containers. Existing TPH messages about eating low mercury fish and flushing drinking water to reduce lead are also important for reducing exposures.

In addition to food-related exposures, other environmental sources for many of these substances may exist in children's daily lives. For example, lead from old paint, pesticides from indoor use and flame retardants or PFCs from consumer products end up in indoor dust, an important exposure pathway for toddlers and young children in particular. Regular hand washing (with plain soap), wet dusting and a "shoes off" policy in playrooms where children are crawling on the floor can reduce exposure to the substances which are higher in indoor dust than in food.

TPH and community and City partners will continue to increase awareness about ways to reduce food-related chemical exposures and to incorporate other environmental health childproofing strategies by developing and disseminating appropriate educational and training material.

Table of Contents

List of Tables

1. Introduction

1.1 History of the Issue

In 2005, Toronto Public Health (TPH) released a lengthy report entitled, *Environmental Threats to Children: Understanding the Risks, Enabling Prevention* which discussed the state of the evidence for children's exposure sources and potential health effects from environmental threats, including both risk and exposure reduction practices related to food contaminants.

In 2007, the Toronto Board of Health (BOH) heard concerns regarding specific chemical substances, including bisphenol A (BPA) and phthalates, which may pose risks to children's health from their presence in food. The focus of the concerns was around exposures that may be present at child care centres. The BOH consequently requested that the Medical Officer of Health report back to them on "the top ten other food additives or container properties which may harm children's health at child care centres".

This report provides a summary of background research on contaminants that can occur in food (including from food packaging, processing or food containers) or drinking water and that are of concern to children's health. This document supports the staff report to the BOH that outlines ways that child care centres might incorporate exposure reduction measures to address these contaminants of concern in food.

1.2 Scope of the Report

The research on certain food-related contaminant exposures and potential associated health effects has expanded substantially since TPH reviewed the literature in 2005. The list of substances reviewed here was initially informed in part by the concerns of some Toronto parents and also by TPH's ongoing work to reduce children's exposure from all sources, not only food. Several criteria were used to determine the list of substances examined: evidence for widespread exposure in humans; differential vulnerability or exposure for children compared to adults; and the potential for effects that are serious and irreversible (e.g. asthma, cancer, reproductive or neurodevelopmental impacts). In all cases, food or water are known exposure pathways which may be amenable to exposure reduction strategies. In addition, the Government of Canada has included these chemicals among a list of "substances of interest" which are deemed priorities for risk assessment and appropriate controls under the Chemicals Management Plan (Government of Canada, no date).

This report builds on the 2005 TPH report and includes up to date information on lead, mercury, pesticides, dioxin-like compounds, BPA, phthalates, perfluorinated compounds (PFCs) and polybrominated diphenyl ethers (PBDEs) (or flame retardants). The report describes what is known about the potential exposure and health effects from each of these substances, or categories of substance.

A review of regulations and policy actions underway for the substances in this report is included where relevant and where important new information was available. The conclusions from this review inform dietary and practice recommendations for parents and for child care practitioners

presented here. While the review of the literature pointed to gaps in scientific evidence that should be addressed, the inclusion of research recommendations regarding this suite of substances was beyond the scope of the current report.

1.3 Why Children are Vulnerable

Children are exposed to environmental substances through many potential pathways. They are often more exposed to environmental contaminants relative to adults and their exposures can differ substantially because children behave and spend their time differently than adults do. Health scientists recognize a number of main environmental media through which contaminants travel to people: air, water, soil, dust and food. Children take in more per unit body weight of the contaminants found in these media compared to adults (NRC, 1993; Selevan et al, 2000; US EPA, 2003). Frequent hand-to-mouth patterns of behaviour in young children mean that soil and dust can be important exposure routes. Children breathe more rapidly than adults do which means that they take in more of the pollutants found in air. Children consume nearly double the amount of water per unit of body weight and relatively much larger amounts of certain foods compared to adults (Selevan et al., 2000).

Windows of vulnerability to harm are not all completely known for individual contaminants but they may span several stages in children's development beginning prior to conception and occurring during prenatal and postnatal periods. The hazards to children of several of the substances included in this report are not fully or equally understood. Thus, the focus here is on precautionary primary prevention and ways to reduce exposure, in light of incomplete information on risks to health. The practices to reduce children's food- related or drinking water exposures outlined in this report also guide parents and child care practitioners on taking action where it is feasible and within their control without compromise to nutrition, safety or other important health protective/disease prevention practices.

2. Food and Food-Related Exposures for Children

2.1 Introduction

Food can be an important, although not always the largest, source of exposure to some contaminants for children. Canadian dietary surveys have previously indicated that exposures to contaminants through food were generally below guideline limits and in some cases, had declined significantly over time (Health Canada, 1998a). In the last ten years, however, there has been greater attention placed on a broader range of environmental chemicals for which recent science indicates exposures for children, including through food, are of possible concern.

Contaminants and chemicals in food are varied and may appear in foods for different reasons. Substances used in agriculture or emissions from industry or vehicles may deposit on food crops. Crops and food animals may also become contaminated from persistent contaminants in the environment and transfer their accumulated load to people. Other contaminants may end up in food because they leach out of food containers such as plastic bottles or other containers, when they are heated. Some types of food packaging may also transfer contaminants to food. Table 1 depicts the child health concerns and varied food-related sources of exposure to the substances

reviewed by TPH. Exposures sources relevant to the child care setting are also identified separately.

Table 1. Health Concerns and Exposure Sources for Substances of Interest

Substance	Child Health Concerns	Exposure Sources
Lead	▪ Large body of literature (epidemiological, toxicological) demonstrating adverse effects at low levels ▪ No safe blood lead level in children has been identified ▪ Neurodevelopmental (lowered intelligence, behavioural problems, and poorer school performance) ▪ The vulnerable windows of exposure are *in utero*, infancy and childhood to early school years	**Possible Food-Related Exposure Source** ▪ Drinking water from homes with lead service lines and lead pipes ▪ Prepared drinks including infant formula made with tap water ▪ Typically found at low levels in all foods due to historical contamination **Relevance to Child Care Setting** ▪ Comparable to exposures at home ▪ Primarily through dust, consumer products, water and food
Mercury	▪ Large body of literature (epidemiological, toxicological) demonstrating adverse effects of mercury ▪ Neurodevelopmental (delayed developmental milestones and cognitive, motor, auditory, and visual deficits) best studied from prenatal exposure ▪ The most important vulnerable window of exposure to mercury is *in utero* ∴ concern to minimize exposure of women (including teenage girls) during childbearing years ▪ Young children are also considered to be at risk from mercury exposure	**Possible Food-Related Exposure Source** ▪ Fish consumption, especially from eating large predatory species such as shark **Relevance to Child Care Setting** ▪ Comparable to exposures at home ▪ Primarily through fish
Pesticides	▪ Pesticide exposure *in utero* or in infancy from parental occupational exposure or from household uses linked to: altered birth outcomes, cancers in childhood, neurodevelopmental impacts ▪ No study of health impacts of pesticides from food exposures ▪ Children given organically grown produce have lower exposure compared to those eating conventionally grown produce	**Possible Food-Related Exposure Source** ▪ Exposure to low levels of pesticides from eating conventionally grown fruits and vegetables, more common with imported produce **Relevance to Child Care Setting** ▪ Indoor and outdoor uses for pest control are generally larger exposure sources. ▪ Other home exposures (e.g. head lice treatments or pet flea treatments) not likely to be relevant to most child care settings

Table 1. Health Concerns and Exposure Sources for Substances of Interest (cont'd)

Substance	Child Health Concerns	Exposure Sources
Dioxin-like Compounds	▪ 2,3,7,8-TCDD characterized as a known human carcinogen; increased risks of cancer in adulthood is a concern ▪ Important vulnerable window of exposure for adverse effects on neurological and immune system, neonate development and endocrine disruption is *in utero*, ∴ concern to minimize exposure of young girls to these persistent substances before reproductive age ▪ Widespread low-level exposure seen in biomonitoring studies. Exposures have decreased over time since the 1980s with reduced environmental emissions	**Possible Food-Related Exposure Source** ▪ Exposure is largely through food ▪ Highest levels in fats from animal-derived foods – meat, poultry, fish, eggs and dairy products ▪ PCBs higher in predatory freshwater fish ▪ Levels declining since 1980s **Relevance to Child Care Setting** ▪ Comparable to exposures at home
Bisphenol A (BPA)	▪ Few human studies ▪ Developmental and reproductive effects at low levels (animal studies). ▪ Continuous widespread exposure to low levels seen in biomonitoring studies ▪ Routes and sources of exposure not fully understood ▪ Vulnerable period of exposure is *in utero* and during early infancy	**Possible Food-Related Exposure Source** ▪ Heating BPA-containing plastic baby bottles during infant formula preparation and from liners of infant formula cans ▪ Consumption of canned food, particularly soups and pastas **Relevance to Child Care Setting** ▪ Comparable to exposures at home (many sources)
Phthalates	▪ Limited epidemiological and adequate animal data ▪ Developmental and reproductive, respiratory and allergic effects ▪ Biomonitoring data show evidence of widespread low level exposure	▪ Found in many products including: some soft plastic toys, plastic floor coverings, scented personal care products ▪ Also present in dust which may be an important exposure route for toddlers **Possible Food-Related Exposure Source** ▪ Found in low levels in many different food items: meat, fish, dairy, vegetables ▪ Processed and high fat foods may have somewhat higher levels **Relevance to Child Care Setting** ▪ Comparable to exposures at home (many sources)

Table 1. Health Concerns and Exposure Sources for Substances of Interest (cont'd)

Substance	Child Health Concerns	Exposure Sources
PBDEs	▪ Little research examining health effects of PBDEs in humans – associations with lower birth weight ▪ Thyroid, hepatic, and neurological system effects (animal and in vitro studies) ▪ E.g. increased incidence of certain liver cancers; associations with hyperactivity, decreased habituation, learning and memory in rat and mouse studies ▪ Widespread exposure ▪ Human exposure has rapidly increased since 1980s	▪ PBDEs used in a wide range of products in the home (furnishings, carpets, electronics) ▪ Dust is the largest source of exposure for young children **Possible Food-Related Exposure Source** ▪ Foods high in animal fats including fish, meat and high fat dairy ▪ Health Canada dietary studies indicate that PBDEs unlikely to be of harm at the current concentrations in food **Relevance to Child Care Setting** ▪ Comparable to exposures at home (many sources)
Perfluorin-ated Compounds (PFCs)	▪ Reproductive and developmental impacts, cancer; suspected endocrine disruptors (animal studies) ▪ Few human studies; links to lower birth weight, lowered sperm quality ▪ Widespread in the environment; some persist, bioaccumulate ▪ Routes and sources of human exposure not fully understood ▪ Widespread human exposure to PFOA and PFOS seen in biomonitoring data; declines in those voluntarily phased out of production and use	▪ Primary source of exposure is likely through foods due to widespread environmental contamination **Possible Food-Related Exposure Source** ▪ Found in low levels in many different food items: meat, fish, dairy, vegetables ▪ Appear in small amounts in some foods with certain grease-resistant paper packaging (e.g. microwave popcorn; fast food french fries and sandwich wrappers, frozen pizza paper liners) ▪ Health Canada dietary studies indicate that PFCs unlikely to be of harm at the current concentrations in food ▪ Non-stick pan surfaces may have small amounts of residues when new but not seen to be a major source of PFCs in foods ▪ Using non-stick pans on high heat (>350^{o}C) may release harmful fumes and degrade the surface coating **Relevance to Child Care Setting** ▪ Comparable to exposures at home (many sources)

The following sections of the report outline the findings of the science evidence review. The scientific information base for substances included in the review suggests that not all are of equal significance to children's health. In addition, while the evidence for impacts on children from some substances may be compelling, food may not be the most important route of exposure.

2.2 Lead

Lead is a well known and well studied neurotoxicant that can harm the developing nervous system. At low levels, lead affects the intellectual and cognitive development of a child, their behaviour, ability to concentrate and irritability. Effects have been demonstrated at exposure levels as low as 2 µg/dL in blood (Jusko et al., 2008; Braun et al., 2006). No threshold for lead effects has been demonstrated (Lanphear et al., 2005; Canfield et al, 2003) and a steeper dose-response curve has been found at blood lead levels below 10 µg/dL (Lanphear et al., 2005).

Exposure to lead is greatest for children ages 1 to 3 when hand to mouth behaviour is the most frequent. Children are believed however, to be at greatest risk from the harmful effects of lead up to age 6 (Cecil et al., 2008; Hornung et al., 2009). This is due to their greater absorption of lead and distinct vulnerability because of their developing brains and nervous systems. Children with inadequate calcium, protein or iron in their diet will also absorb greater amounts of lead, therefore adequate nutrition is important. Ensuring children have an optimal diet with regular meals, low- to modest-fat content and in particular, adequate intake of calcium and iron, reduces susceptibility to lead toxicity (Mahaffey, 1995).

There are small amounts of lead in our food; however, levels have been decreasing[1] since the phase out in the early 1990s of leaded gasoline in Canada, which reduced the widespread environmental contamination (ATSDR, 2007). Lead-solder for food cans, another source of lead in food, has also been substantially eliminated; however some imported foods may still be packaged in lead soldered cans (Health Canada, 2009a).

Lead can enter drinking water from lead service lines, from solder containing lead or from brass fixtures. Older plumbing, in Toronto homes built prior to 1955, may include lead pipes or may still have lead service lines. The use of lead solder for incoming water pipes was banned in Ontario in 1989 (MTTHU and SRCHC, 1995). On June 7, 2007, the Government of Ontario introduced more stringent requirements (O. Reg 243/07) for flushing and testing of drinking water taps in schools, private schools and child care centres (also called “day nurseries”) in response to renewed concerns about potential lead exposure from Ontario drinking water systems. The regulatory requirements for child care centres for example require that facilities a) flush their pipes either weekly or daily and b) perform regular testing for lead in drinking water (MOE, 2007a; MOE, 2009). In November 2009, the MOE amended the regulation to require annual drinking water testing by day nurseries with newer plumbing (post-1989) which were previously exempt from testing requirements (MOE, 2009). If lead levels in drinking water are elevated, the local Medical Officer of Health will assist the facility in determining ways to reduce lead levels and in monitoring the safety of the drinking water (MOE, 2007b).

Where there is some concern is in older homes where lead service lines may still be in place. The City of Toronto is currently engaged in an accelerated long term initiative to replace lead service pipes to homes that were built before 1955 but this process has been slated to take nine years (Toronto Water, 2007) and only applies to the public segment of the service line. In the fall of 2009, Toronto Public Health and Toronto Water distributed over 200,000 brochures to

[1] Since 1969, Health Canada has been testing the lead level in food available in markets across Canada through the Total Diet Study; available at: http://www.hc-sc.gc.ca/fn-an/surveill/total-diet/index-eng.php.

households suspected of having lead service lines, bringing greater awareness to the issue of lead in drinking water and to lead service line replacement (both the public and private segments of the line) as the most effective long-term strategy to reduce lead in drinking water. For households in older homes where there are pregnant women or children under age 6 however, TPH is encouraging these families to use an end-of-tap water filter that is certified to remove lead in the interim (TPH, 2009). This is particularly important if they are preparing artificial baby milk (i.e. infant formula) or beverages for young children. TPH has also incorporated detailed messages about ways to reduce exposure to lead in drinking water into its infant feeding and infant formula preparation factsheets used by public health staff. TPH is also disseminating information on other ways for the public to minimize their own or their children's exposure, with a focus on pregnant women and parents. For example, in revisions to the "Hidden Exposures" fact sheets series for prenatal educators, other service providers and the public, the section on lead is being updated to reflect this new information on lead exposure reduction measures. In the spring/summer of 2009, Toronto Public Health held training sessions with over 100 public health inspectors, public health nurses and other staff on sources of lead exposure and ways to support the public to reduce their exposures to lead.

In summary, lead levels in food have been decreasing since the restrictions on lead in gasoline and in solder for food cans. Lead levels in food are generally low unless imported canned foods are a component of the diet. Following recommendations for an optimal diet as described in *Eating Well with Canada's Food Guide*, particularly ensuring that children have adequate dietary calcium and iron intake, can mitigate the impacts from lead. Lead in drinking water can be a concern where there are still lead service lines such as many houses in Toronto older than 1955. The Ontario Ministry of Environment has new requirements for flushing and testing drinking water that apply to all licensed child care centres and schools. The City of Toronto is working to address the replacement of lead service lines which contribute the most to lead levels in drinking water. Given this initiative will take a number of years, TPH is communicating ways to minimize lead exposure from all sources with a focus on homes with those most vulnerable to impacts from lead.

2.3 Mercury[2]

Mercury in the environment from natural and human sources is transformed through the action of microbes to an organic form of mercury called methylmercury. Methylmercury is a persistent substance which concentrates in organisms found in the aquatic food chain. It can be harmful to the developing brain and nervous system and is another well studied neurotoxicant. Low-level exposure to methylmercury has been associated with problems with attention, fine-motor skills, language development, visual-spatial abilities and verbal memory among children who were exposed while in the womb (NRC, 2000). Therefore the main concern is to avoid or minimize exposure for pregnant women. TPH and other agencies such as Health Canada and the United States Environmental Protection Agency (US EPA) also recommend minimizing exposure to infants and young children because they are also at risk from mercury exposure.

[2] Toronto Public Health reported on a lengthy review of the literature on toxicity and exposure to methylmercury in fish in a 2006 report titled, *Fish Consumption: Benefits and Risks for Women in Childbearing Years and Young Children* found at*:www.toronto.ca/health/hphe*.

The most important and largest source of methylmercury exposure to people is from eating fish and shellfish (Mahaffey 2004). Methylmercury content in fish varies depending on species and the size of the fish. It is highest in large, long-lived predatory fish like shark and swordfish among others. Health Canada and other agencies generally recommend that vulnerable groups restrict how much they eat of these high mercury fish (e.g. see Health Canada 2008a & b; US DHHS/EPA, 2004). Canned tuna is among the most commonly consumed fish in Canada and is a popular choice for children. White canned tuna (made from the Albacore tuna species) is generally much higher in mercury than light canned tuna which is usually made from the Skipjack tuna species (TPH, 2006). TPH considers light canned tuna to be a low mercury fish species acceptable to serve to young children up to two Canada Food Guide servings[3] per week. TPH recommends that people chose "light" over "white" canned[4] tuna when feeding their children.

The 2008 TPH *Guide to Eating Fish for Women, Children and Families* carefully balances messages about the nutritional benefits of fish while letting people know about risks from contaminants and how to minimize these. This resource focuses on the most vulnerable subgroups in the population and encourages consumption of a variety of fish species, while pointing towards the fish species with lower levels of mercury. The guide also identifies fish that have high levels of omega-3 fats and those that may be caught or farmed in a way that is harmful to the environment.

In summary, the largest food source of methylmercury is fish. TPH has studied this issue extensively. The 2008 TPH *Guide to Eating Fish for Women, Children and Families* provides advice on fish consumption for children and other vulnerable subgroups in order to minimize mercury intake while still allowing fish to be included in the diet for its nutritional benefits.

2.4 Pesticides[5]

Pesticides are chemical substances that are used to kill living organisms such as noxious plants, weeds, insects, mould or fungi. Hundreds of different pesticides are used in agriculture and for horticultural (i.e. lawn and garden) purposes. The term "pesticides" therefore refers to a range of many different individual chemicals designed for different purposes and having differing degrees of toxicity.

In recent years, the health effects in children from exposure to pesticides have been studied to a much greater extent, particularly since a 1993 report of the U.S. National Research Council drew attention to the greater vulnerability and exposure to pesticides for children.[6] Evidence from

[3] A Canada Food Guide serving is 75 grams or 2.5 grams or about half a cup. A typical can of tuna is 120 grams (drained weight) which equals 1.6 Canada's Food Guide servings (TPH, 2008).

[4] Bisphenol A (BPA), discussed in section 2.6, is a substance of concern that is used in the epoxy resin lining of food cans. Available data on the average levels of BPA found in cans of tuna indicate that it is present in levels that are low compared to other canned foods. Toronto Public Health considers canned tuna an acceptable and healthy food source for children. Mercury levels should guide the choice of how much light and which (light vs. albacore) canned tuna to serve children rather than concerns about BPA in cans.

[5] Toronto Public Health has previously reviewed the science of exposure and health effects from lawn and garden pesticides in a lengthy report (TPH, 2002). This report updates the information since that 2002 review.

[6] The NRC (1993) study prompted considerable improvements to how children's vulnerabilities are included in the

many studies indicates an elevated risk of certain cancers or neurodevelopmental impacts in infants or children where there was parental occupational exposure to pesticides or exposure from using them in and around the home, especially during pregnancy, rather than from dietary exposure (Infante-Rivard & Weichenthal, 2007; Rauh et al, 2006; Eskenazi et al, 2007; Rosas et al, 2008).

The Canadian Food Inspection Agency (CFIA) monitors pesticide residues on imported and domestically produced food in Canada. Diet does not appear to be a relatively high source of exposure to pesticides for Canadian infants or children based on CFIA data. Pesticides residues are not detected in most foods sampled from Canadian markets (80% or better) and where residues were detected, exceedances of Maximum Residue Levels[7] (MRLs) are infrequent (less than 1% of fresh produce samples exceeded the MRLs) (CFIA, 2006). A 2005 CFIA study of processed[8] fruit and vegetable foods consumed by children ages 2 to 10 years found only 21 out of 594 samples (3.5%) had detectable pesticide residues, none over the MRLs (CFIA, 2005). A U.S. study showed that pesticide residues were lowest on fruits and vegetables that were organically grown[9], intermediate in those produced through integrated pest management (IPM) or certified as containing no detectable residues (NDR) and highest in conventionally grown produce (Baker et al, 2002).

Biomonitoring studies provide information about children's total exposure (that is, from all sources) to pesticides. Some exposure studies have shown that children have higher levels of exposure to certain insecticides such as pyrethroids, from home and garden uses rather than from diet (Lu et al., 2006; Fortin et al., 2008). Children who eat conventionally grown fruit and vegetable produce appear to have low-level exposure to certain agricultural-use insecticides as has been measured in some studies (Fortin et al, 2008). U.S. research indicates that a diet of organic produce reduces children's pesticide exposure measurably (Curl et al., 2002; Lu et al.,

---

ways that government departments, including Canada's Pest Management Regulatory Agency, test, regulate and monitor pesticides. The progressive, child-protective elements of the Pest Control Products Act (PCPA), the law that regulates pesticides in Canada, are discussed in Chapter Six of Environmental Threats to Children (TPH, 2005).

[7] Health Canada determines the maximum amount of pesticide residues, "that are expected to remain on food products when a pesticide is used according to label directions, (and that) will not be a concern to human health" (Health Canada, 2009b: page1). These values are then legally established as maximum residue limits (MRLs) which are now specified and regulated under the PCPA. As of September 2008, the PMRA had completed re-evaluations on 75% of 401 pesticide active ingredients registered before 1995. This re-evaluation program began in 2005. This process has resulted in the discontinuation or phasing out of registrations for over one hundred pesticide active ingredients which were either discontinued by registrants or did not meet requirements under the new PCPA, or both. The process of updating the individual pesticide MRLs is proceeding in tandem. Health Canada states that the re-evaluation program and revisions to the MRLs are applying modern assessment approaches including consideration of exposure risks in sensitive segments of the population, such as infants, children, pregnant women and the elderly; and combined exposure from dietary, residential and drinking water sources. As these assessment procedures continue to be modernized they will also be required to assess the risk of cumulative exposure to pesticides with a common mechanism of action.

[8] Items tested included fruit and vegetable juices, sauces and jams, frozen and canned fruits and vegetables, and condiments such as ketchup, relish, mustard and pickles. Of the 21 samples with detectable residues, 14 were dried fruit bars.

[9] While U.S. organic produce was not always pesticide free, it had significantly lower residue levels and was less likely to have residues of multiple pesticides. Some pesticides may end up in organic (and conventional) produce from soil contaminated by past pesticide use or as spray drift from neighbouring non-organic farms (Baker et al, 2002).

2006; Lu et al, 2008; Fortin et al, 2008). It is not known however, whether serving children organic produce provides health benefits because it is unclear if the observed low level exposures are harmful to begin with. In other words, no studies have linked harmful effects in children from eating conventionally grown produce compared to organic produce. While there are clear benefits[10] in terms of environmental protection, maintenance of biological diversity and sustainability from organic agricultural practices, whether organically grown produce is nutritionally superior to conventionally grown foods is still debated in the literature (Winter & Davis, 2006; Bourn & Prescott, 2002; Zhao et al, 2006).

Among the ways recommended by government agencies to lower pesticide residue levels on fruits and vegetables is thoroughly washing and wiping all produce before eating (U.S. FDA, 1993; Krol et al, 2000; Health Canada, 2009c). A recent review indicates that in most cases large reductions in pesticide residues can be achieved from various processes, but particularly from washing, peeling and cooking foods (Kaushik et al., 2009).

Parents can also choose to purchase locally[11] or domestically grown fruits and vegetables when available as these generally have detectable pesticide residues less frequently and seldom exceed MRLs (CFIA, 2006). Serving children a variety of fruits and vegetables is also advocated as a strategy for lessening exposure to pesticide residues that may be more common on certain types of produce or foods. Finally, as an exposure reduction strategy parents can also integrate some organic produce when it is available or can be afforded.

In summary, there are a number of ways to reduce children's exposure to pesticides in the diet as outlined above. Washing, peeling and cooking conventionally grown produce can reduce intake. Choosing organic foods will minimize food-related exposure to pesticides. Purchasing organic foods is not a low cost option but other factors, such as the environmental benefits from ecologically sustainable organic practices, may influence the decision to serve organic. In addition, choosing local foods when feasible may not significantly reduce pesticide exposure, but it can reduce air pollution impacts from transportation of food and supports the regional economy. TPH maintains that parents should serve children a healthy diet with a variety of fruits and vegetables, choosing fresh over processed whenever possible. Canada's Food Guide emphasizes the importance for reducing the risks of certain cancers and heart disease by eating a diet that is rich in different vegetables and fruit (Health Canada, 2007a). Parents and child care centres may choose to serve organic when it is feasible or affordable.

[10] Organic agricultural practice avoids the use of chemical pesticides (insecticides and herbicides), synthetic fertilizers, sewage sludge or seeds originating from genetically modified organisms (GMO) for crops (Canadian Organic Growers, n.d. http://www.cog.ca/whatisorganic.htm). Organic practice also prescribes that animals are not given growth hormones or antibiotics, and that animal wastes, slaughter by-products or genetically modified organisms (GMOs) are not included in their feed.

[11] Purchasing local foods also can reduce the impact on the environment from greenhouse gas emissions (GHGs) of long distance transportation. Region of Waterloo Public Health has calculated that the environmental impact of 58 food items commonly eaten in the region was about 52,000 tons of GHGs from the nearly 4,500 miles they travelled (Waterloo Region Public Health, 2005). Given our heavy reliance on imported produce, including a portion of the organic produce available in Toronto, this is an important consideration.

2.5 Dioxin-Like Compounds

Dioxin-like compounds are a large group of structurally and chemically related substances that include polychlorinated dibenzodioxins (PCDDs, also known as "dioxins"), polychlorinated dibenzofurans (PCDFs, also known as "furans") and the ''dioxin-like'' polychlorinated biphenyls (PCBs) (Srogi, 2008). Dioxin-like compounds in the environment have different origins. Dioxins are by-products of certain processes; they come largely from incineration of municipal solid waste (Hylander et al., 2003; Chang & Lin, 2001, Chang et al., 2004) or hospital waste (Coutinho et al., 2006), but can also arise from uncontrolled burning of household waste, especially plastics, chlorine bleaching of paper and pulp, the manufacture of some pesticides (Chen, 2004) and from natural processes. PCBs were used predominantly as coolants and lubricants in electrical equipment. While they were never manufactured in Canada, production in the United States ceased in 1977 because of evidence of adverse environmental and human health effects from the manufacture, use and disposal of these chemicals. These compounds are ubiquitous in the environment.

Dioxin-like compounds vary widely in their toxicity. About 30 different types are considered to have significant toxicity (WHO, 2007). The most toxic compound, 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD), is classified by the International Agency for Research on Cancer (IARC) as carcinogenic to humans (IARC, 1997).

The health effects from dioxin-like compounds have been studied in animals and in people, generally from higher exposures (e.g. accidental or occupational) than would be experienced by the general population. There is limited evidence for impacts on pregnancy outcomes, such as being small for gestational age or preterm birth, with low level exposure to dioxin-like compounds (Wigle et al 2007). Studies of impacts from lower level, environmental exposures to dioxin-like compounds have largely focused on exposures during the prenatal period, rather than during childhood (Wigle et al 2007). Where postnatal exposure has been studied, it has focused on infants and exposure to dioxin-like compounds through breast milk. The only known effects in children from postnatal exposure (typically higher level) alone are chloracne, recurrent ear infections and reduced growth in height, however the evidence is variable (Wigle et al 2007).

There have been several birth cohort studies conducted in different parts of the world which have examined impacts from prenatal exposure to PCBs, the most extensively studied of the dioxin-like compounds. Prenatal PCB exposure is associated with an array of neurodevelopmental effects including abnormal reflexes among newborns, reduced motor skills among infants and cognitive deficits in early childhood (Jacobson et al., 2002; Wigle, 2003; Boucher et al, 2008). Children whose mothers regularly ate PCB-contaminated fish from Lake Michigan during and before pregnancy were followed through to age 11 years and the highest exposed in this sample showed poorer performance on various tests of IQ, reading and verbal comprehension, memory and attention (Jacobson and Jacobson, 1996).

Dioxin-like compounds are associated with increased risks of certain cancers in animals and people. They may act like cancer promoters or co-carcinogens (Irigaray et al., 2007). IARC and the U.S. EPA also conclude that PCBs are probable human carcinogens based on evidence from epidemiological studies (in adults but not in children) and experimental animal studies (Wigle et al 2007). There is evidence from *in vitro* and animal studies, and in small human studies, that

dioxin-like compounds are endocrine disrupters (Mocarelli et al., 2008). More research is needed to determine the long-term effects of low-level exposures to dioxin-like compounds on cancer risk, immune function, reproduction and development in humans (IOM, 2003).

Due to their chemical characteristics, dioxin-like compounds are persistent and they accumulate in the fatty tissues of plants and animals. For most people, including children, over 90% of exposure to dioxin-like compounds comes through the diet. Average Canadian dietary intake of dioxins, furans and similar substances is estimated by Health Canada to be 0.62 picograms (pg) per kg of body weight per day (Health Canada, 2005). This value is below the World Health Organization (WHO) guideline level of 2.3 pg/kg bw/day which is considered "tolerable" or not expected to produce health effects. Health Canada currently uses the WHO guideline for tolerable intake of dioxin-like compounds.

Based on 1999 Canadian Total Diet Study data the levels of dioxin-like compounds are highest in food items such as butter, higher fat dairy (such as milk and cheese), ground beef, eggs, wieners/sausages and organ meats (Health Canada, 2006a). Levels of dioxin-like compounds tend also to be higher in freshwater fish than in marine fish or shellfish. A 1999 study of Dutch preschool children examining the relative contribution of different foods to PCB and dioxin exposure found that dairy foods, meat and meat products and processed foods contributed the most to PCB and dioxin intake after weaning (Patandin et al 1999). The levels of dioxin-like compounds in different food groups vary internationally.

Dioxin-like compounds can be measured in the tissues of all people. A 1998 Health Canada exposure study among people in the Great Lakes basin indicated that exposure to dioxin-like compounds such as PCBs was generally higher in infants, children and adolescents than adults at that time (Health Canada, 1998b). The proportionately greater intake of food per unit of body weight of children compared to adults and their greater intake of certain types of food, such as milk, explain the higher exposure measures for younger age groups (Wigle, 2003).

Human body burdens of dioxin-like compounds have continued to decline over time since several countries took regulatory actions to reduce their release to the environment. For example, there has been a 60% decrease in the overall release of dioxins and furans from Canadian sources since 1990 (Health Canada, 2005). Studies of dioxin and furan levels in Canadian breast milk show that the levels decreased by about 50% from the 1980s to the 1990s (Health Canada, 2005). Longterm U.S. biomonitoring data indicate that serum dioxin levels in people have decreased 80% since the 1980s (CDC, 2003). The most recent Canadian data, from the 2008 Alberta Biomonitoring Program, indicate that serum levels of dioxin-like compounds in pregnant women in Alberta were below those measured in U.S. studies (Government of Alberta, 2008).

While the exact risk from intake of low levels of dioxin-like compounds in the diet is not clear, reducing exposure can be achieved through dietary choices and food preparation practices (IOM, 2003; Health Canada, 2005). For example, trimming visible fat, removing the skin from meat or fish and cooking using methods that reduce fat (such as oven broiling) can reduce intake of animal fats. Reducing overall intake of saturated fats such as butter, reducing reliance on high fat animal foods such as eggs, organ meats, wieners and sausages, and including lower fat versions of dairy products such as milk and cheese in a child's diet are also important strategies. The

exception to this advice is the position, currently supported by agencies such as the Canadian Paediatric Society and Health Canada, recommending that a high fat diet (~50% of energy from fat) is appropriate for children up until they are two years of age (see Roy et al, 1995). Choosing marine, canned fish or shellfish over freshwater fish more often is also an exposure reduction strategy. Fruits, vegetables and grains contain lower levels of dioxins, which in some cases can be reduced by washing or peeling (IOM, 2003). Eating a balanced diet, with adequate amounts of fruits, vegetables and grains, therefore, also helps to avoid excessive exposure from a single source (WHO, 2007; Health Canada, 2005). The U.S. Institute of Medicine suggests that such strategies should be a priority for reducing exposure in girls and young women in the years before they become pregnant (IOM, 2003). While breastfeeding can be a large source of dioxin-like compounds for breastfed infants, the many benefits of breastmilk outweigh and in fact, mitigate the risks of contaminant exposure (Landrigan et al, 2002; TPH, 2005; Jorissen, 2007). Finally, households can individually reduce their own contribution to dioxins in the environment which end up in food by not burning residential waste (Health Canada, 2005).

In summary, dioxin-like compounds are persistent substances that are ubiquitous in the environment. Food is the major source of exposure to these substances. While they can be measured in humans globally, levels have decreased in recent decades as regulation to ban their production or limit their releases to the environment has come into place in industrialized nations, including Canada. Health Canada, the U.S. Institute of Medicine and the World Health Organization provide dietary recommendations that can reduce the public's exposure to these persistent compounds. While the precise health effects from children's exposures to dioxin-like compounds need to be better characterized, exposure reduction measures such as reducing intake of saturated fats, red meat and processed foods can be taken to reduce their exposure. Such advice is consistent with healthy eating guidelines, therefore TPH supports these practices as a way to minimize any potential risks.

2.6 Bisphenol A (BPA)

BPA is produced worldwide and used in many consumer products including polymers, resins, dyes, flame retardants and dental sealants. It is used in the lining of food cans and in hard plastic food and water containers. It has been detected in all media, including indoor air, water, dust, and soil (Vanderberg et al., 2007).

General population exposure to BPA is primarily through the diet, but can also be through skin absorption and inhalation of airborne dust. BPA can leach from the epoxy linings of canned foods and polycarbonate water and baby bottles (recycling symbol number 7) into the foods and liquids that they contain. People can also be exposed following treatment with BPA-containing dental sealants. Not all the sources for children's exposures to BPA are known, although food and liquids are presumed to be primary sources. Wilson and colleagues (2007) studied children's exposure to BPA at child care centres and at their homes. They estimated that 99% of the exposure to BPA was from children's diet. Wilson et al., (2007) measured BPA in over 83% and 68% of the children's solid and liquid foods, respectively.

BPA is used in an epoxy resin as the inner coating of food cans to prevent rusting and corrosion. Several studies have explored the conditions that support or enhance leaching of BPA from the

linings of cans. Health Canada's recent exposure assessment found that children ages 1 to 4 years have the highest levels of exposure to BPA, with the majority of this exposure coming from consumption of canned foods (Health Canada, Environment Canada, 2008). The U.S.-based Environmental Working Group (EWG, 2007) tested approximately 100 canned foods and beverages for BPA. Fifty seven per cent (57%) of all the canned food had detectable levels of BPA (range: non detect to 385 ppb; average: 7.9 ppb), however there was considerable variability in levels measured both within and among the different types of canned food, with soups and canned pastas having the highest levels.[12] In the spring/summer of 2008 Health Canada supplemented this dataset and tested BPA in bottled water (average of 1.5 µg/L), canned drinks (average of 0.57µg/L), and baby food products pre-packaged in glass containers with metal lids (average of 0.95 ppb) (Health Canada, 2009 d, e & f).

Health Canada has also assessed BPA levels in infant formula available in Canada finding concentrations ranging from 2.27 to 10.2 ppb (Health Canada, Environment Canada, 2008).[13] Health Canada (2008c) estimates that Canadian infants who are fed infant formula consume an average of 0.2 to 0.5 ug/kg bw/day from canned liquid formula with a worst case scenario estimate of up to 1.35 ug/kg bw/day.

Health Canada also conducted studies on the amount of BPA that leaches out of plastic baby bottles and found this to vary depending on the bottle, the temperature to which it is heated and the length of time that the bottle is heated (Health Canada, Environment Canada, 2008).[14] A series of experimental leaching studies using BPA-based plastic baby bottles found a range of BPA levels (< 0.15 to 8.323 ppb) in the liquid contained therein (Health Canada, Environment Canada, 2008).

Biomonitoring studies show that almost all people sampled in developed countries have detectable levels of BPA in their bodies (Calafat et al, 2005). Many scientists state that current public exposure to BPA, as reflected in biomonitoring studies, is in the range that has been shown to cause adverse effects in laboratory animals (vom Saal, et al., 2007). While BPA is thought to be rapidly detoxified and excreted by adults, the ability to detoxify BPA is not fully developed in the fetus and newborns, resulting in higher levels of the toxic unconjugated or "free" form of BPA in their tissues (Health Canada, Environment Canada, 2008; NTP-CERHR, 2008). Recent research indicates that fetal tissues may in fact also de-conjugate BPA back to the toxic form (Ginsberg and Rice, 2009). These studies challenge the prevailing assumption that fetal and newborn exposure to BPA is insignificant due to rapid conjugation of BPA in pregnant women and newborns (Ginsberg and Rice, 2009).

Health Canada's assessment of BPA concluded that based on existing animal studies, exposure to BPA can affect reproductive, neurological and behavioural development (Health Canada,

[12] Health Canada's review of this information concluded that the EWG data on canned foods were appropriate to apply to Canada (Health Canada, Environment Canada, 2008).

[13] Follow up studies by Health Canada on liquid canned and powered infant formula available in Canada confirm that the liquid canned formula BPA concentrations ranged from 1.14 to 5.44 ppb, with an average of 2.88 pbb (Health Canada, 2008d); whereas, no BPA was detected in any of the powered formula samples (MDL = 0.13 ppb) (Health Canada, 2009g).

[14] Health Canada studies found that the amounts of BPA that leached did not differ substantially depending on whether the bottle was new or used or whether milk or apple juice was the heated liquid.

Environment Canada, 2008). The US National Toxicology Program similarly expressed some concern for the effects of BPA on development of the prostate gland and brain, and for behavioural effects in fetuses, infants and children (NTP-CERHR, 2008).

Epidemiological evidence is emerging that supports the animal studies. Human population studies are indicating evidence for an association between exposure to BPA and reproductive effects (Li et al., 2009; Takeuchi et al., 2004; Sugiura-Ogasawara et al., 2006; Hiroi et al., 2004); cardiovascular disease, diabetes, and liver enzyme abnormalities in adults (Lang et al., 2008); and behavioural effects in toddlers (Braun et al., 2009). These studies are limited and more research is needed before the relevance of epidemiological findings are completely understood. The US National Institute for Environmental Health Sciences recently announced a $30 million research program on the developmental effects of BPA as a first step to achieve this need. Based on the exposure assessment summarized above, Health Canada states that exposures to BPA for the general population, including newborns, infants and children, are well below the current provisional Tolerable Daily Intake (pTDI)[15] and therefore are not expected to pose a health risk. This conclusion is also supported by assessments conducted by the U.S. Food and Drug Administration (FDA) (U.S. FDA, 2009).

Nonetheless, the Canadian federal government stated that, as a precaution, in light of the uncertainty about sources and toxicity of BPA, and in recognition of the sensitivity to newborns and infants to BPA, they would take steps to ensure that exposures for newborns and infants are minimized. The government is currently drafting regulations to prohibit the importation, sale and advertising of polycarbonate baby bottles that contain BPA along with taking action to limit BPA releases to the environment (Government of Canada, 2008). The federal government is also working with manufacturers to continue efforts on the general principle of ALARA (as low as reasonably achievable) to reduce BPA in food packaging applications relevant to infants and newborns, including in can liners, and to explore setting migration targets for canned foods in general.

Therefore, based on a review of the available science, TPH recommends that as a precaution, parents and service providers should reduce infant exposures to BPA through feeding practices where feasible. This can be achieved by exclusive breastfeeding up to age six months (followed by the introduction of appropriate complementary foods and continued breastfeeding for up to two years and beyond) as the healthiest and safest method of feeding infants. TPH also recommends avoiding heating foods or liquids in polycarbonate plastic bottles or containers (that is, those labeled with recycling symbol number 7). This is particularly important where infant formula or expressed breast milk is served in plastic baby bottles. Parents can choose alternatives to plastic baby bottles such as glass, or BPA-free plastic, but if plastic bottles (either BPA-based or BPA-free) are in use, parents and service providers should follow the advice of TPH and Health Canada on how to prepare and warm infant formula or breast milk safely.

[15] The current Canadian reference level (developed in 1996), known as the pTDI, is 25 micrograms per kilogram of body weight per day (ug/kg bw/day) (Health Canada, 2008c). This value is half of the toxicological reference values applied by agencies in the U.S. or Europe. However, these reference levels do not incorporate recent evidence of the low level effects of BPA. Scientists argue that the toxicological reference values are inadequately protective of health (vom Saal and Hughes, 2005).

On the question of reducing exposure to BPA from canned foods in a child's diet, TPH acknowledges that canned food can be an important, and for some (e.g. food bank users) possibly the only, source of nonperishable protein, vegetables and fruit, when refrigeration or fresh or frozen alternatives are not available. While fresh or frozen foods are preferable to canned or highly processed versions, it is not always feasible for the individual to avoid their use. Federal government actions to reduce or remove BPA from canned food liners are seen as the more appropriate approach to further minimize risks from BPA in this context. The Medical Officer of Health included a recommendation to that effect in comments submitted in June 2008 on the government's 2008 Draft Screening Assessment for Bisphenol A to then Minister of Health Tony Clement.

TPH has already incorporated the detailed messages about ways to reduce exposure to BPA into its infant feeding and infant formula preparation factsheets used by public health staff. TPH is also disseminating information on other ways for the public to minimize their own or their children's exposure, with a focus on pregnant women and parents. For example, in revisions to the "Hidden Exposures" fact sheets series for prenatal educators, other service providers and the public, the section on plastics is being updated to reflect this new information on BPA exposure reduction measures.

2.7 Phthalates

Phthalates are a class of synthetic chemicals used in many industrial applications, consumer products, personal care products, and building materials. Some phthalates are used to make polyvinyl chloride (PVC) plastic more flexible and are found in PVC flooring and wall coverings, vinyl shower curtains, cable, wire, and toys, among other uses. Phthalates are used in personal care products, particularly fragranced items like some lotions or perfumes. Although PVC plastics often contain phthalate plasticizers, the American Plastics Council states that phthalates are not used in plastic beverage bottles, plastic food wrap, food containers or any other type of plastic food packaging (Enneking, 2006). A different plasticizer which is not a phthalate, diethylhexyl adipate (DEHA), is commonly used in plastic wrap and containers.

Phthalates are ubiquitous in the environment. They are released into the environment during the manufacture, use and disposal of products. They are not covalently bound into plastics therefore, they do not stay bound within the products in which they are used. They have been measured in the majority of people in biomonitoring studies (Silva et al., 2004). Phthalates do not bioaccumulate but are rapidly broken down in the human body and excreted (Hauser and Calafat, 2005; Wormuth et al., 2006). It is believed that the metabolites of phthalates are more toxic than the parent compounds (Wittassek and Angerer, 2008). Children have higher levels of phthalates and their metabolites in their bodies compared to adults. The reasons for this are not completely understood (Wittassek and Angerer, 2008).

The sources and pathways of exposure to phthalates are not completely understood and vary depending on the type of phthalate. For infants and toddlers exposure may be largely from mouthing plastic objects, or from dust, food or personal care products (Wormuth et al., 2006). The origins of phthalates in foods are diffuse because they are widespread environmental

contaminants. Thus, there is little that consumers can do to reduce their exposure to phthalates in food (Wormuth et al., 2006).

Studies have found that where there are high concentrations of specific phthalates measured in dust, or PVC or vinyl flooring, there is an associated increased incidence of asthma, respiratory symptoms and allergies in children and adults (Bornehag et al., 2004; Jaakkola et al., 2000; Jaakkola & Knight, 2008). Animal test studies and epidemiological studies also indicate that prenatal exposure to phthalates has anti-androgenic and estrogenic effects, particularly on the male reproductive system (Swan et al., 2005; Fisher et al., 2004; Foster, 2006). Hauser and Calafat (2005) note that further epidemiological study on reproductive effects and allergic and airway symptoms are needed.

The federal government is in the process of banning di(2-ethylhexyl) phthalate (DEHP) from products, such as teethers, toys and baby bibs, intended for young children, replacing the current voluntary ban (Health Canada, 2008e). Health Canada monitors phthalates in teethers and rattles, and advises parents and caregivers to restrict children from mouthing PVC toys not intended for sucking and chewing for extended periods.

As phthalates are ubiquitous in the environment, including in food, there are few effective exposure reduction strategies for the public, including child care providers and parents. Strategies to protect children from exposure to phthalates should focus on federal and provincial government restrictions on the use of these chemicals in products, reducing their use in general, and in improving industrial processes and waste disposal.

2.8 Polybrominated diphenyl ethers (PBDEs)

Polybrominated diphenyl ethers (PBDEs) are a class of flame retardants that have been used in consumer products since 1960 (Snedeker, 2007). They are structurally similar to PCBs. They can be found in products containing polyurethane foam, such as couches and mattresses, and in electronics (La Guardia et al., 2006). As PBDEs are released from products like electronics (particularly older electronics) and furniture, they are commonly found in indoor air and dust.

The average PBDE levels measured in North Americans are much higher than concentrations reported in Europeans or Asians and they have increased substantially with time (Hites et al., 2004; Johnson-Restrepo et al., 2007; Environment Canada, 2006). Health Canada (2006b) indicates however, that current levels of PBDE exposure in Canadians are well below those seen to produce health effects in animals.

Although not all the routes of exposure to PBDEs for people are fully understood, PBDEs in food may be important for adult exposure. Data from a 2002 study in Vancouver indicate that fish, meat and butter contain the highest concentrations of PBDEs among the foods sampled. Health Canada states however, that the PBDE levels found currently in any retail foods are not a health concern (Health Canada, 2006b).

Breastfed infants are the highest exposed because of the transfer of PBDEs through human milk fat. Breastfeeding continues to be considered the healthiest way to feed infants nonetheless

(Landrigan et al, 2002; TPH, 2005; Jorissen, 2007). Unintended ingestion of indoor house dust however, is estimated to represent up to 93% or more of Canadian toddler exposure to PBDEs because of their frequency of mouthing events and the great amount of time they spend in close proximity to the floor (Wilford et al., 2005; Jones-Otazo, et al., 2005). Ingestion of dust may also be the major exposure pathway for all life stages except infancy (Jones-Otazo et al., 2005).

Animal and *in vitro* studies show that PBDEs affect the thyroid, hepatic and neurological systems (Costa et al, 2008). Specifically, PBDEs reduce serum T4 levels, increase liver weights and the incidence of hepatocellular carcinomas, and they are associated with hyperactivity, poorer habituation, learning and memory in rat and mouse studies (Costa and Giordano, 2007). There is very little research examining health effects of PBDEs in humans although some Canadian research is underway at the University of British Columbia (UBC, n.d.).

The Government of Canada's Polybrominated Diphenyl Ethers Regulations (PBDE Regulations) came into force on June 19, 2008. These regulations ban the manufacturing of PBDEs and the use, sale or offer for sale, of two types of PBDE mixtures (containing three forms known as tetra, penta and hexaBDE) that had already been voluntarily withdrawn by manufacturers. On March 27, 2009 the Government of Canada announced a proposal to also ban nona-, and deca-BDE from electronics under these regulations (Environment Canada, 2009). These actions were taken on the basis of environmental impacts but will also have the effect of reducing exposure to people in the longterm. Because PBDEs are in so many products commonly used in our society however, they will continue to be a contaminant of concern for some time.

Strategies to reduce exposure to PBDEs, particularly from the diet, are limited because of their widespread use and presence in the environment. Health Canada (2006b) recommends however, that eating lower fat meat, dairy[16] and fish can minimize exposure to PBDEs.

2.9 Perfluorinated Compounds

Perfluorinated compounds (PFCs) are a large class of fluorine-containing substances that are used to make various materials grease, stain and water repellent. They are used in many different consumer items such as carpets, fabrics, electronics, some personal care products and food packaging (Midasch et al., 2006; Kissa, 2001). They are also emitted to the environment from manufacturing processes. A number of the PFCs are very persistent, bioaccumulate and have been found in people and wildlife across the globe, which explains recent attention and action concerning these chemicals. Two particular PFCs, perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) are of greatest concern because of their toxicity, high persistence and widespread abundance in the environment and in people (USEPA, 2009 a and b; Health Canada, 2007b). Few studies have looked specifically at the exposures of children, but they appear to be comparable to those for adults (Fromme et al., 2009).

In Canada, PFOS and related PFCs have been placed on the Schedule 1 Toxic Substances list under the Canadian Environmental Protection Act (CEPA 1999) because of concerns they pose risks to the environment and biological diversity. PFOS was also among nine new persistent

[16] Canadian Paediatric Society and Health Canada, continue to recommend however, that children be served full fat versions of milk products until they are two years of age (see Roy et al, 1995).

organic pollutants (POPs) recently added to the list of substances identified for international action under the Stockholm Convention on POPs in May 2009 (UNEP, 2009).

Based on animal studies, PFCs are known reproductive and developmental toxicants. They are also toxic to the liver and are possible endocrine disruptors (Lau et al., 2007; Jensen et al., 2008). Animal studies indicate that they cause certain cancers and the US EPA has classified PFOA as a known animal carcinogen (Jensen et al, 2008). Evidence of the health impacts in humans from PFOS and PFOA is currently inconclusive (US EPA, 2009a). While studies of workers exposed to high levels of certain PFCs provide some indication of increased risks for cancer of the bladder, the U.S. EPA has yet to decide on how to classify carcinogenicity to humans from PFOA[17] (US EPA, 2009b). Some human studies suggest a possible effect on human fecundity and an association with decreased weight at birth with exposure to PFOS or PFOA (Fei et al., 2009; Calafat et al., 2007).

Though all routes of human exposure are not fully understood, diet appears to be the main source of PFC exposure for Canadians (Tittlemier et al., 2007; Fromme et al., 2009). Exposure to PFCs through water and air appears to be very low for the general population (Fromme et al., 2009). Human exposure to certain PFCs (including PFOS and PFOA) appears to be declining based on recent population biomonitoring studies in the U.S. by the Centers for Disease Control & Prevention (CDC) (Calafat et al., 2007). A recent study of levels of four PFCs (including PFOS and PFOA) in newborns in New York State has similarly shown sharp declines in PFC blood levels since 2000 (Spleithoff, et al., 2008). These temporal trends likely reflect changes in the use of these substances in consumer products brought about by government actions and by voluntary industry phase outs of PFOA and PFOS. Health Canada research notes that current estimated exposure to PFCs of Canadians is not high enough to present a concern to health (Tittlemier et al., 2007; Health Canada, 2009h).

Research is incomplete as to all the food sources of PFCs, however a number of food items such as fish and seafood, meat, poultry, frozen entrées, fast food items, microwave popcorn, fruits, vegetables and dairy, have all been found to contain low levels of PFCs by both Canadian and UK scientists (Tittlemier et al., 2007; UK FSA, 2006). PFCs may end up in meat and fish from transfer through air, water or feed to the food-producing animal. As well, some paper food packaging treated to repel grease, such as microwave popcorn bags, french fry boxes, pizza boxes and fast food wrappers, can or may contain certain PFCs which in experimental studies appear to migrate in small amounts into food oil simulants and water (Begley, et al., 2005; Sinclair et al., 2007). The estimated levels of intake of PFCs by Canadians (age 12 years and older) are well below current toxicological reference values and are not likely to pose a risk to health from dietary exposure according to Health Canada research (Tittlemier et al., 2007). There is currently however, inadequate study of the dietary intake of PFCs by children (Fromme et al., 2009). Because PFCs are measured in many different types of foods and no single food item stands out as having uniquely high levels of PFCs, it is difficult to discern how exposure can be reduced effectively through individual dietary choices alone.

[17] In its peer review of a 2005 draft risk assessment the U.S. EPA's Science Advisory Board suggested that the PFOA cancer data are consistent with the EPA guidelines descriptor "likely to be carcinogenic to humans", however, EPA online documents indicate that they are evaluating additional research and have not yet made definitive conclusions as to the carcinogenicity of PFOA (US EPA, 2009b).

One type of PFC, polytetrafluoroethylene (PTFE) is used commonly as the coating in non-stick cookware. PTFE is described as being relatively non-toxic[18] and chemically inert (Jensen et al, 2008; Health Canada, 1998a). Because PFOA, a more toxic substance than PTFE, is used to make PTFE, new PTFE-coated cookware has been found to contain low level (ng/g or parts per billion) residues of PFOA in the coating as a contaminant from the manufacturing process (Begley et al., 2005). Studies examining the release of PFOA from use of non-stick pans are limited in number. U.S. and U.K. government scientists have concluded from tests of such cookware that the non-stick coating does not appear to be a significant source of PFOA; the levels in new cookware are too low to produce any detectable migration into foods; and, that amounts appear to decline with subsequent uses (Begley et al., 2005; Bradley et al., 2007). Some have measured PFOA in low amounts in air from heating pans to normal cooking temperatures (Sinclair et al, 2007; Consumer Reports, 2007). The amounts vary depending on the brand of pan but typically lessen with use of the pan. Heating pans with non-stick coating to high temperatures (above those that would cause oil to smoke or burn) can cause the PTFE resin to deteriorate (beginning above 260^{o}C) and at very high temperatures (about 350^{o}C) it can degrade to PFOA and release fumes that irritate the respiratory system (Health Canada, 2006c; Dupont, n.d.; Sinclair et al., 2007). There is no evidence to suggest that using a pan where the surface is scratched or flaking will increase exposure to PFC contaminants, however, one source suggests that "(f)laking can cause uneven heating that might accelerate emissions" (Consumer Reports, 2007: 7). Since 2006, the manufacturers of non-stick cookware have been reducing and aiming to eliminate PFOA content in their products as part of the US EPA PFOA Stewardship Program (US EPA, 2009b).

In summary, TPH concludes that PFCs are of concern because of widespread presence in the environment and in people and based on animal studies of their impacts on reproduction, development and on cancer risks. Industry and government actions appear to be reducing the amounts of key PFCs in the environment. Because there is low level exposure to PFCs in many foods and not all exposure sources for children have been adequately characterized, exposure reduction strategies are not specific, but are consistent with all the principles of healthy eating such as in *Eating Well With Canada's Food Guide*. Non-stick coated pans may contain small amounts of PFOA as a residue from manufacturing processes therefore as per the instructions from manufacturers, non-stick coated pans should be washed well before first use and in addition, should not be used on high heat (>350^{o}C) to avoid the formation of harmful fumes or degradation of the surface coating.

3.0 Recommendations for Exposure Reduction

TPH is recommending a best practices approach to help ensure that children's exposures to contaminants in the diet remain low. The recommended food or drinking water related exposure reduction practices specific to each chemical reviewed are found in Table 2. In many cases, the exposures to these contaminants through food occur because of widespread environmental

[18] See also the following Material Data Safety Sheet which describes PTFE itself as unlikely to present a hazard (unless in powdered form which is not likely to be encountered in normal use): http://msds.chem.ox.ac.uk/PO/polytetrafluoroethylene.html

contamination therefore the opportunities to reduce exposure are limited and do not differ whether children are at home or in a child care centre.

Table 2. Food or Water Exposure Reduction Practices for Chemicals of Interest

Substance(s)	Recommended Practices
Lead in drinking water	Follow Ministry of Environment requirement for drinking water for school, private schools and day nurseries including daily flushing if all or part of the plumbing that serves the building was installed before January 1, 1990 and drinking water testing at least once each year for all such facilities (See:http://www.e-laws.gov.on.ca/html/regs/english/elaws_regs_070243_e.htm) In addition, if the child care centre is situated in a house built before the mid-1950s where the lead service line has not been replaced, filtered tap water should be used to prepare artificial baby milk (i.e. infant formula) and when preparing drinks for children under 6 years of age (See TPH 2009 Lead in Drinking Water fact sheet http://www.toronto.ca/health/lead/drinking_water.htm)
Mercury in fish	Follow advice for children in TPH's *Guide to Eating Fish for Women, Children and Families* including: • Choose low mercury, high omega-3 fat fish • Avoid high mercury fish • Choose light over white canned tuna • Choose a variety of fish species
BPA	Choose fresh or frozen foods whenever possible. Avoid microwaving/heating food or beverages in plastic containers. Prepare infant formula with boiling water and allow to cool before adding to plastic bottles. If using plastic bottles to store breast milk or infant formula, re-warm using warm water rather than the microwave.
Pesticides	Where appropriate wash (or peel/trim) fruits and vegetables before giving to children Integrate some organic and choose locally or domestically grown produce when available or affordable
Perfluorinated Compounds (specifically PFOA)	Non-stick coated pans should not be used on high heat (>350^{o}C)
Pesticides Dioxin-like Compounds Phthalates	Follow recommendations in *Eating Well With Canada's Food Guide* for: • Number of servings of a variety of fruits and vegetables • Number of servings for lean meat and meat alternatives, and dairy • Reducing high fat meat and dairy recognizing the higher fat needs of children under age 2; trimming fat from meat and fish • Cooking using methods that reduce saturated fat

This review identifies several best practices that can minimize potential risks from food- or food-container related exposures including: avoiding processed foods of low nutritional quality; serving fresh or frozen foods when feasible; reducing high fat meat and dairy, recognizing the nutritional need for full fat dairy for children under two years of age, and the nutrition benefits of higher fat fish such as salmon; using cooking methods that reduce saturated fat; and, avoiding heating food or drink in plastic containers. Existing TPH messages about eating low mercury fish and flushing drinking water to reduce lead are also important for reducing exposure.

4.0 Conclusions

This review of the evidence for exposure and health impacts indicates that scientific knowledge for individual substances is highly variable. The effects on the developing child from some substances, such as lead and mercury, are well known. Pesticide exposure from home pest control is also known to be associated with health impacts in young children. Effects from dioxin and dioxin-like compounds are also well studied in people who had high exposures or from low level prenatal exposures and while food is the most common source, the levels in food have declined in the last couple of decades. On the other hand, health effects from substances like BPA, PBDEs, phthalates and PFCs are known only or mainly from animal studies. However, exposure studies indicate that most people carry traces of these compounds in their bodies beginning at an early stage of life and that the effects on living organisms or the environment are of potential or actual concern.

The reasons for contaminants being present in food differ, depending on the specific substance. Some persistent substances, for example, dioxin-like compounds or lead, are present in food due to historical environmental contamination. Substances such as phthalates or PFCs may be found in food because of current releases to the environment. In the case of these widespread environmental contaminants, the ability to reduce children's exposure through dietary choices differs depending on the nature of the substance and whether a specific dietary source can be identified. Food containers, food processing practices, packaging and cookware may also contribute in varying degrees to the presence of these substances in food. In some cases, such as for BPA and PFCs, not all the sources for children's exposures are known, although food is presumed to be a main source. Food exposures to pesticides are comparatively much lower than the exposures that are linked to health effects in children such as parental occupational or household uses.

Reducing food-related exposure to contaminants is a complex and challenging issue. Actual exposure data for children in Toronto is not available and it is neither practical nor feasible to gather such data to inform specific recommended actions. As a result, TPH is recommending a best practices approach to help ensure that children's exposures to contaminants through diet remain low as a precaution.

In formulating the set of recommended practices above, TPH considered the severity of the possible health concerns, the potential for exposures in a child care setting, and the feasibility and opportunities to reduce those exposures through reasonable and sustainable actions. The recommended practices also balance nutrition and health benefits against any potential risks.

This review identifies a number of best practices, including several recommendations from *Eating Well with Canada's Food Guide*, that can minimize potential risks from food- or food-container related exposures in a child care setting including: avoiding processed foods of low nutritional quality; reducing overall consumption of high fat meat and dairy (recognizing the nutritional need for full fat dairy for children under two years of age and the nutrition benefits of higher fat fish such as salmon); using cooking methods that reduce saturated fat; serving fresh or frozen foods when feasible; and, avoiding heating food or drink in plastic containers. Existing TPH messages about eating low mercury fish and flushing drinking water to reduce lead are also important for reducing exposure.

References Cited

ATSDR, 2007. Toxicological Profile for Lead. Department of Health and Human Services. Public Health Service. *Agency for Toxic Substances and Disease Registry*. August 2007

Baker BP, Benbrook CM, Groth (III) E, Lutz Benbrook K. 2002. Pesticide residues in conventional, IPM-grown and organic foods: Insights from three U.S. data sets. *Food Addit Contam* 19(5): 427-446. Available at: http://www.consumersunion.org/food/organicsumm.htm

Begley TH, White K, Honigfort P, Twaroski ML, et al. 2005. Perfluorochemicals: potential sources of and migration from food packaging. *Food Addit Contam* (10): 1023-31

Begley TH, Hsu W, Noonan G, Diachenko G. 2008. Migration of fluorochemical paper additives from food-contact paper into foods and food simulants. *Food Addit Contam* 25(3): 384-390

Bornehag CG, Sundell J, Weschler CJ, Sigsgaard T, et al. 2004. The association between asthma and allergic symptoms in children and phthalates in house dust: a nested case-control study. *Environ Health Perspect* 112: 1393–1397

Bourn D, Prescott J. 2002. A comparison of the nutritional value, sensory qualities, and food safety of organically and conventionally produced foods. *Crit Rev Food Sci Nutr* 42(1): 1-34

Bradley EL, Read WA, Castle L. 2007. Investigation into the migration potential of coating materials from cookware products. *Food Addit Contam* 24(3):326-35

Braun JM, Kahn RS, Froehlich T, Auinger P, Lanphear BP. 2006. Exposures to environmental toxicants and attention deficit hyperactivity. *Environ Health Perspect* 114(12): 1904-1909

Braun JB, Yolton K, Dietrich KN, Hornung R, Ye X, Calafat AM, Lanphear BP. 2009. Prenatal bisphenol A exposure and early childhood behaviour. *Environ Health Perspect* Online October 6 2009; DIO: 10.1289/ehp.0900979. Available at: http:dx.doi.org

Calafat AM, Kuklenyik Z, Reidy JA, Caudill SP, et al. 2005. Urinary concentrations of bisphenol A and 4-nonyl phenol in a human reference population. *Environ Health Perspect* 113:391–395

Calafat AM, Wong LY, Kuklenyik Z, Reidy JA, Needham LL. 2007. Polyfluoroalkyl chemicals in the US population: data from the national health and nutrition examination survey (NHANES) 2003-2004 and comparisons with NHANES 1999-2000. *Environ Health Perspect* 115:1596-1602

Canadian Food Inspection Agency (CFIA). 2005. Young children's food chemical residues project report on agricultural pesticides residues, 2003 – 2004. Modified 2005-01-21. Accessed April 30, 2009 at: http://www.inspection.gc.ca/english/fssa/microchem/resid/2003-2004/todenfe.shtml

Canadian Food Inspection Agency (CFIA). 2006. Report on pesticides, agricultural chemicals, environmental pollutants and other impurities in agri-food commodities of plant origin fresh fruit and vegetables. Fiscal Year 2004/2005 Volume 2. http://www.inspection.gc.ca/english/fssa/microchem/resid/2004-2005/plaveg_ffe.shtml

Canadian Partnership for Children's Health and the Environment (CPCHE). 2007. Public health inspectors project. News and Info. December 12, 2007. Accessed September 2, 2009 at: http://healthyenvironmentforkids.ca/english/news/index.shtml?x=3580

Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. 2003. Intellectual impairment in children with blood lead concentrations below 10 micrograms per deciliter. *N Engl J Med* 348:1517-1526

Cecil KM, Brubaker CJ, Adler CM, Dietrich KN, et al. 2008. Decreased brain volume in adults with childhood lead exposure. *PLoS Med* 5(5):e112

Centers for Disease Control and Prevention (CDC). 2003. National report on human exposure to environmental chemicals. http://www.cdc.gov/exposurereport/introduction.htm

Chang MB, Lin JJ. 2001. Memory effect on the dioxin emissions from municipal waste incineration in Taiwan. *Chemosphere* 45:1151–1157

Chang MB, Chang SH, Chen YW, Hsu HCh. 2004. Dioxin emission factors for automobiles from tunnel air sampling in Northern Taiwan. *Sci Total Environ* 325:129–138

Chen HL, Su HJ, Liao PC, Chen HC, Lee CC. 2004. Serum PCDD/F concentration distribution in residents living in the vicinity of an incinerator and its association with predicted ambient dioxin exposure. *Chemosphere* 54:1421–1429.

Consumer Reports. 2007. "Nonstick pans are ok in new tests." *Consumer Reports* 72.6 (June 2007): 7(1). http://www.consumerreports.org/cro/home-garden/kitchen/cookware-bakeware-cutlery/nonstick-pans-6-07/overview/0607_pans_ov_1.htm

Costa LG, Giordano G. 2007. Developmental neurotoxicity of polybrominated diphenyl ether (PBDE) flame retardants. *Neurotoxicology* 28(6):1047-1067

Costa LG, Giordano G, Tagliaferri S, Caglieri A, Mutti A. 2008. Polybrominated diphenyl ether (PBDE) flame retardants: environmental contamination, human body burden and potential adverse health effects. *Acta Biomed* 79(3):172-83

Coutinho M, Pereira M, Rodrigues R, Borrego C. 2006. Impact of medical waste incineration in the atmospheric PCDD/F levels of Porto, Portugal. *Sci Total Environ* 362:157–165

Curl CL, Fenske RA, Elgethun K. 2003. Organophosphorus pesticide exposure of urban and suburban preschool children with organic and conventional diets. *Environ Health Perspect*, 111(3): 377-382

Dupont website. n.d. Key questions about Teflon® non-sticks. Found at: http://www2.dupont.com/Teflon/en_US/products/safety/key_questions.html

Enneking PA. 2006. Phthalates not in plastic food packaging. Correspondence. *Environ Health Perspect* 114(2): A89-90

Environmental Working Group (EWG). 2007. Bisphenol A: Toxic plastics chemical in canned food. A survey of bisphenol A in U.S. canned foods. Environmental Working Group. Available at: http://www.ewg.org/book/export/html/20928. Accessed in June 2009.

Environment Canada. 2006. Risk management strategy for polybrominated diphenyl ethers (PBDEs). http://www.ec.gc.ca/Toxics/docs/substances/PBDE/PBDE_RMS/EN/s4.cfm

Environment Canada. 2009. Revised risk management strategy for polybrominated diphenyl ethers (PBDEs). Last updated: 2009-03-26. http://www.ec.gc.ca/Toxics/docs/substances/PBDE/rrms/en/intro.cfm

Eskenazi B, Marks AR, Bradman A, Harley K, et al. 2007. Organophosphate pesticide exposure and neurodevelopment in young Mexican-American children. *Environ Health Perspect* 115(5): 792-798

Fei C, McLaughlin JK, Lipworth L, Olsen J. 2009. Maternal levels of perfluorinated chemicals and subfecundity. *Human Reprod* 1(1): 1–6

Fisher JS. 2004. Environmental anti-androgens and male reproductive health: focus on phthalates and testicular dysgenesis syndrome. *Reproduction* 127 (3), 305-315

Fortin MC, Bouchard M, Carrier G, Dumas P. 2008. Biological monitoring of exposure to pyrethrins and pyrethroids in a metropolitan population of the Province of Quebec, Canada. *Environ Res* 107(3):343-50

Foster PMD. 2006. Disruption of reproductive development in male rat offspring following in utero exposure to phthalate esters. *Int J Androl* 29 (1): 140-147

Fromme H, Tittlemier SA, Völke W, Wilhem, M, Twardella D. 2009. Perfluorinated compounds–Exposure assessment for the general population in western countries. *Int J Hyg Environ Health* 212: 239–270

Ginsberg G, Rice DC. 2009. Does rapid metabolism ensure negligible risk from Bisphenol A? *Environ Health Perspect* 117(11):1639-1643.

Government of Alberta, Health & Wellness. 2008. Alberta Biomonitoring Program. Chemicals in serum of pregnant women in Alberta. http://www.health.alberta.ca/documents/Chemical-Biomonitoring-2008.pdf

Government of Canada, no date. Chemicals at a Glance. Web page last updated: 2009-08-07. Accessed August 31, 2009 from: http://www.chemicalsubstanceschimiques.gc.ca/interest-interet/index_e.html#interest

Government of Canada, 2008. "Government of Canada protects families with bisphenol A regulations". News Release 2008-167 October 17, 2008 http://www.hc-sc.gc.ca/ahc-asc/media/nr-cp/_2008/2008_167-eng.php

Hauser R, Calafat AM. 2005. Phthalates and human health. *Occup Environ Med* 62:806-818

Health Canada. 1998a. The Health & Environment Handbook for Health Professionals. Food Quality. Ministry of Supply & Services. Cat. No. H49-96/2-1995E

Health Canada. 1998b. Persistent environmental contaminants and the great lakes basin population: an exposure assessment. Health Canada. Minister of Public Works and Government Services, Canada. Cat. No. H46-2/98-218E

Health Canada 2005. It's Your Health – Dioxins and Furans. Original date 2001. Updated September 2005. http://hcsc.gc.ca/iyh-vsv/environ/dioxin_e.html

Health Canada, 2006a. Toxic equivalent (TEQ) concentrations (pg/g whole weight) of dioxin-like chemicals in fatty foods from Total Diet Study in Calgary, 1999. Retrieved May 20, 2009 from http://www.hc-sc.gc.ca/fn-an/surveill/total-diet/concentration/dioxin_conc_dioxine_calgary1999-eng.php

Health Canada, 2006b. PBDE Flame Retardants and Human Health. Last modified: 2006-12-14. http://www.hc-sc.gc.ca/hl-vs/iyh-vsv/environ/pbde-eng.php#mi

Health Canada. 2006c. The Safe Use of Cookware. Found at: http://www.hc-sc.gc.ca/hl-vs/iyh-vsv/prod/cook-cuisinier-eng.php

Health Canada. 2007a. Eating Well with Canada's Food Guide: A Resource for Educators and Communicators. HC Pub.: 4667 Cat.: H164-38/2-2007E-PDF

Health Canada, 2007b. Perfluorooctane Sulfonate (PFOS) and Health. http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/perflurooctane_sulfonate-eng.php

Health Canada. 2008a. Mercury in Fish. Consumption advice: making informed choices about fish. Last modified 2008-02-15. http://www.hc-sc.gc.ca/fn-an/securit/chem-chim/environ/mercur/cons-adv-etud-eng.php

Health Canada. 2008b. It's Your Health: Mercury and Human Health. Original 2004, updated November 2008. http://www.hc-sc.gc.ca/hl-vs/iyh-vsv/environ/merc-eng.php.

Health Canada, 2008c. Health risk assessment of Bisphenol A from food packaging applications. Bureau of Chemical Safety, Food directorate, Health Products and Food Branch. August 2008. http://www.hc-sc.gc.ca/fn-an/securit/packag-emball/bpa/bpa_hra-ers-eng.php. Accessed June 1, 2009.

Health Canada, 2008d. Survey of Bisphenol A in Canned Liquid Infant Formula Products. Bureau of Chemical Safety Food Directorate Health Products and Food Branch. August 2008. Available at: http://www.hc-sc.gc.ca/fn-an/securit/packag-emball/bpa/index-eng.php

Health Canada. 2008e. Proposal for legislative action on di(2-ethylhexyl) phthalate under the Hazardous Products Act. Date Modified: 2008-03-07. http://www.hc-sc.gc.ca/cps-spc/advisories-avis/info-ind/ethylhexyle-eng.php

Health Canada. 2009a. Lead Information Package - Some Commonly Asked Questions about Lead and Human Health. Last updated: April 2009. Accessed on June 2, 2009. from http://www.hc-sc.gc.ca/ewh-semt/contaminants/lead-plomb/asked_questions-questions_posees-eng.php.

Health Canada, 2009b. Maximum Residue Limits for Pesticides. Date Modified: 2009-05-15. http://www.hc-sc.gc.ca/cps-spc/pest/protect-proteger/food-nourriture/mrl-lmr-eng.php

Health Canada. 2009c. Pesticides and Foods. Date Modified: 2009-01-13 http://www.hc-sc.gc.ca/cps-spc/pubs/pest/_fact-fiche/pesticide-food-alim/index-eng.php

Health Canada, 2009d. Survey of Bisphenol A in Bottled Water Products. Bureau of Chemical Safety Food Directorate Health Products and Food Branch. July, 2009. Available at: http://www.hc-sc.gc.ca/fn-an/securit/packag-emball/bpa/index-eng.php

Health Canada, 2009e. Survey of Bisphenol A in Canned Drink Products. Bureau of Chemical Safety Food Directorate Health Products and Food Branch. March, 2009. Available at: http://www.hc-sc.gc.ca/fn-an/securit/packag-emball/bpa/index-eng.php

Health Canada, 2009f. Survey of Bisphenol A in Baby Food Products Prepackaged in Glass Jars with Metal Lids. Bureau of Chemical Safety Food Directorate Health Products and Food Branch. July, 2009. Available at: http://www.hc-sc.gc.ca/fn-an/securit/packag-emball/bpa/index-eng.php

Health Canada, 2009g. Survey of Bisphenol A in Canned Powdered Infant Formula Products. Bureau of Chemical Safety Food Directorate Health Products and Food Branch. July, 2009. Available at: http://www.hc-sc.gc.ca/fn-an/securit/packag-emball/bpa/index-eng.php

Health Canada 2009h. Questions and Answers on Perfluorinated Chemicals in Food. Date Modified: 2009-07-13. http://www.hc-sc.gc.ca/fn-an/securit/chem-chim/environ/pcf-cpa/qr-pcf-qa-eng.php

Health Canada and Environment Canada 2008. Screening assessment for the challenge phenol, 4,4' -(1-methylethylidene)bis- (Bisphenol A) chemical abstracts service registry number 80-05-7. Health Canada. Environment Canada. October 2008. Last updated: 2009-01-31. Last reviewed: 2008-10-29. Available at: http://www.ec.gc.ca/substances/ese/eng/challenge/batch2/batch2_80-05-7.cfm

Hites RA, Foran JA, Schwager SJ, Knuth BA, Hamilton MC, Carpenter DO. 2004. Global assessment of polybrominated diphenyl ethers in farmed and wild salmon. *Environ Sci Tech*, 38, (19), 4945-4949

Hornung RW, Lanphear BP, Cecil KM. 2009. Age of greatest susceptibility to childhood lead exposure: a new statistical approach. *Environ Health Perspect* 117(8): 1309–1312.

Hylander LD, Sollenberg H, Westas H. 2003. A three-stage system to remove mercury and dioxins in flue gases. *Sci Total Environ*, 304:137–144

Infante-Rivard C, Weichenthal S. 2007. Pesticides and childhood cancer: An update of Zahm and Ward's 1998 review. *J Toxicol Environ Health-Part B-Crit Rev* 10(1-2), 81-99.

Institute of Medicine (IOM), 2003. Dioxins and Dioxin-like Compounds in the Food Supply: Strategies to Decrease Exposure. Retrieved May 20, 2009 from http://www.iom.edu/CMS/3788/4607/13097.aspx

International Agency for Research on Cancer (IARC), 1997. Summaries & Evaluations – Polychlorinated dibenzo-para-dioxins. Vol. 69 (1997) p. 33. Retrieved May 20, 2009, from http://www.inchem.org/documents/iarc/vol69/dioxin.html

Jaakkola JJ, Knight TL. 2008. The role of exposure to phthalates from polyvinyl chloride products in the development of asthma and allergies: a systematic review and meta-analysis. *Environ Health Perspect* 116(7):845-53

Jaakkola JJ, Verkasalo PK, Jaakkola N. 2000. Plastic wall materials in the home and respiratory health in young children *Am J of Public Health* 90, 797–799

Jacobson JL, Jacobson SW. 1996. Intellectual impairment in children exposed to PCBs in utero. *N Engl J Med* 335: 783-789

Jacobson JL, Jacobson SW. 2001. Postnatal exposure to PCBs and childhood development. *Lancet* 358(9293):1568-9

Jacobson JL, Jacobson SW, et al. 2002. Breast-feeding and gender as moderators of teratogenic effects on cognitive development. *Neurotoxicol Teratol* 24(3):349-58

Jacobson JL, Janisse J, Banerjee M, Jester J, Jacobson SW, Ager JW. 2002. A benchmark dose analysis of prenatal exposure to polychlorinated biphenyls. *Environ Health Perspect* 110:393-8

Jensen AA, Poulsen PB, Bossi R. 2008. Survey and environmental/health assessment of fluorinated substances in impregnated consumer products and impregnating agents. Danish Environmental Protection Agency. Report. 2008-10-20 http://www2.mst.dk/common/Udgivramme/Frame.asp?http://www2.mst.dk/udgiv/publications/2008/978-87-7052-845-0/html

Johnson-Restrepo B, Addink R, Wong C, Arcaro K, Kannan K. 2007. Polybrominated diphenyl ethers and organochlorine pesticides in human breast milk from Massachusetts, USA. *Journal of Environmental Monitoring* 9(11): 1205-1212

Jones-Otazo HA, Clarke JP, Diamond ML, et al. 2005. Is house dust the missing exposure pathway for PBDEs? An analysis of the urban fate and human exposure to PBDEs. *Environ Sci Technol* 39:5121-30

Jorissen J. 2007. Literature review. Outcomes associated with postnatal exposure to polychlorinated biphenyls (PCBs) via breast milk. *Adv Neonatal Care* 7(5):230-7

Jusko, TA, Henderson CR, Lanphear BP, Cory-Slechta DA, Parsons PJ, Canfield RL. 2008. Blood lead concentrations < 10 microg/dL and child intelligence at 6 years of age. *Environ Health Perspect* 116(2):243-8

Kaushik G, Satya S, Naik SN. 2009. Food processing a tool to pesticide residue dissipation. *Food Res Int* 42:26-40

Kissa E. 2001. A miniature closed vessel technique for testing textile chemicals. *Aatcc Review* 1(3), 27-28

Krol WJ, Arsenault TL, Pylypiw Jr. HM, Incorvia Mattina MJ. 2000. Reduction of pesticide residues on produce by rinsing. *J Agric Food Chem* 48(10): 4666-4670

Landrigan PJ, Sonawane B, Mattison D, McCally M, Garg A. 2002. Chemical contaminants in breast milk and their impacts on children's health: an overview. *Environ Health Perspect* 110(6):A313-5

Lang IA, Galloway TS, Scarlett A, Henley WE, et al. 2008. Association of urinary bisphenol A concentration with medical disorders and laboratory abnormalities in adults. *JAMA* 300(11):1303-1310

Lanphear BP, Hornung R, Khoury J, et al. 2005. Low-level environmental lead exposure and children's intellectual function: An international pooled analysis. *Environ Health Perspect* 113:894-899

La Guardia MJ, Hale RC, Harvey E. 2006. Detailed polybrominated diphenyl ether (PBDE) congener composition of the widely used penta-, octa-, and deca-PBDE technical flame-retardant mixtures. *Environ Sci Technol* 40(20):6247-6254

Lau C, Anitole K, Hodes C, Lai D, Pfahles-Hutchens A, Seed J. (2007). Perfluoroalkyl acids: a review of monitoring and toxicological findings. *Toxicol Sci* 99(2):366-94

Li D, Zhou Z, Qing D, et al. 2009. Occupational exposure to bisphenol-A (BPA) and the risk of self-reported male sexual dysfunction. *Hum Reprod*. Advance access published online on November 10, 2009. doi:10.1093/humrep/dep381. Abstract only retrieved.

Lorber M. 2008. Exposure of Americans to polybrominated diphenyl ethers. *J Expo Sci Environ Epidemiol* Jan;18(1):2-19

Lu C, Barr D, Pearson M, Bartell S, Bravo R, (2006). A longitudinal approach of assessing urban and suburban children's exposure to pyrethroid pesticides. *Epidemiology* 17(6):S406-S407

Lu, C. S., Barr, D. B., Pearson, M. A., & Waller, L. A. (2008). Dietary intake and its contribution to longitudinal organophosphorus pesticide exposure in urban/suburban children. *Environ Health Perspect*, 116(4), 537-542

Mahaffey KR. 1995. Nutrition and lead: strategies for public health. *Environ Health Perspect* 103 Suppl 6:191-6

Mahaffey KR. (2004). Fish and shellfish as dietary sources of methylmercury and the omega-3 fatty acids, eicosahexaenoic acid and docosohexaenoic acid: risks and benefits. *Environ Res* 95:414-428

Midasch O, Schettgen T, Angerer J, (2006). Pilot study on the perfluorooctanesulfonate and perfluorooctanoate exposure of the German general population. *Int J Hyg Environ Health* 209(6):489-496

Ministry of Environment, Ontario (MOE). 2007a. Letter to all schools, private schools, and day nurseries, on the requirements of Ontario Regulation 243/07. http://www.ontario.ca/drinkingwater/158287.pdf

Ministry of Environment, Ontario (MOE). 2007b. Safe drinking water in schools, private schools and day nurseries. http://www.ontario.ca/drinkingwater/178735.pdf

Ministry of Environment, Ontario (MOE). 2009. Updates to lead and drinking water regulations under the Safe Drinking Water Act, 2002. 010-7302. November 19, 2009.

Mocarelli P, Gerthoux PM, Patterson DG Jr, Milani S, Limonta G, Bertona M, et al. (2008). Dioxin exposure, from infancy through puberty, produces endocrine disruption and affects human semen quality. *Environ Health Perspect* 116(1):70-7

MTTHU and SRCHC - Metropolitan Toronto Teaching Health Unit & South Riverdale Community Health Centre. 1995 Why Barns are Red. Health Risks from Lead and Their Prevention.

National Research Council (NRC). 1993. Pesticides in the Diets of Infants and Children. Washington: National Academy Press.

National Research Council (NRC). 2000. Toxicological Effects of Methylmercury. Washington: National Academy Press.

NTP- CERHR, 2008. NTP-CERHR Monograph on the potential human reproductive and developmental effects of bisphenol A. National Toxicology Program U.S. Department of Health and Human Services, Center for the Evaluation of Risks to Human Reproduction. September 2008. NIH Publication No. 08-5994. Accessed June 1, 2009 at: http://cerhr.niehs.nih.gov/chemicals/bisphenol/bisphenol.pdf.

Patandin S, Dagnelie PC, Mulder PG, Op de Coul E, et al. 1999. Dietary exposure to polychlorinated biphenyls and dioxins from infancy until adulthood: A comparison between breast-feeding, toddler, and long-term exposure. *Environ Health Perspect* 107(1):45-51

Rauh VA, Garfinkel R, Perera FP, Andrews HF, Hoepner L, Barr DB, et al. 2006. Impact of prenatal chlorpyrifos exposure on neurodevelopment in the first 3 years of life among inner-city children. *Pediatrics* 118(6): E1845-E1859

Region of Waterloo Public Health. 2005. Food Miles: Environmental Implications of Food Imports to Waterloo Region. Author: Marc Xuereb, Public Health Planner November 2005. http://chd.region.waterloo.on.ca/web

Rosas LG, Eskenazi B, 2008. Pesticides and child neurodevelopment. *Curr Opin Pediatr*. 20(2):191-7.

Roy CC, Bearce-Rogers, J, Chappell J, et al. 1995 Nutrition recommendations update: Dietary fats and children. *Nutrition Reviews* 53(12): 367-75

Selevan SG, Kimmel CA, Mendola P, 2000. Identifying critical windows of exposure for children's health. *Environ Health Perspect* 108(Suppl 3):451-55

Silva MJ, Barr DB, Reidy JA, et al. 2004. Urinary levels of seven phthalate metabolites in the US population from the National Health and Nutrition Examination Survey (NHANES) 1999-2000. *Environ Health Perspect* 112 (3): 331-338

Sinclair E, Kim SK, Akinleye HB, Kannan K. 2007. Quantitation of gas-phase perfluoroalkyl surfactants and fluorotelomer alcohols released from nonstick cookware and microwave popcorn bags. *Environ Sci Technol* 41(4):1180-5

Snedeker, S. 2007. BCERF Briefs: PBDEs. Available online: http://envirocancer.cornell.edu/pbde/brief.cfm Last modified: 2007-12-12

Spliethoff HM et al, 2008. Use of newborn screening program blood spots for exposure assessment: declining levels of perfluorinated compounds in New York State infants. *Environ Sci Technol* 42(14):5361–5367

Srogi K. 2008. Levels and congener distributions of PCDDs, PCDFs and dioxin-like PCBs in environmental and human samples: a review. *Environ Chem Lett* 6, 1-28.

Swan SH, Main KM, Liu F, Stewart SL et al. 2005. Decrease in anogenital distance among male infants with prenatal phthalate exposure. *Environ Health Perspect* 113 (8), 1056-1061

Tittlemier SA, Pepper K, Seymour C, Moisey J, Bronson R, Cao XL, et al. 2007. Dietary exposure of Canadians to perfluorinated carboxylates and perfluorooctane sulfonate via consumption of meat, fish, fast foods, and food items prepared in their packaging. *J Agric Food Chem* 55(8): 3203-3210

Toronto Public Health (TPH). 2002. Lawn and Garden Pesticides: A Review of Human Exposure & Health Effects Research. http://www.toronto.ca/health/hphe/pdf/pesticides_lawnandgarden.pdf

Toronto Public Health (TPH). 2005. Environmental Threats to Children: Understanding the Risks, Enabling Prevention. Technical Report. http://www.toronto.ca/health/hphe/childrens_health_report.htm

Toronto Public Health (TPH). 2006. Fish Consumption: Benefits and Risks for Women in Childbearing Years and Young Children. Technical Report (September 2006). http://www.toronto.ca/health/hphe/fish_mercury.htm

Toronto Public Health (TPH). 2008. A Guide to Eating Fish for Women, Children and Families – Staff Report (April 2008). http://www.toronto.ca/legdocs/mmis/2008/hl/bgrd/backgroundfile-12028.pdf

Toronto Public Health (TPH). (2009) Fact sheet: Lead in drinking water. January 2009. http://www.toronto.ca/health/lead/pdf/factsheet_lead_drinkingwater_0109.pdf

Toronto Water, 2007. Lead water service connection replacement program - Staff Report. http://www.toronto.ca/legdocs/mmis/2007/pw/bgrd/backgroundfile-5459.pdf

United Kingdom Food Standards Agency (UK FSA). 2006. Fluorinated chemicals: UK dietary intakes. Food Survey Information Sheet 11/06, London, UK. http://www.food.gov.uk/multimedia/pdfs/fsis1106.pdf#page=1

University of British Columbia (UBC). n.d. Chemicals, Health and Pregnancy Study (CHirP) webpage http://web.cher.ubc.ca/chirp/

United Nations Environment Program (UNEP). 2009. "Governments unite to step-up reduction on global DDT reliance and add nine new chemicals under international treaty." Media release, May 9, 2009. Found at: http://www.unep.org/Documents.Multilingual/Default.asp?DocumentID=585&ArticleID=6158&l=en&t=long

U.S. Department of Health and Human Services and U.S. Environmental Protection Agency. (US DHHS/EPA) (2004). What you need to know about mercury in fish & shellfish. [electronic version] Retrieved May 31, 2009 www.cfsan.fda.gov/~dms/admehg3.html

U.S. Environmental Protection Agency (US EPA). 2003. Office of Children's Health Protection (OCHP), Children's environmental exposures, 25 pp. Paper Series on Children's Health and the Environment. March, 2003.

U.S. Environmental Protection Agency (US EPA). 2009a. Provisional health advisories for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS). Dated January 8, 2009. http://www.epa.gov/waterscience/criteria/drinking/pha-PFOA_PFOS.pdf

U.S. Environmental Protection Agency (US EPA). 2009b. Perfluorooctanoic Acid (PFOA) and Fluorinated Telomers. Basic Information. Last updated August 12th, 2009. http://www.epa.gov/oppt/pfoa/pubs/pfoainfo.html

U.S. Food and Drug Administration.(US FDA), 2009. Bisphenol A. Message for Consumers. http://www.fda.gov/NewsEvents/PublicHealthFocus/ucm064437.htm. Last updated: August, 31st 2009. Accessed on: November 26th, 2009.

U. S. Food and Drug Administration. FDA Consumer: June 1993. FDA Reports on Pesticides in Foods. http://www.cfsan.fda.gov/~dms/qa-pes4.html

Washburn ST, Bingman TS, Braithwaite SK, Buck RC, Buxton LW, Clewell HJ, et al. 2005. Exposure assessment and risk characterization for perfluorooctanoate in selected consumer articles. *Environ Sci Technol* 39: 3904-3910

Wigle D. 2003. *Child Health and the Environment*, New York: Oxford University Press.

Wigle DT, Arbuckle TE, Walker M, Wade MG, Liu S, Krewski D. 2007. Environmental hazards: evidence for effects on child health. *J Toxicol Environ Health-Part B-Crit Rev* 0(1-2):3-39.

Wigle DT, Arbuckle TE, Turner MC, Bérubé A, Yang Q, Liu S, Krewski D. 2008. Epidemiologic evidence of relationships between reproductive and child health outcomes and environmental chemical contaminants. *J Toxicol Environ Health-Part B-Crit Rev* 11:373-517.

Wilford BH, Shoeib M, Harner T, Zhu JP, Jones KC. 2005. Polybrominated diphenyl ethers in indoor dust in Ottawa, Canada: Implications for sources and exposure. *Environ Sci Tech* 39(18):7027-7035

Wilson NK, Chuang JC, Morgan MK, Lordo RA, Sheldon LS. 2007. An observational study of the potential exposures of preschool children to pentachlorophenol, bisphenol-A, and nonylphenol at home and daycare. *Environ Res* 103(1):9-20

Winter CK, Davis SE. 2006. Organic Foods. *J Food Sci* 1(9): R117-124

Wittassek M, Angerer J. 2008. Phthalates: metabolism and exposure. *Int J Adrol* 31:131-136

World Health Organization (WHO), 2007. Fact sheet No.225, Dioxins and their Effects on Human Health. Retrieved May 20, 2009 from http://www.who.int/mediacentre/factsheets/fs225/en/index.html

Wormuth M,Scheringer M, Vollenweider M, Hungerbuhler K. 2006. What are the sources of exposure to eight frequently used phthalic acid esters in Europeans? *Risk Anal 26* (3):803-824.

Zhao X, Carey EE, Wang WQ, Rajashekar CB. 2006. Does organic production enhance phytochemical content of fruit and vegetables? Current knowledge and prospects for research. *Hort Tech* 16(3): 449-456.