Final Human Health State of the Science Report on Lead

February 2013

Canada

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Executive Summary

Health Canada has conducted an assessment of the most current science on lead and consolidated the information in a State of the Science report.

Lead (Pb) is a naturally occurring element found in rock and soil, yet widespread anthropogenic use has resulted in its ubiquitous presence in the environment. Environmental lead concentrations are extensively measured and reported by national, provincial, and municipal initiatives across Canada. Lead is found in all environmental media in Canada as well as in food and drinking water. Levels of lead in most environmental media have declined significantly over the past few decades.

Canadians are exposed to low levels of lead in food, drinking water, air, dust, soil, and products. Although blood lead levels (BLLs) have declined by over 70% in Canada since 1978–1979, lead is still widely detected in the Canadian population. In 1978–1979, approximately 27% of Canadians aged 6 to 79 years had BLLs at or above 10 micrograms per deciliter (μg/dL) compared with less than 1% of Canadians today. BLLs tend to rise after infancy, peak between 1 and 3 years of age, decline slightly during childhood and adolescence, and rise again with age in adults. BLLs are highest in seniors. Based on the 2007–2009 Canadian Health Measures Survey, geometric mean BLLs are 0.90 μg/dL, 0.80, 1.12, 1.60, and 2.08 μg/dL for the age groups of 6 to 11, 12 to 19, 20 to 39, 40 to 59, and 60 to 79 years, respectively. These findings are consistent with trends reported in the United States. Although national Canadian BLL data for children under 6 years of age are currently lacking, BLLs for this age group are expected to be similar to those reported in the United States, where the geometric mean BLL for children 1 to 5 years of age is 1.51 μg/dL.

After the implementation of measures to reduce exposures to lead through the inhalation route (e.g., prohibition of leaded gasoline), oral exposure from food and water now represents the most significant route of lead intake for the general adult population. For infants and children, ingestion of non-food items contaminated with lead (e.g., house dust, lead-based paint, soil, products) along with dietary intake through food and water are the greatest sources of environmental exposure to lead. Several factors are known to be associated with BLLs. Umbilical cord BLLs are correlated with maternal and infant BLLs. BLLs in infants are correlated with maternal BLLs and lead in breast milk. In children, BLLs are associated with lead levels in water, soil, and household dust. Lead-based paint may be a significant source for those living in older homes. In Canada, BLLs are higher in males than in females; in smokers than in non-smokers; in individuals born outside of Canada than in those born in Canada; in individuals living in older homes (greater than 50 years) than in those living in newer homes; and in residents of households with lower income levels than in those with higher income levels. Elevated BLLs may also result from the increased rate of remobilization of bone lead in blood during life stages such as pregnancy, lactation, menopause, andropause, and post-menopause. Accordingly, at certain life stages, an individual's bone lead stores can represent the single greatest potential source of increased BLLs, as the amount of lead released back into blood for systemic circulation increases.

Effects associated with BLLs below 10 μg/dL, down to 1–2 μg/dL, have been reported in the health effects database and include neurodevelopmental, neurodegenerative, cardiovascular, renal, and reproductive effects. The evidence of an association between health effects with BLLs in the lower range of exposure is strongest for neurodevelopmental effects in children, most commonly assessed as a reduction of intelligence quotient and attention-related behaviours. Developmental neurotoxicity has been associated with the lowest levels of lead exposure examined to date, both in observational studies and in animal experiments. In humans, neurotoxic effects of lead can persist until

the late teen-age years. Dose-response modelling conducted with available observational studies does not currently demonstrate a population threshold for developmental neurotoxicity. Infants and children are a susceptible subpopulation for lead exposure because they have greater gastrointestinal absorption and less effective renal excretion than adults in addition to different behaviour patterns. Identification of infants and children as a susceptible subpopulation and neurodevelopmental effects as the critical health effect is considered protective for other adverse health effects of lead across the entire population.

Although BLLs of Canadians have declined significantly over the past 30 years, health effects are occurring below 10 µg/dL. There is sufficient evidence that BLLs below 5 µg/dL are associated with adverse health effects. Health effects have been associated with BLLs as low as 1–2 µg/dL, levels that are present in Canadians, although there is uncertainty associated with effects observed at these levels. It is considered appropriate to apply a conservative approach when characterizing risk; accordingly, additional measures to further reduce exposures of Canadians to lead are warranted.

This State of the Science report is published along with a Risk Management Strategy for Lead which provides a comprehensive description of the existing management measures and progress to date under the Canadian Federal Risk Management Strategy for Lead. Research and monitoring will continue to support the assessment of lead in Canadians and, where appropriate, assess the performance of potential control measures identified during the risk management phase.

Human Health State of the Science Report

February 2013

Lead

Introduction

In 1994, the Federal–Provincial Committee on Environmental and Occupational Health produced a report on the evidence for low-level effects of lead. The committee recommended a tiered approach to intervention, beginning at blood lead levels (BLLs) above 10 µg/dL, based on evidence that health effects were occurring in the range of 10–15 µg/dL (CEOH 1994). In addition, the report concluded that no clear evidence of a threshold had been shown for the critical neurotoxic effects of lead. Since that time, substantial scientific evidence has been published that demonstrates that health effects occur at BLLs below 10 µg/dL, the current Canadian blood lead intervention level. Current levels of lead in the Canadian environment and the general population along with the scientific evidence for these effects are presented as an update in this State of the Science report.

This State of the Science report was prepared by evaluators within the Contaminated Sites Division and Existing Substances Risk Assessment Bureau of Health Canada. Data relevant to the report were identified in original literature, review and assessment documents, independent contractor reports, stakeholder and academic research reports, and recent literature searches up to February 2012. The report does not represent an exhaustive or critical review of all available data. Rather, it presents a summary of the critical information upon which the evaluation is based. The content of this document was reviewed by senior Health Canada staff for adequacy of data coverage and defensibility of the evaluation. This State of the Science report is published along with a Risk Management Strategy for Lead which provides a comprehensive description of the existing federal management measures and progress to date under the Canadian Federal Risk Management Strategy for Lead.

The critical information and considerations upon which the assessment is based are summarized below.

Substance Identity and Properties

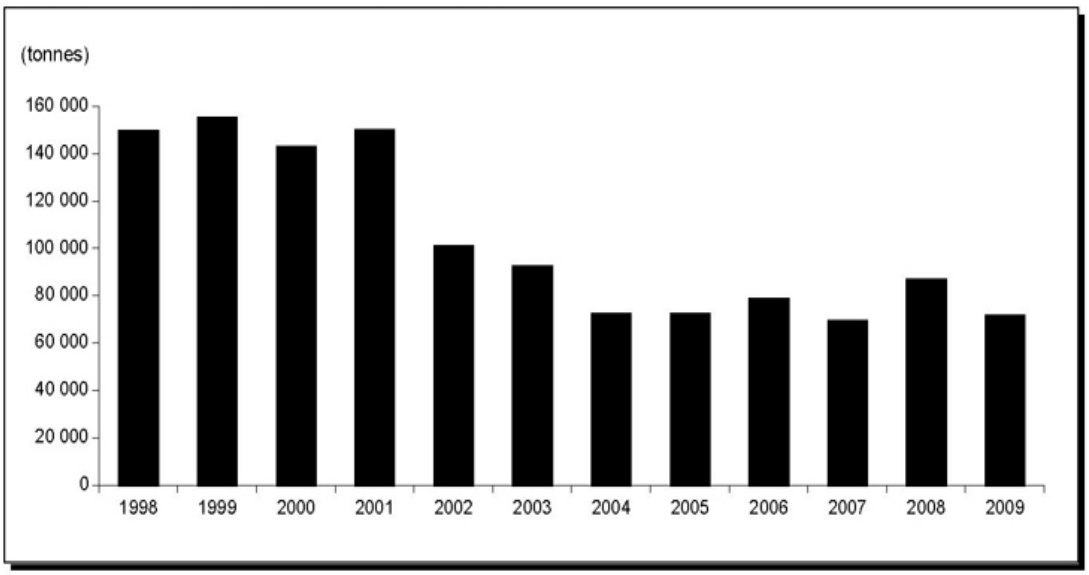
Lead (Pb) is an odourless, bluish-grey, lustrous metal that is malleable, ductile, and resistant to chemical corrosion. Lead has three common oxidation states: $Pb^{(0)}$ (elemental lead), with Chemical Abstracts Service (CAS) Registry Number 7439-92-1, $Pb^{(2+)}$ (plumbous compounds), and $Pb^{(4+)}$ (plumbic compounds); in nature it is the plumbous form that predominates (ATSDR 2007). The melting and boiling points of elemental lead are 327.4°C and 1740°C, respectively (ATSDR 2007). While elemental lead is insoluble in water, salts of the plumbous form may be highly water soluble (e.g., lead(II)nitrate) (ATSDR 2007). Measurements of lead in environmental and biological media rarely identify the form, but rather refer to the lead moiety contained within unspecified substances. Therefore, for the purposes of this report, “lead” will refer to the lead moiety; where relevant, specific lead substances will be explicitly identified by name or CAS registry number.

Production

Canada is a significant global producer and supplier of refined lead, ranking eighth in the world in 2009 in terms of mine production (68 624 tonnes) and sixth in terms of refined lead production (101 484 tonnes) (Panagapko 2009). Summaries of mine production of lead and refined lead metal production in

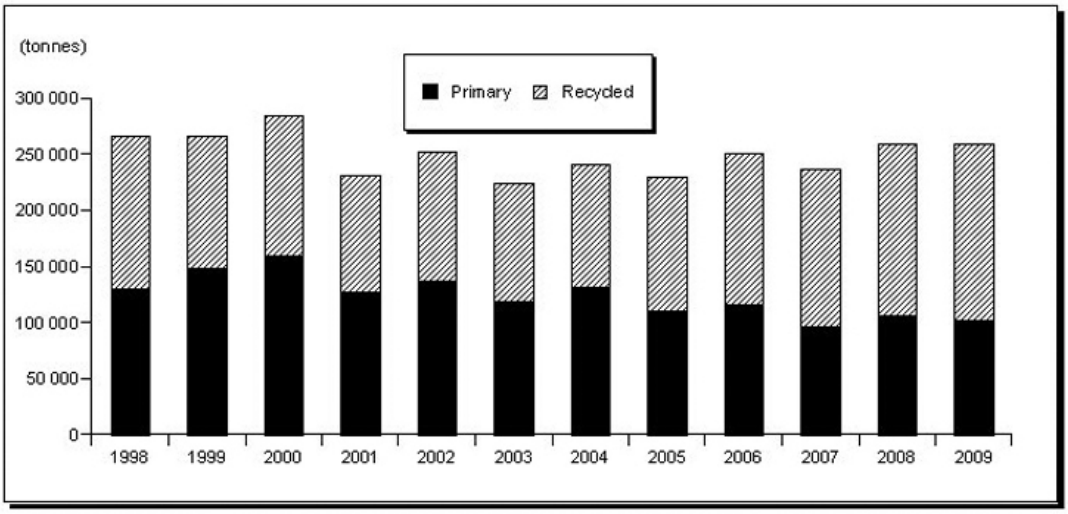
Canada are presented in Figures 1 and 2, respectively. In 2009, Canadian mine output of lead decreased by 31%, whereas world mine output increased by 3.4%. Most lead in Canada is produced as a co-product of zinc mining, while the recycling of lead, mainly from depleted car batteries, represented the primary source of Canada's total refined production (61%) in 2009. Nearly 90% of refined lead produced in Canada is exported to the United States. In 2009, lead concentrates were produced at two mines in Canada. Primary refined lead metal was produced from domestic and foreign concentrates at two smelters, one located in New Brunswick and the other in British Columbia, while secondary lead metal (157 456 tonnes) was produced from recycled lead (primarily car batteries) at four sites in Quebec, Ontario, and British Columbia (Panagapko 2009).

Figure 1. Canadian mine production of lead, 1998–2009



Source: Panagapko 2009.

Figure 2. Canadian refined lead metal production, 1998–2009



Source: Panagapko 2009

Uses

Lead has been used extensively in a variety of applications primarily owing to its low melting point and excellent corrosion resistance in the environment (ATSDR 2007). When lead is exposed to air and water, films of lead sulfate, lead oxides, and lead carbonates are formed that, in turn, act as protective barriers, slowing or halting further corrosion of the underlying metal. For these reasons, lead has been intentionally added to a wide range of components in the built environment, examples including cable sheathing, circuit boards, lining for chemical baths and storage vessels, chemical transmission pipes, electrical components, polyvinyl chloride (as a chemical stabilizer), and radiation shielding. Lead continues to be used extensively in rolled and extruded products in the construction industry; the use of lead sheeting in the building industry has increased in recent years (IARC 2006). Currently, the production of batteries, used predominantly in the automotive industry, comprises the single largest global market for refined lead (75%) since the phase-out of lead in household paints, gasoline additives, and solder in food cans (OECD 1993; Keating and Wright 1994; Keating 1995; Panagapko 2009). However, certain lead compounds are still used in the industrial setting as basic paint primers for iron and steel (Panagapko 2009). According to the International Agency for Research on Cancer (IARC 2006), approximately 100 000 tonnes of lead are used annually in the manufacture of lead shot and ammunition. Lead stampings, pressings, and castings are also still widely used for many weighting applications, including wheel balance weights, weights for analytical instruments, and yacht keels (IARC 2006).

Both soluble and insoluble lead compounds have a variety of industrial uses (NTP 2004). Lead acetate (CAS No. 301-04-2) is used as a water repellent for mildew protection as well as a mordant for cotton dyes. Lead acetate trihydrate (CAS No. 6080-56-4) is used in the production of varnishes and chrome pigments, and as an analytical reagent, while lead chloride (CAS No. 7758-95-4) is used in the manufacture of asbestos clutch or brake linings, as a catalyst, and as a flame retardant. Lead nitrate (CAS No. 10099-74-8) is used as a heat stabilizer in nylon, in the manufacture of matches and

explosives, and as a coating on paper for photothermography, and lead subacetate (CAS No. 1335-32-6) is used in sugar analysis and for clarifying solutions of organic substances (HSDB 2010).

Lead azide (CAS No. 13424-46-9) and lead styphnate (CAS No. 15245-44-0) are both used in the manufacture of munitions. Lead carbonate (CAS No. 598-63-0), lead fluoride (CAS No. 7783-46-2), lead fluoborate (CAS No. 13814-96-5), and lead naphthenate (CAS No. 61790-14-5) are all employed as catalysts, with additional uses in both the electronics and optical industries (lead fluoride), in coatings for thermographic copying (lead carbonate), as a curing agent for epoxy resins (lead fluoborate), and as a varnish drier (lead naphthenate). Both lead phosphate (CAS No. 7446-27-7) and lead stearate (CAS No. 1072-35-1) are used as stabilizers in the plastics industry, while lead iodide (CAS No. 10101-63-0) and lead sulphate (CAS No. 7446-14-2) had previous applications in photography, as well as in thermoelectric materials (lead iodide) and with zinc in the production of galvanic batteries (lead sulfate). Lead oxide (CAS No. 1317-36-8) and lead sulfide (CAS No. 1314-87-0) are used in ceramics manufacturing, as a vulcanizing agent in rubber and plastics (lead oxide), and as a humidity sensor in rockets (lead sulfide). Lead chromate (CAS No. 7758-97-6) is employed as a pigment in paints, rubber, and plastics, while lead tetraoxide (CAS No. 1314-41-6) is used in plasters, ointments, glazes, and varnishes. Lead thiocyanate (CAS No. 592-87-0) is used in the manufacture of safety matches and small-arms cartridges, while lead arsenate (CAS No. 7784-40-9) was historically used as an insecticide and herbicide, but has no current application (NTP 2004).

Organic lead compounds, including tetraethyl lead (CAS No. 78-00-2) and tetramethyl lead (CAS No. 75-74-1), were once widely used as anti-knock additives in motor vehicle fuels in North America before their use in on-road vehicles was prohibited in the 1990s. In Canada, there is currently limited approved use of organic lead in gasoline for piston engine aircraft (avgas) and racing fuels for competition vehicles[1], as an exemption in the *Gasoline Regulations* under the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada 1999).

A summary of Canadian lead use in 2005–2007 is presented in Table 1.

Table 1. Estimate of the uses of lead and lead compounds in Canada between 2005 and 2007 as reported by Natural Resources Canada

LEAD USE[a]	2005			2006			2007		
	Primary	Recycled[b]	Total	Primary	Recycled[b]	Total	Primary	Recycled[b]	Total
Lead used for or in the production of (tonnes):									
Antimonial Lead	X[c]	X	34 241	X	X	30 532	X	X	33 565
Batteries and battery oxides	X	X	X	1366	X	X	X	X	X
Chemical uses; white	X	–[d]	X	3868	–	X	X	–	X

[1] A competition vehicle is defined in the *Gasoline Regulations* as "a vehicle or boat that is used exclusively for competition and does not include a vehicle that is used on a highway or a vehicle or boat that is used for recreational purposes."

LEAD USE[a]	2005			2006			2007		
	Primary	**Recycled[b]**	**Total**	**Primary**	**Recycled[b]**	**Total**	**Primary**	**Recycled[b]**	**Total**
lead, red lead, litharge, tetraethyl lead, etc.									
Copper alloys, brass, bronze, etc.	X	X	12	6	X	9	–	X	X
Lead alloys (tonnes):									
Solders	X	X	X	180	X	X	X	X	X
Others (including babbitt, type metals, etc.)	X	X	X	268	X	X	X	X	X
Semi-finished products (tonnes):									
Pipe, sheet, traps, bends, blocks for caulking, ammunition, etc.	X	X	X	2999	X	X	X	X	X
Other lead products	2496	2119	4615	2154	2704	4858	1491	2512	4003
Total	**28 633**	**39 433**	**68 066**	**18 577**	**29 851**	**48 428**	**19 921**	**36 591**	**56 512**

Source: Panagapko 2009.

[a] Available data, as reported by users.

[b] Includes all remelt scrap lead used to make antimonial lead. Note: this survey is currently suspended by Natural Resources Canada. Numbers may not add to totals owing to rounding error.

[c] X: undisclosed, Confidential Business Information.

[d] –: indicates nil.

Lead service lines have been installed in drinking water systems in many countries including Canada. The National Plumbing Code of Canada permitted the use of lead for service lines until 1975 and in solder until 1986 (Health Canada 2007a). By 1990, the use of solder containing lead in new plumbing and in repairs to plumbing for drinking water supplies was prohibited under the National Plumbing Code (Health Canada 2009a). Therefore, pre-1990 existing distribution and plumbing materials may be a source of lead in domestic tap water.

Until the 1960s, lead was added in significant quantities (ranging from 10% to 50%) to household and industrial paints either as a pigment (lead(II) chromate) or to speed up drying, resist corrosion, and increase durability (lead(II) carbonate) (ATSDR 2007; CMHC 2009). In Canada, the lead content of consumer paints has been steadily decreasing since the introduction of restrictions limiting the amount of lead in indoor paint under the *Hazardous Products Act* (HPA) in 1976 to 5000 mg/kg (0.5%) and through voluntary action on the part of paint manufacturers to limit the amount of total lead in both interior and exterior consumer paints in 1991 to 600 mg/kg. In November 2010, the 600 mg/kg limit was

reduced to 90 mg/kg (0.009%) by amendment to the *Surface Coating Materials Regulations*, now under the *Canada Consumer Product Safety Act* (CCPSA). Also in November 2010, the total lead content limit of applied paints and other surface coatings on children's furniture and other articles, toys, equipment, and other products for use by children in learning or play, and on artists' pencils and brushes was reduced from 600 to 90 mg/kg (Health Canada 2010a). However, despite decreased lead levels in newer paints and surface coatings, opportunities for lead exposure from these sources still exist, as many older buildings may contain older paints and coatings.

Lead may be present in inexpensive jewellery, despite the requirements introduced in Canada in 2005 under the HPA and continued under the CCPSA, which limit the lead content in jewellery products intended primarily for children under 15 years of age to 600 mg/kg of total lead and 90 mg/kg of migratable lead.

Art supplies, such as inks, dyes, paints and pastels, and coloured glazes for pottery or glassware, may also contain inorganic lead pigments. The 90 mg/kg total lead limit under the *Surface Coating Materials Regulations* applies to all children's art paints. General artists' paints are exempt from the 90 mg/kg total lead limit, but if they contain lead, their label must include a lead content warning. Artists' paints contain a much wider range of pigments than do children's paints and are therefore more likely to include lead-based pigments (Health Canada 2009b). The *Glazed Ceramics and Glassware Regulations* (GCGR) under the CCPSA limit the amount of leachable lead in glazes on ceramic and glass products intended for preparing, serving, or storing food. Lead crystalware, which by definition contains lead, is widely used for serving beverages (Health Canada 2009b). The leachable lead content of lead crystal used for serving food or beverages is strictly limited under international industry standards. There are no permissible uses for lead in food in Canada. Tolerance levels for lead in food are listed in Table 1 in Division 15 of the *Food and Drug Regulations*.

Health Canada has introduced several measures to reduce lead in consumer products as part of the Lead Risk Reduction Strategy for Consumer Products (LRRS) (Health Canada 2010a). The *Consumer Products Containing Lead (Contact with Mouth) Regulations* (CPCLR), which came into effect in November 2010, impose a total lead limit of 90 mg/kg for accessible components of products whose normal pattern of use involves mouth contact. These components include all products intended for play and learning by children under 3 years of age, children's crayons, chalks, modelling clays and similar materials likely to be ingested, mouthpieces of musical instruments, and sports mouthpieces.[2]

Lead is present in certain products used in recreational activities, such as in castings used for fishing weights, diving weights, or toy soldiers, soldering, making stained-glass articles, using leaded glazes to make pottery, glass blowing, and screen printing (Grabo 1997). Lead and all its associated products are prohibited ingredients in cosmetic products marketed in Canada under the *Cosmetic Regulations* of the *Food and Drugs Act* (Health Canada 2010b). Tolerance limits exist for lead in Natural Health Products, and the concentration of lead in pharmaceuticals must not exceed the limits specified in Schedule B publications of the *Food and Drugs Act* (Health Canada 2007b). Some traditional products can contain high levels of lead, but these uses are not permissible in Canada. Greta and Azarcon, Hispanic remedies

[2] Health Canada proposes to extend the 90-mg/kg total lead limit under the CPCLR to include all toys for children under 14 years of age, child care articles and equipment, and children's clothing and accessories.[3] The INSPQ study did not measure soil lead concentrations, therefore soil lead concentrations could not be controlled for in the model.

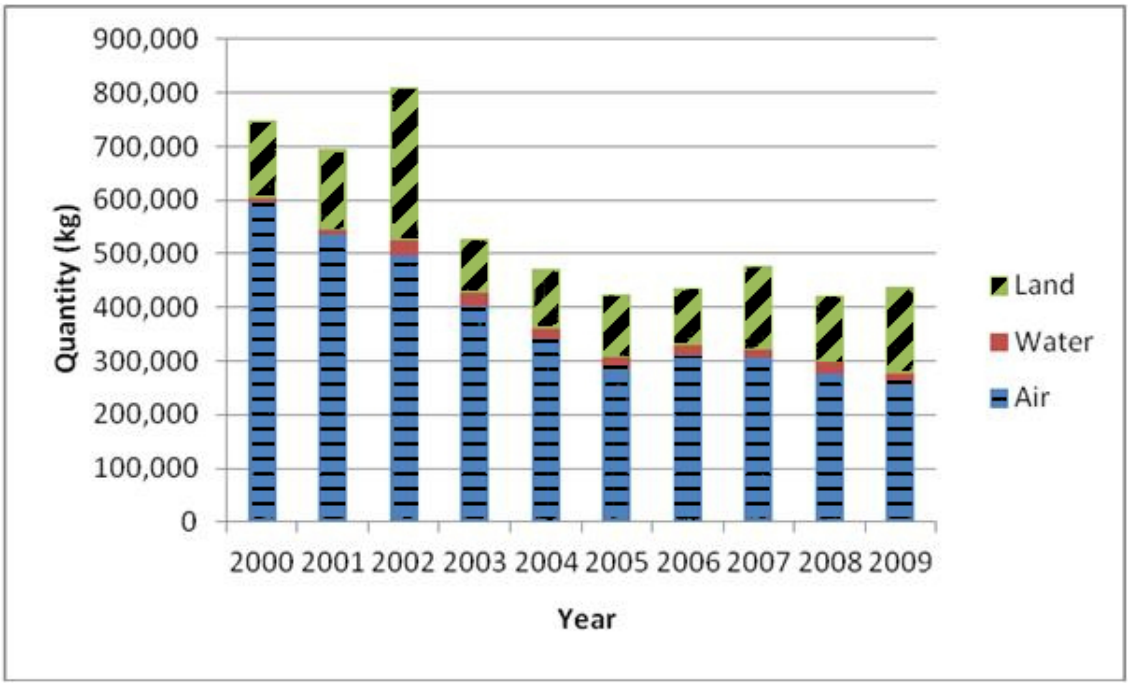
taken for an upset stomach, contain over 90% lead by weight in the form of lead oxide and lead tetraoxide, respectively (Baer and Ackerman 1988; U.S. CDC 2010a). Traditional Kohl eye makeup contains lead in addition to herbs, ashes and other materials; it contains principally lead sulphide but it may also contain lead tetraoxide or lead carbonate (Alkhawajah 1992; Vaishnav 2001; Health Canada 2010c). In Canada, lead may be found as an impurity in a small number of pesticide products, as a formulant impurity in several rodenticides and antifouling paints, and as an impurity in the technical grade of several active ingredients (2010 email from Pest Management Regulatory Agency, Health Canada, to Risk Management Bureau, Health Canada; unreferenced). Owing to lead's ubiquitous nature, it is present in many consumer products as either unintended residues or impurities in other metals. Lead is listed on Schedule 1, List of Toxic Substances, under CEPA 1999.

Releases to the Environment

After the prohibition of leaded fuel for use in on-road vehicles, the primary anthropogenic sources of lead include releases from the mining and smelting of lead ores as well as other ores in which lead is a by-product or contaminant, processing, use, recycling, or disposal (WHO 1995; ATSDR 2007). Electrical utilities also release lead into the environment in flue gas from the burning of fuels such as coal, in which lead is a contaminant (ATSDR 2007).

Releases of lead to the environment are estimated based on Environment Canada's National Pollutant Release Inventory (NPRI). Response to the NPRI is required for those organizations that meet reporting criteria; however, the NPRI does not represent all releases to the environment, and in the case of lead, it does not include some significant sources (e.g., releases to land and water from facilities that are not required to report to the NPRI and lead in products such as shot and sinkers). According to the NPRI database, in 2009, releases of lead and lead compounds into the Canadian environment totalled approximately 436 000 kg: 260 000 kg to air, 16 000 kg to water, and 160 000 kg to land (Figure 3) (Environment Canada 2010a). Of the releases to air, approximately 70% of the total was released by the mining and metals production industries and 17% was released by air transportation; Canadian military bases accounted for approximately 85% of the releases to land (Environment Canada 2010a).

Figure 3. Releases and emissions of lead into the Canadian environment as reported to the NPRI



Sources of Exposure and Concentrations in Environmental Media and Food

Owing to a long history of global anthropogenic use and its naturally occurring presence, lead is ubiquitous in the environment. Canadians may be exposed to lead through environmental media, their diet, and various other sources, including health and consumer products. The main route of exposure to lead for the general adult population currently is oral exposure from food and drinking water, followed by inhalation (ATSDR 2007; EFSA 2010). Inhalation is an important route of exposure for individuals living in the vicinity of point sources of lead (UNEP 2010). The dermal route is not considered to be a significant route of exposure to lead. For infants and children, who have different behaviours from adults, including crawling, greater frequency of hand-to-mouth contact and mouthing behaviour, additional sources of exposure include oral intake of paint chips, house dust and soil contaminated with lead, primarily sourced from older lead-containing paint. Factors that contribute to lead exposure for the general population include oral intake from products which could contain lead, particularly older products, (e.g., costume jewellery, toys, leaded crystal, art supplies), living in or frequently visiting older buildings that contain deteriorating lead paint or that are undergoing renovation activities, and behaviours such as smoking or pica (CoEH 2005; ATSDR 2007, 2010; Bushnik et al. 2010).

Concentrations of lead in Canadian ambient air, soil, indoor air and house dust, food, and drinking water are presented in the following sections. Measurements of lead in environmental media are based on the lead moiety contained within unspecified lead substances and therefore represent exposure to total lead.

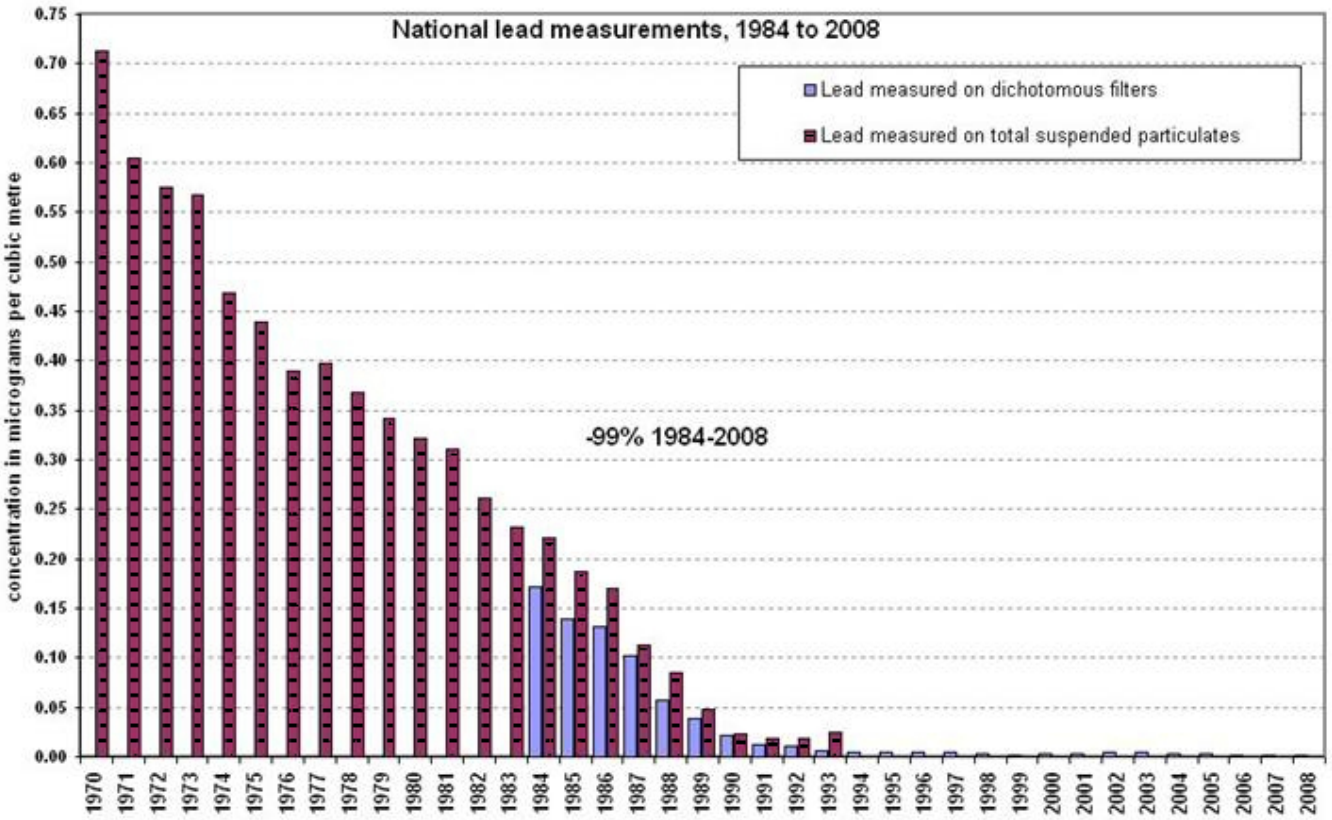
Since the literature cut-off for the draft State of the Science (SOS) report in February 2011, a number of Canadian studies have become available. The Institut national de santé publique du Québec (INSPQ 2011) conducted a cross-sectional study in four boroughs of Montréal, Quebec, from September 2009 to

March 2010. The study sample was randomly selected from the targeted boroughs. This study simultaneously evaluated the BLLs of young children (ages 1 to 5) and the potential sources of exposure to lead (dust, paint, and water) in their home indoor environments in order to determine the impact of these sources on their BLLs. A second study was conducted in St. John's, Newfoundland and Labrador, in the summer and fall of 2010 (Bell et al. 2011). Using a convenience sample, the study investigated the role of housing age in lead exposure of young children (aged 6 months to 6 years) in St. John's. A cross-sectional design was followed, where exposure (lead levels in dust, paint, soil, tap water and garden produce) and children's BLLs were measured over a similar time period. A third cross-sectional study was conducted to examine the distribution of BLLs and the prevalence of BLLs higher than 10 µg/dL in (North) Hamilton, Ontario from 2008 to 2009 (Richardson et al. 2011). A secondary objective was to explore associations between potential lead sources, specific risk and mitigating factors, environmental lead concentrations, and children's BLLs. The study sample was self-selected due to low response in the study area. Lead concentrations were measured in water, dust, and soil samples as well as in the blood of children under 7 years of age. Canada is the first country to design a statistically representative national baseline study for lead concentrations in dust sampled from 1025 urban households, entitled the Canadian House Dust Study (CHDS). Samples for the CHDS were collected between 2007 and 2010 and included wipe sampling (McDonald et al. 2010, 2011) and vacuum sampling (Rasmussen et al. 2011). The above studies have been reviewed and incorporated into the sections that follow. Overall, these data demonstrate that current environmental lead concentrations have a measurable effect on BLLs in children.

Ambient Air

Ambient air concentrations of lead are measured and reported as part of Environment Canada's National Air Pollution Surveillance (NAPS) program at 26 of 286 sites nationwide (Environment Canada 2010b). Under this nationwide program, filter-based samples of particulate matter (PM) having aerodynamic diameter less than 2.5 µm ($PM_{2.5}$) are collected and analyzed for a variety of elements, including lead. $PM_{2.5}$ is respirable and therefore available for systemic absorption. NAPS data demonstrate that ambient air lead concentrations in Canada have declined significantly following the introduction of unleaded gasoline in Canada in 1975 and the prohibition on leaded gasoline for use in on-road vehicles in the 1990s. As shown in Figure 4, in Canada, average ambient air concentrations of lead declined by more than 99% between 1984 (0.16 $\mu g/m^3$) and 2008 (< 0.0015 $\mu g/m^3$) (Environment Canada 2010b). The prohibition on leaded fuels, combined with the imposition of greater controls on lead mining and smelting emissions, has resulted in average ambient atmospheric lead concentrations consistently below 0.02 $\mu g/m^3$.

Figure 4. Average concentration of lead in ambient air in Canada



Source: Data were obtained from Environment Canada (2010b).

Between 2000 and 2009, more than 12 000 individual ambient air measurements were reported. During this 10-year period, the 5th to 95th percentile concentrations of lead $PM_{2.5}$ in Canada ranged from 0.0004 to 0.014 µg/m^3; the 5th, 50th, and 95th percentile concentrations of lead are presented in Table 2.

Table 2. Concentration of lead in $PM_{2.5}$ (µg/m^3) in Canada by percentile

Year	Concentration of lead (µg/m3)[a]		
	5th Percentile	50th Percentile	95th Percentile
2000	0.0004	0.0016	0.014
2001	0.00048	0.0024	0.012
2002	0.00052	0.0027	0.013
2003	0.00081	0.0025	0.011
2004	0.00089	0.0016	0.0084
2005	0.0012	0.0014	0.0084

Year	Concentration of lead (µg/m3)[a]		
	5th Percentile	50th Percentile	95th Percentile
2006	0.00062	0.0014	0.0068
2007	0.00051	0.0014	0.0056
2008	0.00051	0.0012	0.011
2009	0.0023	0.0045	0.014

Source: Data were obtained from Environment Canada (2010c).

[a] Values < Limit of Detection (LOD) were assigned a value LOD/2. Between 2000 and 2008 the median LOD ranged from 0.001 µg/m^3 (2000 and 2001) to 0.003 µg/m^3 (2007). In 2009, the median LOD was approximately 0.007 µg/m^3. The LOD varied across the sampling time frame due to the use of different analytical instruments and protocols over time.

Leaded gasoline is permitted in aviation fuels used in small aircraft with piston engines. This use continues to be a source of lead in ambient air and is currently the largest single source of lead emissions to air, comprising approximately half of the national inventory released in the United States (U.S. EPA 2010a). Lead concentrations in air increase with proximity to airports where piston-engine aircraft operate (U.S. EPA 2010b). In a study conducted around North Carolina, children living within 500 m and 1000 m of an airport where leaded aviation gasoline was used had higher BLLs than other children (Miranda et al. 2011). In Canada, based on the NPRI, as industrial releases have declined, the contribution of lead releases to air from aircraft to total lead releases has increased, rising from approximately 9% in 2000 to approximately 17% in 2009.

Soil

Natural levels of lead in soil reflect the mineralogy of the soil parent (geological) material. Soils and sediments act as primary environmental sinks for lead compounds.

Given its historical dispersive uses, lead is found in virtually all surface soils around the globe, where it can remain indefinitely as a result of its non-volatility and low soil mobility. Due to lead's tendency to strongly adsorb to soils, it is generally retained in the upper soil layers, and its deposition is regarded as irreversible and permanent unless it is removed through remediation (ATSDR 2007).

In Canada, background lead concentrations in soils sourced from various geographical areas are available through Natural Resources Canada's Geological Survey of Canada (GSC). Lead concentrations in glacial till were reported to range from 1 to 152 mg/kg, with an arithmetic mean concentration of 9.65 mg/kg and a 90th percentile of 16 mg/kg, based on 7398 samples collected throughout Canada for the particle size fraction < 63 µm (Rencz et al. 2006). Glacial till is considered to represent the background concentration (i.e., the normal abundance of lead in unmineralized soil that is unaffected by anthropogenic activities) (Rencz et al. 2006).

Lead levels in soil tend to be higher in cities, near roadways, around industrial sources that use or emit lead, near weapon firing ranges, or next to homes, buildings, and structures such as lighthouses where crumbling leaded paint has fallen into the soil (CMHC 2009). Lead-contaminated soil can be tracked into residences and contribute to the lead content of indoor settled dust. In areas with lead-contaminated soils, soil lead appears to be the primary underlying source of variation in both internal floor dust lead and hand-wipe dust lead (Zahran et al. 2011). Several analyses have been conducted that link exterior

lead, measured by soil concentrations around the home or play area, to increased exposure in children (Lanphear et al. 1998; Bell et al. 2011).

Mean concentrations of lead in soil samples collected from residential areas and parklands in Canada range from 35.6 to 766 mg/kg, with individual samples ranging from 2.8 to 24 477 mg/kg (Table 3). Most soil samples have lead concentrations below the current Canadian Council of Ministers of the Environment (CCME) soil quality guideline for human health of 140 mg/kg, which is currently under revision (CCME 1999). Rasmussen et al. (2001) reported concentrations of lead in garden soil sampled during the winter of 1993 in Ottawa, Ontario where lead concentrations ranged from 16 to 547 mg/kg (n = 48) with a median concentration of 34 mg/kg.

Soil samples collected from cities located near point sources generally have higher mean lead concentrations than those collected in residential areas and parklands (Table 3). Average soil concentrations range from 13 to 750 mg/kg overall, with concentrations in individual samples ranging from 3 to 4800 mg/kg. Soil lead concentrations near point release sources, such as those observed in Flin Flon, Manitoba, the site of a copper–zinc smelter, are generally higher than those commonly found in communities not exposed to point release sources. The concentration of lead in Flin Flon's soil was reported to range from 5 to 1447 mg/kg, with a mean concentration of 196.4 mg/kg. Of the 106 sites tested in Flin Flon and neighbouring Creighton, Saskatchewan, 41% exceeded the CCME soil quality guideline for lead of 140 mg/kg (Manitoba Conservation 2007).

A study examining lead concentrations in soil in St. John's, Newfoundland and Labrador, demonstrated that the geometric mean soil lead concentration was significantly different between homes built before 1970 (geometric mean 187 mg/kg) and those built post-1980 (geometric mean 28.5 mg/kg) (Bell et al. 2011). Geometric mean soil lead concentrations declined from 292 mg/kg for houses predating 1946 to 169, 89, 57, 32, and 25 mg/kg for housing cohorts of 1946-1960, 1961-1970, 1971-1980, 1981-2000, and post-2000, respectively. Across all housing ages, soil sampled along the drip line of houses had, on average, higher lead concentrations (103 mg/kg) when compared with soil in play areas and gardens (85.8 and 52.8 mg/kg, respectively). However, BLLs in children in the study were most strongly correlated in a univariate analysis with soil lead concentrations from play areas followed by garden soil, and were not correlated as strongly with lead concentrations in drip line soil.

A 2008–2009 study conducted in (North) Hamilton, Ontario found that the main predictor of soil lead concentrations was proximity to current and historic industry, accounting for 8% of the variance in the soil lead concentrations (Richardson et al. 2011). In a univariate analysis, soil lead concentration was significantly correlated with BLLs in children; however, after controlling for other exposure media (i.e., dust, water) and significant modifiers (e.g., sex, age, and household income) in the regression model, the correlation was no longer significant. Soil lead concentration was found to be a strong contributor to household dust concentrations, suggesting that soil was not making a substantial unique contribution to children's BLLs from direct consumption; instead, soil lead's contribution to children's exposure was primarily through household dust (Richardson et al. 2011). It should also be noted that soil samples were collected approximately 7 to 10 months following the collection of blood samples, further affecting the interpretation of the results.

Table 3. Lead concentrations in soil (mg/kg)

Year	Location	n	Concentration (mg/kg)		Reference
			Arithmetic mean	Range	
Background					
-	Glacial Till, All Canada	7398	9.65	1-152	Rencz et al. 2006
Residential and Parkland					
-	Cariboo Region, BC[a]	-	-	9.5[b]	B.C. MOE 2010
-	Kootenay Region, BC[a]	-	-	75[b]	
-	Lower Mainland Region, BC[c]	-	-	60[b]	
-	Omineca Peace Region, BC[c]	-	-	35[b]	
-	Skeena Region, BC[a]	-	-	15[b]	
-	Thompson Nicola Okanagan Region, BC[c]	-	-	15[b]	
-	Vancouver Island Region, BC[c]	-	-	30[b]	
-	Vancouver, BC[a]	-	-	300[b]	
2003	Victoria, BC[c]	-	90[d]	-	Bowman and Bobrowsky 2003
2010	St. John's, NL (all)	190	91.9	2.8-6800	Bell et al. 2011
2010	St. John's, NL (dripline)	141	103	8.5-6800	
2010	St. John's, NL (play area)	182	85.8	3.8-1900	
2010	St. John's, NL (garden)	31	52.8	2.8-560	
2004-2005	St John’s, NL (all)	1231	446/148[d]	9-24477	Bell et al. 2010
2004-2005	St John’s, NL(dripline)	328	766/194[d]	15-24477	
2004-2005	St John’s, NL(ambient)	514	411/138[d]	9-12,738	
2004-2005	St John’s, NL(road)	389	222/136[d]	16-1765	
2010	Halifax, NS	220	109/43[e]	10-767	Heidary-Monfard 2011
1990-2007	Guelph, ON	91	142	5-420	Wellington-Dufferin-Guelph Public Health 2007
2000	Ottawa, ON	50	64.7/33.8[d]	15.60-547.44	Rasmussen et al. 2001
2009	(North) Hamilton, ON	-	-	<5–790[f]	Richardson et al. 2011
2006	Lake Claire Watershed, QC[e]	2	35.6	23.1-50	Ndzangou et al. 2006
Near / on local anthropogenic source					
1994-1999	Trail, BC	-	750	-	Hilts 2003
2004	Cold Lake, AB	63	13	3-37	Defense Research and Development Canada 2004

Year	Location	n	Concentration (mg/kg)		Reference
			Arithmetic mean	Range	
2006	Creighton, SK	13	92.5	6.3-250	Manitoba Conservation 2007
	Flin Flon, MB	93	196.4	5-1447	
	Cranberry Portage, MB[g]	1	21.9	-	
	Bakers Narrow, MB[h]	1	5.3	-	
2007	Iqaluit, NU	19	-	17-45	Laird 2010
2008	Toronto, ON	30	38	9-210	Aqua Terre Solutions Inc. 2009
2004	Sudbury, ON	365	49.98	3.5-194	Centre for Environmental Monitoring 2004
2000	Port Colborne, ON	17	217	6-1800	OMOE 2002
2010	Oakville, ON	5	-	<10-28.1	Fisher Environmental Ltd 2010
2010	Massawippi and Saint-François rivers, QC	58	-	12.32-149	Saint-Laurent et al. 2010
2009	Buchans, NL	53	729	27-4800	Conestoga-Rovers & Associates 2010
2004	Sydney, NS	55	297/340[d]	52-1700	Lambert and Lane 2004

- not reported
[a] Considered representative of uncontaminated soil concentration for each region.
[b] 95th percentile
[c] Roads, parks, schoolyards
[d] Median
[e] Average of 0-10 cm depth
[f] From homes built prior to 1950
[g] Represents minimally impacted conditions in the greater vicinity of Flin Flon, Manitoba
[h] Represents non-impacted conditions in the greater vicinity of Flin Flon, Manitoba

Indoor Air and House Dust

Canadians spend up to 90% of their time indoors, at home, at school, or in the workplace (Leech et al. 1996). Therefore, indoor environments have the potential to be a significant source of exposure to lead for Canadians.

In the United States, lead-based paint in homes built before 1960 and up to as recently as 1978 represented the most significant source of lead in indoor air (U.S. EPA 2010c). Lead from lead-based paint can be dispersed when the paint degrades and can then contaminate household dust, which, in turn, can become resuspended in air. Lead contamination can also arise from home renovation activities, such as paint removal by scraping, sanding, or open-flame burning. The use of lead in certain indoor activities, such as hobby soldering and stained-glass making, can also contribute to elevated lead concentrations in indoor air. Consistent with these findings on indoor air, studies have frequently implicated household settled dust as a major source of lead exposure through ingestion, particularly for infants, toddlers, preschoolers, and young children (Rabinowitz et al. 1985; Lanphear et al. 1996a, 1996b, 1998; Manton et al. 2000; Roy et al. 2003). The potential sources of lead contamination of indoor settled dust can vary and include exterior contaminated soil, deteriorating paint containing lead, consumer products, various hobbies, such as welding, making stained-glass articles and pottery making, and activities such as smoking (HUD 2001; Jacobs et al. 2002; Sanborn et al. 2002).

Three Canadian studies have been identified that report lead concentrations in indoor air associated with $PM_{2.5}$. Rasmussen et al. (2006) reported lead concentrations in indoor air in homes of non-smokers in

Ottawa, Ontario, from samples collected in 2002. The reported concentrations of lead in $PM_{2.5}$ ranged from 0.0004 to 0.0027 μg/m^3 in rural residences (n = 10, median concentration = 0.0023 μg/m^3, LOD = 0.0002 μg/m^3), and from 0.0010 to 0.0051 μg/m^3 in urban residences (n = 10, median concentration = 0.0015 μg/m^3, LOD = 0.0002 μg/m^3). Matched indoor, outdoor, and personal $PM_{2.5}$ samples were collected in Windsor, Ontario, and analyzed for lead content; the median lead concentration of all the samples was reported to fall in the range of 0.001–0.010 μg/m^3 (LOD = 0.002 μg /filter and 0.15 mg particles/filter) for eight samples collected in 2004 and 37 samples collected during 2005–2006; the highest concentrations were found in outdoor air (Rasmussen et al. 2007, 2009). Information on indoor sources of lead from the Ottawa and Windsor homes sampled was not available.

In 2001, Rasmussen et al. reported multi-element profiles of indoor dust collected during the winter of 1993 from a total of 50 residences of smokers and non-smokers located in 10 neighbourhoods in Ottawa, Ontario. The total lead concentration in settled house dust samples ranged from 50 to 3200 mg/kg (n = 48), with a median concentration of 220 mg/kg; the study did not segregate lead concentrations in dust based on household smoking status. More recently, several studies have examined the concentration of lead in dust on a national level and in several older Canadian communities, where elevated lead concentrations in dust may be expected; results are presented in Table 4.

The CHDS reported that the concentration of bioaccessible lead in vacuum samples ranged from 7.9 to 3916 mg/kg, with median and geometric mean concentrations of 63 and 74 mg/kg, respectively (Rasmussen et al. 2011). Bioaccessible lead concentrations represent the concentrations of lead extracted from the sample under biologically relevant conditions (i.e., in simulated stomach fluids) and typically represent between 63% and 81% of the total lead content of the sample, depending on the solubility of the lead compounds present (Rasmussen et al. 2011). Lead compounds characterized by low solubility (e.g., elemental lead and lead sulfate) can undergo transformation reactions to more soluble forms (e.g., lead carbonate and lead oxide) during weathering under humid conditions (MacLean et al. 2012).

The CHDS identified three distributions of lead in household dust from technician-collected vacuum samples. The first distribution included 90% of homes; the household dust lead concentration in the homes was less than 250 mg/kg, and the geometric mean was 57 mg/kg. The second distribution included 7.5% of homes, which had household dust lead concentrations ≥ 250 and < 975 mg/kg with a geometric mean concentration of 447 mg/kg. The third, and highest, distribution comprised 2.5% of homes; these homes had household dust lead concentrations greater than or equal to 975 mg/kg, with a geometric mean lead concentration of 1730 mg/kg. The second and third groups were, in general, populated with progressively older homes; however approximately 10% of the homes with lead concentrations greater than 250 mg/kg were built after 1980 (Rasmussen et al. 2011).

Sources of lead in household dust were investigated in four homes in the CHDS with lead concentrations greater than 1000 mg/kg (MacLean et al. 2011). The homes were built in 1905, 1982, 1999 and 2000. Minerals commonly found in lead-based paint were identified in the dust of all four homes. However, paint in these homes was not sampled, so it was not possible to attribute paint as the source, particularly in the newer homes, where lead levels in paint are expected to be lower. The other lead species identified in dust included metallic lead, lead species found in soil, and organic lead species. Multiple organic lead species were found in household dust, source identification was not possible. Authors noted that indoor sources should be considered as well as outdoor sources for organic lead in household dust based on the use of organic lead in some consumer products (MacLean et al. 2011).

Speciation of lead in household dust samples collected with a purpose built vacuum system from one 65-year-old two-storey home and one two-storey home of unidentified age in Ottawa, Ontario, was recently reported (Beauchemin et al. 2011; Walker et al. 2011). Information on the types of lead species present in house dust aids in source characterization. Walker et. al. (2011) reported that lead levels from dust in the upstairs bedrooms, where recent renovations had been completed, were substantially higher than in the living room (adult bedroom 14,000 mg/kg versus living room 240 mg/kg). This study reports lead particles in dust from the main floor living room were consistent with lead particles found in garden soil, whereas dust particles in the upstairs bedrooms were primarily consistent with the components of paint (including white lead and lithopone) (Walker et al. 2011). Beauchemin et al. (2011) analyzed samples of paint, plaster, and household dust. The results demonstrated that in this 65-year-old home, paint was a major contributor to the lead content of household dust.

Dust from household vacuum bags of 201 homes in four selected older boroughs of Montréal, Quebec, were reported to contain geometric mean concentrations of 116.54 mg/kg (range 2.90–6897.80 mg/kg) bioaccessible lead and 201.49 mg/kg (range 6.20–8000.00 mg/kg) total lead (Gauvin et al. 2011). The geometric mean concentration of bioaccessible lead in these older homes (116.54 mg/kg) was higher than the geometric mean concentration (74 mg/kg) reported in the nationally representative CHDS. Wipe samples were also collected in these Montréal homes and are presented with other recent Canadian studies and one analysis of U.S. data in Table 4; results obtained in these Canadian studies are similar to those recently reported in the U.S. National Health and Nutrition Examination Survey (NHANES) study.

Wipe sampling involves the movement of a wet wipe over a known area to collect dust. During analysis, the wipe and all adsorbed dust are digested and measured. Results are reported as an amount of lead per area, which is termed “loading”. Wipe dust samples were collected from 222 randomly selected Ontario homes as part of the CHDS; lead loading levels ranged from < LOD to 67 $\mu g/ft^2$ (McDonald et al. 2010). Lead concentrations in wipe dust samples, in addition lead concentration in blood and other environmental media, were measured in three Canadian studies that targeted older homes and communities in Montréal, Quebec (INSPQ 2011), St. John’s, Newfoundland and Labrador (Bell et al. 2011), and Hamilton, Ontario (Richardson et al. 2011); lead loading levels ranged from < LOD to 6101 $\mu g/ft^2$. In the U.S., wipe samples were collected along with measurements of BLLs in children 12 to 60 months of age as part of NHANES from 1999 to 2004 (Gaitens et al. 2009). The geometric mean loading levels were 0.52 $\mu g/ft^2$ (n=2065 homes) for all non-missing floor dust and 7.64 $\mu g/ft^2$ (n=1618 homes) for window dust. The authors noted that the NHANES sampling strategy is representative of the U.S. population and not housing stock. The results obtained in the Canadian studies are within the same order of magnitude to those recently reported in the NHANES study.

Four studies identified a correlation between dust lead loadings and age of the home, with older homes having higher floor dust lead loadings (Gaitens et al. 2009; McDonald et al. 2010; Bell et al. 2011; INSPQ 2011). Three of the studies (Gaitens et al. 2009; Bell et al. 2011; INSPQ 2011) noted a significant difference in lead loading levels between homes built before and after approximately 1950. Gaitens et al. (2009) noted that the difference coincided with reductions in the lead content of paint in the U.S.

Prior to 1940, paint in the U.S. typically contained between 20% and 50% lead. In 1955, a voluntary paint industry standard reduced this to 1%, followed by regulations in 1978 that limited the lead content to 0.06% by weight (Gaitens et al. 2009). In Canada, restrictions limiting the amount of lead in indoor paint under the HPA to 5000 mg/kg (0.5%) were implemented in 1976. The reduction of median lead

concentrations over time was observed in a study conducted by INSPQ (2011); median concentrations of lead in paint chips were 1900 mg/kg (homes built before 1920), 1100 mg/kg (homes built between 1920 and 1949), and 395 mg/kg (homes built between 1950 and 1974). The median concentration of lead in paint chips sampled from homes built in 1975 and later was below the LOD of 10 mg/kg (maximum concentration = 12 mg/kg). The maximum concentration of lead in paint chips (260 000 mg/kg) was measured in a home built between 1950 and 1974. Bell et al. (2011) reported a median concentration of lead in paint chips of 1651 mg/kg for St. John's homes built before 1946, 191 mg/kg (for homes built between 1946 and 1970), 46.1 mg/kg (for homes built between 1971 and 1980), and 27.5 mg/kg (for homes built in 1981 and later). Additionally, a statistically significant correlation was observed between the concentration of lead in paint chips and lead loadings in household dust in St. John's (Bell et al. 2011). In the North Hamilton study, lead concentrations in paint chips were not measured; however, the neighbourhoods with the highest median lead house dust loadings corresponded to the neighbourhoods with the oldest housing stock and higher median yard soil lead concentrations (Richardson et al. 2011).

Table 4. Lead loadings in household dust in Canadian residential homes

Year	Location	n	Sample location	Median/ geometric mean concentration	% < LOD[a]	Concentration range	Reference
Vacuum samples: bioaccessible lead (mg/kg)							
2007–2010	Canada	1025	Whole house, technician collected	63/74	0	7.9–3916	Rasmussen et al. 2011
2010–2011	4 boroughs of Montréal, QC	201	Not stated, participant collected[b]	93/117	0	2.9–6898	Gauvin et al. 2011
Wipe samples (µg/ft^2)							
2008	Ontario[c]	208	Entry	0.52/-	22	<0.086–58	McDonald et al. 2010
		218	Kitchen	0.095/-	49	<0.086–15	
		114	Living room/family room	<LOD/-	49	<0.086–45	
		93	Adult bedroom	0.30/-	43	<0.086–39	
		50	Child bedroom	<LOD/-	60	<0.086–67	
		23	Playroom	0.32/-	26	<0.086–20	
2010–2011	4 boroughs of Montréal, QC[d]	305	Floor	0.70/0.85	—	0.08–90.9	INSPQ 2011
		263	Child bedroom window sill	7.15/7.14	—	< LOD[e]–1216	
2010	St. John's, NL	194	Floor	0.6/0.61	—	0.06–407	Bell et al. 2011
		184	Window sill	1.35/1.1	—	<LOD[f]–1236	
		180	Window trough	17.8/17.8	—	<LOD[e]–6101	
2008–2009	(North) Hamilton, ON	—	Living room floor	—	—	<1–109[g]	Richardson et al. 2011
1999–2004	United States	2065	All floor	-/0.52	—	—	Gaitens et al. 2009
		1618	Window sill	-/7.64			

not reported
[a] Limit of detection.
[b] The vacuum bags or contents of a canister vacuum used by the participant were obtained and submitted for analysis.
[c] Samples collected in 222 Ontario homes during January–March 2008: Barrie (57 homes), Greater Sudbury (86 homes) and Thunder Bay (79 homes).
[d] Samples collected between September 2009 and March 2010.
[e] LOD is reported to be 0.019 µg.
[f] LOD is reported to be 0.125 µg.
[g] From homes built prior to 1950. Report indicates that 196 households participated in environmental sampling; only a small number were built after 1950.

A significant relationship between house dust and BLL has been observed in recent studies (Dixon et al. 2009; Bell et al. 2011; INSPQ 2011; Richardson et al. 2011). In the INSPQ study, children's geometric mean BLLs were significantly different when comparing the first and third tertiles in floor dust and window sill dust lead loadings and when comparing the first and third categories of lead concentrations in paint (INSPQ 2011). For floor dust lead loading, children's BLLs increased from 1.20 µg/dL (first dust loading tertile, ≤ 0.45 µg/ft^2) to 1.48 µg/dL (third dust loading tertile, > 1.22 µg/ft^2). The significant difference between the first and third tertiles of floor dust lead loading was maintained when adjusting for different variables (e.g., age, sex, minority status, season, education). However, the difference was no longer significant when adjusted for other exposure variables, such as lead concentration in kitchen tap water and paint (INSPQ 2011).[3] For paint, the BLLs increased from 1.24 µg/dL (first category; x-ray fluorescence (XRF) surface measurement < 1mg/cm^2) to 1.63 µg/dL (third category; paint chips ≥ 5000 mg/kg). Similarly to floor dust, when lead levels in paint were in the third category, there was a significant relationship between BLL and lead concentration in paint when accounting for confounders, but the relationship was no longer significant when other exposure sources (kitchen tap water and dust) were considered. In the winter samples, for window sill dust lead loading, it was reported that children's BLLs increased from 1.42 µg/dL to 2.45 µg/dL for the first (≤ 3.54 µg/ft^2) and third tertiles (> 14.14 µg/ft^2), respectively. In this case, the association remained significant when controlling for confounders and when controlling for other exposure factors (kitchen tap water and paint) (INSPQ 2011). Bell et al. (2011) reported statistically significant correlations between BLL and lead loadings in household dust; the correlation was strongest for floor dust (r^2 = 0.128) and weakest but still significant for window trough dust (r^2 = 0.055). In the North Hamilton study, based on a univariate analysis, approximately 5 to 8% of the variance in the children's BLLs was accounted for by household dust (r^2 = 0.053–0.078) (Richardson et al. 2011). Richardson et al. (2011) found that yard soil is a strong contributor to household dust lead levels but does not appear to independently influence BLL, and older homes had higher lead concentrations in both dust and soil. A significant association between children's BLLs and floor and window household dust loading as well as renovations over the previous 12 months was found in the U.S. NHANES data for 1999-2004 (Dixon et al. 2009). A model constructed on these data was able to predict BLLs in U.S. children over household floor dust lead loading ranging from 0.25 to 40 µg/ft^2.

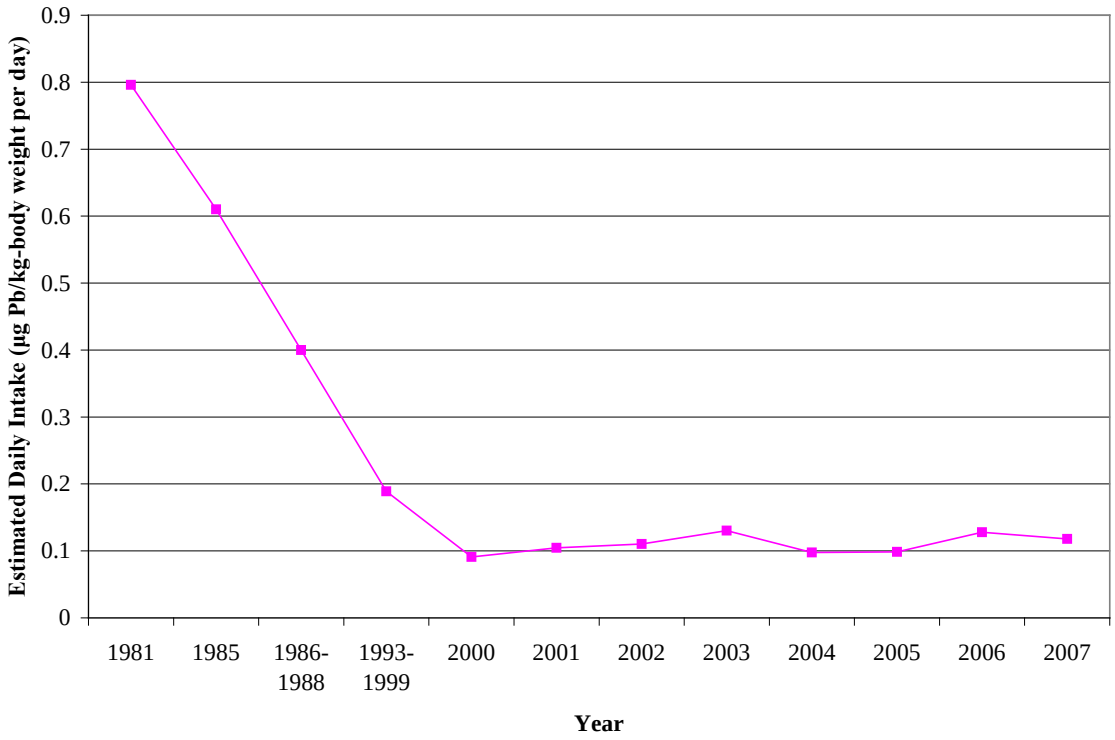
[3] The INSPQ study did not measure soil lead concentrations, therefore soil lead concentrations could not be controlled for in the model.

Food

Before the phase-out of lead in gasoline, inhalation was the predominant route of exposure to lead for the general public (ATSDR 2010). Lead levels in all environmental media (except soil) have since declined, and the predominant route of lead intake for the general adult population is currently oral exposure from food and drinking water (ATSDR 2007, 2010; EFSA 2010). While there are no permissible Canadian food uses for lead, it has been detected in a variety of foods. Lead is primarily introduced to foods through uptake from soil into plants and deposition onto plant surfaces. For example, leafy vegetables grown in lead-bearing soil will contain lead in their leaves and have lead-containing particles on their surface (ATSDR 2007). Fish can absorb lead from water and sediments while other animals may be exposed to lead through the foods they eat (Health Canada 2011a). Additionally, lead may be introduced to foods during transport to market, processing, and kitchen preparation including cooking with water contaminated with lead, or from the use of lead-containing utensils and storage of food in lead-containing vessels, such as lead-glazed ceramic foodware and lead crystal ware (U.S. EPA 1986a; Health Canada 1992; ATSDR 2010). Consumption of wild game that has been shot with lead bullets is another potential source of dietary lead exposure (Tsuji et al. 2008; Health Canada 2011a). The European Food Safety Authority identified uptake from soil as the primary source of lead in foods (EFSA 2010).

Since 1969, Health Canada has carried out a Total Diet Study (TDS) to estimate levels of exposure to chemicals through the food supply for Canadians in different age and sex groups and cities (Health Canada 2010d). To date, six separate studies have been undertaken. Five were carried out between 1969 and 1973, 1976 and 1978, 1985 and 1988, 1992 and 1999, and 2000 and 2004; the most recent TDS was initiated in 2005, and data are available up to 2007. Lead concentrations in foods in the Canadian marketplace are currently low. Health Canada TDS results show that the dietary intake of lead (in units of μg/kg body weight [bw] per day) by average Canadians of all ages and sexes has decreased since 1981 (Figure 5). The major reason for this decrease has been the replacement of lead-soldered cans for food storage with lead-free cans.

Figure 5. Estimated dietary intake of lead by average Canadians (all ages and sexes)



Estimated dietary intake of lead from food for all ages of the general Canadian population is approximately 0.1 µg/kg body weight per day. Overall, dietary exposures are generally higher for children and decrease with age (Health Canada 2011b). Based on data collected as part of the First Nations, Food, Nutrition and Environment Study (FNFNES), average daily intake of lead from food and tap water for BC First Nations people living on reserve was estimated to be 0.23 µg/kg body weight per day (Chan et al. 2011).

Data on lead levels in table-ready food assessed as part of the Canadian TDS conducted from 2003 to 2007 are presented in Appendix 1. Lead levels ranged from less than 0.1 µg/kg in natural spring water to 392 µg/kg in herbs and spices. During the years 2003 to 2007 lead levels were consistently highest in herbs and spices (ranging from 292 to 392 µg/kg), with the next highest food product being salt (ranging from 41.5 to 202 µg/kg). The food groups contributing most to the dietary intake of lead since 2004 in Canada are beverages (e.g., beer, wine, coffee, tea, soft drinks), cereal-based foods, and vegetables (Health Canada 2011a). The largest lead contributor to European dietary exposure is cereal products (EFSA 2010).

Lead concentrations in traditional food samples consumed by BC First Nations people living on reserve have recently been measured as part of the FNFNES. Lead concentrations in all food items were at background level except for beaver heart, Canada goose, deer, and grouse meat. The highest concentration of lead was reported in grouse meat, at 61 µg/kg; the source of this lead was likely from

lead shot. Heavy consumption of some game meat may be associated with an increased risk of lead exposure due to lead shot contamination (Chan et al. 2011).

In addition to the TDS and FNFNES, lead is also measured in food through the Canadian Food Inspection Agency's (CFIA) National Chemical Residue Monitoring Program (NCRMP) and Children's Food Project. The Children's Food Project Report on Sampling for 2007–2008 reported lead concentrations in 836 processed food samples (CFIA 2011). The samples included grain, dairy, and processed fruit and vegetable products, as well as miscellaneous products, such as ready-to-eat meals (Table 5). The highest lead levels were found in grain-based products; 162 of the 365 samples had lead levels above the detection limit. For those 162 samples, the mean lead concentration was 25 μg/kg and the range was from 2 to 977 μg/kg. The Children's Food Project Report on Sampling for 2006–2007 (Table 5) measured 350 samples, with 11 results above the detection limit (CFIA 2009). The highest lead concentration was reported in organic vegetable baby food (140 μg/kg). Concentrations of lead in foods from the most recent NCRMP annual report for 2005–2006 are presented in Appendix 2; the highest concentration of 2040 μg/kg reported came from 1 of the 81 samples of chicken muscle (CFIA 2010).

Table 5. CFIA Children's Food Chemical Residues Project results

Metal analyte	Number of Samples			Concentration (μg/kg)		
	Total no. samples	Total no. negative	Total no. positive	Minimum	Maximum	Mean
Sampling years 2006–2007 (CFIA 2009)						
Total	350	339	11			
Baby food, fruit	34	33	1	60	60	60
Baby food, fruit, organic	10	9	1	50	50	50
Baby food, meat	10	10	0			
Baby food, poultry	14	14	0			
Baby food, poultry, organic	2	1	1	40	40	40
Baby food, vegetable	20	20	0			
Baby food, vegetable, organic	10	9	1	140	140	140
Fruit/vegetable juice	132	132	0			
Fruit/vegetable juice, organic	18	18	0			
Cookie	94	89	5	40	100	70
Cookie, organic	6	4	2	40	40	40
Sampling years 2007–2008 (CFIA 2011)						
Total	836	445	391			
Fruit- and vegetable-based	375	187	188	2	344	22

Metal analyte	Number of Samples			Concentration (µg/kg)		
	Total no. samples	Total no. negative	Total no. positive	Minimum	Maximum	Mean
Grain-based	365	203	162	2	977	25
Dairy-based	38	30	8	2	42	15
Miscellaneous	58	25	33	0	159	23

Drinking Water

Drinking water is a source of exposure to lead. Lead may be introduced into drinking water as a result of dissolution from lead service connections, old lead-based solders used to join copper pipes within homes and buildings, and plumbing fittings, faucets, and components containing lead.

The amount of lead leaching from the plumbing system is affected by a number of factors, including the age of the plumbing system, the chemistry of the water (e.g., water temperature, pH, buffering capacity/alkalinity), and the length of time the water sits in the pipes (Health Canada 2009a). Seasonal variations in temperature between the summer and winter months have been correlated with changes in lead concentrations, with the warmer temperatures of the summer months increasing lead concentrations (Britton and Richards 1981; Karalekas et al. 1983; Colling et al. 1987, 1992; Douglas et al. 2004). Douglas et al. (2004) reported a strong seasonal variation in lead concentration in Ottawa, Ontario, with the highest lead concentrations seen from May to November.

The introduction of the disinfectant chloramine to water systems with lead-containing pipes, fixtures, or solder may increase the amount of dissolved lead in water due to changes in water chemistry. In Washington, D.C., a change of disinfectant from free chlorine to chloramine in the early 2000s caused an elevation in drinking water lead concentrations which resulted in elevated BLLs for children (≤ 1.3 years) consuming that water (Edwards et al. 2009). A similar trend was observed in North Carolina, where the use of chloramine disinfectant was a significant predictor of BLL in children (Miranda et al. 2007a). In this study, lead concentrations in water were not measured, and the authors suggested that the increase in BLL was caused by an increase in lead concentration in drinking water resulting from the switch to chloramine. The impact of chloramines on children's BLL was mitigated in newer housing, where the presence of lead service lines and lead solder was less likely. The increase in BLL for children where chloramine was used was greatest in houses built before 1926, followed by houses built in 1926–1950 and 1951–1975 (Miranda et al. 2007a).

A study conducted by the U.S. Centres for Disease Control and Prevention examined the relationship between lead service lines and BLLs in children in Washington, D.C. between 1998 and 2006. The study found that children tested after a partial replacement of their lead service lines (the length of the water service line from the water main to the corporation valve was replaced) were more than 3 times as likely to have a BLL ≥ 10 µg/dL compared with children who had never had a lead service line (odds ratio [OR] 3.3, CI 2.2-4.9) (Brown et al. 2011). In addition, there was no significant difference between children's BLLs in homes with an intact (full) lead service line (the entire length of the service line from the water main to the water meter in the residence) and in those homes with a partially replaced one. Based on these results, the study authors concluded that partially replacing lead service lines may not decrease the risk of elevated BLLs associated with lead service line exposure (Brown et al. 2011).

However, the study was not designed to quantify the impact of lead concentrations in drinking water on children's BLLs.

There is no national database for lead concentrations in Canadian drinking water; however, many municipalities and provinces maintain databases on the results of water quality analyses, including the concentration of lead (Table 6). The Province of Ontario has published results of its Drinking Water Surveillance Program, which include data on the concentrations of lead in treated water as it leaves the water treatment facility (OMOE 2011). These data indicate that average concentrations of lead in treated water leaving the water treatment facility are currently less than 1 µg/L, and historical data suggest that they have been at this level since 2000. Average concentrations are well below Health Canada's Maximum Acceptable Concentration (MAC) of lead in drinking water of 10 µg/L and are consistent with lead concentrations in drinking water at the tap, resulting primarily from the contribution of materials in the distribution system (e.g., lead service lines) and plumbing system.

Table 6. Concentrations of lead (µg/L) in treated water exiting treatment facilities in Canada

Year	Location	n	Concentration (µg/L)			Reference
			Median/ mean	Minimum	Maximum	
2005	Victoria	2	1.6/-	-	-	CRD 2010
2006		3	<0.5/-	-	-	
2007		10	<0.5/-	0.3	<0.5	
2008		9	0.3/-	<0.2	0.9	
2009		10	0.4/-	<0.2	0.5	
2009	Calgary	-	<0.5/-	-	-	Calgary 2010
2000	Ontario	451	0.1/-	<0.05	4.7	OMOE 2011
2001		435	0.05/-	<0.05	8.7	
2002		375	0.04/-	<0.05	4.88	
2003		377	<0.05/-	<0.05	6.83	
2004		256	0.07/-	<0.05	10.3	
2005		356	0.04/-	<0.01	20.8	
2006		328	0.03/-	<0.01	5.14	
2007		329	0.02/-	<0.01	29	
2006	Montréal	-	-/<1	<1	<1	Montréal 2010
2007		-	-/<1	<1	<1	
2008		-	-/<1	<1	<1	
2009		-	-/1.03	0.78	1.27	
2008–2010	Halifax and Dartmouth	-	Typical, <0.5	-	-	Halifax 2011
2009–2010	Halifax and Dartmouth	-	Typical, <0.5	-	-	Halifax 2011
2005	Yukon	-	-/-	<0.1	0.4	Yukon 2011

Year	Location	n	Concentration (µg/L)			Reference
			Median/ mean	Minimum	Maximum	
2006		-	-/-	<1.0	1.8	
2007		-	-/-	<0.1	0.3	
2008		-	-/-	<0.05	0.98	
2009		-	-/-	<0.2	0.5	
2010		-	-/-	<0.1	7.6	
2005	Manitoba	248	-/-	<0.2	11	Manitoba 2011
2006		136	-/-	<0.2	14	
2007		144	-/-	<0.2	11	
2008		142	-/-	<0.2	19	
2009		102	-/-	0.09	10.6	
2010		108	-/-	0.09	9.44	
- not reported						

Concentrations of lead in drinking water distribution systems from the National Survey of Disinfection By-Products and Selected Drinking Water Contaminants in Canadian Drinking Water (2009–2010) (Tugulea 2011), the Ontario Drinking Water Surveillance Program (years 2000–2007), Saskatchewan (2005–2010), and Charlottetown, Prince Edward Island (2009) are presented in Table 7. The national survey sampled 65 sites in all provinces and territories in the summer and winter seasons during 2009–2010. However, the results are not statistically representative of Canadian population exposure, and samples were collected from the system after 10 minutes of flushing, which represented the distributed water after it left the water treatment facility and before it reached the service line or plumbing. The median concentration in winter was less than the method detection limit of 0.5 µg/L (range: < 0.5−8.2 µg/L) and was 0.6 µg/L (range: < 0.5−24 µg/L) for summer samples.

Table 7. Concentrations of lead (µg/L) in drinking water distribution systems in Canada

Year	Location	n	Concentration (µg/L)			Reference
			Median	Minimum	Maximum	
2009–2010	Various locations across Canada	65[a]	0.6	<0.5[b]	24	Tugulea 2011
		31[c]	<0.5[d]	<0.5[d]	8.2	
2000	Ontario	445	0.32	<0.05	18.5	OMOE 2011
2001		447	0.28	<0.05	29	
2002		369	0.28	<0.05	8.66	
2003		376	0.23	<0.05	13.5	
2004		390	0.18	<0.05	359	
2005		362	0.19	<0.01	44.9	

Year	Location	n	Concentration (µg/L)			Reference
			Median	Minimum	Maximum	
2006		330	0.18	<0.01	30.4	
2007		321	0.16	<0.01	108	
2005	Saskatchewan	237	0.45	0.01	11	Saskatchewan 2011
2006		209	0.2	0.001	89	
2007		222	0.20	0.001	17	
2008		267	0.30	0.01	31	
2009		276	0.20	0.00021	68.8	
2010		366	0.40	0.01	186	
2009	Charlottetown		Typical, <0.2			Charlottetown 2009
2005	Manitoba	18	-	<0.2	0.3	Manitoba 2011
2006		11	-	<0.2	0.3	
2007		10	-	<0.2	0.6	

- not reported
[a] Samples collected during summer.
[b] 30 of 65 samples were below the method detection limit (MDL): 0.5 µg/L; LOD/2 was used to impute values for these samples.
[c] Samples collected during winter.
[d] 18 of 31 samples were below the MDL: 0.5 µg/L; LOD/2 was used to impute values for these samples.

The median concentrations of lead measured in Ontario and Saskatchewan distribution systems ranged from 0.16 to 0.32 µg/L and from 0.2 to 0.45 µg/L, respectively. In Saskatchewan between 2005 and 2010, 1577 measurements of lead were reported, and concentrations ranged from 0.0002 to 186 µg/L; of these, 32 measurements (2%) exceeded 10 µg/L. In Ontario between the years 2000 and 2007, lead concentrations ranged from below 0.01 to 359 µg/L; three samples were taken from the same location as the maximum (359 µg/L) 2 weeks later and were significantly lower (1.59, 1.62, and 1.68 µg/L). Overall, only 13 of 3038 measurements (0.4%) of lead in drinking water distribution systems in Ontario between 2000 and 2007 resulted in lead concentrations greater than 10 µg/L.

Lead concentrations in over 10 000 samples collected from 2005 to 2010 from private drinking water wells in Prince Edward Island, were provided to Health Canada and are presented in Table 8 (PEI 2011). Approximately 90% of the samples had measured lead concentrations below the detection limit of 2 µg/L, and the average concentration for this period was 9 µg/L with levels ranging from < 2 to 335 µg/L.

Table 8. Concentrations of lead (μg/L) in private wells used for drinking water in Prince Edward Island

Year	n	% non-detectable	Average concentration (μg/L)	Maximum concentration (μg/L)
2005[a]	1077	84	0.9	68.0
2006[a]	2086	87	0.8	99
2007[a]	2491	82	1.6	335
2008[b]	2083	91	1	35.4
2009[c]	1760	98	0.2	64
2010[c]	526	99	0.02	5.0

Source: PEI 2011.
[a] Prince Edward Island data represent unfiltered samples.
[b] Data represent both filtered and unfiltered samples.
[c] Data represent filtered samples.

Concentrations of lead in residential tap water samples were identified for a number of municipalities and for the Provinces of Saskatchewan, Ontario, Quebec, and Newfoundland and Labrador (Table 9). Regulatory sampling for these provinces includes flushing before collecting the water sample for lead analysis. In the municipalities of Edmonton, Alberta, and Winnipeg, Manitoba, the median concentrations of lead reported were < 0.5 and < 1 μg/L, respectively. In Saskatchewan, of the 176 measurements reported, the median lead concentration was 6.7 μg/L, and concentration ranged from < 0.1 to 60 μg/L. Quebec supplied information on the concentration of lead in tap water covering the years 2005 through 2010; during this time frame, the median lead concentration ranged from 2 to 5 μg/L (range 0.06–530 μg/L) in more than 13 000 samples (Quebec 2011). Between 2005 and 2010, 27 of 5331 tap water samples were reported to have lead concentrations in excess of 10 μg/L in residences in Newfoundland (Newfoundland and Labrador 2011).

In a corrosion study, Capital Health and EPCOR in Edmonton, Alberta, reported that 11 of 35 homes receiving water through a lead service line and sampled between June and November 2007 had lead concentrations exceeding 15 μg/L in first-draw stagnant water (water that was present in the pipes for at least 6 hours), and 60% of those 11 had lead concentrations that remained above 15 μg/L after 5 minutes of flushing (EPCOR 2008).

In June 2007, Ontario changed the regulatory requirements for sampling of lead. Ontario Regulation 170/03 (Ontario 2010) requires that stagnant samples (after 30 minutes of stagnation) be taken from homes known or suspected to have lead service lines. In 2007–2008, the Province of Ontario conducted a Community Lead Testing Program; lead levels in more than 37 000 samples were collected in two sampling campaigns (OMOE 2009). These results indicated that 2.3% of the measurements exceeded the OMOE standard and Health Canada's MAC during the winter and 3.1% exceeded this level during the summer. The next priority is to test those homes known or suspected to contain lead solder. Therefore, these measurements represent exposures for the subpopulation with the highest probability of lead exposure via drinking water. For the eight Ontario cities for which lead concentrations in residential plumbing were identified in 2009, the concentrations were reported to range from < 0.02 to 1320 μg/L

(Table 9). Of the 3159 samples, 175 were reported to exceed the Ontario Ministry of the Environment (OMOE) guideline value of 10 μg/dL.

A study covering four boroughs of Montréal, Quebec, conducted by INSPQ, measured lead in water samples in 313 homes during the fall and winter of 2009–2010. Based on the average of five 1 L kitchen tap water samples collected per household (1 L collected after a 5 minute flush and four 1 L samples collected sequentially following 30 minutes of stagnation), the geometric mean lead concentration was determined to be 1.60 μg/L, with a range of 0.06–27.98 μg/L (INSPQ 2011). A comparison between Montréal homes built before 1970 with and without lead service lines was published by Beausoleil and Brodeur (2007). Of the 130 homes sampled in that study in the summer of 2006, 111 homes with lead service lines and 19 without were sampled. Data from two sampling protocols were reported; the first (composite sample) was a composite of stagnant and flushed water collected following 30 minutes of stagnation (average of first litre, second litre and a third one litre after 1 minute of flushing), and the second (flushed sample) was 1 L collected after a further 4 minutes of flushing (corresponding to 5 minutes of total flushing). The results indicated that the average lead concentration in homes with lead service lines (composite: 20 μg/L, flushed: 11 μg/L) was substantially higher than those without lead service lines (composite: 1 μg/L, flushed: 0.7 μg/L). In 2007, Deshommes et al. (2010) measured lead concentrations in 45 Montréal homes with lead service lines. The study reported both dissolved and particulate lead concentrations; dissolved lead concentrations (maximum 114 μg/L) were significantly higher than particulate lead concentrations (maximum 12 μg/L) (Table 9).

Lead was measured in household drinking water collected from approximately 20 different households in each of 21 B.C. First Nations communities from 2008 to 2009 as part of the FNFNES. The maximum concentration measured was 20.4 μg/L; a total of 3 (first draw samples) of 568 samples exceeded the drinking water guideline of 10 μg/L (Chan et al. 2011).

Stagnation time has been identified as one of the most important physical factors in the consideration of a monitoring program (AWWARF 2004; Health Canada 2009a). Concentrations of lead can increase significantly following a few hours of water stagnation in the distribution system (Lytle and Schock 2000). Schock et al. (1996) concluded that lead concentrations increase exponentially upon stagnation, but ultimately approach a fairly constant value after overnight stagnation. As such, sampling with little or no stagnation (i.e., flushed samples) may underestimate lead concentrations in the drinking water (Schock and Lemieux 2010).

Table 9. Concentrations of lead (μg/L) in residential tap water in Canada

Sample location	Year	n	% > 10 μg/L	Concentration (μg/L)			Reference
				Median	Min[a]	Max[a]	
British Columbia, First Nations reserves	2008-2009	568	0.5	-	-	20.4	Chan et al. 2011
Edmonton[b]	2009	-	-	<0.5	-	-	EPCOR 2009
Saskatoon[b]	2009	8	0	<2	-	-	Saskatoon 2009
Saskatchewan[b]	2009	176	-	6.7	<0.1	60	Saskatchewan 2011
Winnipeg[b]	2009	-	-	<1	<1	<1	Winnipeg 2009

Sample location	Year	n	% > 10 µg/L	Concentration (µg/L)			Reference
				Median	Min[a]	Max[a]	
Ontario[c]	2007/8[j]	37 517	2.3	Average: 2.0	-	-	OMOE 2009
	2008[k]	37 895	3.1	Average: 1.9	-	491	
Hamilton[c]	2009	634	8	-	<1	97	Hamilton 2010
(North) Hamilton[c]	2008	-	-	-	<0.5[n]	28[n]	Richardson et al. 2011
Waterloo[c]	2009	121	0	-	<1	4	Waterloo 2009
Barrie[c]	2009	512	0.2	-	0.11	23.9	Barrie 2010
Kingston[c]	2009	198	8	-	<0.02	48.1	Kingston 2009
Ottawa[c]	2009	264	4	-	<0.5	38	Ottawa 2011
St. Catharines[c]	2009	2	0	-	<0.02	0.03	St. Catharines 2009
Sudbury[c]	2009	790	0.5	-	1	17	Sudbury 2009
Thunder Bay[b]	2009	454	21	-	<1	1320	Thunder Bay 2009
Montréal[d, e]	2006	111	53	Average: 11	3	33	Beausoleil and Brodeur 2007
Montréal[e, f]	2006	19	-	Average: 0.7	1	2.7	
Montréal[d, g]	2006	111	-	Average: 20	3	95[m]	
Montréal[e, f]	2006	19	-	Average: 1	0.3	7[m]	
Montréal[h]	2007	135	-	14, dissolved Pb	2.1	114	Deshommes et al. 2010
	2007	135	-	0.39, particulate Pb	<0.02	12	
Montréal[l] (4 selected boroughs)	2009/10	313	1.6 (5 homes)	2.08	0.06	27.98	INSPQ 2011
Province of Quebec	2005	1009	0.7	2	1	20	Quebec 2011
	2006	2220	0.3	3	1	90	
	2007	2573	0.8	5	1	370	
	2008	2649	0.4	3	0.5	45	
	2009	2730	0.5	2	0.2	530	
	2010	2678	0.6	2	0.06	154	
Newfoundland and Labrador[i]	2005	839	0.4	<1.0	<1.0	46.0	Newfoundland and Labrador 2011
	2006	1009	0.6	<1.0	<1.0	45.0	
	2007	817	0.6	<1.0	<1.0	43.0	
	2008	1148	0.6	0.20	<1.0	49.0	
	2009	1029	0.4	<1.0	<1.0	54.0	
	2010	489	0.2	<1.0	<1.0	52.0	
St. John's	2010	194	-	0.51[e]	0.11[e]	19.1[e]	Bell et al. 2011
	2010	194	-	0.84[c]	0.15[c]	58.2[c]	

- not reported
[a] Min: minimum; Max: maximum.
[b] No flushed sample protocol.
[c] Water collected after 30 minutes of stagnation.
[d] Homes with lead service lines.
[e] Flushed sample (5 minute).
[f] Homes without lead service lines.
[g] Composite sample.
[h] Water drawn upon entering home with no specified or known stagnation period.
[i] Values below the method detection limit (MDL) are reported as 0.
[j] Sampling 15 December 2007 to 15 April 2008 (cold water, winter sampling).
[k] Sampling 15 June to 15 October 2008 (warm water, summer sampling).
[l] Based on average of five 1L kitchen tap water samples collected per household. 1 L collected after 5 minute flush, four 1L sequential samples collected following 30 minutes of stagnation. Sampled September 2009 to March 2010 (fall sampling and winter (cold water) sampling). Approximately 56% of these households likely had lead service lines. Note: The column reporting % > 10 ug/L includes only samples collected after 5 minute flush.
[m] Average of maximum values
[n] From homes built prior to 1950

Although average lead concentrations in Canadian drinking water are considered to be low (i.e., below 10 µg/L), drinking water is considered to be an important source of exposure to lead for Canadians when lead service lines or other lead-bearing materials are present in the distribution and plumbing systems. As water lead concentrations increase, water becomes an increasingly important source of exposure for children (Miranda et al. 2007a; Edwards et al. 2009; Renner 2009; INSPQ 2011).

In the INSPQ 2011 study, a multivariate analysis was completed that controlled for confounding factors (e.g., age, sex, visible minority status, total water consumption by body weight) and exposure variables (e.g., lead concentration in floor and window dust and wall paint). This multivariate analysis demonstrated that children who consumed kitchen tap water containing higher lead concentrations (> 3.274 µg/L) had mean BLLs significantly higher than those of children who consumed kitchen tap water with lower lead concentrations (≤ 0.748 µg/L). The presence of lead service lines also resulted in significantly higher blood lead levels in children. The study authors determined, based on a multiple linear regression analysis controlling for confounding factors and exposure variables, that a 10-fold increase in the concentration of lead in water would result in a 23% increase in children's BLLs (INSPQ 2011).

The relationship between the age of the house and water lead concentrations was examined in studies conducted in St. John's, Newfoundland and Labrador (Bell et al. 2011), Montréal, Quebec (INSPQ 2011), and Hamilton, Ontario (Richardson et al. 2011). In the St. John's study, average lead concentrations in flushed kitchen tap water were slightly higher in pre-1970 housing compared with post-1980 housing, but the difference was not statistically significant ($p = 0.234$) (Bell et al. 2011). However, concentrations of lead in kitchen tap water samples from the INSPQ study were significantly higher in homes built before 1920 (geometric mean concentration 2.05 µg/L) than in those built after 1975 (geometric mean concentration 0.55 µg/L, $p < 0.005$) (St-Laurent et al. 2012). In the North Hamilton study, houses constructed prior to 1920 and between 1920 and 1944 were significant predictors of kitchen water lead concentration ($p \leq 0.001$), accounting for approximately 13% of the variation in tap water lead concentrations (Richardson et al. 2011).

Exposure to Lead Levels in the Canadian Environmental Media and Food

Recent concentrations of lead in Canada were identified for all environmental media including indoor and outdoor air, soil, house dust, food, and drinking water.

Many factors, including the concentration of lead in environmental media and food, contribute to total lead exposure. There has been extensive research investigating the primary sources of exposure to lead over time. However, many existing exposure studies were conducted at a time when lead concentrations in both environmental media and people were much higher. As such, it is unclear whether the results of those studies are still relevant today. Many existing studies did not consider all sources of exposure, particularly dietary exposure. Many studies that identified dust and paint as major sources of exposure for children were conducted in areas that had low lead concentrations in water, so the importance of water as a source may have been underestimated (Edwards et al. 2009). In children, there has historically been a strong association between BLLs and both soil lead concentrations (Mielke et al. 1997, 2007; Mielke 1999) and pica behaviour (LaGoy 1987; Mielke et al. 1989). Lead contamination of indoor settled dust can arise from outdoor sources, such as lead contaminated soil (Hertzman et al. 1990; Adgate et al. 1998). Houses in proximity to point sources of lead, such as smelters or contaminated sites, can display elevated concentrations of lead in household dust (Hertzman et al. 1990; von Lindern et al. 2003; Spalinger et al. 2007). Dust contaminated with lead can be generated during renovations in which lead-based paint is removed (Farfel and Chisolm 1990; HUD 2001); hence, renovation activities can directly affect BLLs in children (U.S. CDC 2009a). Removal or remediation of the source of lead contamination has been demonstrated to result in reduced indoor dust lead concentrations, which directly reduced BLLs in resident children (Rhoads et al. 1999; Hilts 2003; Lanphear et al. 2003; B.C. MOE 2009).

Three recent Canadian studies, conducted from 2008 to 2010 in Montréal, Quebec (INSPQ 2011), St. John's, Newfoundland and Labrador (Bell et al. 2011), and Hamilton, Ontario (Richardson et al. 2011), examined relationships between children's BLLs and specific sources of exposure. In the INSPQ (2011) study, the authors found that overall, lead concentrations were low in tap water, household dust, and paint. Lead concentrations were highest in older homes and there was a clear association between year of construction and concentrations of lead in water, paint, and dust (INSPQ 2011; St-Laurent et al. 2012). Twenty-seven percent of residences sampled (primarily older residences) had high lead concentrations in paint chips (up to 260 000 mg/kg), exceeding the 5000 mg/kg criterion, whereas 31% of residences sampled had high lead concentrations in paint on walls and various surfaces, exceeding the 1 mg/cm^2 criterion. Although lead concentrations in water, household dust, and paint were generally low, the authors found that both residential tap water and window sill dust (winter samples) had a statistically significant impact on the BLLs of children participating in the study. Drinking water was the most constant and significant source associated with higher BLLs in children. Lead service lines were considered to be the primary source of tap water lead contribution. As the concentration of lead in water, paint, and dust increased, so did BLLs in children. The contribution of lead in household dust and paint to the BLLs of the children in the study was smaller than that from water, but within the same order of magnitude (INSPQ 2011).

The St. John's study found significant correlations between children's BLLs and lead concentrations in indoor dust (floor, window sill and window trough), flushed kitchen tap water, and residential soil, specifically from play areas (Bell et al. 2011). Concentrations of lead in water were generally low and were highest in older homes, although this difference was not statistically significant. The highest environmental concentrations of lead were associated with paint chips from inside or outside the house in which the child lived, and concentrations were significantly higher in older homes, pre-1970. However, there was no correlation between BLLs and lead concentrations in paint chipping from indoor or outdoor surfaces or dripline soil. Lead concentrations were quantified in above-ground and below-ground garden produce from 34 participating households; however, due to insufficient data, the authors

were unable to test for correlations with children's BLLs. Further analysis of the study results to determine which environmental factors best explain the BLLs is underway by the study team (Bell et al. 2011).

In the (North) Hamilton, Ontario, study, environmental sources of lead exposure, including tap water, household dust, and yard soil were significant predictors for children's BLLs based on univariate regression analysis (Richardson et al. 2011). When considering all three media and controlling for modifiers in a multivariate analysis, tap water was the only significant predicator of children's BLLs. Water lead concentrations were generally below the MAC of 10 µg/L. Yard soil appeared to be a strong contributor to household dust lead leads, but did not appear to be a strong contributor to BLLs (Richardson et al. 2011). Neighbourhoods with higher median living room dust levels corresponded to areas with higher median yard soil lead concentrations, which were also the areas with very old housing stock. The authors investigated the contribution of a given source to BLL by doubling the lead concentration in one medium and keeping the concentration of lead in other media constant. Based on this modelling exercise, a doubling of the tap water lead concentration resulted in a 4 to 15% increase in children's BLL. A doubling of lead concentrations in household dust and yard soil resulted in an 8 to 22% and 0 to 12% increase in children's BLL, respectively. The study authors were unable to determine one sole factor that was most responsible for determining a child's BLL (Richardson et al. 2011).

The results of these Canadian studies support conclusions from earlier work, which identified drinking water, household dust, lead-based paint, and residential soil as important contributors to lead exposure for children. As concentrations of lead in water, dust, paint or soil increase, these media become increasingly influential contributors to BLLs. However, in all of these recent Canadian studies, there was no single predominant source of lead exposure for Canadian children. In two Canadian studies, exposure sources such as water, soil, household dust, and lead-based paint accounted for a moderate proportion (≤ 22%) of the total variability in children's BLLs based on modelling (INSPQ 2011; Richardson et al. 2011). It should be noted that these studies did not examine lead exposure from food or consumer products, which are considered to be meaningful contributors to lead exposure for the Canadian population.

Overall, food, water, and ingestion of non-food items contaminated with lead including dust, lead-based paint, soil, and products, are all considered to be important sources of exposure. Other risk factors, including age, sex, age of house, and socioeconomic status, are also important contributors to the variability in BLLs.

Lead Concentrations in the General Population

Toxicokinetics

Gastrointestinal absorption of lead depends on physiological and physicochemical properties, such as nutritional status (fasting, calcium, and iron status), age, particle size, and solubility (ATSDR 2007; EFSA 2010). Absorption of lead ingested with food is 3–10% in adults and increases up to 40–50% in children (Alexander et al. 1974; Ziegler et al. 1978). Fasting significantly increases the absorption of water-soluble lead (ATSDR 2007). Nutritional iron and calcium deficiencies in children both appear to increase lead absorption (EFSA 2010). Respiratory absorption of up to 95% of particles smaller than 1 µm has been demonstrated (Hursh et al. 1969, as cited in EFSA 2010), while less than 1% of inorganic lead is taken up into the systemic compartment after dermal exposure (ATSDR 2007; EFSA 2010).

Once lead has entered the body, its kinetics are largely governed by calcium dynamics, as lead mimics the behaviour of calcium. Consequently, any inorganic lead present in the body is distributed essentially in the same manner regardless of the route of exposure (Chamberlain et al. 1978, 1979; Kehoe 1987). Lead circulates in the bloodstream and either accumulates in bone or is excreted from the body. Under steady-state conditions, 96 to 99% of the lead is bound to proteins in red blood cells (Schutz et al. 1996; Bergdahl et al. 1997, 1998, 1999; Hernández-Avila et al. 1998; Manton et al. 2001; Smith et al. 2002; ATSDR 2007) and thus is not available to cross into other tissues or organ systems (Goyer 1990). Bones act as a reservoir for lead and accumulate up to 90% and 70% of the absorbed lead in adults and children, respectively (Barry 1975). Circulating blood lead represents less than 1% of the total body burden, while soft tissues represent about 8% (EFSA 2010). The half-life for lead in blood and soft tissue is approximately 30 days, and in bone is approximately 10 to 30 years (Rabinowitz 1991; EFSA 2010). Owing to the profound difference in half-lives, there is a continual exchange of lead between the bones and the blood and soft tissue so that the concentration in blood and soft tissue remains relatively constant. More importantly, lead in bone can, under certain conditions, be mobilized at an increased rate and released back into the systemic circulation. Pregnancy, lactation, menopause, andropause, extended bed rest, hyperparathyroidism, and osteoporosis are all conditions that result in increased blood lead concentrations from bone stores (Silbergeld et al. 1988; Franklin et al. 1997; Gulson et al. 1997, 1999a, 2003). Lead released from maternal bone can serve as a source of exposure of the fetus when calcium is mobilized and used for production of the fetal skeleton (Silbergeld et al. 1988; Franklin et al. 1997; Gulson et al. 1997, 1999a, 2003).

Multiple conditions can result in the remobilization of lead stored in bone and, hence, significantly increase blood lead levels (BLLs), even with the declining environmental exposures evident in North America during the past few decades (Korrick et al. 2002).

Regardless of the route of exposure (oral, inhalation, dermal), the major excretory pathways for lead in the human body are urinary and intestinal (faecal). These two pathways account for up to 75% and 25% of the total excretion of absorbed lead, respectively (Klaassen 2008). Sweat, saliva, hair, nails, and human milk have also been identified as minor routes of excretion (Hursh and Suomela 1968; Hursh et al. 1969; Griffin et al. 1975; Rabinowitz et al. 1976; Chamberlain et al. 1978, 1979; Kehoe 1987; Stauber et al. 1994; ATSDR 2007).

Biomarkers of Exposure

The concentration of lead in whole blood is the most widely used biomarker of exposure both for general population surveillance and as the principal exposure metric in epidemiological studies investigating associations between lead exposure and health outcomes. BLLs reflect a combination of recent exposure and, to some extent, chronic exposure due to the transfer of lead between bone and blood (EFSA 2010). BLLs are strongly influenced by recent exposures over the previous 30 days based on the half-life of lead in blood.

In contrast, bone lead, because of its extremely long half-life, is a biomarker of chronic exposure or total body burden; under normal physiological conditions, bone can be considered a comparatively stable compartment where the vast majority of the lead in the body is stored. In practice, however, bone lead is not the preferred biomarker of exposure to lead owing to the limited availability of x-ray fluorometers for non-invasive bone assessment.

Lead can also be measured in other biological matrices, such as breast milk, cord blood, urine, hair, and teeth. Most epidemiological and experimental studies preferably use blood and bone measurements making the interpretation of data using other biological matrices more difficult. Hence, the present assessment focuses on data measuring lead in bone and blood. Hanning et al. (2003) examined maternal, cord, and infant BLLs, and lead in breast milk. Maternal and cord BLLs were significantly correlated, breast milk lead concentration and maternal BLLs were significantly correlated, and infant BLL was correlated with matched cord BLL. Thus, lead concentration in breast milk and cord blood are good biomarkers of maternal and infant exposure to lead. Maternal bone may account for approximately 80% of the lead in cord blood (Gulson et al. 1997, 1999b, 2003). Lead in breast milk and maternal BLLs are significantly correlated with infant BLLs (Ettinger et al. 2004; Koyashiki et al. 2010). In one study, maternal BLL and breast milk accounted for 30% and 12% of the variability in infant BLLs, respectively (Ettinger et al. 2004). Ettinger et al. (2004) found that although concentrations of lead in breast milk were low, they have a strong influence on infant blood lead levels even when considering the influence of maternal blood lead (Ettinger et al. 2004; Koyashiki et al. 2010). Urinary lead is an indicator of recent exposure to lead, and represents the amount of lead being eliminated but, in general, has not been strongly correlated with observed health effects. Accordingly, urinary lead is not a preferred biomarker to assess the effects of lead in humans. The measurement of lead in hair has potential for external contamination and thus is not considered to be a useful biomarker of exposure.

Lead in Human Milk

Breast milk not only is a biomarker of exposure for lactating women but also is a source of exposure for breast-fed infants. Maternal bone and diet are the dominant sources of lead in breast milk (Gulson et al. 1998). In one Canadian study, concentrations of lead in breast milk were significantly less than lead concentrations in formula and evaporated milk; however, BLLs were significantly higher in infants receiving breast milk and formula than in those receiving evaporated milk (Hanning et al. 2003). A longer duration of breast-feeding is associated with higher infant blood lead concentrations (Lozoff et al. 2009).

In a 1981 survey concentrations of chemical residues, including lead, were measured in the milk of 210 mothers across Canada. Lead concentrations ranged from < 0.025 to 15.8 µg/L, with a geometric mean concentration of 0.566 µg/L (Dabeka et al. 1986). Lead concentrations in human milk from 25 Cree women averaged 2.08 ± 1.67 µg/L (mean ± standard deviation) with a range of 0.41–8.33 µg/L (Hanning et al. 2003). Based on an analysis conducted by Koyashiki et al. (2010) of 11 international studies, the maternal milk:blood ratio for lead ranges between 0.01 and 0.48.

Although there are no recent Canadian data on lead concentrations in human milk, lead will be measured in breast milk in Canadian women as part of the Maternal–Infant Research on Environmental Contaminants Study (MIREC). MIREC is a national 5-year study recruiting approximately 2000 women from 10 sites across Canada and is expected to be completed in 2012. In addition to breast milk, MIREC

will measure a variety of contaminants in maternal blood, urine, and hair as well as cord blood and meconium[4].

Lead in Blood

Blood lead levels, or BLLs, represent aggregate exposure to lead from all routes and all sources, including environmental media, products, and lead remobilized from bone. As there are sufficient BLL data to quantify lead exposure in the Canadian population, estimates of daily intake of lead were not derived.

In August 2010, Health Canada and Statistics Canada released national BLL data collected as part of the Canadian Health Measures Survey (CHMS). Blood lead was measured in 5600 Canadians aged 6 to 79 years at 15 sites across the country from March 2007 to February 2009. The sample represented 96.3% of the population; full-time members of the Canadian Forces and residents of Crown lands, Indian reserves, institutions and certain remote regions were excluded. Lead was detected in blood in almost 100% of the population with a geometric mean BLL of 1.34 μg/dL. BLLs in Canadians have declined by over 70% since 1978–1979, when the geometric mean BLL was approximately 4.79 μg/dL among people aged 6 to 79 years (Bushnik et al. 2010). This decline is attributed to the successful phase-out of lead in gasoline, lead-based paints and lead solder in food cans, in addition to other government regulations and industry action over this time period.

BLLs in Canadians exhibit characteristic age trends. From birth up until 6 months of age, an infant's BLL will reflect that of its mother (Schell et al. 2003). The transfer of maternal lead, either through the placenta or later through breast milk, is the dominant source of an infant's total lead body burden (Manton et al. 2000). As children become more active and mobile at around 6 months of age, environmental lead exposures gradually increase. Settled dust and soil start to represent an increasing portion of overall exposure to lead through hand-to-mouth behaviour and the mouthing of non-food objects that may contain lead, which is often exacerbated during teething (Manton et al. 2000; Tulve et al. 2002; U.S. CDC 2009a). Studies of lead-exposed children have confirmed an increase in BLLs beginning in late infancy, with peak childhood BLLs being reached between 1 and 3 years of age (Baghurst et al. 1987; Dietrich et al. 1993, 2001; Canfield et al. 2003a; U.S. CDC 2009b; Bell et al. 2011; INSPQ 2011; Richardson et al. 2011; Zahran et al. 2011). In a study conducted in St. John's, Newfoundland and Labrador, children under 3 years of age had significantly higher geometric mean BLLs than did children older than 3 years (Bell et al. 2011). In studies conducted in Montréal, Quebec and Hamilton, Ontario children aged 24 to 35 months and 18 to 36 months, respectively, had higher geometric mean BLLs than did younger and older children; however, BLLs were not statistically different (INSPQ 2011; Richardson et al. 2011). BLLs tend to decline slightly during childhood and adolescence and then begin to rise again with age in adults. Seniors typically have the highest BLLs which result from exposure to higher environmental lead concentrations in the past and the remobilization of lead that has been accumulating in bone over time into the blood stream.

[4] Meconium represents the earliest faeces, i.e., material ingested during gestation *in utero*, and allows evaluation of *in utero* exposure via the oral route.

According to the CHMS data, BLLs in Canadians in the youngest age group measured, 6–11 years are 0.90 μg/dL, decrease slightly from 12 to 19 years (0.80 μg/dL), and then increase with age to 1.12, 1.60, and 2.08 μg/dL for the age groups of 20–39, 40–59, and 60–79 years, respectively (Health Canada 2010e). Males have significantly higher blood lead concentrations than do females in all age groups except 6–11 years (Bushnik et al. 2010).

Fewer than 1% of Canadians aged 6 to 79 years have BLLs at or above 10 μg/dL. Approximately two percent have BLLs from 5 to < 10 μg/dL, 23% have concentrations between 2 to < 5 μg/dL, and 74% of the population have BLLs below 2 μg/dL. At least 95% of people younger than 20 years of age have BLLs below 2 μg/dL, but by ages 60–79, 53% have BLLs exceeding 2 μg/dL (Bushnik et al. 2010).

A comparison of historic and recent data on human BLLs between Canada and the United States shows that BLLs and trends in these two North American populations are comparable. Recent analyses of 2007–2008 National Health and Nutrition Examination Survey (NHANES) data for the United States (U.S. CDC 2011) found similar BLLs as those found in Canada. The U.S. geometric mean BLLs for the age groups 6–11 and 12–19 years were 0.988 and 0.800 μg/dL, respectively, compared with 0.90 and 0.80 μg/dL for the same age groups in Canada (Bushnik et al. 2010; U.S. CDC 2011). The geometric mean and 95th percentile BLLs in the US population over 1 year of age were 1.27 and 3.70 μg/dL respectively, whereas the geometric mean and 95th percentile BLLs for Canadians aged 6–79 were 1.34 and 3.79 μg/dL respectively.

BLLs in Canadian children have continued to decline significantly over the past few decades of monitoring; data collected from 1987 to 1990 indicate that average BLLs in Canadian children at that time were predominantly in the 5 to 7 μg/dL range (CEOH 1994). Currently there are no national Canadian BLL data for children under 6 years of age. Given the similarity of BLLs between Canada and the U.S. for other age groups, NHANES BLL data are a reasonable surrogate for BLLs in Canadian children under 6 years of age. The geometric mean and 95th percentile BLLs for children aged 1 to 5 in the 2007–2008 NHANES were 1.51 and 4.10 μg/dL, respectively (U.S. CDC 2011).

Studies in three Canadian cities measured BLLs in children under 7 years of age between 2008 and 2010. A study was conducted during the fall and winter in four boroughs of Montréal, Quebec (INSPQ 2011). The geometric mean BLL for children aged 1–5 years was 1.35 μg/dL, with concentrations ranging from 0.37 to 19.06 μg/dL; only one child had a BLL exceeding 10 μg/dL. These boroughs were selected based on certain criteria (e.g., older houses, presence of lead service lines) to investigate the impact of residential sources of lead on the BLLs of young children. Data from a second study of children aged 6 months to 6 years living in older homes in St. John's, Newfoundland and Labrador, measured a geometric mean BLL of 1.12 μg/dL, with BLLs ranging from 0.21 to 7.5 μg/dL (Bell et al. 2011). In a study conducted in Hamilton, Ontario, the geometric mean BLL for children aged 0 to 6 years was 2.21 μg/dL, with concentrations ranging from < 1.0 to 19.4 μg/dL; less than 1% of children had BLLs exceeding 10 μg/dL (Richardson et al. 2011). It should be noted that the North Hamilton study quantified lead in capillary blood using graphite furnace atomic absorption spectrophotometry with a relatively high LOD (1.0 μg/dL) which could have resulted in higher lead estimates than in studies where quantification of blood lead was in venous blood using inductively coupled plasma mass spectrometry (ICP-MS) with much lower LODs (0.01-0.1 μg/dL), a more commonly employed technique. BLLs for children living in the region of Nunavik, northern Quebec, reported in Turgeon-O'Brien et al. (2010), have declined significantly over the past decade and are similar to levels in the United States and elsewhere in Canada (see Table 10 for details). The results from these studies further demonstrate that children who participated in these Canadian studies have BLLs similar to those of U.S.

children. Although there are currently no national BLL data for Canadian children under the age of 6 years, BLLs in children aged 3 to 5 years are currently being collected as part of Cycle 2 of the CHMS.

A health survey that serves to complement the CHMS is the First Nations Biomonitoring Initiative. This national survey targets First Nations people over the age of 20 living on reserves (south of 60^{o}N). A pilot project was conducted in 2010–2011, followed by the full survey in 13 additional First Nations communities across Canada in 2011–2012. This survey provided for the first time the opportunity to determine national-level data for BLLs of First Nations peoples living on reserves and serves to elucidate additional risks to vulnerable subsets of the general population. Subsequent to this study, and contrary to the exposure profile for the general Canadian population, it has been hypothesized that a major source of lead exposure is through harvesting of wild game.

Several studies completed under the Northern Contaminants Program have surveyed BLLs in communities north of 60^{o}N (Donaldson et al. 2010). Blood lead was measured in Inuit, Dene/Metis, and non-Aboriginal mothers across several regions of the Canadian Arctic. Baseline studies, completed in the mid- to late- 1990s, found higher geometric mean BLLs in Inuit (1.9-5.6 µg/dL) and Dene/Metis (3.5 µg/dL) mothers than in the non-Aboriginal mothers (1.3 µg/dL). In follow-up studies conducted between 2004 and 2007, geometric mean BLLs had decreased markedly to between 0.69 and 1.6 µg/dL. The proportion of Aboriginal mothers with BLLs above 10 µg/dL also decreased from between 3.2% and 19% in the 1990s to no exceedances of 10 µg/dL in the follow-up studies (Donaldson et al. 2010). BLLs decreases in Nunavik have been attributed to the reduced use of lead shot for hunting of traditional foods (Levesque et al. 2003).

Elevated BLLs that result from local point source contamination of lead in soil have been measured in children living in Canadian communities that adjoin smelters. Geometric mean BLLs in these communities ranged from 2.70 to 5.6 µg/dL (Government of New Brunswick 2005; Trail Health and Environment Committee 2007, 2009; Intrinsik 2010).

Blood lead levels in the general population are presented in Table 10.

Table 10. Blood lead levels (BLLs, µg/dL) from Canada and the United States and in Canadian adults and children

Location	Sampling year	Age	n	BLL (95% CI) (µg/dL)		Reference
				Geometric mean	95th percentile	
National level data						
Canada	1978–79	6–79 years	4142	4.79		Canada Health Survey: Bushnik et al. 2010
Canada	2007–09	6 –79 years	5319	1.34 (1.24–1.44)	3.79 (3.32–4.26)	CHMS: Health Canada 2010e
		6–11 years	910	0.90 (0.81–0.99)	1.95 (1.65–2.26)	
		12–19 years	945	0.80 (0.74–0.85)	1.64 (1.47–1.82)	
		20–39 years	1165	1.12 (1.04–1.21)	3.12 (2.75–3.49)	
		40–59 years	1220	1.60 (1.46–1.75)	3.87 (3.16–4.57)	
		60–79 years	1079	2.08 (1.90–2.29)	5.19 (4.20–6.18)	
United States	2007–08	1+ year	8266	1.27 (1.21–1.34)	3.70 (3.50–3.90)	NHANES: U.S. CDC 2011
		1–5 years	817	1.51 (1.37–1.66)	4.10 (3.40–5.19)	
		6–11 years	1011	0.988 (0.914–1.07)	2.50 (2.10–2.88)	
		12–19 years	1074	0.800 (0.744–0.859)	1.9 (1.70–2.32)	
		20 years and older	5364	1.38 (1.31–1.46)	3.90 (3.68–4.23)	
Cord blood						
Mushkegowuk Territory, Ontario	Not reported	Newborn	79	2.08 ± 1.67[a]	0.30–11.08 (range)	Hanning et al. 2003
Nunavik, Quebec	1993-1998	Newborn	193	3.7[c]	0.8–20.9 (range)	Cord Blood Monitoring Program: Boucher et al. 2012
Children's data						
Mushkegowuk Territory, Ontario	Not reported	4 months	31	1.67 ± 1.04[a]	0.50–5.50 (range)	Hanning et al. 2003
Nunavik, Quebec	2000–02	4–6 years	109	4.1 ± 5.0	1.0-37.1 (range)	Després et al. 2005
Nunavik, Quebec	2005-2007	9–13 years	193	2.0[c]	0.4-12.8 (range)	Boucher et al. 2012
Nunavik, Quebec	2006–08	10 months–4.5 years	129	1.7 (1.4–1.9)	Not reported	Turgeon-O'Brien et al. 2010
Belledune, New Brunswick	2004	3–6 years	10	3.54	12.5 (maximum)	Government of New Brunswick 2005
		7–15 years	13	2.70	19.2 (maximum)	

Location	Sampling year	Age	n	BLL (95% CI) (µg/dL)		Reference
				Geometric mean	95th percentile	
(North) Hamilton, Ontario	2008	0–6 years	643	2.21[b] (2.11–2.32)	<1.0–19.4 (range)	Richardson et al. 2011
		0–18 months	120	2.15[b] (1.95–2.40)		
		18 months–<3 years	154	2.34[b] (2.15–2.57)		
		3 - 6 years	369	2.18[b] (2.07–2.30)		
Trail, British Columbia	2007	6 months–5 years	132	5.1	14.1 (maximum)	Trail Health and Environment Committee 2007, 2009
	2009	6 months–3 years	100	5.6	22.7 (maximum)	
Flin Flon, Manitoba	2009	0–6 years	202	2.75 (2.51–2.95)	Not reported	Intrinsik 2010
Montréal, Quebec (4 selected boroughs)	2009–10	1–5 years	306	1.35 (1.27–1.43)	3.11 range: 0.37–19.06	INSPQ 2011
		1-<2 years	50	1.32	3.94 range: 0.50 – 4.77	
		2-< 3 years	66	1.41	3.52 range: 0.39 – 7.46	
		3-5 years	190	1.34	2.90 range: 0.37 – 19.06	
St. John's, Newfoundland and Labrador	2010	6 months -6 years	257	1.12 (1.04–1.2)	2.71 (2.70–3.38) range: 0.21 – 7.5	Bell et al. 2011
		6 months - < 3 years	105	1.29 (1.14–1.46)	range: 0.21 – 7.5	
		3 – 6 years	144	1.00 (0.91 – 1.01)	2.5 (2.25 – 3.0) range: 0.21 – 4.58	
Adult data						
Mushkegowuk Territory, Ontario	Not reported	Pregnant Women	83	2.29 ± 1.25[a]	0.66–6.04 (range)	Hanning et al. 2003
Québec City	2001	18–65 years	441	2.1 (2.0–2.2)	11.1 (maximum)	INSPQ 2004
Province of Quebec	2003	Adult men	125	3.1[c]	20.9 (maximum)	Abdelouahab et al. 2008
		Adult women	87	1.74[c]	12.5 (maximum)	
Nunavik, Quebec	2004	18–74 years	917	3.9 (3.7–4.1)	49.7 (maximum)	Dewailly et al. 2007; Fontaine et al. 2008

Location	Sampling year	Age	n	BLL (95% CI) (µg/dL)		Reference
				Geometric mean	95th percentile	
British Columbia	2004–05	Men 30–65 years	33	2.5	4.6	Clark et al. 2007
		Women 30–65 years	28	1.7	3.7	
Inuvik, Northwest Territories	2005-06	Dene/ Métis Pregnant women 16-36 years	17	1.3	0.48-3.5 (range)	Donaldson et al. 2010
		Inuvialuit Pregnant women 17-38 years	52	1.3	0.44-5.6 (range)	
		Non-aboriginal Pregnant Women 23-40 years	6	0.69	0.46-1.0 (range)	
Baffin, Nunavut	2005-07	Pregnant women 15-39 years	99	1.4	0.41-7.1 (range)	Donaldson et al. 2010
Nunavik, Quebec	2007	Pregnant women 18–37 years	42	1.6	0.66–7.7 (range)	Donaldson et al. 2010

CI: confidence interval.
[a] Arithmetic mean.
[b] Capillary sampling (versus venous sampling), LOD of 1.0 µg/dL
[c] 50th percentile.

Several factors are known to be associated with increased human exposure to lead and consequential increased BLLs. Maternal bone and blood lead are the major source of exposure for the developing fetus. Cord BLLs are correlated with maternal and infant BLLs. Maternal exposure to lead in occupational settings, such as lead smelting, battery manufacturing, and recycling, can result in elevated BLLs in neonates and infants (ATSDR 2007). Lead concentrations in breast milk are significantly correlated with infant BLLs, however maternal BLLs have a stronger influence on infant BLLs (Ettinger et al. 2004). Seasonal variations in BLLs have been reported in the literature, with higher BLLs observed in warmer periods (e.g., summer) (Yiin et al. 2000; Haley and Talbot 2004). Seasonal variation was also observed in the INSPQ study in which the BLLs of children sampled in the fall (geometric mean concentration 1.50 µg/dL) were significantly higher than those of children sampled in the winter (geometric mean concentration 1.27 µg/dL) (INSPQ 2011).

In the United States, BLLs have been reported to be strongly dependent on time elapsed since immigration of individuals from their country of origin to the U.S., with BLLs being highest in those

who had immigrated most recently (U.S. CDC 2006). This trend was also seen in Canada where individuals born outside Canada had a significantly higher BLL (1.54 μg/dL) than did Canadian-born individuals (1.29 μg/dL) (Bushnik et al. 2010). In the INSPQ study, children with visible minority status had significantly higher BLLs than did children without visible minority status (INSPQ 2011). Infants born in the United States of parents who originated from countries with less stringent regulations controlling the industrial use and release of lead may also be exposed to elevated BLLs derived from the mobilization of maternal lead stores during gestation (Rothenberg et al. 1999a, 1999b; ATSDR 2007). This finding can be attributed to continued use of lead in other countries, such as leaded gasoline and lead solder in canned foods, and less stringent regulatory measures mitigating the impact of industrial pollution.

BLLs among Canadians aged 12 to 79 years, as part of the CHMS, were associated with smoking behaviour and alcohol consumption regardless of age and sex. BLLs are higher in Canadians residing in households with lower household income levels compared with higher income levels (Bushnik et al. 2010). Continued elevated exposure to lead for all age groups often occurs among the economically disadvantaged in the United States and Canada as a result of poor housing and other socioeconomic conditions (ATSDR 2010; Bushnik et al. 2010).

In older communities, several exposure sources and risk factors may converge. Older communities are more likely to have lead service lines, lead-containing fittings, and lead solder in the plumbing systems, lead-contaminated soil from current or historical industry or from fallout from historical use of leaded gasoline on major roads, and lead-based paint in the home. In addition, these older areas may have a higher proportion of new immigrants or families with lower incomes. Older homes may be more likely to undergo renovations. Results from the CHMS confirm that BLLs are significantly higher in individuals living in older homes (greater than 50 years) compared with newer homes (Bushnik et al. 2010). In the INSPQ study, BLLs in children living in residences built before 1920 were significantly higher than those of children living in residences built after 1975, and concentrations of lead in water, floor dust, and paint chips were all correlated with the age of home; levels were higher in homes built before 1950 (INSPQ 2011; Levallois 2011). The objective of the study in St. John's, Newfoundland and Labrador was to investigate whether there is increased lead exposure for children living in older housing stock in St. John's (Bell et al. 2011). Downtown areas of St. John's have high soil lead levels resulting from urbanization and the traditional practices of painted clapboard housing and coal burning. In particular, high soil lead concentrations have been found along the drip line of residences built before 1960 and anywhere on properties that date to 1920 or earlier. Housing age cohort was a significant predictor of BLLs and children aged 6 to 72 months living in residences built before 1946 had significantly higher geometric mean BLLs than did children living in younger residences (Bell et al. 2011). In the North Hamilton, Ontario, study, older housing and proximity to historic and current lead-emitting industries were also good predictors of children's BLLs (Richardson et al. 2011).

Health Effects

Health effects of lead have been extensively studied. The adverse health outcomes of lead exposure have been well documented for a wide variety of tissues, organs, and organ systems. This assessment focuses on chronic health effects in humans where there is evidence to indicate that effects are occurring below the current blood lead intervention level of 10 μg/dL including neurodevelopmental, neurodegenerative, cardiovascular, renal, and reproductive effects. Human epidemiological studies are presented along with relevant *in vivo* animal evidence in support of findings in human studies. Therefore, this document is not a review of all the toxicity data on lead or intended to establish reference values; rather, it is a

presentation of the most sensitive and best characterized health effects. Comprehensive reviews of the health effects database of lead are provided in review documents published by international agencies, including WHO (1995), IARC (2006), the U.S. EPA (2006), ATSDR (2007), NTP (2012). A draft review is also available from the U.S. EPA (2012).

Developmental Neurotoxicity

Epidemiological studies have reported an association, after adjusting for confounders, between early-life lead exposure and adverse developmental effects on a variety of neurological, neurophysiological, cognitive, and behavioural endpoints, including:

- neuromotor function (Dietrich et al. 1993; Wasserman et al. 2000; Ris et al. 2004; Després et al. 2005; Fraser et al. 2006; Boucher et al. 2012);
- academic achievement and reading or math skills (Needleman and Gatsonis 1990; Fergusson et al. 1997; Lanphear et al. 2000; Al-Saleh et al. 2001; Wang et al. 2002; Miranda et al. 2007b; Chandramouli et al. 2009);
- delinquent or antisocial behaviour (Fergusson et al. 1993; Bellinger et al. 1994b; Needleman et al. 1996, 2002; Dietrich et al. 2001);
- attention and executive function (Bellinger et al. 1994a; Canfield et al. 2003b; Chiodo et al. 2004, 2007; Ris et al. 2004; Braun et al. 2006; Nigg et al. 2008, 2010; Wang et al. 2008; Bouchard et al. 2009; Froehlich et al. 2009; Ha et al. 2009; Cho et al. 2010 ; Kim et al. 2010; Nicolescu et al. 2010);
- auditory function (Schwartz and Otto 1991; Dietrich et al. 1992; Osman et al. 1999); and
- visual function (Fox et al. 1997, 2008; Rothenberg et al. 2002b; Laughlin et al. 2008).

In the past decade, many of these effects have been associated with blood lead concentrations less than 10 µg/dL (Osman et al. 1999; Lanphear et al. 2000, 2005; Canfield et al. 2003a, 2003b; Chiodo et al. 2004, 2007; Després et al. 2005; Fraser et al. 2006; Tellez-Rojo et al. 2006; Miranda et al. 2007b; Chandramouli et al. 2009) and several studies have modelled a dose–response relationship that extends down to the lowest blood lead concentrations studied (1–2 µg/dL) (Canfield et al. 2003a; Miranda et al. 2007b; Jusko et al. 2008; Jedrychowski et al. 2009).

BLLs have long been reported to be associated with attention-related behaviours. The consistency of the effects is most evident from recent studies in children 3 to 18 years old, and effects have been reported at BLLs below 5 µg/dL in multiple studies (Chiodo et al. 2004, 2007; Braun et al. 2006; Nigg et al. 2008, 2010; Wang et al. 2008; Ha et al. 2009; Froehlich et al. 2009; Cho et al. 2010; Kim et al. 2010; Nicolescu et al. 2010; Plusquellec et al. 2010). Two studies using the same NHANES data set (1999-2002) reported similar results when comparing attention deficit hyperactivity disorder (ADHD) in children 4-15 (Braun et al. 2006) and 8-15 years of age (Froelich et al. 2009), with a mean BLL around 2 µg/dL. Odd ratios were, respectively, 4.1 (CI 1.2-14) and 2.3 (CI 1.5-3.8) compared with the reference population (mean BLL 0.8 µg/dL). Nigg et al. (2010) found an association between BLL and ADHD even after controlling for covariates including IQ in a group of children 6-17 years of age with a mean BLL of 0.73 µg/dL and a maximum BLL of 2.2 µg/dL. Overall, there are multiple cross-sectional studies in human that consistently report an association between attention related behaviours, and the

evidence is sufficient to support the observation of effects below 5 µg/dL. In June 2012, the NTP concluded that there is sufficient evidence that BLLs < 5 µg/dL are associated with various indices of reduced cognitive function, and increased incidence attention-related behaviours and problem behaviours in children (NTP 2012).

The developmental neurotoxicity endpoint that has been most studied, and for which there is the greatest weight of evidence of a causal relationship, is the adverse consequences of early-life lead exposure on psychometric tests of intelligence (IQ) among school-aged children. This body of research demonstrates an association between chronic lead exposure in early life, as measured by various biomarkers, and decrements in school-aged children's IQ. Although some studies of early-life lead exposure and childhood IQ did not demonstrate statistical significance, the cognitive effects are generally indicative of effects of concern at exposure levels below 10 µg/dL (Ernhart et al. 1987, 1989; Cooney et al. 1989a, 1989b, 1991; Bellinger et al. 1991, 1992; Dietrich et al. 1992; Schnaas et al. 2006). Many of the individual epidemiological studies model a dose-response relationship for developmental neurotoxicity down to the lowest blood lead concentration range measured, 1–2 µg/dL (Schwartz 1994; Lanphear et al. 2000, 2005; Canfield et al. 2003a; Chiodo et al. 2004, 2007; Schnaas et al. 2006; Tellez-Rojo et al. 2006), but not all studies report this pattern of results (e.g., Surkan et al. 2007; Chandramouli et al. 2009). The overall weight of human evidence supporting developmental neurotoxicity following lead exposure, at levels below the current blood lead interventional level, is strong when considered in the context of potential measurement error; the potential for overcontrol of modifying rather than confounding variables; the relative insensitivity of IQ decrement as a measure of brain injury; and the complex interdependence of effects on magnitude, duration, and timing of exposure. There is also evidence indicating persistence, until at least the late teenage years, of the changes in neurological systems associated with childhood blood lead concentrations including effects on memory, learning–IQ, attention, visual construction,[5] and fine-motor coordination (Fergusson et al. 1997; Ris et al. 2004). However, longitudinal studies in adults (not highly exposed during childhood versus highly exposed during childhood with cessation in adulthood) are lacking to confirm irreversibility.

The pooled analysis study by Lanphear et al. (2005) has been established as the critical study for the risk characterization of the effects of lead on children's IQ score. The pooled analysis combines data from seven longitudinal studies. It contains the highest number of subjects (n = 1333), is diverse as it includes studies from the United States, Mexico, Australia, and Yugoslavia, but not Canada, and contains a sufficient number of pre-school and school-aged children with BLLs below 10 µg/dL. The pooled analysis contains a sufficient number of subjects to provide the statistical power needed to best characterize the relationship between BLL and IQ score at low exposure levels.

Lanphear et al. (2005) pooled data from seven longitudinal prospective studies initiated before 1995 and followed subjects from birth or infancy until 5 to 10 years of age. The data retrieved were from Boston, Massachusetts (Bellinger et al. 1992), Cincinnati, Ohio (Dietrich et al. 1993), Cleveland, Ohio (Ernhart et al. 1989), Rochester, New York (Canfield et al. 2003a), Mexico City, Mexico (Schnaas et al. 2000), Port Pirie, Australia (Baghurst et al. 1992), and Kosovo, Yugoslavia (Wasserman et al. 1997). The analysis involved a total of 1333 children with complete data on requisite covariates. A subject's IQ was assessed at primary school age (mean age of assessment 6.9 years, range 4.8–10 years) and five of the

[5] Assessed through Block Design Subtest of the Wechsler Intelligence Scales for Children (WISC-III) and the Rey-Osterrieth Complex Figure – Accuracy Score.

seven studies assessed IQ at 6 or 7 years of age. The assessments were conducted with an age- and language-appropriate version of the Wechsler Intelligence Scales for Children (WISC-R, WISC-III, WPPSI, or WISC-Spanish).

Children's blood lead was sampled by either venous or capillary sampling, depending on the protocols of the individual participating studies. Four blood lead indices were used in the analyses: (1) concurrent, defined as BLLs measured closest to the time of cognitive testing (2) maximum, (3) lifetime average, and (4) early childhood, defined as the 6-month to 2-year mean blood lead. Data on cord blood lead were also available for 696 subjects. The pooled median concurrent, maximum, lifetime average, and early childhood BLLs were 9.7, 18.0, 12.4, and 12.7 μg/dL, respectively. Eighteen percent (n = 244) of subjects had maximum blood lead concentrations of less than 10 μg/dL and 8% (n = 103) of subjects had maximum blood lead concentrations below 7.5 μg/dL.

Data on the following covariates were included in the pooled analysis: maternal IQ, education, marital status, and prenatal alcohol and tobacco use; Home Observation and Measurement of the Environment (HOME) inventory score; and subject sex, birth order, and birth weight. The influence of ethnicity was investigated for the subset of U.S. data. Potentially important covariates that were not included in the pooled analysis included socioeconomic status (SES), nutritional status, and paternal IQ. Lanphear et al. (2005) found an inverse relationship between concurrent BLL and IQ score. IQ point decrements of 3.9, 1.9, and 1.1 were associated with concurrent BLLs of 2.4 to 10 μg/dL, 10 to 20 μg/dL, and 20 to 30 μg/dL, respectively. Lanphear et al. (2005) in a pooled-analysis based on studies conducted in times of higher lead exposure concluded that intellectual deficits are associated with maximum BLLs of less than 7.5 μg/dL in children. However, since BLLs have declined considerably since the Lanphear (2005) study was conducted, there is uncertainty regarding the extrapolation of this dose-response curve to the current Canadian population.

The health effects literature from experimental studies in laboratory animals supports the findings of the observational studies in humans. There is no equivalent to an IQ test in animals, but adverse behavioural outcomes that reflect learning and memory have been demonstrated at the lowest blood lead concentrations studied (approximately 10 μg/dL) in multiple species, including non-human primates (Cory-Slechta and Thompson 1979; Rice 1984, 1985; Rice and Gilbert 1985; Gilbert and Rice 1987; Rice and Karpinski 1988). Studies in laboratory animals indicate that the developmental neurotoxicity of lead is not reversible and persists after exposures cease and blood and brain lead concentrations return to normal (Rice 1984, 1985; Cory-Slechta 1995; Rice and Barone 2000). To date, no exposure threshold for lead-induced behavioural deficits in laboratory animals has been established.

Key studies that have investigated the association between blood lead levels and neurodevelopmental effects in humans and in experimental animals are summarized in Table 11. Considering the technological limitations of the measurement methods, BLLs identified in the table should not be interpreted as clear thresholds of effect. It should also be emphasized that the effects and BLLs reported in this and subsequent tables in this section are based on the study authors' conclusions.

Table 11. Neurodevelopmental effects and associated BLLs (µg/dL)[a]

Study type	Effect	BLL associated with the effect (µg/dL)	Reference
Human studies	Increased ORs for ADHD in children 4–15 years of age	>1.3	Braun et al. 2006
	Significant association with ADHD scores in children 8–11 years of age	1.9	Cho et al. 2010
	Decreased performance on standardized tests of reading and math in grade 4	2	Miranda et al. 2007b
	Increased ORs for ADHD in children 8–15	>2	Froelich et al. 2009
	Non-linear analysis indicates that BLL as low as about 2 µg/dL may be associated with decline in IQ	2.1	Jusko et al. 2008
	Association with ADHD both as a diagnosis and as a symptom dimension	<2.2	Nigg et al. 2010
	Increased inattention and hyperactivity in children 8–10 years of age	≥2.2	Kim et al. 2010
	IQ decrements in 10-year olds	2.5	Schnaas et al. 2006
	Major depressive disorder, panic disorder, and generalized anxiety disorder in young adults (aged 20–39 years)	3	Bouchard et al. 2009
	Increased inattention	≥3	Chiodo et al. 2004; 2007
	Association with ADHD and with mediated hyperactivity	3.47	Nigg et al. 2008
	Positive association with ADHD in children (6–17 years) with increasing BLLs between 0.1 and 10 µg/dL	positive trend with levels <3.5	Ha et al. 2009
	IQ decrements and hyperactivity in children 8 to 11 years of age	3.74	Kim et al. 2010
	No IQ decrements in children aged 6 to 10 years	3–4	Surkan et al. 2007
	No effect on tests of standardized educational outcomes in children aged 7–8 years	2–5	Chandramouli et al. 2009
	Increased ADHD-related behaviour in children 8–12 years of age	3–5	Nicolescu et al. 2010
	Decreased math and reading skills in 6–16-year olds from NHANES III	<5	Lanphear et al. 2000
	Decreased score on Fagan Test of Infant Intelligence in infants 7 months of age	<5 (maternal blood)	Emory et al. 2003
	Mild association with deficit in cognitive function in children 12 to 36 months of age	<5 (cord blood)	Jedrychowski et al. 2009
	Prevalence of ADHD in Chinese children 4–12 years of age	<5	Wang et al. 2008

Study type	Effect	BLL associated with the effect (µg/dL)	Reference
	IQ decrements and lower academic achievement at 4–11 years of age (verbal and non-verbal)	5	Min et al. 2009
	IQ decrements and other neurological impairment in children aged 7–9 years	5.4	Chiodo et al. 2004
	Higher ADHD and inattention scores, greater hyperactivity and poor attention in children 7–9 years of age	5.4	Chiodo et al. 2004, 2007
	IQ decrements in 10-year olds	7	Bellinger et al. 1992
	IQ decrements in 3–5-year olds No threshold for effects	7.4–7.7 3.4–9.7	Canfield et al. 2003a
	IQ decrements in children 5–10 years of age; Pooled-analysis (7 studies)	<7.5 (maximal) 2.4–10 (concurrent)	Lanphear et al. 2005
	IQ decrements in 11–13-year olds	7.9	Tong et al. 1996
	IQ decrements in children aged 6 to 10 years	5–10	Surkan et al. 2007
	Decrements in Bayley Mental Development Index (MDI) in 2-year olds	5–10	Tellez-Rojo et al. 2006
	Decreased performance on tests of standardized educational outcomes in 7–8-year olds	5–10	Chandramouli et al. 2009
Animal studies	Neurobehavioural impairments in rats	10	Cory-Slechta and Thompson 1979
	Neurobehavioural impairments in monkeys	12	Rice 1984, 1985; Rice and Gilbert 1985; Rice and Karpinski 1988

[a] As reported by study authors. Note that for many of the studies, the LOD was approximately 1 µg/dL and the limit of quantification (LOQ) was approximately 3 µg/dL. This limit of analytical capacity causes difficulty in interpreting study results in this low range of exposures.

Neurodegenerative Effects

Neurodegenerative effects (e.g., cognitive decline) have not been as well studied as developmental neurotoxicity. The observational and experimental evidence that exists supports an association between lead exposure and increased rate of neurological decline. Associations between bone lead concentrations and neurodegenerative effects have been more consistently reported than have associations between blood lead and neurodegenerative effects (Shih et al. 2006).

While there is observational and experimental evidence to support an association between lead exposure and increased rate of neurological decline, the weight of evidence is limited for BLLs below 10 µg/dL. Associations between BLLs and age-related cognitive decline have been observed in several studies (Wright et al. 2003; Stewart and Schwartz 2007; Khalil et al. 2009; Weuve et al. 2009). The likelihood of older men scoring less than 24 on the Mini-Mental State Exam (MMSE) was inversely associated with blood lead concentrations among subjects of the U.S. Veterans Affairs Normative Aging Study (NAS) with a mean blood lead concentrations of 5 µg/dL (Wright et al. 2003). These results, however,

are in contrast to findings from other cohort studies examining both men and women with similar levels of exposure (Muldoon et al. 1996; Nordberg et al. 2000; Krieg et al. 2005).

Tibia lead concentration, but not blood lead concentration, has been associated with cognitive decline in a cohort of older women with mean blood lead concentrations of 2.9 µg/dL (Weuve et al. 2009). Tibia lead level, but not blood lead, was similarly associated with lower current cognitive performance and cognitive decline in a longitudinal occupational cohort study (Khalil et al. 2009).

Occupational cohort studies have relatively consistently reported a number of central nervous system and peripheral nervous system effects at blood lead concentrations higher than 20 µg/dL, with few data available at lower blood lead concentrations. Effects reported in occupational cohort studies include abnormal postural sway, abnormal visual-evoked potential and brainstem auditory-evoked potential (BAEP), peripheral sensory nerve impairment, neuromotor impairment, and neurological symptoms (ATSDR 2007).

There are few experimental data on the effects of lead-induced neurodegeneration on animal behaviour. Studies in both rats and non-human primates at blood lead concentrations of about 20 µg/dL demonstrate that older animals are more susceptible to the adverse neurobehavioural effects of lead than are younger animals at comparative doses and durations of exposure (Rice 1990, 1992a, 1992c; Cory-Slechta and Pokora 1991).

Key studies that have investigated the association between neurodegenerative effects and BLLs are presented in Table 12.

Table 12. Neurodegenerative effects and associated BLLs (µg/dL)[a]

Study type	Effect	BLL associated with the effect (µg/dL)	Reference
Human studies	Increased risk for essential tremor	3	Louis et al. 2003
	No association with neurobehavioral test scores in NHANES III	3.3	Krieg et al. 2005
	No association with MMSE score	3.7	Nordberg et al. 2000
	Increased risk of MMSE score < 24	5	Wright et al. 2003
	No association with neuropsychological test scores (urban)	5.4	Muldoon et al. 1996
	Impaired neuropsychological test scores of older women (rural)	7	Muldoon et al. 1996
Animal studies	Neurobehavioural impairments in adult monkeys	11	Rice and Karpinski 1988
	Increased beta-amyloid proteins in older rats exposed only as infants	20	Basha et al. 2005
	Neurobehavioural impairments (increased fixed-interval and variable-interval response rates) in older rats	20	Cory-Slechta et al. 1991
	Neurobehavioural impairments in adult monkeys	25	Rice 1990, 1992b, 1992c
	Increased beta-amyloid proteins and plaques in 23-year-old monkeys exposed only as infants	32–36	Wu et al. 2008

[a]As reported by study authors

Cardiovascular Effects

There are multiple lines of evidence, including human epidemiological studies, *in vivo* animal assays, and *in vitro* experiments, that demonstrate that chronic lead exposure can result in adverse cardiovascular effects in humans by various mechanisms. Lead exposure has been associated with several cardiovascular endpoints, including cardiovascular mortality, stroke mortality, myocardial infarction (MIA) mortality, inotropic and chronotropic cardiotoxicity, and peripheral arterial disease, and there is evidence for several of these effects at blood lead concentrations of less than 10 μg/dL (Lustberg and Silbergeld 2002; Navas-Acien et al. 2004, 2007, 2008; Menke et al. 2006; Schober et al. 2006). The endpoint that has been most studied and for which there is the greatest weight of evidence of a causal relationship is lead-induced increases in blood pressure, particularly systolic blood pressure (SBP), or risk of hypertension.

There have been a large number of epidemiological studies of blood lead and blood pressure or risk of hypertension in adults as well as in pregnant women, and many report a significant, but modest, association (Rothenberg et al. 2002a; Glenn et al. 2003; Nash et al. 2003; Vupputuri et al. 2003; Chen et al. 2006; Yazbeck et al. 2009; Wells et al. 2011). Several meta-analyses have also reported a significantly positive, but small, association (Staessen et al. 1994; Schwartz 1995; Nawrot et al. 2002). The modest effect size and the inconsistency of results can be attributed, in part, to measurement error in both lead exposure and blood pressure. Biological variations such as age, diet, gender, and ethnicity (which are all risk factors of cardiovascular disease) may also explain the inconsistency in the results, since these co-variates were not consistently controlled for in the above-noted studies. In contrast to studies that have relied on blood lead as a measure of exposure, bone lead concentration has more consistently been associated with increased blood pressure or risk of hypertension in the aged (Cheng et al. 2001).

Glenn et al. (2003) reported on the longitudinal association between adult BLLs and annual change in blood pressure in occupationally exposed males. The average blood lead at baseline was 4.6 μg/dL, with a range of 1 to 20 μg/dL. Vupputuri et al. (2003) reported on the cross-sectional relationship between BLLs and SBP among 14 952 adults in the Third National Health and Nutrition Examination Survey (1988–1994) (NHANES III). Blood lead was significantly associated with higher SBP and diastolic blood pressure (DBP) among African-American men and women, but not Caucasians. The mean BLLs were 5.4 and 3.4 μg/dL for African American men and women, respectively. Other studies also observed higher BLLs, higher blood pressures and higher prevalence of hypertension among non-Caucasians compared with Caucasians (Den Hond et al. 2002; Scinicariello et al. 2010). Nash et al. (2003) examined a study group of females aged 40 to 59 from the NHANES III data set for increases in SBP and/or DBP and general risk of hypertension associated with increases in BLLs. An increase from the lowest BLL quartile (mean, 1.0 μg/dL; range 0.5–1.6 μg/dL) to the highest (mean, 6.4 μg/dL; range 4.0–31.1 μg/dL) was associated with a 1.7- and 1.4-mm Hg increase in SBP and DBP, respectively.

Glenn et al. (2006) followed 575 subjects in a lead-exposed occupational cohort in the Republic of Korea between October 1997 and June 2001. Blood pressure changes in relation to tibia lead concentration were evaluated, as well as changes in concurrent BLL. The mean BLL in the cohort was 31.4 μg/dL and the mean tibia lead concentration was 38.4 μg/g tibia bone mineral at first presentation. Change in SBP was statistically associated with concurrent BLL. Cheng et al. (2001) performed a cross-sectional and a longitudinal study on the relationship between tibia lead concentration and SBP in 519 participants of the U.S. Veterans Affairs Normative Aging Study (NAS) from Greater Boston, Massachusetts. They reported a significant positive association with a 0.1 mm Hg increase in SBP per

µg lead/g tibia bone. Gerr et al. (2002) assessed the association between tibia lead concentration and blood pressure in young adults (19-29 years old) who were heavily exposed or not exposed to lead in their childhood. When comparing the four bone lead concentration groups (< 1 µg/g, 1-5 µg/g, 6-10 µg/g and > 10 µg/g), SBP and DBP were respectively 4.3 and 2.8 mm Hg higher ($p < 0.05$) in the highest bone lead concentration group compared with the lowest. The corresponding BLL for the high bone lead group was 3.15 µg/dL. Blood lead was not statistically significantly associated with blood pressure suggesting that the observed increase in blood pressure was related to the long-term effects of past exposure.

Lead exposure has been associated with increased SBP or risk of hypertension among environmental cohorts with average blood lead concentrations as low as 3–5 µg/dL (Vupputuri et al. 2003; Martin et al. 2006). Other studies have disputed the presence of a relationship between low-level lead exposure and hypertension (Staessen et al. 1994; Chu et al. 1999; Nawrot et al. 2002). There have been few analyses on the shape of the blood lead concentration–response relationship for cardiovascular effects; some show evidence of an attenuation of the slope over lower ranges of current blood lead concentrations for SBP and MIA mortality (Schwartz and Stewart 2000; Martin et al. 2006; Menke et al. 2006), but not stroke mortality (Menke et al. 2006).

Both chronic and subchronic oral exposure to lead in laboratory animals results in elevated blood pressure. This effect has been demonstrated in multiple species and has been repeatedly demonstrated in multiple rat strains. Three-month oral lead exposures that produced a mean blood lead concentration of 2.4 µg/dL resulted in approximately a 30% increase in blood pressure in rats (Attri et al. 2003). Similarly, when rats were exposed to varying concentrations of lead in drinking water for 60 days, exposure that led to a BLL of 2.15 µg/dL resulted in a 10% and 11% increase ($p < 0.01$) in SBP and DBP, respectively, compared with controls (Tsao et al. 2000).

As a whole, meta-analyses of the epidemiological findings have identified a persistent trend in the data that supports a relatively mild, but statistically significant association between BLL and SBP in adults (ATSDR 2007; ILZRO and EBRC 2008; EFSA 2010).

Key studies that have investigated the association between BLL and cardiovascular effects are summarized in Table 13.

Table 13. Cardiovascular effects and associated BLLs (µg/dL)[a]

Study type	Effect	BLL associated with the effect (µg/dL)	Reference
Human studies	Increased risk of pregnancy-induced hypertension in EDEN cohort study	2.2	Yazbeck et al. 2009
	Stress-induced increase in total peripheral vascular resistance in children aged 9 years	2.8	Gump et al. 2005
	Increased risk of peripheral arterial disease	2.9	Navas-Acien et al. 2004
	No association with ambulatory SBP	2.9	Staesson et al. 1996
	Increased risk of cardiovascular mortality	3.6	Menke et al. 2006
	Increased SBP among Caucasian males	4.6	Glenn et al. 2003

	Increased SBP among African-Americans in NHANES III	3.4–5.4	Vupputuri et al. 2003
Animal studies	Increased blood pressure in rats	2.4	Attri et al. 2003
	Increased blood pressure in rats	3.2	Ding et al. 1998
	Increased blood pressure in rats	2.15	Tsao et al. 2000
EDEN: Etude des Déterminants pré et post natals du développement et de la santé de l'Enfant [a] As reported by study authors			

Renal Effects

In humans, the general pattern of lead nephrotoxicity indicates severe deficits in function and pathological changes when BLLs > 50 μg/dL, enzymuria and proteinuria becoming evident when BLLs > 30 μg/dL, and reduced glomerular filtration (measured as a decrease in creatinine clearance or increase in serum creatinine) when BLLs < 20 μg/dL (ATSDR 2007). A review of the epidemiological literature conducted by Ekong et al. (2006) concluded that lead contributes to nephrotoxicity at BLL < 5 μg/dL and this is particularly true in susceptible populations, such as those with hypertension, diabetes, and/or chronic kidney disease (CKD).

An inverse association between blood lead and glomerular filtration rate (GFR) has been reported, after adjusting for confounders, in most environmental cohort studies, and this has been observed in cohorts with mean blood lead concentrations as low as 2 μg/dL (Akesson et al. 2005). A 10-fold increase in blood lead is associated with decreases in creatinine clearance ranging from 10 to 30 mL/minute (Staessen et al. 1992; Payton et al. 1994). Increases in serum creatinine of 0.08 mg/dL are associated with a 10-fold increase in blood lead (Kim et al. 1996).

Several significant studies and reports spanning a decade examine the relationship between blood lead and renal function. Longitudinal studies from subsets of the NAS found associations between decreased creatinine clearance and BLL (Payton et al. 1994) and increased serum creatinine (Kim et al. 1996). When the results were stratified by diabetes and hypertension status, significant associations between serum creatinine concentration and lead levels (blood and bone) were found in diabetic and hypertensive groups, which suggest the possibility of interactions between lead exposure, glomerular function, and diabetes or hypertension (Tsaih et al. 2004; ATSDR 2007). As part of the Belgian Cadmibel Study, decreased creatinine clearance was significantly associated with blood lead (Staessen et al. 1992). Mean BLLs were 11.4 and 7.5 μg/dL for males and females, respectively.

Muntner et al. (2003) analyzed serum creatinine concentrations as a function of GFR and BLL as part of NHANES III (1988–1994). The mean BLL was 3.3 μg/dL in the normotensive group and 4.2 μg/dL in the hypertensive group. There was a strong, dose-dependent association between BLLs and elevated serum creatinine and chronic kidney disease (CKD) among persons with hypertension. In a follow-up analysis of the NHANES III and NHANES 1999–2002 data, there was a clear dose–response relationship between the quartiles of BLL and the prevalence of hypertension (Muntner et al. 2005). In an analysis of associations between joint exposure to low-level cadmium and lead from NHANES (1999–2006), a statistically significant trend was found between the quartiles of BLL and blood cadmium and reduced estimated GFR (Navas-Acien et al. 2009). The authors concluded that both cadmium level and lead should be considered as risk factors for CKD. The relationship between BLL and kidney function in adolescents (12–20 years of age) was investigated by Fadrowski et al. (2010)

from NHANES III data. Higher BLLs were associated with lower estimated GFRs, and the median BLL was 1.5 μg/dL.

There are few *in vivo* data on environmentally relevant lead exposures and renal function in animals. A blood lead concentrations of 26 μg/dL in rats produced decreased creatinine clearance and accelerated renal microvascular and renal tubulointerstitial injury (Roncal et al. 2007). A blood lead concentrations of 29 μg/dL in rats produced transient hyperfiltration, a finding consistent with similar data from relatively high-exposure occupational cohort studies. This exposure level also produced mild renal tubular atrophy and interstitial fibrosis (Khalil-Manesh et al. 1993).

Key studies that have investigated the association between BLL and renal effects are summarized in Table 14.

Table 14. Renal effects and associated BLLs (μg/dL)[a]

Study type	Effect	BLL associated with the effect (μg/dL)	Reference
Human studies	Reduced GFR and creatinine clearance, Women's Health in Lund Area Study	2.2	Akesson et al. 2005
	Risk of elevated serum creatinine and chronic kidney disease among hypertensives, NHANES III	2.5–3.8	Muntner et al. 2003
	Reduced creatinine clearance, Belgian Cadmibel Study	7.5	Staessen et al. 1992
	Reduced creatinine clearance, NAS 1988–1991	8.1	Payton et al. 1994
	Increased serum creatinine, NAS 1991–1994	9.9	Kim et al. 1996
Animal studies	Reduced creatinine clearance, microvascular and tubulointerstitial injury in rats	26	Roncal et al. 2007
	Transient hyperfiltration, tubular atrophy, and interstitial fibrosis in rats	29	Khalil-Manesh et al. 1993

[a]As reported by study authors

Reproductive Effects

Key reproductive effects observed in females that have been associated with low-level exposure to lead include delays in sexual maturation, risk of spontaneous abortion, and low birth weight/pre-term birth. Epidemiological evidence in African American and Mexican American girls based on an analysis from NHANES III data, demonstrated an association between delayed puberty in adolescent girls and blood lead concentrations as low as 3 μg/dL (Selevan et al. 2003). These findings have been replicated in two of the three additional studies that have examined this effect (Wu et al. 2003; Denham et al. 2005; Wolff et al. 2008) and are supported by recent and replicated mice *in vivo* studies that reported a 30% delay in onset of puberty at blood lead concentrations of 2–3 μg/dL compared with 0.7 μg/dL (Iavicoli et al. 2004, 2006). These low-dose *in vivo* studies also report a dose–response relationship, with no evidence

of attenuation in the relationship as blood lead concentrations approach 0.7 μg/dL. Delayed onset of puberty and reductions in circulating levels of insulin-like growth factor 1 (IGF1), luteinizing hormone (LH), and estradiol (E2) were associated with female rats exposed to lead during gestation only, with a peak blood lead concentration of about 14 μg/dL (Dearth et al. 2002). Altered levels of progesterone were reported in monkeys with blood lead concentrations of 25–30 μg/dL (Foster et al. 1996).

Older epidemiological studies have reported associations between spontaneous abortion and occupational lead exposure (Hertz-Picciotto 2000). Findings from more recent environmental cohort studies are inconsistent, but methodological issues limit the strength of the conclusions that can be drawn from these studies. One well-designed, but not yet replicated, study from Mexico City (1994–1996) reported a concentration–effect gradient over blood lead concentrations of 3 to 29 μg/dL with an increased odds ratio of spontaneous abortion of 1.8 (CI 1.1–3.1) for each 5-μg/dL increase in blood lead concentration for women ranging from 14 to 43 years of age (Borja-Aburto et al. 1999). In another study of pregnant women from Mexico City, a 0.1% increment in the maternal plasma to blood ratio was associated with a 12% greater incidence of reported history of spontaneous abortion (Lamadrid-Figueroa et al. 2007). However, in a study conducted with 351 women in Iran from 2006 to 2008, mean BLLs did not differ significantly between spontaneous abortion cases and ongoing pregnancies (Vigeh et al. 2010). The mean BLL was 3.8 μg/dL. Overall, there is limited evidence to conclude that there is increased risk of spontaneous abortions with BLLs < 30 μg/dL (U.S. CDC 2010b).

The epidemiological evidence for an association between maternal blood lead and birth weight and pre-term birth is inconsistent, probably owing to differences in study design, sample size, and control for confounders (U.S. CDC 2010b). In a birth cohort study from Mexico City, Gonzalez-Cossio et al. (1997) reported that bone lead burden is inversely related to birth weight. Infants from mothers with tibia lead concentrations in the highest quartile (> 15 μg/g) were, on average, 156 g lighter than infants from mothers in the lowest quartile (< 4.5 μg/g). From the same cohort, Hernández-Avila et al. (2002) reported a positive association between bone lead concentration and shorter birth length. In a separate study (Mexico City 1997–1999), maternal whole-blood lead levels measured during the first and second trimesters yielded an inverse association with length of gestation and increased risk of prematurity (Cantonwine et al. 2010).

In males, most reported effects on the reproductive system have been observed at BLLs > 10 μg/dL and include decreased sperm count, morphological aberrations, and an increased risk of infertility (Alexander et al. 1996; Sallmen et al. 2000; Bonde et al. 2002). However, one study from the Russian Federation reported an association of BLLs as low as 3 μg/dL with decreased growth and differences in pubertal onset in periadolescent boys (Hauser et al. 2008).

In experimental animals, lead adversely affects offspring development at maternal blood lead concentrations that do not produce maternal clinical toxicity. Effects reported range from fetal mortality to reduced birth weight and postnatal growth. *In vivo*, in rats and monkeys, fetal mortality and reduced birth weight and postnatal growth have been associated with maternal blood lead concentrations of 30–50 μg/dL. Maternal blood lead concentrations of 13 μg/dL and higher have resulted in increased fetal resorptions in dams and maternal blood lead concentrations of 10 μg/dL and above have resulted in increased external malformations in pups (Flora and Tandon 1987).

Key studies that have investigated the association between BLL and reproductive effects are summarized in Table 15.

Table 15. Reproductive effects and associated BLLs (µg/dL)[a]

Study type	Effect	BLL associated with the effect (µg/dL)	Reference
Human studies	Delayed puberty in females	3	Selevan et al. 2003
	Delayed puberty in females	3	Wu et al. 2003
	Delayed puberty in males	3	Hauser et al. 2008
	No increased risk of spontaneous abortion	3.8	Vigeh et al. 2010
	Increased risk of spontaneous abortion	5	Borja-Aburto et al. 1999
	Decrease in gestational length and increased risk of pre-term delivery	7.2	Cantonwine et al. 2010
	Increased time to pregnancy	10	Shiau et al. 2004
	Increased relative risk of infertility	10–15	Sallmen et al. 2000
	Decreased sperm concentrations	15–24	Alexander et al. 1996
Animal studies	Delayed onset of puberty in female mice	0.7	Iavicoli et al. 2004
	Altered structure of Sertoli cells and spermatids in male rats	7	Murthy et al. 1995
	Decreased number of spermatozoa in male rats	7	Barratt et al. 1989
	Increased external malformations	10	Flora and Tandon 1987
	Delayed onset of puberty in female mice	8–13.0	Iavicoli et al. 2006
	Delayed onset of puberty and altered hypothalamic–pituitary–gonadal (HPG) axis in female rats	14	Dearth et al. 2002
	Structural damage to seminiferous tubules and reduced prospermatogonia in male rats	14	Corpas et al. 1995

[a]As reported by study authors

Mode of Action

Overall, the mode of action for lead is attributed to its affinity for thiol groups (-SH) and other organic ligands in proteins (Vallee and Ulmer 1972). The molecular characteristics of lead are very similar to those of calcium, which results in the kinetics of lead being greatly influenced by calcium dynamics. This is well illustrated by the observation that gastrointestinal absorption of lead is influenced by dietary calcium intake, such that adults and children with low dietary calcium will absorb more lead (Ziegler et al. 1978; Heard and Chamberlain 1982; Blake and Mann 1983; Mahaffey et al. 1986). Lead can substitute for calcium (and perhaps other essential metals) and this is considered to factor into the mode of action as well (Bressler and Goldstein 1991; Zawia et al. 1998; Hanas et al. 1999; ATSDR 2007; EFSA 2010). Like calcium, lead accumulates in bones. Lead can opportunistically cross cellular membranes through the voltage-regulated Ca^{2+} channel (Calderon-Salinas et al. 1999) or by "piggybacking" on the active transport system such as Ca^{2+}–Mg^{2+}-adenosine triphosphatase (Simons 1988).

Consequently, the health effects associated with lead are considered to occur via common modes of action, such as lead's ability to mimic other biologically essential metals (calcium, iron, and zinc), the

most notable of which is calcium (Kosnett 2006). In the nervous system (central and peripheral), calcium-dependent reactions and calcium homeostasis disruptions are thought to be the most important pathways leading to overt toxicity (EFSA 2010). In the cardiovascular system, the increases in intracellular Ca^{2+} triggered by the presence of lead (along with other effects) cause increases in vascular smooth muscle tone, resulting in increased microcirculation resistance, and thus blood pressure (Piccinini et al. 1977; Kramer et al. 1986; Watts et al. 1995; Hwang et al. 2001). ATSDR (2007) has postulated that the effects of lead, elevation in blood pressure, and decrements in GFR may be related.

In vivo, lead interferes with the activities of key enzymes in the synthesis of heme, a critical component of hemoglobin, and adversely affects erythrocyte morphology and life span (Klaassen 2008; EFSA 2010). It is the inhibition of delta-aminolevulinic acid dehydratase (ALA-D), in addition to other enzymes required for heme synthesis, that results in the increased production of zinc protoporphyrin (detectable in both blood and urine) and ultimately in lower amounts of hemoglobin in red blood cells. This effect of systemic lead exposure is seen clinically as anemia (U.S. EPA 1986b; Goering 1993).

Risk Characterization

There is evidence of health effects associated with blood lead concentrations under 10 μg/dL, down to 1–2 μg/dL, which include neurodevelopmental, neurodegenerative, cardiovascular, renal, and reproductive effects. Of these, the strongest association at the lower levels is for neurodevelopmental effects, specifically the reduction of IQ score and attention-related behaviours. IQ score is the most widely measured endpoint to assess developmental neurotoxicity from exposure to lead. The weight of evidence from observational and *in vivo* experimental animal studies supports the following conclusions on neurotoxicity:

- Developmental neurotoxicity has been associated with the lowest levels of lead exposure reported, both in observational studies and in animal experiments.
- In humans, the developmental neurotoxic effects of lead can persist until the late teen-age years.
- In laboratory animals, the developmental neurotoxic effects of lead can persist after exposures have ceased and blood and brain lead concentrations have returned to normal or control levels.
- Currently, dose-response modelling conducted with data from observational studies does not demonstrate a population threshold for developmental neurotoxicity over the lower ranges of current environmental lead exposures. For some endpoints, such as IQ deficits, the preponderance of evidence indicates that the dose–response relationship is curvilinear, with a steeper slope over the lower ranges of current environmental lead exposures.
- Lead can interact with multiple cell types in the central nervous system, and potential modes of action supported by experimental evidence have been developed to explain the observed developmental neurotoxicity of lead. These modes of action are considered relevant to humans.

Infants and children are a susceptible subpopulation for lead exposure because they have greater gastrointestinal absorption and less effective renal excretion than adults. Selection of infants and

children as a susceptible subpopulation and neurodevelopmental effects as the critical health effect is considered protective for other adverse effects of lead across the entire population.

The most comprehensive analysis for developmental neurotoxicity was conducted by Lanphear et al. (2005) who pooled and analyzed data from seven longitudinal studies. The Lanphear et al. (2005) analysis contains the highest number of subjects (1333 children), is diverse in including studies from the United States, Mexico, Australia, and Yugoslavia, but not Canada, and contains a sufficient number of pre-school and school-aged children with blood lead levels below 10 µg/dL. The pooled analysis contains a sufficient number of subjects to provide the statistical power needed to best characterize the relationship between BLL and IQ score at low exposure levels. The pooled analysis study by Lanphear et al. (2005) is identified as the critical study for the risk characterization of neurodevelopmental effects in children since there is no comparable analysis available for attention-related behaviours.

Dose–response analyses have been conducted by some agencies, such as the European Food Safety Authority (EFSA 2010) and the Office of Environmental Health Hazard Assessment of the California Environmental Protection Agency (OEHHA 2007), to characterize risk. Once the dose–response relationship was characterized, a benchmark dose was determined and used to calculate margins of exposure. These margins do not reflect a safe level but rather serve as an indicator of exposure producing a specified effect. Both EFSA (2010) and JECFA (2010) concluded that the existing provisional tolerable weekly intake (PTWI) for lead of 25 µg/kg-bw is no longer appropriate to protect human health. In addition, JECFA (2010) concluded that, based on current data, it was not possible to establish a new PTWI that would be health protective. Based on the dose–response modelling conducted by California OEHHA (2007) and EFSA (2010), each incremental increase in BLL of 1 µg/dL is associated with approximately a 1-point deficit in IQ. More details on the various dose–response assessments reported in the literature are presented in Table 16.

Canadians are exposed to low levels of lead through food, drinking water, air, dust, soil, and products. Although blood lead levels (BLLs) have declined by over 70% in Canada since 1978–1979, lead is still widely detected in the Canadian population. In 1978–1979, approximately 27% of Canadians aged 6 to 79 years had BLLs at or above 10 µg/dL compared with less than 1% of Canadians today. BLLs tend to rise after infancy, peak between 1 and 3 years of age, and decline slightly during childhood and adolescence before rising again with age in adults. BLLs are highest in seniors, followed by children under 6 years. Based on the 2007–2009 Canadian Health Measures Survey (CHMS), geometric mean BLLs are 0.90 µg/dL, 0.80, 1.12, 1.60, and 2.08 µg/dL for the age groups of 6 to 11, 12 to 19, 20 to 39, 40 to 59, and 60 to 79 years, respectively. This is consistent with the trends reported in the United States. Although a data gap for national Canadian BLL data in children under 6 years currently exists, the geometric mean BLL for children 1 to 5 years of age in the U.S. population is 1.51 µg/dL.

Since the implementation of measures to reduce exposure to lead through the inhalation route (e.g., prohibition of leaded gasoline), oral exposures from food and water now represent the most significant sources of lead intake for the general adult population. For infants and children, ingestion of non-food items contaminated with lead (e.g., household dust, lead-based paint, soil, products) along with dietary intake through food and water are the greatest sources of environmental exposure to lead. Several factors are known to be associated with BLLs. Umbilical cord BLLs are correlated with maternal and infant BLLs. BLLs in infants are correlated with maternal BLLs and lead concentrations in breast milk. In children, BLLs are associated with lead levels in water, soil, and household dust. Lead-based paint may be a significant source for those living in older homes. In Canada, BLLs are higher in males than in females; in smokers than in non-smokers; in individuals born outside of Canada than in those born in

Canada; in individuals living older homes (greater than 50 years) than in those living in newer homes; and in residents of households with lower income levels than in those with higher income levels. Elevated BLLs may also result from increases in the rate of remobilization of bone lead in blood during life stages such as pregnancy, lactation, menopause, andropause, and post-menopause. Accordingly, at certain life stages, an individual's bone lead stores can represent the single greatest potential source of increased BLLs, as the amount of lead released back into blood for systemic circulation increases.

Effects associated with BLLs below 10 μg/dL have been reported in the health effects database and include neurodevelopmental, neurodegenerative, cardiovascular, renal, and reproductive effects. In humans, neurodevelopmental effects of lead can persist until the late teen-age years. The evidence of an association between health effects and BLLs below 5 μg/dL is strongest for neurodevelopmental effects in children, as indicated by a reduction of IQ score and attention-related behaviours. Developmental neurotoxicity has been associated with the lowest levels of lead exposure examined to date both in observational studies (down to 1-2 μg/dL) and in animal experiments. It must be noted that for many of the studies, the LOQ was approximately 3 μg/dL, making it difficult to interpret the lower end of the dose response curve. Uncertainty in the accuracy of the measured BLLs increases at or below the LOQ. Dose-response modelling conducted with available observational studies does not currently demonstrate a population threshold for developmental neurotoxicity.

Although BLLs of Canadians have declined significantly over the past 30 years, health effects are occurring below 10 μg/dL. There is sufficient evidence that blood lead levels below 5 μg/dL are associated with adverse health effects. Health effects have been associated with BLLs as low as 1–2 μg/dL, levels that are present in Canadians, although there is uncertainty associated with effects observed at these levels.

Table 16. Summary of dose–response assessments by international agencies and regulatory authorities of BLLs versus IQ deficits based on Lanphear et al. (2005)

Organization	Endpoint assessed	Assessment results					Reference
OEHHA (California)	IQ deficits in children (based on Lanphear et al. 2005)	Calculated the average change in IQ over a range of 1 to 10 μg/dL based on the best-fit log–linear model presented in Lanphear et al. (2005) from 2.4 to 30 μg/dL (the 5th and 95th percentiles, respectively, for the pooled data set) to develop a child-specific health guidance value: benchmark incremental change in blood lead concentration (ΔPbB) of 1 μg/dL of blood in children's blood that would reduce IQ by up to 1 point (*de minimus*)					OEHHA 2007
EFSA	IQ deficits in children (based on Lanphear et al. 2005)	BLL	Logarithmic model		Piecewise linear model		Budtz-Jørgensen 2010; EFSA 2010;
			BMD_{01} (μg/dL)	$BMDL_{01}$ (μg/dL)	BMD_{01} (μg/dL)	$BMDL_{01}$ (μg/dL)	
		Concurrent	0.35	0.26	1.8	1.20	
		Peak	0.39	0.27	1.03	0.69	
		Life-time	0.36	0.25	1.48	0.97	
		Early childhood	0.56	0.34	3.77	1.61	

Organization	Endpoint assessed	Assessment results				Reference
		Concluded the piecewise linear model, using the segment fit to the lower BLLs, provided less uncertain estimates of the $BMDL_{01}$. Determined the 95th percentile lower confidence limit of the benchmark dose (BMD) of 1% extra risk ($BMDL_{01}$) of 1.2 µg/dL as a reference point for the risk characterization of lead when assessing the risk of intellectual deficits in children measured by the Full Scale IQ score				
JECFA	IQ deficits in children	Based on a dose–response analyses (not provided), estimated that the previously established PTWI of 25 µg/kg-body weight is associated with a decrease of at least 3 IQ points in children. The Committee concluded that the PTWI could no longer be considered health protective and withdrew it.				JECFA 2010
U.S. EPA	IQ deficits in children (based on Lanphear et al. 2005)	BLL (µg/dL)	Incremental IQ loss (IQ points per µg/dL)			U.S. EPA 2007
			Log-linear with cut point	Log-linear with low-exposure linearization	Two-piece linear	
		<2	0.94	2.29	0.45	
		<5	0.87	1.41	0.45	
		<7.5	0.73	1.09	0.45	
BMD: Benchmark Dose. BMDL: Benchmark Dose (lower confidence limit).						

Level of Confidence and Uncertainties

The level of confidence in the health effects database is high. The database is well populated, contains numerous human observational studies, and assesses multiple organs or systems. Furthermore, the critical health effects identified are based on well-established endpoints and are supported by mechanistic data as well as studies conducted in laboratory animals. Despite some uncertainties, the overall findings from the literature are relatively strong and clear, particularly regarding the most critical health effect identified in children.

The following uncertainties were identified. The use of BLLs as the biomarker of exposure is well correlated with health effects, but does not represent whole body burden (e.g., lead sequestered into bone). Also, confounders such as socioeconomic factors, which can influence IQ score results, were not always accounted for in human studies. Effects may be related, in part, to co-exposures to other chemicals. In addition to the inherent uncertainties related to analytical limitations, at the time when many of the blood analyses were conducted there was no standardized methodology for measurement of blood lead concentrations below 10 µg/dL. The variance on those blood measurements was determined by the U.S. Centers for Disease Control and Prevention to be 4 µg/dL (U.S. CDC 2007). For many of the studies, the LOQ was approximately 3 µg/dL, making it difficult to interpret the lower end of the dose-response curve. Pooled analyses incorporating recent published epidemiological data at BLLs under 5 µg/dL have not been reported. Biological variations such as gender, diseases state, nutritional state, genetics, and ethnicity can also influence the absorption of lead or the severity of the effects observed. Hence, the following individuals are considered to be part of more susceptible subgroups: children, males, aging adults, pregnant women, non-Caucasians and people suffering from hypertension, chronic kidney disease, diabetes and nutritional deficiencies. Finally, it should be noted that some of the effects identified are based on population observations as opposed to individual effects.

Confidence in the data on levels of lead in the Canadian environment and Canadians is high. National surveys and programs report recent data on concentrations of lead in Canadians and environmental media (ambient air, house dust, and food), and levels of lead in drinking water were supplied by several provinces and identified in municipal and provincial publications. Furthermore, the national data are supplemented by studies comparing measurements of lead in air and house dust in several communities. Three recent studies conducted in Canada have examined the contribution of lead in environmental media to BLLs in children. Gaps exist for Canadian national blood level data in children less than 6 years of age. The second cycle of the Canadian Health Measures Survey (CHMS 2009–2011) will include children aged 3 to 5 years. However, a gap will remain for children under 3 years of age, who are expected to have elevated exposures compared with older children. The U.S. NHANES includes children under 6 years of age and is considered an acceptable surrogate for Canadian data owing to the similarity and consistency of BLLs between Canada and the United States for other age groups.

Additional research and monitoring, including exposure studies and measurements in Canadians and effects at low-level exposures, will continue to support the assessment and management of lead.

Conclusion

In infants and children, neurodevelopmental effects are considered to show the strongest association, with a decrease in IQ score and attention-related behaviour being the most sensitive endpoints. Developing infants and children are more susceptible to lead's toxic effects than are adults, owing to a higher gastrointestinal absorption rate and less effective renal excretion in addition to different behaviours. The relationship between neurodevelopmental effects and blood lead levels represents the strongest evidence of adverse health outcomes from exposures to lead concentrations below 10 µg/dL in this vulnerable population. Identification of infants and children as a susceptible subpopulation and neurodevelopmental effects as the critical health effect is considered protective for other adverse effects of lead across the entire population.

In summary, although BLLs of Canadians have declined significantly over the past 30 years, health effects are occurring below the current Canadian blood lead intervention level of 10 µg/dL. There is sufficient evidence that blood lead levels below 5 µg/dL are associated with adverse health effects. Health effects have been associated with BLLs as low as 1–2 µg/dL, levels that are present in Canadians, although there is uncertainty associated with effects observed at these levels. It is considered appropriate to apply a conservative approach when characterizing risk; accordingly, additional measures to further reduce exposures of lead to Canadians are warranted.

The outcome of this State of the Science report is consistent with conclusions from the larger scientific community (OEHHA 2007; EFSA 2010; JECFA 2010; U.S. CDC 2010b; WHO 2010; NTP 2012). Both EFSA (2010) and JECFA (2010) concluded that the provisional tolerable weekly intake (PTWI) for lead could no longer be considered to be health protective, as there is no evidence of a threshold for critical lead-induced health effect.

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Appendices – Concentrations of Lead Measured in Food

Appendix 1. Concentrations of lead in food reported in Canadian cities as part of the Canadian Total Diet Study

FOOD GROUP Composite	Concentration (µg/kg)				
	2007 (Vancouver)	2006 (Halifax)	2005 (Toronto)	2004 (Winnipeg)	2003 (Montréal)
DAIRY PRODUCTS					
Milk, whole	<0.22	0.24	0.19	<0.37	<0.33
Milk, 2%	<0.24	0.41	0.28	<1.08	<0.42
Milk, 1%	0.24	0.20	0.21	<1.33	<0.40
Milk, skim	0.30	0.17	0.26	<1.71	<0.33
Evaporated milk	0.23	0.76	0.69	5.46	0.52
Cream	<0.19	0.25	0.19	<0.32	<0.22
Ice cream	2.52	2.35	2.14	<1.87	2.81
Yogurt	0.49	0.72	0.60	<1.82	<0.39
Cheese	7.63	5.54	5.33	5.93	8.07
Cheese, cottage	1.11	0.95	0.89	<0.36	0.57
Cheese, processed	9.80	5.10	4.11	6.00	13.5
Butter	1.51	0.42	1.41	<0.85	4.39
Chocolate milk, 1%	N/A	1.22	1.34	N/A	N/A
Buttermilk, 1%	N/A	0.64	0.20	N/A	N/A
MEAT AND MEAT PRODUCTS					
Beef, steak	1.21	2.85	4.89	3.24	12.3
Beef, roast	1.42	2.29	7.60	1.36	4.28
Beef, ground	4.93	3.44	2.51	3.40	1.61
Pork, fresh	1.60	1.92	1.62	3.12	2.25
Pork, cured	4.95	9.10	3.98	7.39	9.37
Veal	3.53	2.46	5.34	2.24	2.67
Lamb	1.87	4.27	2.33	1.63	1.74
Luncheon meat, cold	4.20	7.42	7.46	4.02	7.73
Luncheon meat, canned	3.19	3.24	3.66	5.97	1.88
Organ meats	10.60	12.8	32.6	18.3	28.5
Wieners, sausage	6.35	7.19	4.25	5.42	10.5
POULTRY AND POULTRY PRODUCTS					
Eggs	<0.91	2.15	1.25	1.95	1.67
Poultry, chicken and turkey	1.98	2.27	2.13	2.16	2.11
Poultry, liver pâté	6.95	3.65	4.20	8.34	5.64
FISH AND FISH PRODUCTS					
Fish, marine	2.50	3.56	5.38	5.12	20.7

FOOD GROUP Composite	Concentration (µg/kg)				
	2007 (Vancouver)	2006 (Halifax)	2005 (Toronto)	2004 (Winnipeg)	2003 (Montréal)
Fish, freshwater	<1.03	0.50	2.55	1.41	0.83
Fish, canned	3.69	1.70	2.92	1.88	2.13
Shellfish	4.48	8.00	3.87	6.23	5.92
SOUPS					
Soups, meat canned	4.69	3.65	3.84	1.69	3.11
Soups, creamed canned	3.25	5.74	2.94	2.96	6.94
Soups, broth canned	3.73	4.69	3.06	1.89	2.18
Soups, dehydrated	5.56	6.53	3.48	3.64	2.32
CEREAL AND CEREAL PRODUCTS					
Bread, white	2.15	5.45	2.61	3.89	6.12
Bread, whole wheat	3.72	3.44	2.31	5.02	6.75
Bread, rye	2.52	10.0	7.01	2.96	9.66
Cake	10.60	7.35	8.59	10.1	14.1
Cereal, cooked wheat	10.30	15.9	4.23	4.89	4.26
Cereal, corn	0.86	1.59	3.17	0.77	1.31
Cereal, oatmeal	10.50	8.86	2.12	5.16	5.98
Cereal, wheat, rice and bran	6.22	2.83	2.93	6.26	7.05
Cereal, mixed	1.93	1.47	2.60	2.21	3.20
Cookies, chocolate chip	8.73	8.33	14.7	11.8	11.0
Crackers	2.01	2.95	2.50	2.91	3.69
Danish, doughnuts and croissants	3.30	7.25	4.46	3.47	5.76
Flour, white	1.27	1.25	1.07	1.33	2.94
Muffins	5.27	3.66	2.47	4.88	5.04
Pancake and waffle	2.45	2.83	5.18	3.08	3.25
Pasta	5.53	7.49	7.27	4.83	3.74
Pasta, plain	4.66	7.75	3.09	3.45	2.38
Pie, apple	2.63	<2.72	2.88	6.33	3.40
Pie, other	3.97	<3.16	2.82	2.33	3.31
Rice	11.30	5.14	6.57	2.18	3.48
Buns and rolls	3.81	5.69	2.72	3.03	9.01
Bread, other	3.49	3.55	4.63	3.33	5.38
VEGETABLE AND VEGETABLE PRODUCTS					
Baked beans, canned	3.46	5.77	6.82	5.53	4.17
Beans, string	7.88	4.31	5.61	4.06	4.79
Beets	8.73	4.52	4.99	3.40	4.62
Broccoli	4.65	4.13	2.39	2.51	5.75

FOOD GROUP Composite	Concentration (µg/kg)				
	2007 (Vancouver)	2006 (Halifax)	2005 (Toronto)	2004 (Winnipeg)	2003 (Montréal)
Cabbage	2.47	4.27	5.22	4.32	4.63
Carrots	4.34	27.7	6.24	3.46	5.70
Cauliflower	5.76	5.97	1.06	3.18	3.59
Celery	2.00	4.19	3.81	2.49	2.30
Corn	2.68	2.03	3.32	1.86	2.74
Cucumbers	2.27	3.13	4.18	3.42	9.36
Lettuce	3.50	6.28	2.30	4.36	2.81
Mushrooms	3.96	2.00	3.22	6.15	2.54
Onions	4.07	4.05	1.89	2.99	3.94
Peas	4.46	3.74	2.12	2.57	2.38
Peppers	2.13	1.75	3.64	1.08	1.12
Potatoes	6.86	2.98	2.02	4.68	5.42
Potato chips	2.09	2.12	1.41	1.79	1.14
Rutabagas or turnip	2.50	1.16	1.51	1.30	2.83
Vegetable juice, canned	1.58	1.17	1.14	1.33	1.56
Tomatoes	1.19	1.20	1.91	0.54	1.01
Tomato sauce	11.2	8.58	5.54	5.26	5.84
Spinach	18.8	15.5	23.8	N/A	N/A
Asparagus	8.34	5.36	3.91	N/A	N/A
Brussel sprouts	10.4	3.93	2.43	N/A	N/A
Potatoes, baked with skin	2.41	N/A	N/A	N/A	N/A
Corn chips	2.34	N/A	N/A	N/A	N/A
FRUIT AND FRUIT PRODUCTS					
Apple juice, canned	2.07	1.68	1.29	1.68	3.85
Apple sauce	7.43	1.00	1.46	1.14	1.52
Apple, raw	7.64	5.76	1.98	2.63	4.28
Bananas	<0.55	0.82	0.68	0.26	1.56
Blueberries	4.90	2.36	2.07	3.66	3.37
Cherries	3.32	2.33	1.44	2.27	2.49
Citrus fruits	0.84	1.98	0.78	5.47	<0.62
Citrus juice, frozen	1.40	1.94	2.60	4.35	7.26
Citrus juice, canned	0.82	0.39	0.25	0.50	<0.34
Grape juice, bottled	6.33	9.79	11.90	10.2	12.9
Grapes	1.81	0.85	1.75	1.83	0.95
Melons	1.65	6.90	0.81	0.73	0.55
Peaches	5.54	11.2	14.4	7.00	5.63
Pears	1.53	1.02	2.07	2.99	<2.20

FOOD GROUP Composite	Concentration (µg/kg)				
	2007 (Vancouver)	2006 (Halifax)	2005 (Toronto)	2004 (Winnipeg)	2003 (Montréal)
Pineapple, canned	9.71	9.19	33.5	36.7	23.1
Plums and prunes	2.02	2.92	9.02	3.55	83.4
Raisins	20.1	23.2	29.7	9.80	23.3
Raspberries	3.29	1.97	1.98	2.21	1.78
Strawberries	2.73	1.76	1.71	1.51	<1.75
Kiwi fruit	2.93	0.95	0.04	1.51	<1.64
Apricot	25.60	38.5	20.8	N/A	N/A
FATS AND OILS					
Cooking fats and salad oil	0.84	1.85	<0.18	0.25	0.92
Margarine	0.81	<0.56	<1.23	0.67	<3.87
Mayonnaise	1.52	2.80	4.20	3.59	3.04
MISCELLANEOUS					
Chocolate bar	8.92	10.20	9.21	9.41	10.7
Candy	3.54	5.41	4.71	5.27	5.68
Gelatin dessert	5.61	4.70	1.62	4.77	2.29
Honey, bottled	6.57	10.70	5.59	6.93	9.63
Jams	2.87	3.55	5.14	1.56	2.50
Peanut butter	4.37	5.98	3.65	3.03	2.85
Puddings	1.64	2.17	2.09	3.48	3.92
Sugar	0.42	0.39	<0.29	0.49	<1.26
Syrup	4.67	3.44	0.36	0.96	<1.60
Seeds, shelled	0.64	1.00	N/A	1.02	<1.86
Nuts	3.50	6.37	5.06	8.12	19.1
Popcorn	12.0	4.39	4.05	2.35	<29.3
Chewing gum	92.3	47.4	109	32.6	62.8
Condiments	4.62	5.23	3.36	2.28	5.89
Desserts	1.79	2.86	1.54	2.47	1.97
Salt	41.5	158	202	102	116
Baking powder	19.2	17.0	19.8	12.2	17.9
Yeast	41.2	30.1	23.3	30.1	38.7
Vanilla extract	3.29	2.65	1.81	0.73	<0.82
Herbs and spices	392	358	292	378	367
Soya sauce	17.7	17.5	17.5	10.8	1.15
Tap water, kitchen	0.38	<2.55	0.65	0.44	33.9
Tap water, area	0.57	<2.60	0.38	0.42	<0.72
Water, natural spring	<0.17	<2.52	<0.10	N/A	N/A
Water, mineral	<0.15	<2.48	<0.14	N/A	N/A

FOOD GROUP Composite	Concentration (µg/kg)				
	2007 (Vancouver)	2006 (Halifax)	2005 (Toronto)	2004 (Winnipeg)	2003 (Montréal)
Dinner: cereals, vegetables, meats	2.19	4.60	1.37	1.28	<9.38
Dinner: cereal	N/A	N/A	N/A	N/A	1.26
Dinner: vegetables, meats, poultry	1.05	2.29	1.40	N/A	N/A
Formulae, milk	0.50	5.01	1.53	2.07	1.43
Formulae, soya	5.42	9.10	3.99	5.48	2.50
Fruit, apple peach	8.74	6.80	2.05	2.12	<8.82
Meat, poultry or eggs	1.43	3.27	1.06	1.64	<10.5
Vegetable, peas	1.66	2.72	0.92	2.11	<7.21
BEVERAGES					
Alcoholic drinks, beer	0.30	<0.25	0.44	0.36	0.51
Alcoholic drinks, wine	9.78	12.6	15.2	14.4	14.1
Coffee	4.04	2.34	1.96	1.44	4.04
Soft drinks, canned	0.37	<0.25	0.23	0.71	0.32
Tea	5.32	7.71	4.62	2.63	2.54
Soy beverage, fortified	1.24	0.77	N/A	N/A	N/A
FOODS TO BE COOKED IN PACKAGE					
Frozen entrées	4.08	4.01	3.94	2.68	<6.90
FAST FOODS					
Pizza	8.24	6.93	7.42	11.9	9.36
French fries	2.77	1.32	1.86	2.16	<6.12
Hamburger	4.70	4.89	9.97	6.82	<8.03
Chicken burger	3.47	3.18	4.48	2.60	5.10
Hot dog	4.87	6.5	2.46	4.34	5.41
Chicken nuggets	2.76	2.06	2.90	3.36	3.97
Other	Beef chow mein, carry-out: 4.31	Beef chow mein: 4.63	Beef chow mein: 2.64	N/A	N/A

Source: Health Canada 2010d.

N/A: data not available.

Appendix 2. Lead concentrations in food products reported in the National Chemical Residue Monitoring Program (NCRMP) Annual Report 2005–2006

Food type	No. of detections per total no. of samples	Concentration range (ppm)	Mean concentration (ppm)	Origin of product
Raw milk	24/322	1.0–22.0	2.5	Domestic
Butter	0/11	ND	ND	Argentina, Chile, New Zealand, U.S., Uruguay
Cheese	1/12	40	40	Italy
	1/15	40	40	Netherlands
	1/20	10	10	Switzerland
	0/50	ND	ND	Denmark, El Salvador, France, Germany, Greece, Israel, Norway, UK, U.S.
Egg	2/219	40	40	Domestic
	1/162	70	70	U.S.
Honey	19/130	40–1450	166.3	Domestic
	3/5	60–110	83.3	Argentina
	4/7	130–230	185.0	Australia
	1/1	80	80	Brazil
	2/4	130–240	185	Bulgaria
	1/1	50	50	Croatia
	1/3	210	210	Greece
	1/3	230	230	Iran
	1/1	0.05	0.05	Malaysia
	1/1	40	40	Romania
	1/4	180	180	Switzerland
	1/1	150	150	Turkey
	2/4	200–210	205.0	U.S.
	2/2	50–850	450	Vietnam
	0/7	ND	ND	France, Hungary, Israel, Moldova, Russian Federation, Slovenia
Beef, muscle	5/168	110–1270	294.2	Domestic
Buffalo, muscle	0/79	ND	ND	Domestic
Chicken, muscle	1/81	2040	2040	Domestic
Cow, muscle	9/149	6.0–140.0	37	Domestic
Deer, muscle	0/2	ND	ND	Domestic
Duck, muscle	0/32	ND	ND	Domestic
Elk, muscle	1/1	18.0	18	Domestic
Fowl, muscle	3/84	80.0–160	130	Domestic

Food type	No. of detections per total no. of samples	Concentration range (ppm)	Mean concentration (ppm)	Origin of product
Game bird, muscle	1/37	10.0	10.0	Domestic
Goat, muscle	0/5	ND	ND	Domestic
Goose, muscle	0/14	ND	ND	Domestic
Horse, muscle	5/180	7.0–180	64.8	Domestic
Mutton, muscle	0/168	ND	ND	Domestic
Ostrich, muscle	0/29	ND	ND	Domestic
Pork, muscle	1/186	210	210	Domestic
Rabbit, muscle	0/32	ND	ND	Domestic
Sow and boar, muscle	3/179	11.0–140	67.0	Domestic
Turkey, muscle	0/91	ND	ND	Domestic
Veal, muscle	0/178	ND	ND	Domestic
Wild boar, muscle	0/6	ND	ND	Domestic
Beef, cooked	0/11	ND	ND	Brazil
Goat meat, muscle	0/1	ND	ND	Australia
Mutton meat, muscle	0/3	ND	ND	New Zealand
Pork meat, muscle	0/7	ND	ND	Chile, Spain, U.S.
Pork, cooked	6/6	40–90	63.3	China
Salami	0/1	ND	ND	Hungary
Turkey meat, muscle	0/1	ND	ND	U.S.
Fresh fruit/vegetable products	Found in 50 samples (total no. samples unknown)	50–1230	180	Domestic
	Found in 29 samples (total no. samples unknown)	40–780	168	Imported
Apple, fresh	1/39	60	60	Domestic
Apricot, fresh	0/2	ND	ND	Domestic
Asparagus, fresh	0/4	ND	ND	Domestic
Bean sprouts, fresh	0/10	ND	ND	Domestic
Bean, fresh	0/34	ND	ND	Domestic
Beet, fresh	0/29	ND	ND	Domestic
Blueberry, fresh	0/34	ND	ND	Domestic
Broccoli, fresh	0/38	ND	ND	Domestic
Brussels sprouts, fresh	0/4	ND	ND	Domestic
Cabbage, fresh	1/72	650	650	Domestic
Cabbage, fresh Chinese	0/10	ND	ND	Domestic
Carrot, fresh	2/49	70–90	80	Domestic
Cauliflower, fresh	0/25	ND	ND	Domestic
Celeriac, fresh	0/3	ND	ND	Domestic

Food type	No. of detections per total no. of samples	Concentration range (ppm)	Mean concentration (ppm)	Origin of product
Celery, fresh	0/21	ND	ND	Domestic
Cherry, fresh	0/67	ND	ND	Domestic
Coriander, fresh	2/2	110–210	160	Domestic
Corn, fresh sweet	0/31	ND	ND	Domestic
Cranberry, fresh	0/6	ND	ND	Domestic
Cucumber, fresh	0/47	ND	ND	Domestic
Cucumber, fresh (greenhouse)	0/24	ND	ND	Domestic
Dill, fresh	1/1	70	70	Domestic
Eggplant, fresh	1/12	80	80	Domestic
Garlic, fresh	0/4	ND	ND	Domestic
Grape, fresh	0/4	ND	ND	Domestic
Leaf lettuce, fresh	14/66	50–860	211	Domestic
Leek, fresh	3/14	50–380	197	Domestic
Lettuce, fresh	0/19	ND	ND	Domestic
Melon, fresh	0/9	ND	ND	Domestic
Mint, fresh	0/2	ND	ND	Domestic
Mushroom, fresh	0/42	ND	ND	Domestic
Nectarine, fresh	0/4	ND	ND	Domestic
Onion, fresh green	2/23	110–150	130	Domestic
Onion, fresh sweet	0/28	ND	ND	Domestic
Oriental vegetable	0/1	ND	ND	Domestic
Parsley, fresh	0/4	50–120	78	Domestic
Parsnip, fresh	1/22	160	160	Domestic
Pea, fresh	0/9	ND	ND	Domestic
Peach, fresh	0/8	ND	ND	Domestic
Pear, fresh	0/17	ND	ND	Domestic
Pepper, fresh hot	0/1	ND	ND	Domestic
Pepper, fresh sweet	1/49	80	80	Domestic
Plum, fresh	0/9	ND	ND	Domestic
Potato, fresh	3/106	50–1230	59	Domestic
Pumpkin, fresh	0/5	ND	ND	Domestic
Radish, fresh	4/29	50–240	127	Domestic
Raspberry, fresh	0/12	ND	ND	Domestic
Rhubarb, fresh	0/7	ND	ND	Domestic
Roquette, fresh	1/1	90	90	Domestic
Rutabaga, fresh	0/25	ND	ND	Domestic
Shallot, fresh	0/1	ND	ND	Domestic

Food type	No. of detections per total no. of samples	Concentration range (ppm)	Mean concentration (ppm)	Origin of product
Snowpea, fresh	0/1	ND	ND	Domestic
Spinach, fresh	6/17	60–180	98	Domestic
Squash, fresh	0/27	ND	ND	Domestic
Strawberry, fresh	0/21	ND	ND	Domestic
Swiss chard, fresh	3/4	50–260	123	Domestic
Tomato, fresh	0/19	ND	ND	Domestic
Tomato, fresh (greenhouse)	0/34	ND	ND	Domestic
Turnip, fresh	0/1	ND	ND	Domestic
Zucchini, fresh	0/20	ND	ND	Domestic
Apple, fresh	0/47	ND	ND	Chile, China, New Zealand, South Africa, U.S.
Apricot, fresh	0/5	ND	ND	U.S.
Artichoke, fresh	0/9	ND	ND	France, U.S.
Asparagus, fresh	0/13	ND	ND	Mexico, Peru, U.S.
Avocado, fresh	0/19	ND	ND	Mexico, Peru, U.S.
Banana, fresh	0/53	ND	ND	Colombia, Costa Rica, Ecuador, Guatemala, Honduras, Mexico, Peru, U.S., Vietnam
Bean, fresh	0/16	ND	ND	Costa Rica, Dominican Republic, Kenya, Mexico, U.S
Beet, fresh	0/4	ND	ND	Mexico, U.S.
Bittermelon, fresh	0/1	ND	ND	Dominican Republic
Blackberry, fresh	0/1	ND	ND	Mexico
Blueberry, fresh	0/10	ND	ND	Argentina, Chile, U.S.
Broccoflower, fresh	0/3	ND	ND	U.S.
Broccoli, fresh	0/13	ND	ND	Mexico, U.S.
Brussels sprout, fresh	0/11	ND	ND	Mexico, U.S.
Cabbage, fresh	0/13	ND	ND	China, Netherlands, Taiwan, U.S.
Cabbage, fresh Chinese	0/6	ND	ND	China, U.S.
Cactus pear, fresh	0/4	ND	ND	Italy, Mexico, South Africa
Carrot, fresh	1/4	100	100	China
	0/35	ND	ND	Mexico, U.S.
Cauliflower, fresh	0/18	ND	ND	U.S.
Celery, fresh	0/28	ND	ND	Mexico, U.S.

Food type	No. of detections per total no. of samples	Concentration range (ppm)	Mean concentration (ppm)	Origin of product
Cherry, fresh	0/18	ND	ND	Chile, U.S.
Chive, fresh	1/2	50	50	China
Coconut, fresh	0/2	ND	ND	Dominican Republic, Thailand
Corn, fresh sweet	0/9	ND	ND	U.S.
Cranberry, fresh	0/2	ND	ND	U.S.
Cucumber, fresh	1/10	100	100	Mexico
	0/21	ND	ND	Honduras, The Netherlands, Spain, U.S.
Date, fresh	0/3	ND	ND	Israel, Jordan, Tunisia
Dragonfruit, fresh	0/6	ND	ND	China, Israel, Vietnam
Eggplant, fresh	0/11	ND	ND	Belgium, Dominican Republic, Honduras, Mexico, The Netherlands, U.S.
Endive, fresh	2/3	50–80	65	U.S.
Fig, fresh	0/13	ND	ND	Argentina, Greece, Turkey, U.S.
Gai choy, fresh	1/1	60	60	Mexico
Garlic, fresh	0/17	ND	ND	China, Hong Kong, Republic of Korea, Mexico, Philippines, Thailand, Turkmenistan, U.S.
Ginger, fresh	1/1	80	80	Thailand
	0/4	ND	ND	Brazil, China
Gooseberry, fresh	0/1	ND	ND	Colombia
Grape, fresh	0/75	ND	ND	Chile, Italy, Mexico, South Africa, UK, U.S.
Grapefruit, fresh	0/65	ND	ND	Argentina, Cuba, Jamaica, Mexico, South Africa, Swaziland, U.S., Zimbabwe
Guava, fresh	0/14	ND	ND	Brazil, Costa Rica, Mexico, Taiwan
Italian parsley, fresh	1/1	130	130	Ethiopia
Jicama, fresh	0/1	ND	ND	Mexico
Kiwi fruit, fresh	0/27	ND	ND	Chile, China, Italy, New Zealand, U.S.
Kumquat, fresh	0/3	ND	ND	Brazil, Israel
Leaf lettuce, fresh	2/5	50–90	70	U.S.

Food type	No. of detections per total no. of samples	Concentration range (ppm)	Mean concentration (ppm)	Origin of product
	1/1	ND	ND	China
Leek, fresh	1/4	40	40	U.S.
	0/1	ND	ND	Guatemala
Leek flower, fresh	0/1	ND	ND	China
Lemon, fresh	0/31	ND	ND	Argentina, Chile, South Africa, U.S.
Lettuce, fresh	1/1	120	120	Mexico
	2/38	60–560	310	U.S.
	0/1	ND	ND	China
Lime, fresh	0/11	ND	ND	Brazil, Mexico, U.S.
Lobok, fresh green	0/1	ND	ND	China
Longan fruit, fresh	0/3	ND	ND	Thailand
Loquat, fresh	0/1	ND	ND	Vietnam
Lotus root, fresh	0/2	ND	ND	China
Lychee nut, fresh	0/6	ND	ND	China, South Africa, Taiwan, Thailand
Malanga root, fresh	0/1	ND	ND	Nicaragua
Mango, fresh	0/18	ND	ND	Brazil, Cuba, Haiti, Mexico, Peru, South Africa
Melon, fresh	1/3	60	60	Brazil
	0/49	ND	ND	China, Costa Rica, Ecuador, France, Guatemala, Honduras, Republic of Korea, Mexico, Nicaragua, Panama, U.S.
Mushroom, fresh	0/5	ND	ND	China, U.S.
Mustard, fresh	0/1	ND	ND	U.S.
Nectarine, fresh	0/18	ND	ND	Chile, U.S.
Okra, fresh	0/1	ND	ND	Egypt
Onion, fresh green	1/7	40	40	Mexico
	0/6	ND	ND	China, U.S.
Onion, fresh sweet	0/9	ND	ND	Mexico, U.S.
Orange, fresh	1/20	270	270	China

Food type	No. of detections per total no. of samples	Concentration range (ppm)	Mean concentration (ppm)	Origin of product
	0/120	ND	ND	Argentina, Australia, Brazil, Chile, Cuba, Cyprus, Dominican Republic, Egypt, Italy, Jamaica, Japan, Republic of Korea, Morocco, Panama, Peru, South Africa, Spain, Switzerland, Taiwan, Thailand, U.S., Uruguay
Papaya, fresh	0/32	ND	ND	Belize, Brazil, Costa Rica, Cuba, Denmark, Guatemala, Jamaica, Mexico, U.S.
Parsnip, fresh	0/2	ND	ND	U.S.
Passion fruit, fresh	0/2	ND	ND	Colombia
Pea, fresh	0/20	ND	ND	China, Guatemala, Kenya, U.S.
Peach, fresh	0/22	ND	ND	Chile, U.S.
Pear, fresh	1/21	670	670	U.S.
	0/26	ND	ND	Argentina, Chile, China, Italy, Republic of Korea, New Zealand, Portugal, South Africa, Spain
Pear, fresh Asian	0/8	ND	ND	China, Republic of Korea
Pepper, fresh hot	0/9	ND	ND	Dominican Republic, Netherlands, Trinidad and Tobago, U.S.
Pepper, fresh sweet	0/29	ND	ND	Israel, Mexico, The Netherlands, Spain, U.S.
Persimmon, fresh	0/6	ND	ND	Chile, China, Israel, Democratic People's Republic of Korea, South Africa
Pineapple, fresh	0/27	ND	ND	Costa Rica, Ecuador, Equatorial Guinea, Guatemala, Honduras, Mexico, U.S.
Plantain, fresh	0/4	ND	ND	Costa Rica, Dominican Republic, Ecuador, Guatemala
Plum, fresh	0/26	ND	ND	Chile, Italy, Spain, U.S.

Food type	No. of detections per total no. of samples	Concentration range (ppm)	Mean concentration (ppm)	Origin of product
Pomegranate, fresh	0/1	ND	ND	U.S.
Potato, fresh	2/73	50	50	U.S.
Pummelo, fresh	0/16	ND	ND	Argentina, China, Thailand
Pumpkin, fresh	0/1	ND	ND	U.S.
Radish, fresh	0/10	ND	ND	China, Mexico, U.S.
Raspberry, fresh	0/20	ND	ND	Chile, Mexico, U.S.
Snowpea, fresh	0/9	ND	ND	China, Guatemala
Spinach, fresh	1/1	50	50	Egypt
	9/29	60–1250	222	U.S.
	3/4	40–70	60	Mexico
	3/12	80–780	460	U.S.
Squash, fresh	0/13	ND	ND	Costa Rica, Mexico, U.S.
Starfruit, fresh	0/8	ND	ND	Malaysia, Taiwan
Strawberry, fresh	0/13	ND	ND	New Zealand, U.S.
Sweet potato, fresh	1/2	60	60	China
	1/22	120	120	U.S.
	0/6	ND	ND	Costa Rica, Honduras, Jamaica
Tangerine, fresh	0/2	ND	ND	China
Taro, fresh	0/1	ND	ND	Thailand
Tomato, fresh	0/82	ND	ND	Belgium, Cuba, Israel, Mexico, The Netherlands, U.S.
Ugli fruit, fresh	0/3	ND	ND	Jamaica
Watermelon, fresh	0/18	ND	ND	Chile, Guatemala, Mexico, U.S.
Yam, fresh	0/5	ND	ND	Brazil, Japan, U.S.
Yuchoy-sum, fresh	0/1	ND	ND	Mexico
Zucchini, fresh	0/2	ND	ND	Mexico
Child’s cereal-based foods	2/87	40–50	45	Domestic
Child’s fruit and vegetable-based foods	0/47	ND	ND	Domestic
Children’s foods (miscellaneous)	7/198	50–140	74	Domestic
Bottled water	0/16	ND	ND	Domestic
Apple drink	0/1	ND	ND	U.S.
Apple juice	0/1	ND	ND	U.S.

Food type	No. of detections per total no. of samples	Concentration range (ppm)	Mean concentration (ppm)	Origin of product
Apricot product	1/1	86	86	China
	2/2	70–80	75	South Africa
	2/3	12–15	13.5	Turkey
	0/4	ND	ND	Morocco, U.S.
Artichoke product	0/3	ND	ND	Spain
Asparagus, canned	0/1	ND	ND	China
Asparagus, frozen	2/2	1.0	1.0	Chile
	1/1	11	11	China
Bean product	1/1	80	80	China
	1/1	1.0	1.0	U.S.
	0/6	ND	ND	France, India, Mexico, Portugal, Thailand, Turkey
Brussels sprout, frozen	1/1	1.0	1.0	Belgium
Cabbage, canned	1/1	130	130	Taiwan
Carrot, canned	0/1	ND	ND	The Netherlands
Carrot, frozen	1/1	30	30	Belgium
Cauliflower, frozen	1/1	5.0	5.0	China
	1/1	7.0	7.0	Mexico
Cherry, canned	0/1	ND	ND	U.S.
Children’s cereal-based foods	1/2	40	40	Brazil
	3/21	40–90	60	China
	1/4	60	60	Hong Kong
	1/4	70	70	Italy
	1/12	40	40	Japan
	1/7	60	60	Thailand
	1/30	40	40	U.S.
	0/65	ND	ND	Australia, Austria, Czech Republic, Egypt, Germany, Iran, Israel, Republic of Korea, Malaysia, The Netherlands, Pakistan, Poland, Singapore, Spain, Taiwan, Turkey, UAE
Children’s fruit and vegetable-based foods	2/4	40	40	China
	1/5	130	130	Egypt
	1/4	120	120	Iran
	1/4	60	60	Thailand

Food type	No. of detections per total no. of samples	Concentration range (ppm)	Mean concentration (ppm)	Origin of product
	1/1	50	50	UK
	0/44	ND	ND	Australia, Croatia, Japan, Lebanon, Malaysia, The Netherlands, Philippines, Poland, Singapore, U.S.
Children's foods (miscellaneous)	12/23	40–150	71.7	China
	2/20	80	80	Hong Kong
	12/45	50–200	104	India
	1/5	50	50	Singapore
	1/15	50	50	Thailand
	5/59	40–110	64	U.S.
	0/81	ND	ND	Australia, Indonesia, Italy, Japan, Republic of Korea, Malaysia, The Netherlands, Pakistan, Philippines, Taiwan, UK, Vietnam
Corn, canned	1/2	1.0	1.0	Thailand
	0/1	ND	ND	U.S.
Fruit cocktail	1/1	10	10	Chile
	1/1	6.0	6.0	Mexico
	1/1	47	47	South Africa
	0/1	ND	ND	Thailand
	1/1	5.0	5.0	Vietnam
Mango product	0/3	ND	ND	Mexico, Thailand
Miscellaneous products	1/1	43	43	China
	1/2	15	15	Ecuador
	1/2	7.0	7.0	France
	1/1	40	40	Lebanon
	1/1	9.0	9.0	Mexico
	1/4	8.0	8.0	Thailand
	0/6	ND	ND	Germany, Japan, Republic of Korea, Taiwan, U.S.
Mushroom, canned	10/17	1.0–40	25.4	China
	1/5	17	17	Taiwan
	1/2	370	370	Vietnam
Mushroom, frozen	1/1	37	37	Italy
Pea product	0/4	ND	ND	U.S.

Food type	No. of detections per total no. of samples	Concentration range (ppm)	Mean concentration (ppm)	Origin of product
Peach, canned	0/1	ND	ND	China
	2/2	22–31	26.5	Greece
	1/2	29	29	South Africa
Pear product	0/1	ND	ND	Spain
Pear, canned	0/3	ND	ND	China
	1/1	110	110	South Africa
	1/2	22	22	U.S.
Pepper, canned	1/1	10	10	Mexico
Pepper, frozen	1/1	2.0	2.0	Chile
Pineapple product	1/2	38	38	Thailand
Pumpkin, canned	1/1	10	10	China
Raspberry, frozen	1/1	4.0	4.0	Chile
Spinach, canned	0/5	ND	ND	U.S.
Strawberry product	1/1	4.0	4.0	Chile
Tomato juice	2/4	1.0–3.0	2.0	U.S.
Tomato, canned	1/1	9.0	9.0	Italy
Vegetable, canned	1/1	24	24	Denmark
	1/1	3.0	3.0	France
	1/1	39	39	Germany
Vegetable, processed	1/1	14	14	Belgium
	8/8	3.0–60	15	China
	3/4	1.0–8.0	4.0	Ecuador
	1/1	63	63	Hong Kong
	3/3	7.0–31	17	India
	1/1	11	11	Israel
	1/1	135	135	Jamaica
	5/6	1.0–14	4.6	Mexico
	3/3	8.0–61	35	Spain
	5/5	1.0–31	7.6	Thailand
	1/1	3.0	3.0	U.S.
	0/1	ND	ND	Guatemala
Maple syrup	80/148	180–670	286	Domestic

Source: CFIA 2010

ppm: parts per million.
ND: not detectable.